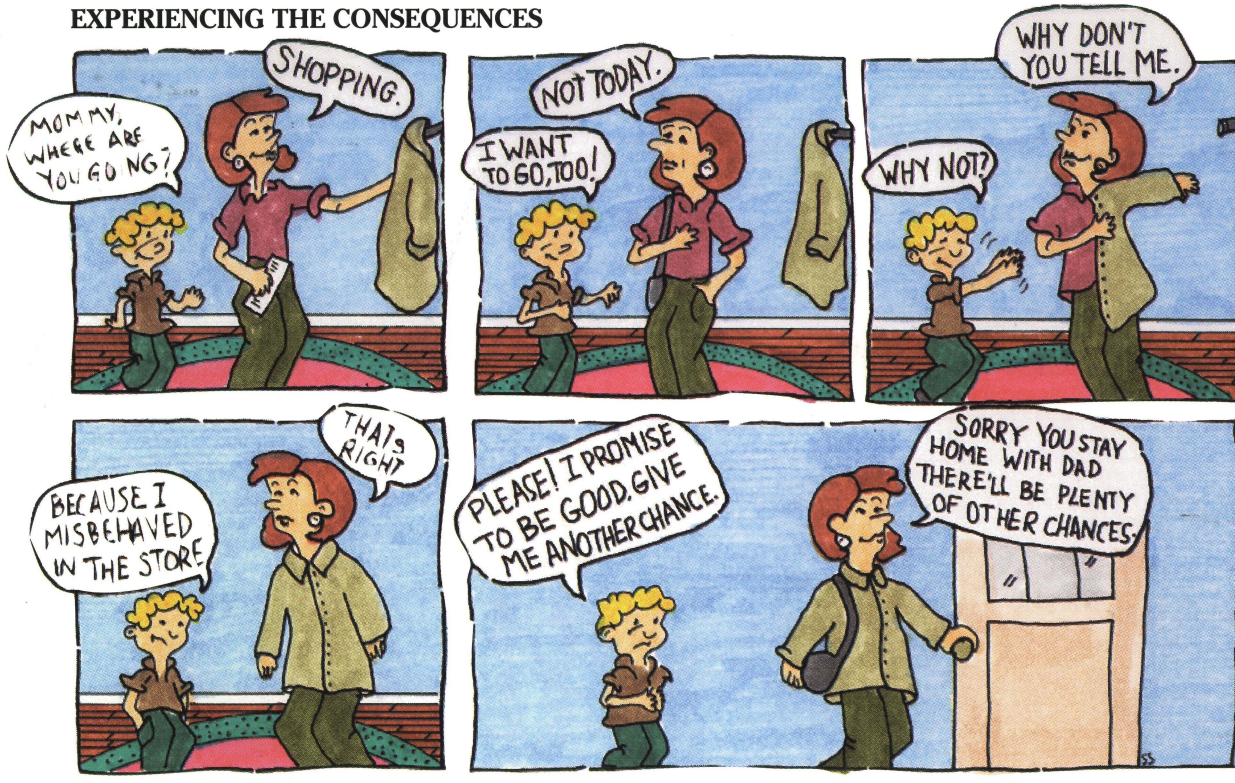
ASDC
AMERICAN SOCIETY OF DENTISTRY FOR CHILDREN
JOURNAL OF DENTISTRY FOR CHILDREN
SEPTEMBER—OCTOBER 1996

Misbehavior serves a very valuable function for all children. It is the way they test the world and learn to trust the people they love. Viewed in this way, misbehavior has a positive purpose in a child's life. This does not mean we should condone or encourage children to misbehave — quite the contrary. We can seize these opportunities to teach our children important lessons about the world and about their relationship with us. We do not punish a child for "being bad" or reward another child for "being good." We use discipline to lead our children out of ignorance into more self-fulfilling and socially constructive behavior. Goodness has nothing to do with what we discipline children for, but by careful teaching, we can help a child learn to do good things in the world.

—Peter Williamson, Ph D

EXPERIENCING THE CONSEQUENCES



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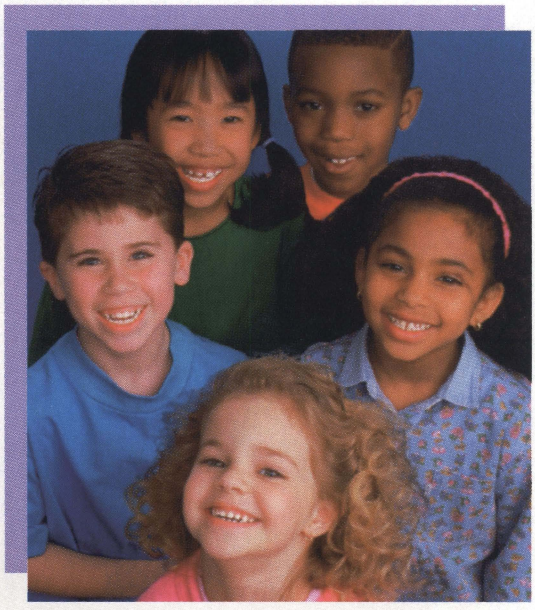

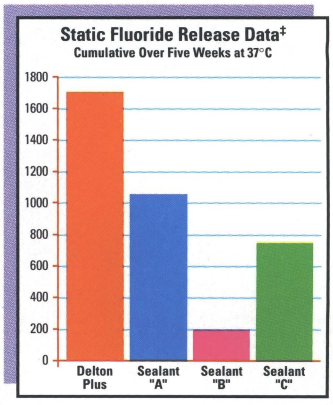

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* E.J. Mertz-Fairhurst and J.W. Ergle, *J Dent Research* 73 (IADR Abstract #2513, 1994) 416.
E.J. Mertz-Fairhurst et al., *JADA* 109 (August 1984) 252.
† Dental Research Associates, "U.S. Dental Merchandise Sales Report," 3rd quarter, 1994.
‡ Data on file

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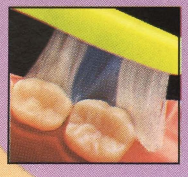

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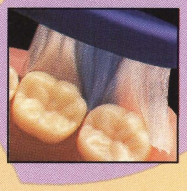

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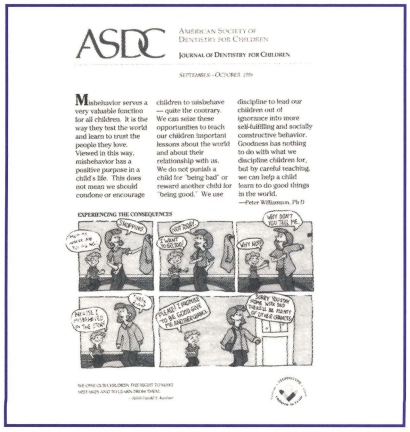
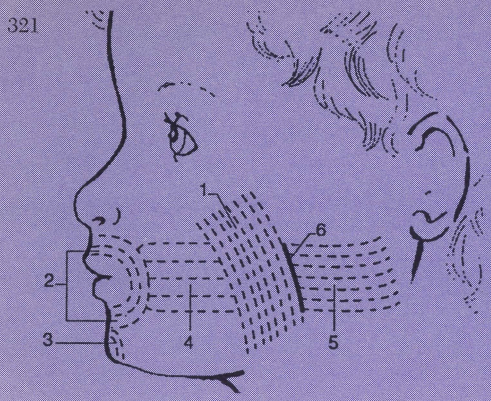
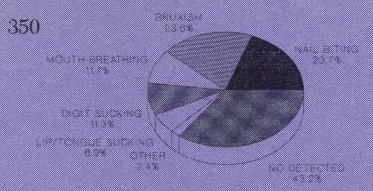


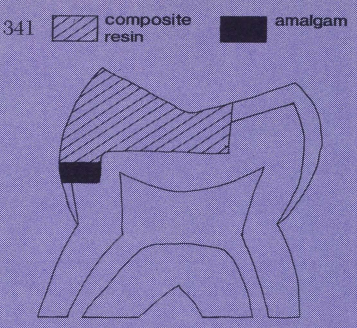
We use discipline to lead our children out of ignorance into self-fulfilling and socially constructive behavior
Art and design by Sharlene Nowak-Stellmach

REVIEWS

CLINIC







DEMOGRAPHICS

REPORTS

Calendar

1996

ASDC Annual Meeting, Westin Canal Place, New Orleans, LA, October 23-27
ASDC Regional Seminar, September 7, La Mansion del Rio, San Antonio, TX 78205
Evolving Issues in Child Behavior Management: Legal, Ehtical, and Practical Considerations. The University of North Carolina, October 18.

1997

ASDC Annual Meeting, Registry Resort, Naples, FL, October 22-26

For The Busy Reader

Nutritive and nonnutritive sucking habits: A review—page 321

Children who neither receive unrestricted breastfeeding nor have access to a pacifier may satisfy their need with alternative habits such as finger-sucking.

Requests for reprints should be sent to: Dr. Huguette Turgeon-O'Brien, Faculté des Sciences de l'agriculture et de l'alimentaiton, Université Laval, Québec, Québec, Canada G1K 7P4.

Dens evaginatus: A review—page 328

Several methods of treatment have been advocated previously, including extraction, selective grinding, prophylactic pulp capping, and sealants. The authors review the literature and discuss the clinical management of *dens evanginatus*.

Requests for reprints should be sent to: Dr. Dean S. Uyeno, Heidelberg Dental Activity, Unit 29225, APO AE 09102.

Craniomandibular dysfunction in children with clefts and noncleft children with and without unpleasant life events: A comparative study—page 333

The author investigated the differences in the prevalence of signs and symptoms of this disorder between children with and without clefts, in different emotional states caused by unpleasant life-events.

Requests for reprints should be sent to: Dr. Apostole P. Vanderas, 11 Makedonias Str., 14561 Kifisssia, Athens, Greece.

The effectiveness of preventive resin restorations in pediatric patients—page 338

It is unfortunate that so many dentists have not been convinced of the usefulness of this technique for the prevention of fissure caries. It was the purpose of this study to determine the effectiveness of composite sealant.

Requests for reprints should be sent to: Dr. Jerry D. Walker, Department of Pediatric Dentistry, College of Dentistry, The University of Iowa, 201 Dental Science Building S, Iowa City, IA 52242-1001.

Clinical evaluation of class II combined amalgam-composite restorations in primary molars after 6 to 30 months—page 341

These studies showed that microleakage at the cervical margin has been reduced significantly, when compared to conventional composite restorations; the long-term effect of the amalgam on the appearance of the restorations, however, could not be evaluated.

Requests for reprints should be sent to: Dr. Gideon Holan, Department of Pediatric Dentistry, Hadassah Faculty of Dental Medicine, Jerusalem, Israel, POB 1172.

Effect of heat treatment on stainless steel lingual arch appliances—page 346

Heat treatment of wire can be accomplished at comparatively low temperatures (370-480 degrees C). The purpose of the study was to evaluate the effect of heat treatment on 0.036" diameter stainless steel wire.

Requests for reprints should be sent to: Col. Gregory P. Mathieu, Director, Pediatric Dentistry Residency Program, U.S. Army Dental Clinic Command, Ft. Meade, MD 20755.

Etiology of oral habits—page 350

The treatment of pernicious oral habits on the basis of the utilization of orthodontic appliances or occlusal therapy has brought about unsatisfactory results. Inadequate knowledge about the source of the problem, whether on the part of the dentist, parents or teachers, may en-

courage poor conduct by the child, when the soothing effects of oral habits are interrupted. This could trigger rebelliousness, sleep and nourishment problems, enuresis, and learning problems, resulting in a persistence of the habit.

Requests for reprints should be sent to: Dr. Ruben E. Bayardo, Centro Universitario Ciencias de la Salud, Facultad de Odontologia. Col. Independencia, Universidad de Guadalajara, Mexico 44340.

Low birth-weight and the relationship to developmental problems—page 354

At school age, children who were born with low birth-weight are more likely than children of normal birth-weight to have mild learning disabilities, attention disorders, developmental impairment, and breathing problems such as asthma.

Without a thorough medical history, few dentists might consider the association of low birth-weights with difficulties in providing dental services for some youngsters. It is in an effort to provide dental practitioners with a greater awareness of the causes, prevalence and consequences of low birth-weight that the following introduction to the subject is provided.

Requests for reprints should be sent to: Dr. H. Barry Waldman Professor, Dental Health Services, General Dentistry, State University of New York at Stony Brook, Stony Brook, NY 11794-8706.

Poor children remain poor—page 358

While the official poverty estimates of the number of poor persons rose from 35.7 million to 36.9 million between 1991 and 1992, (a "...relatively small...change in the total number and percent of persons in poverty..."), a most significant finding from the study is the frequent transition into and out of poverty. Almost half (48 percent) of the surveyed population spent only two to four months living in poverty and more than two-thirds (68 percent) spent less than nine months living in poverty.

Requests for reprints should be sent to: Dr. H. Barry Waldman Professor, Dental Health Services, General Dentistry, State University of New York at Stony Brook, Stony Brook, NY 11794-8706.

Neonatal sublingual traumatic ulceration (Riga-Fede disease): Reports of cases—page 362

One complication of neonatal teeth is an ulceration on the ventral surface of the tongue caused by the tooth's sharp incisal edge. Constant trauma may create sufficient ulceration to interfere with proper suckling and feeding, and put the neonate at risk for nutritional deficiencies.

Requests for reprints should be sent to: Dr. Curt Goho, DENTAC Unit 26610, Box 5, APO AE 0-9244.

Transposition and fusion in the primary dentition: Report of case—page 368

Fusion is defined as the partial or complete union of two adjacent tooth germs, resulting in a bifid crown and two root canals. By contrast, germination arises from a single tooth germ and results in a bifid crown with a single root or root canal.

Requests for reprints should be sent to: Dr. Karen Duncan, University of Bristol, Division of Child Dental Health, Dental School, Lower Maudlin Street, Bristol BS1 2LY England.

Talon cusp in permanent dentition associated with other dental anomalies: Reports of seven cases—page 368

The etiology remains unknown. Similar to other defects in tooth form, talon cusp originates during the morphodifferentiation stage of tooth development.

Requests for reprints should be sent to: Dr. Faiez N. Hattab, Departments of Restorative and Pediatric Dentistry, Faculty of Dentistry, Jordan University of Science and Technology, Irbid, Jordan.

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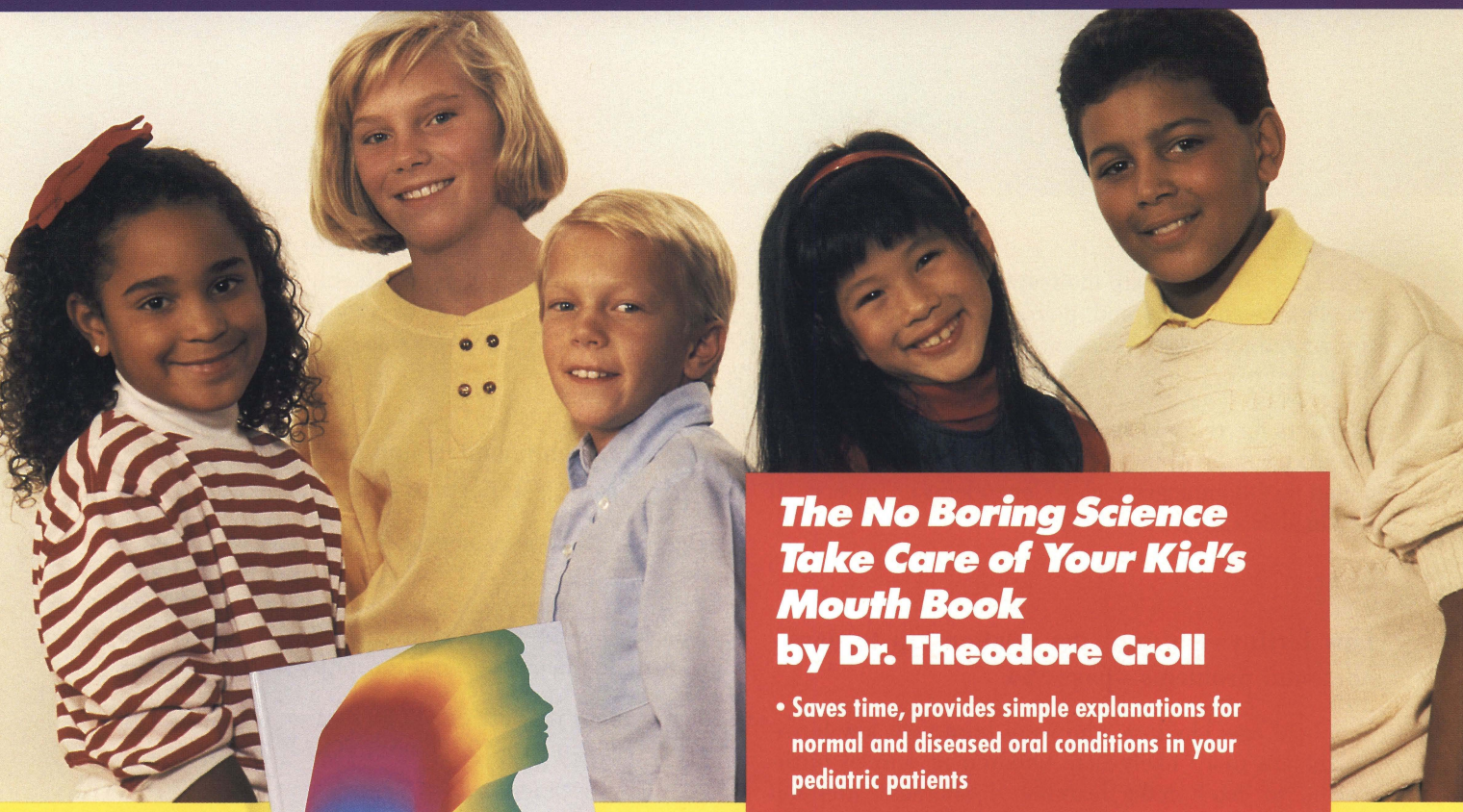

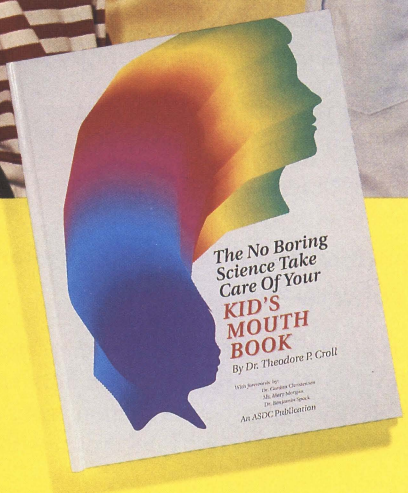

Dr. Theodore Croll, the author, is a board-certified pediatric dentist with an intuitive ability to manage children.

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ABSTRACTS

Turgeon-O'Brien, Huguette; Lachapelle, Diane; Gagnon, Pierre F. *et al*: Nutritive and non-nutritive sucking habits: A review. J Dent Child, 63: 321-327, September-October 1996.

The habit of sucking is the first coordinated muscular activity of the infant. There are essentially two forms of sucking: the nutritive form which provides essential nutrients, while non-nutritive sucking insures a feeling of warmth and a sense of security. This review gives a description of the anatomy and physiology of sucking together with the influence of breastfeeding and bottle-feeding (conventional or orthodontic nipples) on the dentofacial structures of the infant. Factors involved in the choice of feeding are also discussed. Children who do not have access to unrestricted breastfeeding or bottle-fed children may satisfy their instinctive sucking urge with a pacifier. This paper presents the different types of pacifiers (conventional or orthodontic) along with the beneficial effects provided by pacifiers. Detrimental effects caused by incorrect use of pacifiers or digit-sucking habits are also summarized. Health professionals should inform expectant mothers about the dentofacial advantages of breastfeeding.

Sucking habits; Breastfeeding; Bottle-feeding; Pacifiers

Uyeno, Dean S. and Lugo, Alberto: Dens evaginatus: A review. J Dent Child, 63:328-332, September-October 1996.

A review of the developmental dental anomaly, dans evaginatus, and a discussion of its clinical management is presented. Fracture or wear of the tubercle of dens evaginatus frequently results in pulp necrosis and early periapical infection, often prior to completion of root formation. Early recognition of this anomaly, followed by timely treatment can prevent the loss of pulp vitality in these teeth. Patients presenting with nonvital teeth and incomplete root development have been successfully treated by apexification followed by conventional endodontic therapy.

Dens evaginatus; Pulp vitality

Vanderas, A.P.: Craniomandibular dysfunction in children with clefts and noncleft children with and without unpleasant life events: A comparative study. J Dent Child, 63: 333-337, September-October 1996.

An epidemiologic study was conducted to investigate any difference in the prevalence of signs and symptoms of craniomandibular dysfunction on children with clefts and noncleft children with and without unpleasant life events. The experimental group consisted of thirty white children six to ten years of age with unilateral cleft lip and cleft lip and palate. On the basis of the sex and age distribution of the experimental group 30 out of 25 white children without unpleasant life events and 30 out of 105 white children with unpleasant life events were selected as control group I and control group II, respectively.

The results showed statistically significant difference in the prevalence of temporomandibular joint tenderness between experimental and control group I, while the difference between experimental and control group II was not significant. Statistically significant difference in the frequency of temporomandibular joint tenderness was also revealed between the control groups. With respect to the severity of the symptoms, the only statistically significant difference was that of the anamnestic dysfunction index with severe symptoms (AiII) between the experimental and control group I. The other differences were not significant. The present study suggests that children with clefts and those with unpleasant life events run a greater risk of developing temporomandibular joint tenderness.

Craniomandibular dysfunction; Orofacial clefts

Walker, J.; Floyd, K.; Jakobsen, J. *et al*: The effectiveness of preventive resin restorations in pediatric patients. J Dent Child, 63:338-340, September-October 1996.

In a six-year, eight-month period, 5,185 preventive resin composite sealants (preventive resin restorations) were placed in permanent molars and premolars in children, five through eighteen years of age in the University of Iowa Pediatric Dentistry Clinic. The purpose of this investigation was to determine the frequency of failure or retreatment of these teeth. The median observation time was for 18.5 months (the longest was for 79.2 months); 88.3 percent required no additional restorative procedures. The median time for resealing was one year after placement, in 6.2 percent of the cases. Three hundred and sixty-four (8 percent) of these teeth required retreatment consisting of one-surface amalgam or additional composite material.

In conclusion it would appear that the composite/sealant/restoration in the permanent dentition may require further evaluation in an attempt to reduce the incidence of repair or replacement of these restorations.

Composites; Sealants; Effectiveness

Holan, Gideon; Chosack, Aubrey; Eidelman, Eliecer: Clinical evaluation of Class II combined amalgam-composite restorations in primary molars after six to thirty months. J Dent Child, 63:341-345, September-October 1996.

Composites are claimed to be inappropriate for Class II restoration due to polymerization shrinkage. The present study evaluated the clinical and radiographic appearance of Class II combined amalgam composite restorations in primary molars. Conventional cavities (groups A & B) were restored with 1mm thick amalgam at the cervical floor covered by a posterior composite (Estilux Posterior). In group A Amalgambond was placed between the layers; in group

Continued page 316

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Interested individuals should communicate with Dr. Milton Houpt, Chair, Department of Pediatric Dentistry, UMDNJ-New Jersey Dental School, 110 Bergen Street, Newark, New Jersey 07103.

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New Jersey-Newark—The UMDNJ-New Jersey Dental School is seeking an additional full-time staff dentist for the Center for Treatment of the Handicapped. Responsibilities include the provision of dental care with and without conscious sedation in the Center, and treatment under general anesthesia in the adjoining University Hospital. Responsibilities also include instruction to predoctoral and postdoctoral students. Qualified applicants for the position must be licensed or licensable in the State of New Jersey and must have experience in parenteral conscious sedation. Experience in the management of dental problems for the handicapped, including the performance of treatment under general anesthesia in the operating room environment is essential.

Interested individuals should communicate with Dr. Milton Houpt, Chair, Department of Pediatric Dentistry, UMDNJ-New Jersey Dental School, 110 Bergen Street, Newark, New Jersey 07103.

The University of Medicine and Dentistry of New Jersey is an affirmative action/equal opportunity employer. Women and minorities are encouraged to apply.

New York-Bronx—Montefiore Medical Center/Albert Einstein College of Medicine offers a full-time position as Associate Director of the Division of Pediatric Dentistry. Responsibilities include postdoctoral teaching, administration, scholarly per research activities and patient care in a faculty practice. Applicants must have DDS/DMD, be eligible for licensure in New York, and be Board certified or eligible. Previous hospital-based experience and experience in the management of medically compromised patients is desirable. Salary and academic appointment are commensurate with qualifications and experience. Montefiore Medical Center is an Equal Opportunity/Affirmative action Employer. Please send curriculum vitae and the names and address of three references to: Steven Chussid, DDS, Director, Division of Pediatric Dentistry, Department of Dentistry, Montefiore Medical Center, 111 East 210 Street, Bronx, NY 10467.

Oregon-Portland—A full-time tenure-track position will be available January 1, 1997. Primary responsibilities of the position are didactic and clinic teaching, in the undergraduate curriculum. Two or three days per week will be committed to undergraduate clinical teaching and associated didactic duties. Preference will be given to candidates who have the potential to establish and maintain a productive research program. A DDS/DMD degree from an ADA accredited dental school is required. Board eligibility in pediatric dentistry and a PhD is a relevant biologic discipline are desired. Specialty licensure is available in Oregon and dental licensure is required for the position. One day per week intramural faculty practice is required. Academic rank and salary will be commensurate with credentials. The Oregon Health Sciences University is a EEO employer. Send a curriculum vitae and name, address, and phone number of three references to George Riviere, DDS, PhD, Chair, Department of Pediatric Dentistry, School of Dentistry, Oregon Health Sciences University, Portland, OR 97201-3097.

Pennsylvania-Danville—The Geisinger Medical Center invites applicants for a full-time staff, associate position in the Department of Dental Medicine and Surgery. Geisinger is a tertiary care hospital with a large out-patient clinic facility and Children's Hospital located in rural central Pennsylvania. Applicants will assume responsibility for an established pediatric Dentistry practice in a modern, multispecialty dental clinic. A highly motivated, productive individual with hospital experience and skills in treating medically compromised patients is desired. Applicants must have a Pennsylvania license or eligible for licensure by the Pennsylvania Board of Dental Examiners. Individuals must be board eligible or certified by the American Board of Pediatric Dentistry.

A competitive salary and attractive benefit package are offered. Interested individuals should forward a CV to Kurt Scott (PD-DU), Director Physician Recruitment, Geisinger, 100 N. Academy Avenue, Danville, PA 17822-1528 or FAX to 800-622-2515. Phone inquiries may be directed to 800-845-7112. E.O.E. M/F/H/V.

FELLOW PROGRAM

Pennsylvania-Philadelphia—Applications are now being accepted for a one-year Pediatric Dentistry Fellow Program. The program includes extensive clinical and hospital experience—in-patient, out-patient, and operating room—and is based at The Children's Hospital in Philadelphia. Lectures, seminars, and rounds will be offered at Children's Hospital, as well as the University of Pennsylvania. Participants may also spend a portion of their time teaching undergraduate dental students from the University of Pennsylvania School of Dental Medicine. The Fellowship offers vast experience and opportunities to treat special patients as well as healthy children. Sedation techniques and treatment in the operating room are emphasized. Candidates completing a Pediatric Dentistry Specialty Program will find this an excellent opportunity to practice and at the same time to augment and complement their clinical skills and knowledge base. Candidates who are now graduating from an accredited dental school will find the program offers exceptional exposure to all aspects of Pediatric Dentistry and care of children. The Fellowship provides outstanding preparation for future Pediatric Dentist specialty training. All candidates must be graduates from an ADA-accredited dental school. Send curriculum vitae, dental school transcript, Postgraduate/Residency transcript, if applicable, and three references to Dr. Mark L. Helpin, Chairman, Pediatric Dentistry, University of Pennsylvania, School of Dental Medicine, 4001 Spruce Street, Philadelphia, PA 19104-6003. Application deadline is February 17, 1997.

NEW PEDIATRIC DENTAL PUBLICATION

The Southeastern Society of Pediatric Dentistry is pleased to announce a new publication, The Journal of the Southeastern Society of Pediatric Dentistry (JSSPD). Its mission is to promote and improve the practice of pediatric dentistry. For a complimentary copy and subscription information contact: Dr. Edward S. Nacht, Editor, JSSPD, 7400 N.W. 5th Street, Plantation, FL 33317.

INDEX TO ADVERTISERS

ABSTRACTS *Continued from page 312*

B conventional enamel bond was applied. Vertical increments of Estilux Posterior over enamel bond restored cavities of group C. A fluted carbide bur and Sof-lex discs finished all restorations. Criteria developed by Cvar & Ryge (1971) were used for clinical evaluation of 39 restorations (12, 16 and 11 of Groups A, B and C respectively). No complaints of pain or discomfort were reported during the 6-30 months (mean 15.3) follow-up period. All groups presented excellent surface appearance. Ninety-two percent of group A and 100 percent of groups B & C presented excellent marginal adaptation. Anatomic form was excellent in 92 percent, 8 percent and 100 percent of groups A, B and C respectively. The underlying amalgam was visible through the composite of groups A & B reducing the percentage of excellent ratings of color match to 33 percent and 38 percent respectively, with 9 percent in Group C. This difference was significant ($p<0.02$). Secondary caries was observed in two teeth (group A & C). Radiographs presented radiolucent area at the amalgam-composite interface only in one restoration (group A) and one at the tooth-composite interface (group C). Bubbles were found in 6.6 percent of group A; 12.5 percent of group and 64 percent of group C ($p<0.01$). This study detected differences between the groups only in color match and the presence of bubbles.
Amalgam-composite restorations; Primary molars

Nagatani, Scott S.; Fisher, Jon G.; Hondrum, Steven O.: Effect of heat treatment on stainless steel lingual arch appliances. J Dent Child, 63: 346-349, September-October 1996.
This study evaluated the effect of heat treatment on 0.036" diameter stainless steel wire. Forty wires were bent into arch forms (20 experimental and 20 control). The arch width changes were measured and the forces generated were determined over an eight week period. Heat treatment resulted in immediate and significant expansion ($P< 0.001$) followed by stabilization of the arch width. The control wires continued to expand throughout the study. The force generated by the control group and experimental group expansion was capable of producing tooth movement.
Orthodontic wires; Orthodontic appliances; Space maintenance; Orthodontics, arch width

Bayardo, R.E.; Mejia, J.J.; Orozco, S. *et al*: Etiology of oral habits. J Dent Child, 63:350-353, September-October 1996.
The pedodontic admission histories of 1600 Mexican children were analyzed, to determine general epidemiologic factors or oral habits, as well as their relationship with identifiable biopsychosociologic factors.

Fifty-six percent of the children gave evidence of an oral habit, with a significant predisposition among female patients, single children, subjects in poor physical health (particularly from allergies), as well as children with histories of chronic health problems.

Oral habits should be considered a major health hazard because of their high incidence. Successful treatment requires a multidisciplinary approach to the basic cause of the problem.
Oral habits; Epidemiologic factors; Biopsychosociologic factors

Waldman, H.B.: Low birth-weight and the relationship to developmental problems. J Dent Child, 63: 354-357, September-October 1996.
A general introduction is provided into the causes, prevalence and long term consequences of low birth-weights. Although the vast majority of low birth-weight children have normal outcomes, as a group they have higher rates of subnormal growth, illnesses and neurodevelopmental problems. An increased awareness of the consequences of low birth-weight is needed as dentists provide services for this population of youngsters.
Low birth weight; Developmental problems

Waldman, H.B.: Poor children remain poor. J Dent Child, 63:358-361, September-October 1996.
A longitudinal national study emphasizes the greater levels and longer terms of poverty experienced by children.
Poverty; Children

Goho, Curt: Neonatal sublingual traumatic ulceration (Riga-Fede disease): Reports of cases. J Dent Child, 63:362-364, September-October 1996.
Neonatal sublingual traumatic ulceration (Riga-Fede Disease) presents as a painful ulceration of the ventral tongue caused by the sharp incisal surfaces of neonatal teeth. It can interfere with infant suckling and neonatal nutrition. Ideal treatment resolves the soft tissue ulceration and maintains the tooth, although in severe cases neonatal tooth extraction may be required. Case reports present different presentations of tongue ulcerations, dictating different treatments.
Neonatal teeth; Traumatic ulceration

Duncan, Karen and Crawford, Peter J.M.: Transposition and fusion in the primary dentition: Report of case. J Dent Child, 63:365-367, September-October 1996.
Transposition of teeth in the primary dentition is said not to occur. By contrast, fusion of teeth is the commonest anomaly of number or form to affect this dentition. A case is reported where there was both transposition and fusion of the primary upper right central and lateral incisors. Details of subsequent anomalies in the permanent dentition are also documented.
Primary dentition; Transposition; Fusion

News items

Frank Kihn recognized

Frank Kihn was recognized by the Maryland Society of Dentistry for Children for his many years of leadership and service to the Maryland unit and to children's causes in general. Dr. Kihn is an ASDC life member and in his 39th year of membership.

Speaking at the same occasion was Dr. Pat Fosarelli, a pediatrician with a Master's degree in Theology. Her topic was "Raising moral children in an immoral world." She explored how children think and learn and how sensitive they are to everything in their environments. Guidelines for parents: (1) Know your child's friends and families; (2) Believe what your child says; (3) Model the best behavior possible; (4) Make rules easy to understand; (5) Beware of your behavior, practice civility at all times.

Steven P. Perlman elected

The Academy of Dentistry for Persons with Disabilities elected Dr. Steven P. Perlman of Lynn, MA, as president for 1996-1997. He is in private practice and on the faculty of Boston University. Dr. Perlman is founder and dental director of the Special Athletes, Special Smiles program of the Special Olympics.

Parents Beware!

Perhaps you've never heard of "huffing"—the inhaling of the propellant in aerosol cans. According to an article in May-June *The Occlusal Review*, aerosol products are attractive to children as a way to get high and are the most abused substance among grade school children.

Author Richard Heiss, MD, knows the tragedy of huffing firsthand: he lost his 12-year-old son to aerosol propellants in December 1995. His comments appeared in the newsletter of the Kern County (California) Dental Society.

Aerosol propellants, Heiss says, cause oxygen starvation to body tissues. This can be followed by unconsciousness and cardiac arrest.

Heiss urges parents, grandparents and teachers to educate themselves about this problem and to teach children to avoid such substances. He says concerned citizens can begin to fight this problem by removing aerosols from their homes and replacing them with pumps, roll-ons and non-aerosol products.

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This offer expires December 31, 1996 and is limited to one free sample pack per person.

President's Message

As I write this President's Message I am finishing my final preparations for the 1996 ASDC Annual Meeting. This is a bittersweet time in my life. In a few weeks I will be ending the highlight of my dental career, my year as ASDC President. This is a position I desired since the early 1980s when I was a member of the ASDC Executive Council. I worked hard for ASDC since that time with the hope that I could lead this great organization and "make a contribution" to ASDC and dentistry for children. Well, my year is almost over and I have been reflecting on whether or not I have made this contribution. This is a question that I cannot answer, but I can say that I have continued to work for the objectives that are mentioned in the ASDC mission. I guess only time will tell whether I have made a contribution.

In a way I am grateful to pass the gavel to my friend John Willis. The responsibility of being President of an organization in existence for almost 70 years is very humbling. The pressure of this responsibility is greater than I imagined. The ability to implement programs and policies adopted by the Board of Trustees is a power which is accompanied by many hours of worry and anticipation. I am very fortunate to have had a hard working Executive Committee, all of whom will be better Presidents than I. The National Office Staff, including our editor George Teuscher, have made this year very enjoyable. But, I am ready to pass the mantel on to others.

I have had the occasion to talk to many colleagues over the past few months about ASDC, its direction, and its future. Some people, both ASDC members and non-ASDC members, are uncertain about ASDC's future and its place in organized dentistry. Well, for all those who are concerned about the "health" of ASDC allow me to state its health status. ASDC is a sprightly 69-year-old, who is looking forward to "life" in the 21st Century. ASDC's place in dentistry has not changed since its inception in 1927. ASDC is an organization composed of ALL dentists who treat children and is focused on the health and well-being of all children. ASDC's reasons for being created still drive it 70 years later. The dissemination of knowledge to ALL dentists, parents, and society about the dental care of children is its paramount mission. ASDC has the unique position of counting general dentists and pediatric dentists in its membership and leadership. It's place is to continue to fulfill its mission established in 1927.

To update you on past and upcoming activities, allow me to present this short synopsis. The second ASDC Educational Seminar of this year was presented in San Antonio on Saturday September 7th. As was the case with our first seminar, presented in May in Chicago, this seminar proved to be a grand success. The San Antonio seminar, which was presented in collaboration with the Texas Unit of ASDC, had over 100 participants. Of this number, 50 participants were new members. This amounts to a total of approximately 200 participants, and over 60 new members, at both educational seminars. Kevin Donly and Bob Henry deserved a great thank you for their efforts. I would like also to thank Ted Croll, Catherine Flaitz, Bob Henry, and Carolyn Wilson for presenting at the seminar.

I hope that many of you attend the 1996 ASDC Annual Meeting in New Orleans. In addition to my swan song, the meeting will be one of the best ASDC has ever presented. In addition to the main scientific program, featuring C. Richard Bennett, Kevin Donly, and Leonard Carapezza, other presentations are planned. Breakfast with the Experts will again to be a part of the program, featuring Martin Davis, Bill Posnick, Bob Musselman, Jimmy Pinkham, and John Nathan. In addition, Mini-clinic and table clinic presentations will to be a meeting highlight. Meeting dates are October 23-27, with the Westin Canal Place serving as our meeting hotel.

The ASDC Strategic Plan is now completed. Hallelujah!!! The ASDC Strategic Plan Steering Committee worked very diligently on this project. Ten past presidents and several members have also reviewed the plan and their insights have been incorporated into the final product. I would like to thank all of you who have participated in this project. I realize that planning exercises are less than enjoyable and I sincerely appreciate your assistance. The completed plan will to be presented to the House of Delegates at the upcoming Annual Meeting. I hope that this plan will serve as a guidebook for the near future of ASDC.

Before I close this President's Message, I must mention something which is personally very sad. This coming organizational year will be the last year that George Teuscher will serve as editor of the *Journal of Dentistry for Children*. The Journal has been the cornerstone of ASDC and under George's editorship it has become the prominent journal in dentistry for children. A George Teuscher cannot be replaced. But, his influence will be felt for many years to come. A search committee is now being planned to begin a nation-wide search for a new editor. George is a member of this committee, so I feel assured that the new editor will be very competent.

I hope that I see you in New Orleans.

Peter J. Fos

EDITORIAL

Place the problem where it belongs

If we accept the premise that a child's misbehavior presents the opportunity to teach and to discipline without punishment, we are in a position to instruct the child in the necessary skills for independent living. Acquiring those skills comes naturally to some children, while others need a great deal of assistance. Williamson asks "How will your child learn to get along with other children, get organized for school, pay attention to the teacher, recognize danger, be responsible at home? These things need to be taught, and for your child, they are as important and as difficult as any academic subject. As a parent, your great gift is that you are the one who is able to help your child learn these necessary skills." (Williamson, Peter: *Good kids, bad behavior.* New York: Simon and Shuster, 1990, p 62)

As adults we should consider a child's tantrums and rudeness as signs that he has much to learn about life and about living happily with other people. None of us would tolerate tantrum behavior from another adult. It should not be tolerated from the child, in the child's best interest. Temper tantrums are not uncommon in the offices of pediatric dentists. Tantrums and other forms of bad behavior often occur in bizarre forms. Lives there a pediatric dentist who has not had to cope with the child who vomits or threatens to do so, in protest to examination or treatment?

These children are unable to regulate their behavior appropriately, and are in need of help, preferably from a parent or some other caring adult. These children are manipulating their respective environments to conform to their desires. As long as they remain comfortable in them, they will have no wish to change their behavior.

The parent must supply the impetus to change, and to help the child feel the need for change. The child who vomits in the dental office will see little need to change, if he succeeds in avoiding treatment and if someone other than he cleans up the mess. The problem of cleaning up should be the child's problem—not the dentist's, not his assistant's, not the parent's. As adults (parents, teachers, health professionals) we can make the consequences of his behavior either highly desirable or highly undesirable. The child must make the choice.

Williamson points out that "Behavior management works only when children feel a sense of urgency to accomplish some goal.... If your child feels the urgency and is ready to work, then all is going well. If you start feeling the urgency to "help" your child succeed, however, stop and immediately examine what you are doing. When you feel the child's urgency slipping away, you can fall into a trap by trying to replace it with your own....the more you express your own urgent feelings, the more you can feel his urgency fade into bland indifference." (ibid pp 65,66)

It is important that as adults attempting to help children learn, we keep complete control of our own emotions: Do not use your own emotions to persuade the child to behave more properly; do not give vent to your own negative feelings, thus allowing the child to remain in the spotlight; and do not allow frustration and anger to affect your attitude toward the child.

And of great importance: Do not confuse rewards with bribes. They differ and the difference is of great importance. Bribery is doomed. Positive reinforcement will work, if approached with the proper attitude.

In the course of a day, adults working with children have several opportunities to teach them important lessons about life, such as getting along with other people, and building moral conduct and developing character. John Dewey said that moral education is most potent when lessons are taught to children in the course of real events, not just as abstract lessons - the mode of emotional literacy (Moral lessons in *John Dewey: Religious faith and democratic humanism.* New York: Columbia University Press, 1991).

Character development is the foundation of democratic societies and the foundation of character is self-discipline and self-control. In teaching these important lessons to children, however, we need to put aside self-centered impulses, thus opening the way to empathy, and real listening. If we can develop in our children the attitudes of caring and mutual respect, we will be on the road to a more democratic society.

GWT

REVIEWS

Nutritive and nonnutritive sucking habits: A review

Huguette Turgeon-O'Brien, PhD
Diane Lachapelle, MSc
Pierre F. Gagnon, MSc
Isabelle Larocque, MSc
Léo-François Maheu-Robert, DMD

ANATOMY AND PHYSIOLOGY OF SUCKING

The habit of sucking is a reflex occurring in the oral stage of development and disappears during normal growth between the ages of 1 and 3 1/2 years.[1,2] It is the first coordinated muscular activity of the infant. In the newborn, the tongue is relatively large and positioned forward during normal sucking. The tip of the tongue actually protrudes through the anterior gum pads and takes part in the anterior lip seal. This type of swallowing is termed infantile or visceral swallowing.[3] As indicated in Figure 1, many muscles are involved in sucking and swallowing. The musculature includes mainly the two parts of the orbicularis oris, the buccinator, and its continuation beyond the pterygomandibular raphe into the superior pharyngeal constrictor.[4]

With eruption of the incisors at approximately six months of age, the tongue position starts to retract from the anterior gum pads, shifting to adult or somatic swallowing.[3] It is characterized by decreased use of the perioral muscles, because the teeth and the lips are in contact and the tongue remains within the confines of the dental arches.

Dr. Turgeon-O'Brien and Ms. Larocque are with the Groupe de Recherche en Nutrition Humaine and Dr. Maheu-Robert, Mr. Gagnon and Ms. Lachapelle are with the Faculté de Médecine Dentaire, Université Laval.
The authors acknowledge with gratitude the participation of Suzette Patry from the "Service des Ressources Pédagogiques, Université Laval" for the drawings and illustrations.
This work was supported by the Fonds Emile-Beaulieu, Faculté de médecine dentaire, Université Laval, Québec, Canada.

There are essentially two forms of sucking: the nutritive form (breastfeeding, and bottle-feeding) which provides essential nutrients, and the nonnutritive form, which ensures a feeling of well-being, warmth, and a sense of security (Figure 2). Nonnutritive sucking is probably the earliest sucking habit adopted by infants in response to frustration and to satisfy their urge and need for contact.[5,6] Children who neither receive unrestricted breastfeeding nor have access to a pacifier may satisfy their need with alternative habits such as finger-sucking or sucking of other objects (a blanket or toy), which might be detrimental to their dentofacial development.

NUTRITIVE SUCKING

Breastfeeding

Nutritive sucking includes breastfeeding and bottle-feeding. Each of these types of feeding influence differently the dentofacial structures of the infant. During breastfeeding, the baby bites the short gland of the mother's breast as a flat plane. The baby is able to regulate the flow by biting and releasing the gland. In the infantile or visceral swallowing, the tongue exerts a progressive pressure (see arrows on Figure 3) toward the rear in order to drain the milk out of the lacteal ducts. The mother's milk is then released into the oral cavity, where it is predigested by the saliva and then enters the digestive tract. According to Woolridge, breastfed babies are better oxygenated and less stressed by this sucking rhythm than babies who are bottle-fed.[7] Meier and Anderson reported from research with five preterm infants,

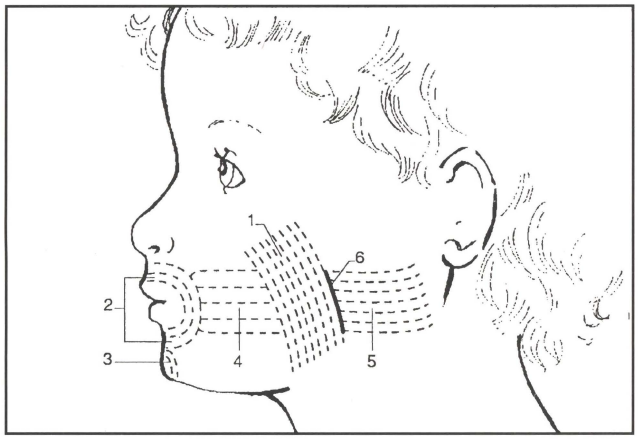

Figure 1. The main muscles involved in sucking and swallowing: (1) masseter, (2) orbicularis oris, (3) chin muscle, (4) buccinator, (5) superior pharyngeal constrictor, (6) pterygomandibular raphe.

who served as their own controls, that they learned the coordinated rhythm of breastfeeding faster than they learned to bottle-feed.[8]

Breastfeeding also promotes a better orofacial development. There is a more rapid mandibular protrusion movement in the breastfed baby than in the bottle-fed baby.[9] In certain infants, the infantile swallowing persists well beyond six months of age. According to Graber *et al*, it appears that several factors can account for the persistence of infantile swallowing patterns.[3] Separately or in combination they may be due to finger-sucking, bottle-feeding, and mouth-breathing. The data from the 1981 National Health Interview Survey (Child Health Supplement) were analyzed to assess the association between breastfeeding and malocclusion.[10] The authors noted that there is a strong indication that breastfeeding, especially if prolonged, does protect against malocclusion. They concluded that "oral facial development may be one health issue on which breastfeeding may have measurable impact."[10] The extent to which health educators integrate these preventive dental concepts in their teaching, however, could be improved. A pilot-study including 127 community health centers in the province of Quebec, indicated that 43 percent of the respondents made no recommendation concerning proper infant oral sucking practices.[11]

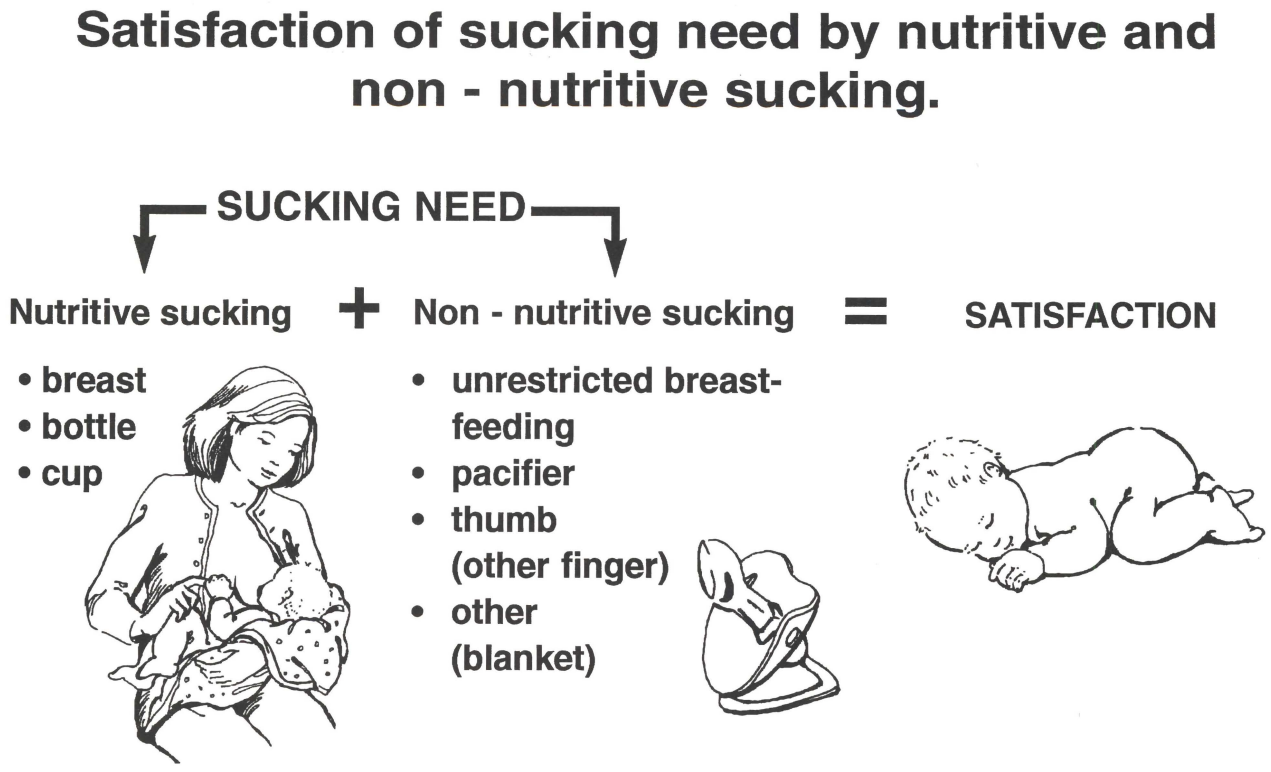

Figure 2. Satisfaction of need for sucking by nutritive and nonnutritive sucking.

Figure 3. Normal swallowing patterns. The tongue exerts a progressive pressure (see arrows) toward the rear in order to drain the milk from the lacteal ducts.

N.B.: Figure 3 is adapted from : Woolridge M. The anatomy of sucking. Midwifery 2; 167–171, 1986.

BOTTLE-FEEDING

The effects of bottle-feeding on dentofacial development vary according to the type of nipples used. As indicated in Figure 4, artificial nipples are available in various sizes and shapes. A comparison between existing nipples reveals important differences in the length and form of the nipple, the location of the holes, the rate of liquid flow, and the flexibility. Also, some nipples are physiologically designed and referred to as "orthodontics."

When a nonphysiologically designed nipple is used (Figure 5a), the end of the nipple is almost against the pharyngeal wall.[7,8] The liquid is then released almost directly into the digestive tract, decreasing the period of predigestion. Also, the flow of milk may be too rapid because of the size of the openings or the number of holes in the nipple. Moreover, the holes may be enlarged by parents, in order to increase the milk flow. Consequently, the infant will either force the nipple out of his mouth, or regulate or stop the flow with the tip of his tongue. Thus, certain muscles involved in breastfeeding are either immobilized (orbicularis oris, masseter), overactive (chin muscle) or malpositioned (the tongue is pushed backward) (Figure 1) and may produce an abnormal dentofacial development in the child.[10] Davis and Bell found a highly significant association between bottle-feeding and incorrect relationship of the teeth in the anteroposterior plane.[12]

With the physiologically designed nipple (Figure 5b), there is a forward movement of the tongue under the flat surface of the nipple that draws it backward and upward against the hard palate of the infant. Consequently, the child has to work and exercise the lower jaw. The posterior part of the tongue then awaits the milk and pushes it down into the esophageal area (Figure 5b). Thus, the milk flows due to the peristaltic-like action of the tongue and the cheeks, instead of being squirted into the throat, which occurs when an inadequate nipple is used. The flat shape of the physiologically designed nipple also improves the labial seal.[8]

The physiologically designed nipples seem better adapted to the anatomy and physiology of sucking. According to Gegovic and Ostric, nonphysiologic nipples do not require the same degree of activity of oral and perioral muscles as required in breastfeeding and more adequate nipples.[12] More studies are needed, however, to determine the influence of different types of nipples on dentofacial development.

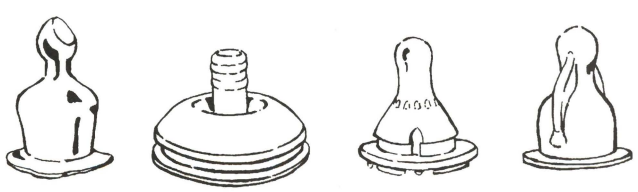

Figure 4. Nipples used for bottle-feeding.

FACTORS INVOLVED IN THE CHOICE OF FEEDING

Factors found to be related to breastfeeding or artificial feeding are diversified. In a recent study carried on with 3,285 infants living in Shangai, it was found that lower and higher birth weights, delivery by caesarian, high level of parental schooling, and male infants were significant predictors of artificial feeding.[14] Nonsmoking in the mother was also found to be positively associated with

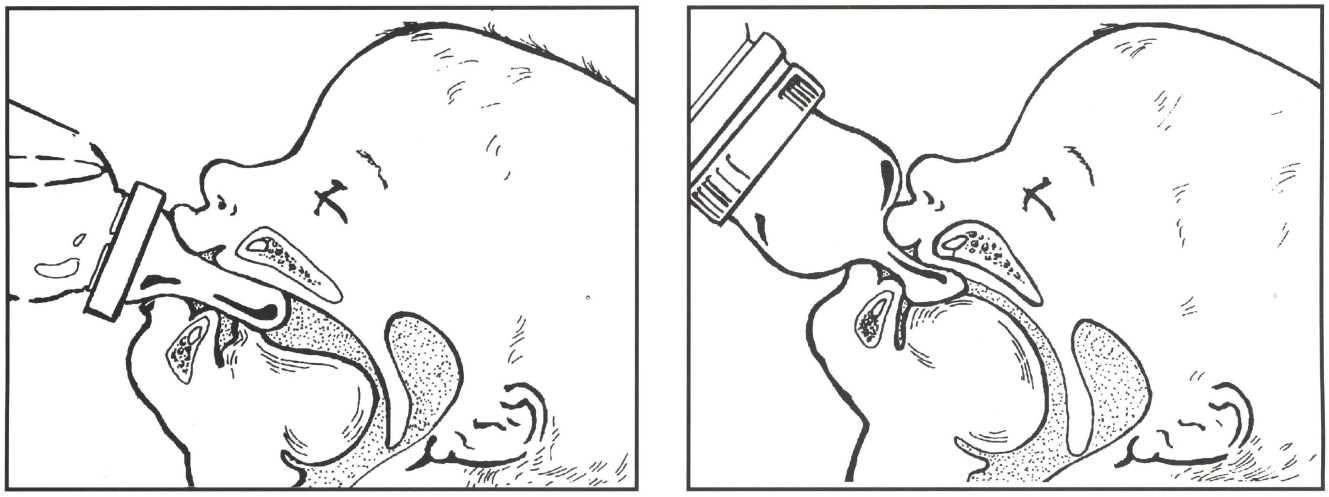

Figure 5. Bottle-feeding. (left) Nonphysiologically designed nipple: improper labial seal, incorrect tongue position, physiologic action of the tongue is impossible. (right) Physiologically designed nipple: improved labial seal, more natural tongue position, physiologic action of the tongue is also impossible.

breastfeeding.[15,16] During a study done in Quebec City, a telephone questionnaire was administered by nurses to 879 mothers of newborns.[16] Among the 666 babies whose mothers did not smoke, 62.6 percent were breastfed. This proportion declined respectively to 37.7 percent, 17.7 percent and 14.6 percent among babies whose mothers smoked one to ten, eleven to twenty and more than twenty cigarettes per day.

NONNUTRITIVE SUCKING

Pacifiers and finger-sucking

Children who do not have access to unrestricted breastfeeding or who are bottle-fed may satisfy their instinctive sucking urge with a pacifier. Figure 6 illustrates pacifiers available on the market. There are many sizes and shapes from which to choose, depending on the baby's age. The physiologically designed pacifier also called "orthodontic pacifier," offers, according to the manufacturer, many advantages: it conforms to the baby's lips, while the smooth and adapted contour promotes nasal breathing. Also because of its flat nipple, it simulates the shape of the mother's breast, allowing the tongue to touch the palate in a more natural sucking position and improving lip seal.

Very few scientific data are presently available to substantiate the desirability of the functionally or physiologically designed nipple over the conventional one. A recent study showed some minor differences between the occlusions of ninety-five children, twenty-four to fifty-nine

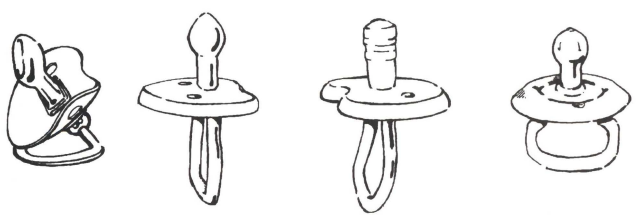

Figure 6. Pacifiers available on the market: (left to right) "Orthodontic" model or "Physiologically" designed model; three conventional models.

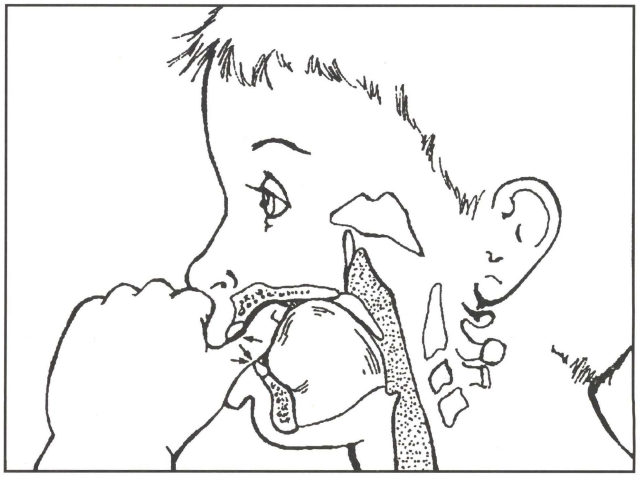

Figure 7. Thumb-sucking. Thumb, lips and tongue pressures (see arrows) can cause detrimental effects on dentofacial development.

months of age, who used orthodontic or conventional pacifiers and a control group composed of children who had no sucking habits.[17]

Few data are available on the percentage of utilization of the different types of pacifiers. According to some studies carried on in England and Finland, about 20 percent of infants use a pacifier, largely discontinued by school age.[18,19] To our knowledge, we have no information concerning the proportion of Canadian infants using a pacifier, nor the length or frequency of utilization.

With preterm infants, Anderson showed that the ones who used a pacifier lost a lower percentage of birth weight; had earlier and more organized sucking patterns; greater weight gain with equal calories; earlier easier and more rapid bottle-feeding; more rapid gastrointestinal transit; more bowel movements; earlier discharge home; and a higher weight and head circumference at one year.

There is also a relationship between the use of pacifiers and the prevalence of finger-sucking. During the last decade, according to studies with Swedish children, the prevalence of dummy suckers has increased greatly, while at the same time the number of children with finger- or thumbsucking habits has diminished.[5,21-25]

The influence of the type of feeding (breastfeeding or bottle-feeding) on the acquisition of a finger sucking habit has been studied by some investigators. Studies carried on between 1949 and 1971 reveal that bottle-feeding is positively correlated with finger-sucking, whereas other studies failed to find such a relationship.[26-31] According to Bishara, this controversy might be partly explained by the fact that there are no longitudinal studies describing the modifications in the dental arches of children fed by different methods. Nevertheless, the long-term effects of finger-sucking habits are important, because children tend to maintain this habit up to the age of seven or more, whereas children using a pacifier stop this habit during the first five years of life.

During the last forty years, studies have been carried on to determine the incidence of fingersucking habits in children. According to some studies, the incidence of digit-sucking is about 30 percent in one-year-old children, with approximately equal numbers in both sexes.[32] According to Brenchley, this percentage decreases to 12 percent by nine years of age and 2 percent by 12 years of age.[18] On the contrary, other studies indicate that about 50 percent of those who start a finger-sucking habit still do so at seven years of age.[21,25,33] According to Larsson, the prevalence of prolonged finger sucking habits varies greatly for nine-year-old children, prevalences of 30-40 percent have been reported in some studies.[2] Also, lower incidence is found in black children, and the habit seems to endure longer in girls.[18] More studies are needed to determine the prevalence of finger-sucking habits, and the extent of involvement of many factors, because of the potentially detrimental effects of the habits on dentofacial development.

Detrimental effects of sucking habits

Deleterious effects caused by certain sucking habits or the incorrect use of pacifiers, as when the shield is partly positioned inside the lower lip, can cause undesirable effects on the dentition and even affect the bone of the anterior part of the mandible.[21]

Also the more or less continuous presence of the thumb or finger in the oral cavity can exert sufficient pressure to deform the maxillary arch, or palate, or both.[34] There is a strong agreement that digital sucking habits that persist while permanent teeth erupt can have detrimental effects on dentofacial development.[34-37] For the first three to four years of life, studies indicate that damage to the occlusion is confined largely to the anterior segment.[21,23,38] These damages are usually temporary, provided that the child starts with a normal occlusion. Researchers mentioned that if the sucking habits are discontinued before the age of six years, dental changes are often transitory, whereas those children who stop after six years of age invariably have malocclusions at twelve years of age.[39,40]

The possible effects of sucking habits on the oral structures are determined by the direction and the nature of the force that can vary greatly (Figure 7). The effects of sucking habits on maxillary and mandibular development and on the dental arches depend upon the frequency and the intensity with which the habits are practiced, the duration of the habit, the osteogenic development, and the genetic endowment of the child.[35,39,41] The etiology of an abnormal swallow is not yet well understood. It can be a learned habit caused by improper feeding techniques during infancy, prolonged pacifier use, finger-sucking or a combination of these factors.[3,27,42] Whatever the cause of the problem, it is commonly accepted that abnormal tongue posture and strong tongue thrusting can have detrimental effects on normal skeletal development and dental positioning.[1-3,27,40,42,43]

Numerous studies on the relationship between nutritive and nonnutritive sucking and malocclusion have been performed, but the results are not conclusive. Also, a direct cause-and-effect relationship between tongue posture/tongue thrust swallowing and malocclusion needs further research.[1,41,44] Up to now, studies have determined significant correlation only in the primary dentition between digit sucking and class II malocclusion, and be-

tween digit sucking and posterior lingual crossbites.[22,24,33,34,42,44] Anterior open-bite and nonocclusion can affect the periodontium. It may be related to habits such as finger-sucking and/or tongue thrusting, when swallowing leads to open bite.[45] Other possible damages have been reported in the literature in relation with the general health of the children.[5,21,24,35,36,45] Also, chronic thumb-sucking in school-age children, which is sometimes associated with strong attachment to an object (e.g. blanket, toy), may also reduce peer social acceptance, an important contributor to social development.[46,47]

Health professionals should consider the teaching of the subject of oral habits as part of dental prevention programs. Specific recommendations must be addressed to the parents on the child's sucking need and it's role in oral muscular activity. Breastfeeding must be considered as an infant's best method of feeding, and the prevention of malocclusion is one additional benefit of breastfeeding. It seems that physiologically designed nipples or pacifiers would be better than conventional ones, but more research is necessary to substantiate the desirability of one design over another.

The effectiveness of prenatal dental education has been reported.[48] Factors affecting oral health behaviors and interventions are numerous, and many of them are issued from the family environment.[49,50] Multidisciplinary studies involving psychologists, sociologists and allied health professionals are necessary to understand better all the factors involved in oral health.[50]

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FLUORIDE AND CARDIOVASCULAR DISEASE

One of the most striking developments in recent public health history is the sharp decline in the standardized death rate due to cardiovascular disease, particularly ischemic heart disease, in the USA [Gordon and Thom, 1975; Taves, 1978] and several other countries [Guberan, 1979]. The decline first became apparent in the early 1960s, some 20 years after controlled water fluoridation had been started in the USA and, while the phenomenon continues, its cause has yet to be determined. The decline is not attributable to population changes in any of the major risk factors. There are both epidemiological [Bernstein et al., 1966; Taves, 1978; Luoma, 1980] and laboratory data [Taves and Neuman, 1964; Zipkin et al., 1970], however, which suggest the involvement of fluoride through its ability, at relatively low levels, to inhibit soft tissue calcification. There is a clear need for in-depth research in this important area.

In addition to its established cariostatic effect and its possible preventive or therapeutic roles in other major diseases, fluoride is a hazardous substance when large doses are taken acutely or when lower doses are taken chronically. Its effects range from dental fluorosis [Fejerskov et al., 1977; DenBesten and Thariani, 1992], reversible gastric disturbances [Jowsey et al., 1979] and transient reductions in urinary concentrating ability [Goldemberg, 1931; Whitford and Taves, 1973] to skeletal fluorosis [Singh and Jolly, 1970] and death [Hodge and Smith, 1965; Church, 1976; Dukes, 1980; Eichler et al., 1982; Gessner et al., 1994].

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Dens evaginatus: A review

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Dens evaginatus is a developmental dental anomaly that can be defined as a tubercle or protuberance from the involved surface of the affected tooth. It consists of an outer layer of enamel, a core of dentin and may contain a slender extension of pulp tissue.[1-4] This condition is clinically important because fracture or wear of the tubercle frequently leads to the major complication of *dens evaginatus*, pulp necrosis and early periapical infection, often before completion of root formation.[1-21] Several methods of treatment have been advocated previously, including extraction, selective grinding, prophylactic pulp capping, and sealants. The purpose of this article is to review the literature and discuss the clinical management of *dens evaginatus.*

DESCRIPTION

Since 1974 this anomaly has usually been referred to in the literature as *dens evaginatus*. Stewart and associates believed that the term *dens evaginatus* most accurately describes this anomaly from the standpoint of developmental, morphological, and histological considerations and it is the term that will be used in this article.[1] *Dens evaginatus* has also been referred to as: the simplest type of dilated composite odontoma, occlusal tubercle, Leong's premolar, odontomas of the axial core type, tuberculated premolar, evaginated odontoma, evaginated odontoma and interstitial cusp, supernumerary cusp, or occlusal enamel pearl.[2-11]

Dens evaginatus is most often reported in premolars, but can also occur in molars, canines, and incisors.[5,12,13]

Clinically the tubercle of *dens evaginatus* appears as a pronounced elevation on the occlusal surface of posterior teeth and may occur in the form of a drop, nipple, a pointed or cylindrical cone.[4] In anterior teeth a conically shaped extension of the cingulum is observed.[12]

Oehlers and associates identified five variants of pulpal horns in *dens evaginatus*.[9] These are:

- ☐ Wide pulpal horns.
- ☐ Narrow pulpal horns.
- ☐ Horns constricted pulpally.
- ☐ Isolated segments of pulp.
- ☐ Absence of pulpal horns.

Dens evanginatus has been reported to occur predominately in people of Mongoloid racial stock: Chinese, Thai, Malaysians, Japanese, Indians, Eskimos and Filipinos.[1-7,11,14-20] There are isolated reports of its occurrence in African-Americans and Caucasians.[8,10,13,21]

The prevalence of *dens evaginatus* ranges from 1.09 percent to 4.3 percent.[4,16] Merrill examined 650 American Indian, Eskimo, and Aleut students and reported a prevalence of 4.3 percent. He also compared the results obtained by Kato, Wu and Lau in their studies of Japanese and Chinese populations. Kato reported a prevalence of 1.09 percent among 1,467 Japanese evaluated. Lau and

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The views expressed in this article are those of the authors and do not reflect the official policy or position of the Department of Defense or the United States Government.

Wu reported rates of 1.29 percent and 1.52 percent, respectively, in Chinese populations.[4] Yong states that the prevalence may be higher than reported in the literature as many are likely to be undetected, if they present with the tubercles lost by fracture.[16] Bedi and Pitts sought to overcome this bias by the selection of twelve-year-old patients for their prevalence study. They state that selection of twelve-year-old patients are the most appropriate choice to determine the prevalence of *dens evaginatus*, because the premolars are erupting or have recently erupted. The risk of the tubercle fracturing away before the clinical examination and a false negative recording is minimized. Bedi and Pitts reported a prevalence of 3 percent among the Hong Kong Chinese population.[20] Yip found a prevalence of 3.6 percent among Chinese children.

Dens evaginatus frequently occurs bilaterally.[4,11] Bedi and Pitts reported this finding in over a half of the children they examined. They state that whenever *dens evaginatus* is observed, a careful examination should be done to rule out bilateral occurrence.[20]

PATHOGENESIS

The exact mechanism for the formation of the tubercle of *dens evaginatus* is not known; the theory first proposed by Tratman and echoed by subsequent authors is, however, presented. The tubercle of *dens evaginatus* is believed to be due to proliferation and evagination of part of the inner enamel epithelium into the stellate reticulum of the enamel organ during tooth development. Tooth development proceeds with dentin and enamel formation and apposition creating a tubercle of enamel and dentin containing a horn of pulp tissue.

The familial occurrence of *dens evaginatus* has been reported previously and several authors have suggested a hereditary component in its etiology.[1,4,5,7,22] Stewart and associates documented the occurrence of *dens evaginatus* in several members of a family of Guatemalan Indian descent.[1] The authors state that although the precise mechanism of genetic transmission is not entirely clear, autosomal dominant inheritance is probable.

DENS EVAGINATUS AND TALON CUSP

Previous authors have treated *dens evaginatus* and *talon cusp* as separate anomalies, depending on tooth location. Stewart states that *dens evaginatus* describes an anomaly of posterior teeth.[1] Mellor and Ripa and subsequent authors have described *talon cusp* as an anomaly of the anterior teeth.[23,34] Certain cases previously reported as *dens evaginatus* of anterior teeth have been considered to be *talon cusps* by some authors.[24,35]

Mellor provided a description of *talon cusp* stating that it resembles an eagle's talon in shape and is a cusp-like structure projecting from the cingulum area of maxillary or mandibular incisors. He stated that radiographically it is similar to a normal tooth in that it is composed of enamel and dentin and contains a horn of pulp tissue.[29] Other authors report that the pulpal horn is not always present.[24-27]

Talon cusp has been reported in Chinese, Caucasians and African-Americans.[24-32] There are also reported cases of *talon cusp* in a Hispanic and a Japanese.[33,34]

The reported prevalence of *talon cusp* ranges from 0.17 percent to 7.7 percent.[27,28] This figure is clouded somewhat by the use of different criteria by different authors. Davis and Brook have suggested the following definition: "an additional cusp that projects from the lingual surface of primary or permanent anterior teeth, is morphologically well delineated and extends at least half the distance from the cemento-enamel junction to the incisal edge."

It is proposed that *talon cusp* and *dens evaginatus* are the same anomaly and that *talon cusp* is a subset of *dens evaginatus*. This can be evaluated from a morphological and histological basis.

The morphology of both *talon cusp* and *dens evaginatus* have been reviewed previously and attention is called to their similarity. Histological descriptions of ground sections of *dens evaginatus* and *talon cusp* depict a tubercle of dentin and enamel that frequently contains a slender extension of pulpal tissue.[3,4,6,24,27] Little has been written concerning the pathogenesis of *talon cusp* other than the possible hyperproductivity of the dental lamina.[36] A pathogenesis identical to *dens evaginatus* is postulated; i.e. a proliferation and evagination of part of the inner enamel epithelium into the stellate reticulum of the enamel organ during tooth development. This would be expected if, as proposed, *dens evaginatus* and *talon cusp* are the same anomaly. This would also account for the similar morphological and histological appearance.

DENTAL COMPLICATIONS

The primary dental complication of *dens evaginatus* is fracture or wear of the tubercle which leads to pulp exposure, pulpal necrosis and periapical infection .[1-8,10-12,14-18,21,22,37-39] Pathosis of the pulp can occur before complete root formation with cessation of root development.[4,8]

When the anomaly occurs in anterior teeth it may be

mistaken for a supernumerary tooth and an unwarranted surgical procedure may be done.[23] If a surgical procedure is attempted, the tubercle may be fractured off as a result, and exposure of the pulp may occur.

Interference with the eruption of teeth and displacement of teeth is another complication that has been reported.[4,8,23,25] This could adversely affect the positioning of teeth in the arch, resulting in malocclusion. Sharp, prominent tubercles on anterior teeth may interfere with and irritate the tongue during mastication and speech.[25] Anomalous root patterns such as dilacerations may develop, due to the presence of *dens evaginatus*.[6] Another possible complication is the formation of caries in the fissures and grooves surrounding the tubercle.[25]

REVIEW OF METHODS FOR CLINICAL MANAGEMENT

Several methods of treatment of *dens evaginatus* have been reported in the literature. As early as 1949 Tratman advocated "early recognition of the condition before the pulps are severely infected and the placement of small fillings immediately."[2] This treatment approach has been repeated with various refinements by subsequent authors.

Tratman recommended extraction of the tooth when the patient presented with a fractured or worn tubercle, resulting in periapical infection. He reasoned that endodontic treatment was contraindicated, due to the wide open apex of the affected tooth.[2] Palmer thought that endodontic therapy is often contraindicated, due to abnormal root patterns associated with teeth exhibiting *dens evaginatus*.[8] Other authors have presented additional reasons to avoid endodontic treatment of teeth with *dens evaginatus*. Villa and associates reported two cases of *dens evaginatus* where the patients presented with pain, purulent exudate, and apical rarefaction. In both cases the involved teeth were extracted, due to extensive apical root resorption.[3] In 1974 Senia and Regezi reported a case of bilateral mandibular *dens evaginatus* with periapical involvement. The teeth were extracted, due to incomplete root development and advanced local periodontitis.[15]

Poyton and Vizarra and Curzon and associates have recommended routine endodontic therapy, however, either before or after suspected exposure of the pulpal horn in cases of *dens evaginatus*.[7,40] Senia and Regezi state that "early recognition followed by appropriate therapy can prevent loss of these otherwise normal teeth."[15] They recommend the maintenance of pulp vitality to allow root-end closure, as this would permit uncomplicated root canal therapy later if necessary.

In 1967 Oehlers and associates attempted to treat *dens evaginatus* prophylactically by careful grinding of the intact tubercles of twenty-two vital teeth to stimulate formation of secondary dentin.[9] The results were variable and the authors concluded that this was an unreliable method of treatment. Shey and Fytel thought that grinding the tip of the tubercle would stimulate only a few odontoblasts and would not result in sufficient reparative dentin formation. They advocate grinding the entire lingual surface of the tubercle to increase the surface area of stimulated odontoblasts thus producing more reparative dentin.[12] A possible problem with this method is the thinning of the tubercle, which could weaken it and predispose it to fracture and pulpal exposure.

Hill and Bellis described a case in 1984 where three vital teeth with *dens evaginatus* were treated by removing the enamel on the tubercles to the level of the dentin.[18] Calcium hydroxide was placed in the resulting occlusal cavity and a preventive resin restoration as described by Simonsen was placed. At ten months after treatment, the teeth remained vital. In 1984 Chen described another method of treatment for *dens evaginatus* of vital teeth that involved selective grinding.[37] With Chen's technique the tip of the tubercle is ground in small increments using a high speed handpiece at six-month intervals until the tubercle is clearly free of occlusal interference, when full eruption is achieved. At the first appointment the enamel is removed until the dentin is just exposed. The exposed dentin is desensitized with 8 percent stannous fluoride for five minutes. At subsequent appointments dentin is removed, being careful not to expose the pulp. Following radiographic evidence of dentin deposition, a shallow cavity is prepared and amalgam placed. Chen reported zero incidence of pulp exposure or necrosis with this method. Davis and Brook also reported a case that was successfully treated using a similar method.[24]

Pledger and Roberts state several disadvantages of treating *dens evaginatus* by grinding the tubercle.[29] One disadvantage is sensitivity of the tooth. If the tubercle is reduced gradually, then many of the problems associated with its presence may develop before the reduction is completed. Also the patient may need to be seen a large number of times, which could prove very inconvenient. The authors conclude that grinding of the tubercle may only be successful in cases where there is no pulpal extension into it.

In 1967 Oehlers and associates advocated pulp capping of teeth with *dens evaginatus*, but no cases were presented.[9] In 1974 Yong reported the results of thirty-nine teeth treated by pulp capping.[16] All teeth treated in this manner were vital and asymptomatic preopera-

tively. The teeth were isolated with rubber dams and occlusal cavity preparations were performed. The cavity was lined with calcium hydroxide paste followed by a zinc oxide-eugenol base and an amalgam restoration. Follow-up evaluations were done at one week, one month, six months, and annually. Pulp vitality testing and periapical radiographs were performed at six months and annually. The follow-up period extended to thirty months. The thirty-nine teeth remained vital and asymptomatic throughout this period.

In 1981 Ciechanowski and Sonnenberg reported a case of *dens evaginatus* in a twelve-year-old African-American female with three mandibular premolars affected.[21] The authors utilized a different method of treatment for each affected tooth. One tooth was treated by grinding the tubercle at six-month intervals followed by application of 1.23 percent acidulated phosphate fluoride. The second tooth was treated by Yong's method described previously. The third tooth was treated by application of pit and fissure sealant. These three teeth remained vital and asymptomatic a year after treatment. Bazan and Dawson reported a case of *dens evaginatus* in a vital tooth treated by application of pit and fissure sealant over and around the tubercle to prevent fracture.[14] The authors state that an occlusal cavity preparation was considered unfavorable, because of the difficulty in performing an ideal pulp capping on the resulting large pulp exposure. They were of the opinion that a pulp capping if needed could be done in the future, if the pulpal horn had receded. The authors also note that the tooth has remained vital and asymptomatic six months after treatment. Richardson and Knudson report success using a similar technique.[35]

As noted earlier, when teeth with *dens evaginatus* become pulpally involved before root closure, some authors have advocated extraction as the treatment of choice. In 1976 Priddy and associates recommended apexification followed by conventional endodontic therapy.[22] Stewart and associates made a similar recommendation in 1978; neither group, however, reported any cases treated utilizing this technique.[1] In 1984 Hill and Bellis reported a case that was successfully treated, using the calcium hydroxide apexification technique and followed by conventional nonsurgical endodontic therapy.[17] Chen reported success in three of four cases treated by calcium hydroxide apexification.[37] In the fourth case the apical radiolucency became larger over a six-month period and the tooth was extracted. Shay and Su also reported cases successfully treated using apexification, followed by endodontic therapy.

DISCUSSION

Several methods of treating *dens evaginatus* have been reviewed. An obstacle facing clinicians seeking to evaluate these methods is summarized by Bedi and Pitts.[20] They state that 'the management of *dens evaginatus* is based upon empirical evidence, mostly in the form of single case reports and retrospective reviews of clinical notations. Despite the occasional report of several cases there has, to date, been no controlled clinical trial conducted to evaluate the effectiveness of a particular type of treatment for this anomaly.' The validity of this statement is acknowledged; nevertheless, case reports are all that are available at the present time. An evaluation of these reports is presented.

Stewart and associates grouped treatment of *dens evaginatus* into those techniques employed on vital teeth and those on nonvital teeth.[1] Among the methods reviewed for vital teeth, prophylactic pulp capping as described by Yong and selective grinding appear to be the most effective.[16] Yong treated a relatively large number of teeth compared to other investigators and reported success in all of the teeth that were treated. Several methods of selective grinding were reviewed; the method described by Hill and Bellis, however, seems to be the least cumbersome and may avoid some of the disadvantages of grinding that were described earlier by Pledger and Roberts.[18,29] An additional disadvantage was mentioned by Senia and Regezi, namely the risk of mechanical exposure of the pulp.[15] In light of this consideration, the placement of calcium hydroxide on the floor of the cavity preparation as advocated by Hill and Bellis may serve to increase the success rate of selective grinding, since it is conceivable that there can be a minute exposure of the pulpal extension, undetected at the time the procedure is performed. The placement of the calcium hydroxide acts as a pulp capping in this instance. Taking the above into consideration, a rational and conservative approach to the management of *dens evaginatus* in vital teeth includes early diagnosis and treatment to prevent fracture or attrition of the tubercle that could result in pulpal pathosis. This treatment would include careful selective grinding after the manner described by Hill and Bellis, with rubber dam isolation and pulp capping in the event of direct pulp exposure.[18] A preventive resin restoration has been advocated by Hill and Bellis to restore the tubercle of *dens evaginatus* after selective grinding is completed. The preventive resin restoration would help to prevent caries developing in the fissures and grooves surrounding the tubercle. Use of dentinal

bonding agents may have application here in the prevention of microleakage.

When presented with a case of *dens evaginatus* in a nonvital tooth with incomplete root development, the treatment of choice has been extraction. Several authors have reported successfully treating these types of cases, however, with apexification followed by conventional endodontic therapy.[18,37-39] Extraction may be considered, if the apexification procedure is unsuccessful or contraindicated, or if the tooth is to be removed for orthodontic purposes.

CONCLUSIONS

- ☐ *Dens evaginatus* and *talon cusp* are names for the same anomaly. *Talon cusp* is a subset of *dens evaginatus*.
- ☐ Based on a review of the literature, the most favorable method of treatment of *dens evaginatus* in vital, asymptomatic teeth is selective grinding as described by Hill and Bellis, using rubber dam isolation. The clinician should be prepared to perform a pulp capping, if a pulp exposure is observed.
- ☐ The treatment of choice of *dens evaginatus* in nonvital teeth with incomplete root development is apexification, followed by conventional endodontic therapy.
- ☐ Clinical trials are needed to determine adequately the best method for treating *dens evaginatus*. At the present time, this information is based primarily on case reports.

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CLINIC

Craniomandibular dysfunction in children with clefts and noncleft children with and without unpleasant life events: A comparative study

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The prevalence of craniomandibular dysfunction (CMD) in children with clefts and without clefts has been reported to be high.[1-5] A comparative study, however, to investigate any difference in the prevalence of signs and symptoms of craniomandibular dysfunction between children with clefts and those without clefts, but with different emotional states, has not been published previously.

The purpose of this study is to investigate any difference in the prevalence of signs and symptoms of craniomandibular dysfunction between children with clefts and noncleft children with different emotional states caused by unpleasant life events.

MATERIAL AND METHODS

The sample consisted of 386 white children without clefts and thirty white children with either unilateral cleft lip or unilateral cleft lip and palate, ages six to ten years, selected from the school of Dental Medicine and Children's Hospital of Pittsburgh and the Cleft Palate Center of the University of Pittsburgh respectively. The children were interviewed for symptoms and examined clinically for signs of craniomandibular dysfunction by the same investigator. A questionnaire was distributed to the parents before the examination to collect information related to organic diseases that can cause craniomandibular dysfunction, history of trauma, upper respiratory infection, toothache, and orthodontic treatment. A question was included in the questionnaire of the subjects without clefts to identify children with different emotional states, based on unpleasant life events. On the basis of the answers to the questionnaire, the sample of the children without clefts was divided into the following groups:

- ☐ Subjects without unpleasant life events rated by the parents as calm; this group was referred to as calm group (N=250).
- ☐ Subjects with unpleasant life events rated by the parents as tense, anxious, or under stress; this group was referred to as not calm group (N=105).
- ☐ Subjects with dentofacial injuries either with or without unpleasant life events (N=25).

Six subjects with upper respiratory infection and/or toothache were excluded from the sample. The investigator was unaware of the subjects' classifications during the clinical examinations and interviews. Table 1 shows the unpleasant life events as well as the number and distribution of the subjects.

Regarding the cleft sample, subjects with associated malformations and syndromes were excluded.[6-8] The cleft-lip group had no alveolar clefts.

The clinical examination of the masticatory system included limited maximal opening, temporomandibular joint (TMJ) sounds, TMJ and muscle tenderness. The symptoms recorded in this study were headaches occurring once or more a week; pain in the temple region; or

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Table 1 ☐ Life events and the number and percentage of the examined subjects

Life events	Number of subjects	Percentage
School performance	37	35.2
Divorce	30	28.5
Marital separation	17	16.2
Single parent	10	9.5
Illness in family members	7	6.6
Drinking problems in family	3	2.8
Esthetics	1	.9

when the mouth was opened wide or during chewing, difficulties in opening wide, and clicking. The interview and the clinical examination, related to the function of the masticatory system, and the classification of the subjects without clefts were explained in detail in previous studies.[1,9,10]

With respect to the noncleft children, statistically significant differences in the prevalence of clinical signs of craniomandibular dysfunction were found among subjects with and without dentofacial injuries.[11] The subjects with dentofacial injuries were excluded, therefore, from the study. Also, statistically significant differences in the prevalence of clinical signs of craniomandibular dysfunction were revealed among children with and without unpleasant life events.[11,12] In this investigation, therefore, these groups were studied separately. The children with unilateral cleft lip or cleft lip and palate were considered as one group, since no statistically significant differences in the prevalence of signs and symptoms of craniomandibular dysfunction were found between them.[1]

On the basis of the age and sex distributions of the cleft children called experimental group, a control group I of thirty calm subjects (twenty-two males, eight females) and a control group II of thirty not-calm subjects (twenty-two males, eight females) were identified for comparison in the prevalence of signs and symptoms of craniomandibular dysfunction. In cases where the number of children from the control groups exceeded the number required for precise matching, the selection criterion in each control group was the closest date of child's birth to the respective experimental subject. Table 2 shows the distribution of the subjects by age and sex in the experimental and control groups. The respective differences in the severity of craniomandibular dysfunction between members of the groups were investigated by using Helkimo's clinical and anamnestic dysfunction indices.[13]

Table 2 ☐ Distribution of the subjects by age and sex in the experimental and control groups.

	Age										
	6		7		8		9		10		
Groups	M	F	M	F	M	F	M	F	M	F	Total
Experimental	7	2	6	0	7	1	0	1	2	4	30
Control I	7	2	6	0	7	1	0	1	2	4	30
Control II	7	2	6	0	7	1	0	1	2	4	30
Total	21	6	18	0	21	3	0	3	6	12	90

Table 3 ☐ Prevalence of each sign in the experimental and control groups.

Groups	Limited maximal opening %	TMJ sounds %	TMJ tenderness %	Muscle tenderness %
Experimental	13.3 (4)	20.0 (6)	26.6 (8)	60.0 (18)
Control I	0.0 (0)	13.3 (4)	3.3 (1)	40.0 (12)
Control II	3.3 (1)	13.3 (4)	26.6 (8)	66.7 (20)

*TMJ stands for temporomandibular joint
**The number in parenthesis shows the subjects with the respective sign.

Statistical methods

The data were computerized and the SPSS/PC+ statistical package was used for their analysis. The prevalences of all clinical signs and symptoms, as well as the clinical and anamnestic dysfunction indices, were calculated in percentages. Differences were tested by the chi-square test. The 95 percent probability level was used.

RESULTS

Differences in the prevalence of signs

The frequency of each sign of craniomandibular dysfunction for the experimental and control groups is presented in Table 3. Statistically significant differences were found in the prevalence of TMJ tenderness between the experimental group and control group I ($X^2 = 4.70$, $p = .03$); while the difference between the experimental group and control group II was not significant. Also, the difference in the frequencies of TMJ tenderness was significant ($X^2 = 4.70$, $p = .03$) between the control groups. The differences in the prevalence of other signs were not significant, either between the experimental and control groups, or between the control groups.

Differences in the prevalence of symptoms

Table 4 reveals the prevalence of each symptom for each group. Statistically significant differences were not found

Table 4 ☐ Prevalence of each symptom in the experimental and control groups.

Groups	Headaches %	Difficulties in opening wide %	Pain in Temple region %	Pain in opening wide and chewing %	Reported clicking %
Experimental	16.6 (5)	26.6 (8)	10.0 (3)	36.6 (11)	13.3 (4)
Control I	3.3 (1)	6.7 (2)	3.3 (1)	26.6 (8)	6.7 (2)
Control II	3.3 (1)	20.0 (6)	6.7 (2)	13.3 (4)	6.7 (2)

*The number in parenthesis shows the subjects with the respective symptom.

Table 5 ☐ Clinical and anamnestic dysfunction indices in the experimental and control groups.

	Clinical index				Anamnestic index		
Groups	DiO %	DiI %	DiII %	DiIII %	AiO %	AiI %	AiII %
Experimental	26.7 (8)	16.7 (5)	43.3 (13)	13.3 (4)	53.4 (16)	3.3 (1)	43.3 (13)
Control I	50.0 (15)	30.0 (9)	20.0 (6)	0.0 (0)	73.3 (22)	10.0 (3)	16.7 (5)
Control II	33.3 (10)	20.0 (6)	36.7 (11)	10.0 (3)	70.0 (21)	6.7 (2)	23.3 (7)

*The number in parenthesis shows the subjects with the respective index.

in any of the symptoms, either between the experimental and control groups or between the control groups.

Differences in the severity of signs and symptoms

The frequency of the clinical and anamnestic dysfunction indices is shown in Table 5. The only statistically significant difference was that of the anamnestic dysfunction index, with severe symptoms (AiIII) between the experimental group and control group I ($X^2 = 3.88$, $p = .04$). The frequency was higher in the experimental group. The other differences in the prevalences of the clinical and anamnestic dysfunction indices of the groups were not statistically significant.

DISCUSSION

The composition of the sample in the cleft group reflects the frequency of clefts in males and females. Cleft lip with or without cleft palate is twice as prevalent in males as in females.[14,15] To make groups comparable, therefore, a matching by age and sex was performed. Also, the various criteria for inclusion of the subjects in the study were the same in all groups, minimizing sample heterogeneity.

The methods used in this study (clinical examination, questionnaire, and interview) showed high reproducibility in the children without clefts.[9,10] In addition, the criteria used to identify craniomandibular dysfunction were the same for all groups and the subjects were examined and interviewed by the same investigator, which increased the comparability of the groups.

The present study showed a statistically significant difference in the prevalence of TMJ tenderness between the experimental group and control group I; while the difference was not significant between the experiment group and control group II. This finding implies that the etiologic factor of TMJ tenderness can be the same in children with clefts and with unpleasant life events. Clefts are considered as stress factors of long standing.[17] Jones found significant differences between cleft lip and palate and noncleft subjects with respect to behavior, school status, popularity, happiness and satisfaction, and physical attributes and appearance.[17] Since the correlations between different types of malocclusion and TMJ tenderness were not significant in these groups, it is likely that the emotional states caused by the unpleasant life events, as well as by the impairment of the masticatory system due to clefts, are the etiologic factors of this clinical sign.[18,19] Also, a statistically significant difference in the prevalence of TMJ tenderness was found between control groups I and II. This finding was reported by other studies conducted on the same groups, but with different compositions of the samples. The con-

sistency of this result confirms the conclusion of a previous study: that children with emotional states run a greater risk of developing temporomandibular joint tenderness.[12]

The differences in the prevalence of muscle tenderness were not significant between the experimental group and control groups I and II, respectively, as well as between the control groups. In other studies conducted on the same noncleft children with different compositions of the samples, the difference was highly significant.[11,12] The inconsistency of this finding can be attributed to the fact that emotional states present fluctuations in terms of intensity and duration. It is likely, therefore, that muscle changes produced by a given level of intensity of emotional states and expressed by slight tenderness are reversible, when the level of etiologic factor is dropped. As a result, subjects of this study with levels of emotional states within the range of tolerance did not present muscle signs during the examinations. In contrast, initial changes in temporomandibular joint, like elongation of discal or capsular ligaments expressed by slight tenderness are not reversible.[20] It is also likely that the adaptability of joint tissues to the new conditions is completed later than in the muscles where the level of etiological factor is dropped or eliminated. This may explain the consistency of the significant difference in the frequency of TMJ tenderness found in the present and in previous studies as well.[11,12] The lack of significant differences in the prevalence of TMJ sounds can be explained by the fact that at these young ages the alterations in the joints were at an early stage and joint sounds had not yet developed.

In this study, the minimal value of maximal opening was that determined statistically in noncleft children without craniomandibular dysfunction.[21,22] Four subjects of the experimental group and one of control group II had values of maximal mouth-opening, less than the statistically determined values. The differences among the groups were not significant. It should be pointed out, however, that limited mouth-opening may be dysfunctional, whereas a dysfunctional opening may not be limited.[23] In addition, it has been reported that it is not possible to fix minimal values of the mandibular movements for all patients, since age and body height have a considerable influence upon these values.[23] Future epidemiologic studies should measure, therefore, the dysfunctional maximal-opening, instead of the limited one.

Regarding the symptoms, although their frequency was higher in the experimental groups, no statistically significant differences were found between the study groups. This result should be attributed to the fact that most of the signs detected in the subjects were in the subclinical phase of the disease and, therefore, symptoms had not yet developed.

Finally, the severity of craniomandibular dysfunction was measured in this study by Helkimo's index as mild, moderate, and severe. No statistically significant differences were found in the prevalence of clinical dysfunction index between the groups. Statistically significant difference between the experimental and control group I, however, was revealed in the frequency of anamnestic dysfunction index with severe symptoms (AiII). This finding shows that children with clefts reported more severe symptoms than those without unpleasant life events. Nevertheless, the reported symptoms were occasional and mild. Helkimo's dysfunction index was an important development because of its ability to quantify the severity of craniomandibular dysfunction. Unfortunately, this index does not help much in epidemiologic studies, since it is not sensitive to chronicity and etiology of craniomandibular dysfunction.

The multifactorial etiologic approach of craniomandibular dysfunction has been noted by some investigators.[2,3,24-26] Trauma, oral parafunctions, malocclusion, and emotional states are considered as the etiologic factors. A recent review study, however, reported that malocclusion cannot be "necessary" and/or "sufficient" etiologic factor of craniomandibular dysfunction.[27] The present study showed that children with clefts and those with unpleasant life events run a greater risk of developing TMJ tenderness. It is likely, therefore, that emotional states caused by the impairment of the masticatory system due to clefts as well as by the reported unpleasant life events are the etiologic factor of TMJ tenderness in these groups.

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FLUORIDE INTAKE

The fluoride intake of most persons derives chiefly from dietary sources. Several recent studies, however, have indicated that substantial amounts, which may approach or exceed dietary fluoride intake, may come from inadvertent swallowing during and after the use of fluoride-containing products [Whitford et al., 1987; Burt, 1992]. Freshwater fluoride levels vary widely, ranging from less than 0.1 ppm to over 100 ppm depending on the amounts and solubilities of the compounds available locally in the earth. Fresh or unprepared foods in the USA generally have fluoride concentrations that range from 0.01 to 1.0 ppm. The major exceptions include fish and tea. The oceans have a fluoride concentration of approximately 1.5 ppm which continuously exposes marine plants and animals to large amounts of fluoride. The fluoride concentrations of dry tea leaves vary widely (approx. 4–400 ppm) while those of brewed tea range from 1 to 6 ppm depending on the amount of dry tea used, the water fluoride level and the brewing time [Cremer and Buttner, 1970; Wei et al., 1989]. Thus, persons who live on ocean islands or in coastal regions and/or who frequently consume tea may have relatively high fluoride intakes.

Whitford, G.M.: The metabolism and toxicity of fluoride. Basel: Karger, 1996 p 3.

The effectiveness of preventive resin restorations in pediatric patients

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Preventive resin restorations (PRR) were introduced in the dental literature by Simonsen in 1977.[1] A preventive resin restoration consists of resin-composite filling of the instrumented preparation with the addition of an occlusal sealant to the undecayed pits and fissures. The great advantage of this approach is the conservation of tooth structure as opposed to extension for prevention. There have been numerous reported studies on the effectiveness of this technique.[2-8] The acceptance of the sealant as a preventive measure must be recognized, to justify this technique. It was discouraging to learn from the 1987 NIDR survey that so few children (8 percent) had permanent teeth sealed, although an improvement was observed in the Third National Health and Examination Survey (NHANES III - Phase 1) where it was reported that 18.5 percent of children and adolescents had one or more sealed teeth.[10] It is unfortunate that so many dentists have not been convinced of the usefulness of this technique for the prevention of fissure caries. It was the purpose of this study to determine the effectiveness of composite/sealant (preventive resin restoration, PRR) placed in pediatric patients at the University of Iowa College of Dentistry.

METHODOLOGY

Information from Collegiate Visit Slips regarding procedures performed at the University of Iowa College of Dentistry is stored on the Collegiate Patient Management System. All procedures performed are recorded by an ADA code. When an individual permanent molar was identified by the computer as having a one-surface or Class I restoration, the following information was retrieved about the tooth and the patient.

- ☐ Tooth number.
- ☐ Initial date of placement.
- ☐ If any further treatment was performed on the tooth, the date of placement and procedure performed were noted.
- ☐ The last date the patient was seen in the clinic.

These data were collected for 5,185 composite/sealants (PRR), placed on posterior permanent teeth in the Pediatric Dental Clinic at the University of Iowa College of Dentistry.

RESULTS

Five thousand one hundred and eighty-five preventive resin restorations were placed in permanent teeth of pediatric dental patients, ranging in age from six to eighteen. Of these teeth, 4,314 had not required replacement (Table 1) and 323 required replacement of the sealant only. The teeth not requiring retreatment were observed up to 6.5 years; the median observation time was 1.1 year.

In comparing the premolars with the first and second permanent molars, it was observed that the molars required a statistically significant increase in the size of the restoration after the placement of a preventive resin restoration (Table 2). A large number of the replacement restorations included two or more surfaces (186). The remaining 359 were one-surface restorations. Although

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Table 1 ☐ Replacement of original restoration.

Procedure	Frequency	Percent	Cumulative frequency	Cumulative percent
No replacement	4314	83.2	4314	83.2
Sealant	323	6.2	4637	89.4
1-surface amal.	52	1.0	4691	90.5
2-surface amal.	75	1.4	4766	91.9
3-surface amal.	12	0.2	4778	92.2
4-surface amal.	2	0.0	4780	92.2
1-surface composite	289	5.6	5069	97.8
1-surface glass ionomer	17	0.3	5086	98.1
2-surface composite	16	0.3	5102	93.4
(PRR) 1-surface	1	0.0	5103	93.4
(PRR) 2-surface	73	1.4	5177	99.8
(PPR) 3-surface	8	0.1	5185	100.00

Table 2 ☐ Comparisons of procedures on premolars against certain teeth.

Baseline	Comparisons to baseline (probability)		
(1st premolar)	(2nd molar)	(1st molar)	(2nd premolar)
#5	#2	#3	#4
	.000	.000	.090
#12	#15	#14	#13
	.000	.000	.530
#21	#18	#19	#20
	.000	.000	0.83
#28	#31	#30	#29
	.000	.000	.0124

Chi square test was used to determine differences between number of replacement procedures for each type of tooth.

replacement of these restorations, for any reason, is of concern for the clinician, approximately a third were restored due to proximal-surface caries.

DISCUSSION

The preventive resin restoration technique is simple. The reluctance of many to abandon traditional concepts of "extension for prevention" is understandable, given historical backgrounds in restorative dentistry and adherence to the principles of G.V. Black. We now have, however, a proven material, bonded resin pit and fissure sealant, that obviates the need for extension for prevention. In addition, resin-composite can restore tooth structure with much less need for mechanical retention. Perhaps the chief advantage of the resin bonding systems is repairability of the restorations.

At the University of Iowa Pediatric Dentistry Clinic we observed a necessity to replace pit and fissure sealants in approximately 13 percent of the sealed teeth and another approximately 8 percent of molar teeth became carious. The 10 percent of these teeth that required further restoration compared favorably with the previously observed decay-rate in our clinic. That 186 of the 548 teeth required proximal restorations and had nothing to do, therefore, with defects of the previously placed bonded resin-composite or sealant should be considered, when evaluating the overall failure rate of this restorative procedure. It was also observed during a survey of the chronological record of treatment of failed restorations that the intervention was usually needed as a result of the loss of perhaps the failure to place sealants in the distal pits and fissures of maxillary molars and buccal pits of mandibular molars. The latter may not have been accessible during the original procedure because of an operculum or gingival margin.

Conservation of the tooth structure associated with the preventive resin restoration is undeniable. Conry *et al* measured the occlusal surface areas of preventive resin cavity preparations in lower first permanent molars of thirty-three pediatric patients and found them to average 19 percent of the gross surface.[11] Welbury *et al* compared amalgams and preventive resin restorations on occlusal surfaces over a five-year period. Their findings demonstrated an even greater conservation of occlusal tooth structure. Resin-composites occupied only 5 percent of the occlusal surface compared to amalgam restorations occupying about 25 percent of the surfaces. There was no significant difference in the median survival times.

CONCLUSIONS

In conclusion it was determined that the majority of composite/sealants (PRR) failures were due to factors other than the composite restoration. A review of clinical records verified failures were due to:

- ☐ Failure to replace the sealants.
- ☐ Smooth surface caries at a distant coronal site.
- ☐ Restoration placed in partially erupted teeth.
- ☐ Placement where the teeth were compromised, i.e. hypoplastic enamel was indicated as present.

In summary the preventive resin restoration is an effective restoration that conserves tooth structure when it is correctly placed and examined at regular intervals.

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RECOMMENDATIONS FOR USING FLUORIDES

In order to reduce the ingestion of fluoride from fluoride-containing dental products, many recent recommendations for modification of product formulation and usage have been made [Bawden, 1992]. Included among the recommendations are: (1) strict adherence to the current recommendations for dietary fluoride supplementation; (2) continued monitoring of the fluoride concentrations of water (including bottled water) and foods for young children; (3) parental supervision of brushing or mouthrinsing; (4) the use of the smaller amounts of dentifrices or mouthrinses; (5) the use of products with lower fluoride concentrations (as is being done in several European countries), and (6) placing the fluoride concentration on the dentifrice container. Because young children often swallow materials that are placed in their mouths, parental supervision should include the use of a 'pea-sized' portion or a 'smear layer' of dentifrice on the brush and assuring that expectoration after brushing or rinsing is as complete as possible. Further, fluoride-containing products for home use should be kept out of the reach of children.

Whitford, G.M.: The metabolism and toxicity of fluoride. Basel: Karger 1996, p 9.

Clinical evaluation of class II combined amalgam-composite restorations in primary molars after 6 to 30 months

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Composite materials are widely used in dentistry to restore posterior teeth with proximal cavities. Microleakage at the cervical margins and secondary caries, however, are still major concerns for the dental practitioner.[1-3] Recently, in-vitro studies combined the sealing capacity of amalgam with the esthetic appearance of composite materials. A thin layer of amalgam has been condensed to the gingival floor of the proximal box before insertion of the composite material.[4,5] These studies proved that microleakage at the cervical margin has been significantly reduced, when compared to conventional composite restorations; the long-term effect of the amalgam on the clinical appearance of the restorations, however, could not be evaluated.

The purpose of this study was to assess the influence of a thin layer of amalgam placed at the cervical floor of class II composite restorations in primary molars, on the clinical and radiographic findings and esthetic appearance of these restorations.

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METHODS

Experimental design

To participate in this study the children had to have at least one primary molar with small to moderate proximal caries, and proximal contact with an adjacent tooth. The children had to be available for recall appointments every six months until shedding of the teeth, and have parental consent to participate in this study. Medical history was recorded and a treatment plan developed based on clinical examination and bite-wing radiographs.

A total of forty-two cavities were filled in eighteen patients (six girls and twelve boys); their ages at time of the restorations ranged between six years, six months, and twelve years with a mean of eight years, one month.

Clinical procedure

Following local anesthesia, a rubber dam was placed, and conventional class II cavities were prepared, using a #330 carbide bur under coolant spray. The axial and pulpal walls were protected with Dycal (L.D. Caulk), a transparent celluloid matrix (Howe-Neos Dental, CH 6925 Gentilino, Switzerland) was placed and adapted with a Tofflemire matrix holder and a clear wedge with light reflecting surfaces (Howe-Luciwedges, Howe-Neos Dental, CH 6925 Gentilino, Switzerland). Each tooth was then randomly assigned to the study group or one

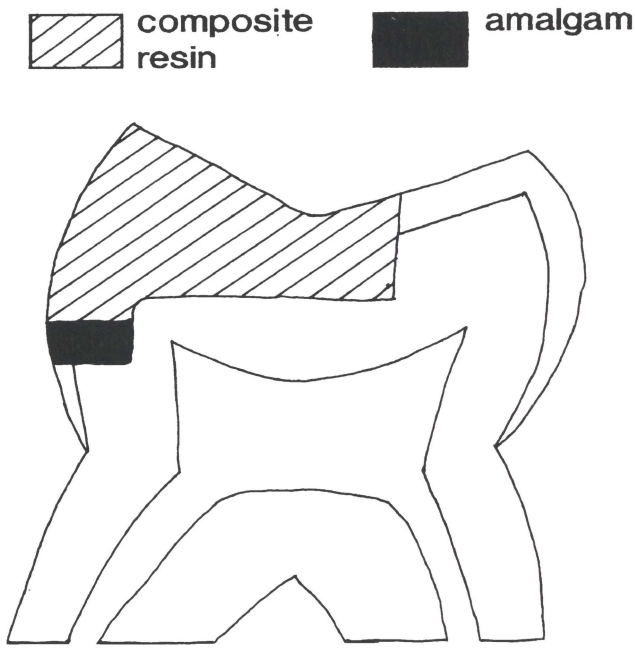

Figure 1. Diagram of a "sandwich" restoration showing an approximately 1mm thick cervical layer of amalgam covered with a composite resin.

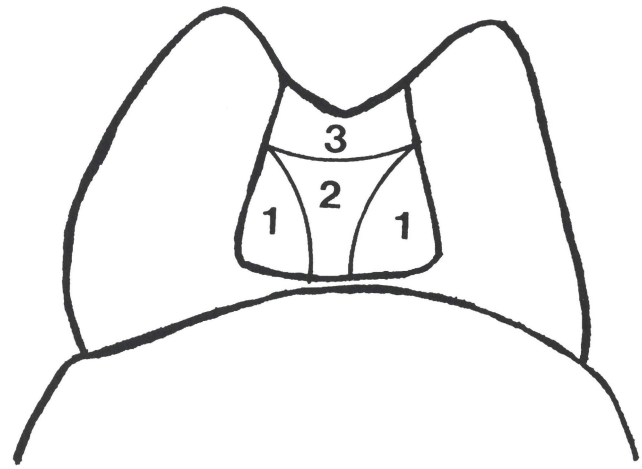

Figure 2. Diagram showing the incremental method used for the composite restoration (approximal view). The numbers indicate the order of placement of the increments and light curing.

of two control groups. A layer of approximately 1mm thick amalgam (Silmet, Gyvatayim Israel) was condensed on the gingival floor of the proximal box of the study group (group A) (Figure 1). Amalgambond (Parkell, Bio-Material Division, Farmingdale, NY 11735) was used as a bonding material and placed over the amalgam following the manufacturer's instructions. This means that an acid activator has been applied for thirty seconds to the enamel and ten seconds to the dentin of the proximal box (the activator was first applied to the enamel only and added to the dentin after twenty seconds). The acid was washed and the cavity dried with an air stream. Then, a thin film of an adhesive agent and Amalgambond were brushed on the cavity walls and on the amalgam. The rest of the cavity was filled in one bulk with Estilux Posterior (Kulzer & Co Gmbh, Wehrheim, W. Germany), and light cured for twenty seconds from each of the buccal, lingual and occlusal aspects. The same steps were applied for the cavities assigned for one of the control groups (group B), except that conventional enamel bond supplied with the composite material, instead of Amalgambond, was applied to the etched enamel on the buccal and lingual walls and to the amalgam. In group C the enamel of the cavities was etched and a conventional enamel bond applied and cured. Three vertical increments (buccal, lingual and middle) were used to fill the box with Estilux Posterior, and a fourth increment filled the occlusal part of the cavity, as described in Figure 2. Each increment was cured separately for twenty seconds. All restorations were polished and finished with a fluted carbide bur (#4205L Composhape Finishing System - Densco) and a set of Sof-lex discs (3M Company, St. Paul, MN). Occlusal disturbances, if present, were detected with an articulating paper and eliminated. The total of forty-two restorations were randomly divided between the study and control groups as shown in Table 1.

In those cases where adjacent proximal surfaces of two teeth were included in this work, the cavities were prepared, calcium hydroxide bases were placed and matrix bands were adapted in both teeth. One of the cavities was filled, hardened and finished first, however, and then the adjacent cavity was restored.

Evaluation

The restorations were evaluated at baseline and every six months for surface appearance, color match, marginal adaptation, marginal discoloration, anatomic form, and secondary caries, using the criteria described by Cvar and Ryge.[6] Evaluation at baseline and recall examination were done by at least two of the three examiners in each case, and a consensus reached in case of disagreement.

Bite-wing radiographs taken at the recall examination

Table 1 ☐ Experimental design. Distribution of restored teeth.

Group	# of teeth	Type of restoration
A	14*	** Amalgam + Amalgambond + Estilux Posterior
B	17*	** Amalgam + Enamel bond + Estilux Posterior
C	11	Enamel bond + Estilux Posterior (increments)

* Two teeth of group A and one of group B were not available for any follow-up evaluation.
** Sandwich restoration.

Table 2 ☐ Distribution of evaluated teeth by follow-up period in months.

Follow-up period (months)	Group A	Group B	Group C	Totals
6 - 8	2	2	1	5
9 - 11	2	6	5	13
12 - 14	3	2	0	5
15 - 17	2	0	1	3
18 - 20	1	2	0	3
21 - 23	1	1	0	2
24 - 26	0	2	1	3
27 - 30	1	1	3	5
Totals	12	16	11	39

Table 3 ☐ Distribution of the evaluated teeth according to type and location.

	Maxillary	Mandibular	Totals
First Molar	12	7	19
Second Molar	10	10	20
Totals	22	17	39

Table 4 ☐ Clinical evaluation of the restorations at the latest follow-up examination (6-30 months).

		Groups					
		A		B		C	
Evaluation Criteria	Rating*	N	%	N	%	N	%
Surface appearance	Alpha	12	100	16	100	11	100
Color match	Alpha	4	33	6	38	10	91
	Bravo	8	67	8	50	1	9
	Charlie	0		1	6	0	
	Delta	0		1	6	0	0
Marginal adaptation	Alpha	11	92	16	100	11	100
	Bravo	1	8	0		0	
Marginal discoloration	Alpha	10	83	14	88	11	100
	Bravo	2	17	2	12	0	
Anatomic form	Alpha	11	92	13	81	11	100
	Bravo	0		2	13	0	
	Charlie	1	8	1	6	0	
Secondary caries	No	11	92	16	100	10	91
	Yes	1	8	0		1	9

*Irrelevant ratings were deleted from the table.

Table 5 ☐ Distribution of different radiographic defects by groups.

	GROUPS		
	A	B	C
Total number of restorations	12	16	11
Type of radiographic defect			
Tooth - Amalgam interface	0	0	N.R.*
Amalgam - Composite interface	1	0	N.R.*
Tooth - Composite interface	0	0	0
Amalgam overhang	3	2	N.R.*
Bubbles in composite	2	2	7
Pooling	1	0	1
Voids in contour	0	0	2
Free of any radiographic defects	7	12	4

*Not Relevant

were examined for the presence of radiolucent areas at the tooth-amalgam (T-A) interface, amalgam-composite (A-C) interface, tooth-composite (T-C) interface, and defects at the cervical margins, overhang of amalgam, and bubbles in the body of the composite restorations. Each investigator evaluated all radiographs and any disagreement was discussed until consensus was achieved.

RESULTS

Of the forty-two restorations thirty-nine (12 of group A; 16 of group B and 11 of group C) were available for follow-up evaluation after six to thirty months (mean 15.1 months) (Table 2). The distribution of teeth according to type and location is presented in Table 3. One tooth exfoliated naturally before the six-month recall, and one patient (two restorations) did not appear for recall examinations.

Clinical evaluation

None of the patients complained of any pain or discomfort associated with the restorations placed in this study. The results of the clinical evaluation of the teeth are summarized in Table 4. Color match was rated Alpha in 33 percent of group A, 38 percent of group B and 91 percent of group C. This difference was significant ($p<0.02$).

The investigators realized that using a conventional enamel bond is much simpler and less time consuming than the multistep technique of Amalgambond. In addition, the adaptation of a preformed transparent celluloid matrix with a Tofflemire matrix holder to primary molars was found to be difficult. The investigators found it difficult to use the incremental method to fill the relatively minute proximal box. The incremental method requires also four twenty-second periods of light expo-

Figure 3. Bitewing radiograph exposed eleven months post-treatment, showing a mesioocclusal restoration in the mandibular second primary molar. A layer of amalgam fills the cervical part of the cavity covered with a composite material. The first primary molar presents a distoocclusal composite restoration.

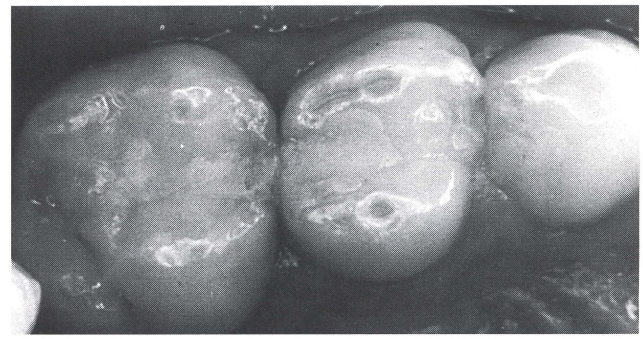

Figure 4. A clinical view of the occlusal surface of two "sandwich" restorations in maxillary primary molars. Note the dark discoloration of the composite at the marginal ridge that reflects underlying amalgam.

sure to cure the various increments, which add more working time to the procedure.

Radiographic evaluation

None of the teeth presented internal or external resorption, or interradicular or periapical radiolucency. The results of the radiographic evaluations are shown in Table 5. Only one tooth of group A presented a radiolucent area at the A-C interface. None of the other interfaces presented any radiolucency. Amalgam overhang was observed at the gingival margin of the box of four restorations; none, however, exceeded 1mm length. Seven (63.6 percent) restorations of group C presented bubbles, compared to only two (16.6 percent) in group A, and two (12.5 percent) in group B. This difference between the groups was significant ($p<0.01$). Pooling of bonding material was observed under the composite of two restorations.

DISCUSSION

The clinical and radiographic results of the restorations of all three groups were found to be very good. None of the patients complained of any pain or discomfort associated with the restorations; no radiographic pathological evidence was observed; and no replacement of any restoration or early tooth extraction was necessary. Initial secondary caries at the occlusal margins was observed clinically in two restorations and was treated conservatively, without removal of all the restorative material. Esthetics is the main reason for using composite materials for tooth restoration. In the present study, however, this was only partially achieved. The underlying amalgam was visible through the composite of the experimental restorations (groups A and B) reducing the percentage of excellent ratings of color match to 33 percent and 38 percent, respectively. It is justified, therefore, to prefer an opaque bonding agent to mask the silver amalgam.[7] The percentage of excellent color match in group C dropped to 91 percent, due to mismatch of the shade of the composite material to that of the tooth. Radiolucent defects in the composite material observed on the radiographs were interpreted as air bubbles trapped between the layers. These were detected mainly in group C restorations in which four increments were used to fill the cavities. Composite restorations in the present study presented more bubbles (63.6 percent) than in a previous clinical study.[8] The same technique was used in both studies to insert the composite material. The difference may arise from different qualities of the composite materials used in the two studies; P-30 was used in the previous study and P-50 in this investigation. A layer of approximately 1mm-thick amalgam was condensed on the gingival floor of the proximal box of groups A and B. In many cases the amalgam on radiographs appeared thicker, however, than 1mm. This can happen when the X-ray beam is not parallel to the horizontal axis of the amalgam layer. The

wider the buccolingual width of amalgam, the thicker will the amalgam appear on the radiograph. Another possible reason for the amalgam to appear radiographically thicker than intended could be due to extension of amalgam along the buccal and lingual walls of the proximal box. This makes the radiographic evaluation of the amalgam-composite interface of limited validity.

The use of a transparent matrix band and wedge is recommended in order to allow better access of the light and polymerization of the composite material at the cervical margins first. The celluloid matrix with Toffelmire matrix holder was found very difficult to adapt to the tooth, however, and time consuming. The use of a thin layer of amalgam at the gingival floor of the box may eliminate the need of a transparent matrix, allowing the use of the more convenient metal matrix band with a wooden wedge. Two different adhesive materials were used in the present study to bond the composite material to the underlying amalgam (Amalgambond in group A and conventional enamel bond in group B). No clinical difference was observed between the teeth restored with the two adhesive materials.

It is obvious that the clinical and radiographic criteria used in this study are not sensitive enough to evaluate microleakage in the short term. A longer follow-up period, however, may allow detection of caries developing at the cervical margins as it is routinely used for amalgam restorations. It is not yet clear whether any of the bonding agents used, adequately seals the different interfaces, and only a laboratory study of the teeth after exfoliation will serve as better indicator of the sealing capacity of these methods.[8] Any attempt to cope, however, with cervical marginal leakage of Class II composite restorations using a thin layer of amalgam will have to take into consideration the esthetic factor and cover the amalgam with an opaque bonding agent.

CONCLUSION

Since none of the restorations caused pain, discomfort or pulp involvement and none needed replacement or presented secondary caries, a major shortcoming that needs further improvement is the color match. A suitable opaque adhesive should cover the amalgam layer hiding its silver shade. If no leakage will be detected in the examination of the retrieved teeth, the combination of amalgam and composite for the restoration of Class II cavities in molars can be recommended.

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GASTROINTESTINAL ABSORPTION OF FLUORIDE

Several reviews of the gastrointestinal absorption of fluoride [Smith, 1966; Cremer and Buttner, 1970] indicate that, in the absence of relatively high levels of ions such as calcium, magnesium and aluminum that can combine with fluoride to form insoluble salts, the rate of absorption is unusually rapid and that the degree of absorption is nearly complete. . . . . The half-time for absorption is about 30 min. As stated previously, the elimination rate of fluoride from plasma exceeds the absorption rate after the bulk of the fluoride has been absorbed. The extent of absorption from the stomach is inversely related to the pH of the contents and may account for up to 40–50% of the amount ingested. Most of the remainder will be absorbed from the upper small intestine.

Whitford, G.M.: The metabolism and toxicity of fluoride. Basel: Karger, 1996 pp. 10, 11.

Effect of heat treatment on stainless steel lingual arch appliances

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One use of the lingual arch appliance is as a space maintainer following the premature loss of primary teeth.[1] When used this way, the lingual arch should be made passive, to prevent undesirable movement of the teeth.[1] Active appliances can tip molars, resulting in a posterior crossbite.

Heat treatment or annealing to relieve stress is intended to eliminate residual stresses in a wire subsequent to bending.[3] Heat treatment can be accomplished at comparatively low temperatures (370-480 degrees C).[4] Marcotte recommends stress-relieving stainless steel at 399 degrees C for 11 minutes in a dental oven.[5] A popular technique devised by Funk utilizes a color index to determine optimum heating.[6] A straw color is obtained after the wire has been heat-treated at 454 degrees C.

Durr *et al* used three methods (dental oven, electric current, and flame) to stress-relieve U-shaped 0.036" cobalt-chromium orthodontic wires.[7,8] The arch width of the heat treated wires increased and then stabilized. The dimensional change in nonheat-treated wires continued throughout the experimental period. After six weeks, Durr *et al* readjusted their control and experimental wires to the baseline widths and observed that the majority did not re-expand in one week.[8] Expanded wires had less change than after the initial bending.

The purpose of this study was to evaluate the effect of heat treatment on 0.036" diameter stainless steel wire bent to simulate a lingual arch appliance.

MATERIALS AND METHODS

Straight, stainless steel, round 0.036" diameter orthodontic wire (Unitek/3M Permachrome Medium-Hard, Monrovia, CA) was cut into forty 10.0 cm lengths. To facilitate the measurement of the interarch distance, lines were scribed with a scalpel blade 15.0 mm from the ends of each wire. A custom-made mold and a universal testing machine (Instron, model 1011, Canton, MA) was used to form archwires of approximate dimensions (Figure 1).

The experimental group consisted of twenty standard archwires. These were heat-treated in 2 cm segments

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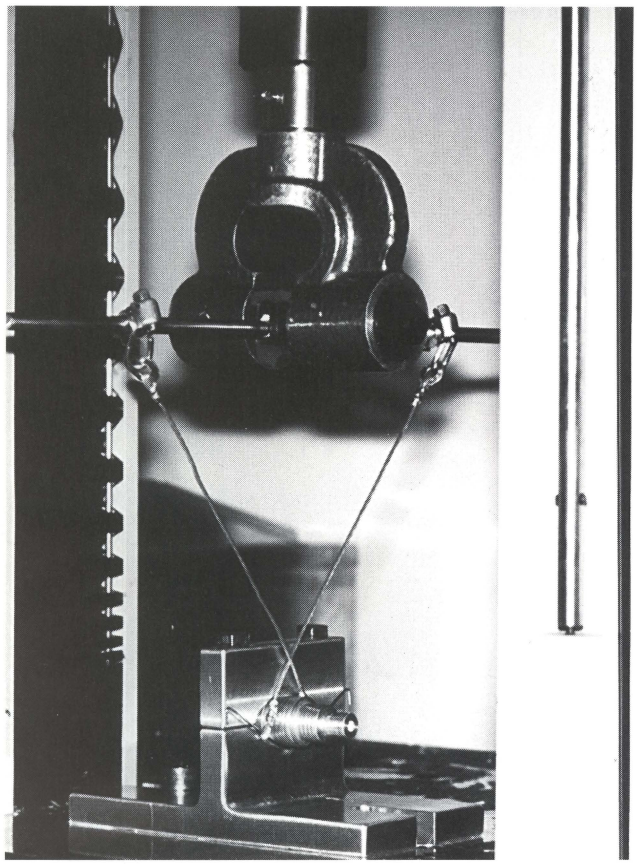

Figure 1. Wire bent into arch form using universal testing machine.

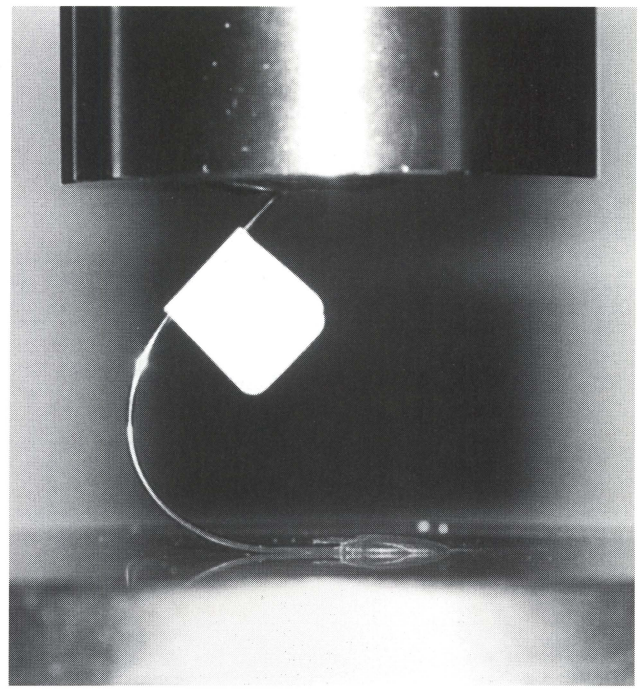

Figure 2. Determination of force generated by expanded wires using universal testing machine.

with an orthodontic spot welder (Model 660, Rocky Mountain Orthodontics, Denver, CO), utilizing 510 degrees C. temperature-indicating paste (Rocky Mountain Orthodontics, Denver, CO). The control group was twenty nonheat-treated wires.

The interarch width of all wires was measured at the scribed marks with a microscope (Nikon Measurescope Model II, Japan). Experimental group interarch width was measured immediately after forming and immediately after heat treatment. The interarch width of the control wires was measured immediately after forming. Arch wires were measured at one week, four weeks, and eight weeks and were stored at room temperature throughout the study.

After the eight-week measurement, the dimensional change in the interarch distance of each wire was calculated. The wires were cut at the scribed mark and stabilized on the compression platens of the universal testing machine (Figure 2). The amount of compression delivered to each wire equated to the overall expansion. The compression force was interpreted as an indication of the force caused by expansion of the wires.

STATISTICAL ANALYSIS

Repeated measures analysis of variance and paired t-tests were used to determine the significance of dimensional changes in the control and experimental wires. Significance was established at $P < 0.05$. The Pearson Correlation Coefficient was employed to determine whether a correlation existed between the amount of force generated and the amount of wire expansion.

RESULTS

The vast majority of expansion occurred as a result of the heat treatment in the experimental group. To study the effect of the heat treatment, the experimental wires were evaluated, using two baselines: Baseline 1 was the initial arch width as determined before heat treatment and Baseline 2 was the initial width as determined immediately after heat treatment. Table 1 lists the means and standard deviations of the changes in interarch dis-

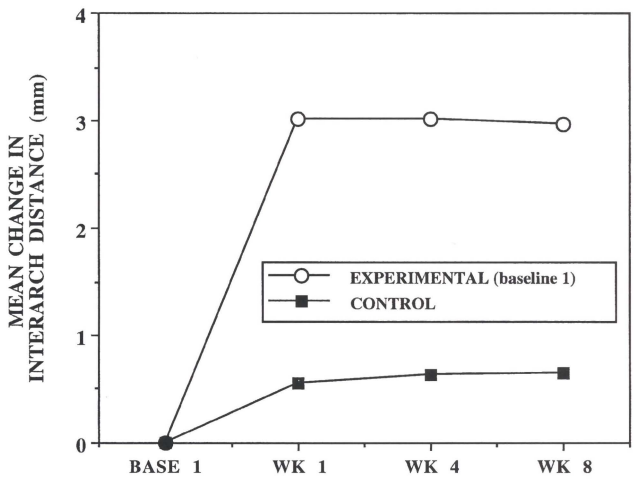

Figure 3. Mean change in arch width utilizing Baseline 1 (baseline width determined before heat treatment).

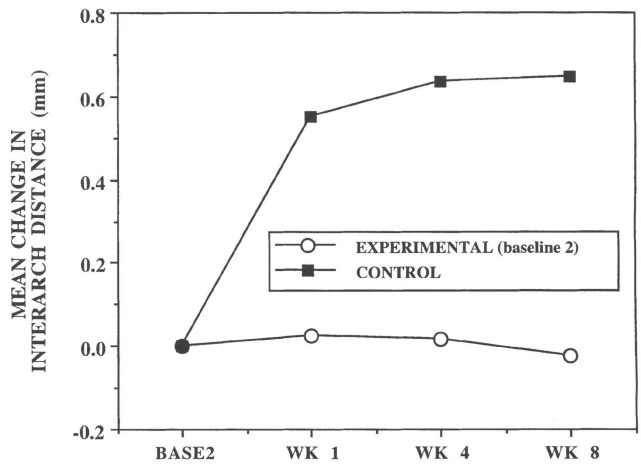

Figure 4. Mean change in arch width utilizing Baseline 2 (baseline width determined after heat treatment).

Table 1 ☐ Change in arch width from baseline.

Time interval	Control	Experimental (Baseline 1)	Experimental (Baseline 2)
1 Week	0.55mm (0.44)*	3.02mm (0.52)	0.02mm (0.10)
4 Weeks	0.64mm (0.42)	3.01mm (0.50)	0.02mm (0.12)
8 Weeks	0.65mm (0.50)	2.97mm (0.51)	−0.02mm (0.13)

* Mean (Standard Deviation)

Table 2 ☐ Expansion and force generated.

	Control	Experimental
Overall expansion	0.66 mm (0.50)*	2.97 mm (0.51)
Percent of expansion	1.80% (1.33)	7.71% (1.51)
Force generated	35.0 g (24.1)	273.4 g (74.3)

* Mean (Standard Deviation)

tance at one, four, and eight weeks for control and experimental wires. Figure 3 is a plot of arch width changes as measured from Baseline 1, while Figure 4 is a plot of arch width changes measured from Baseline 2. Note that Figures 3 and 4 are the same for the control wires since their measurements are not affected by choice of baseline.

The control wires significantly changed in dimension during the first four weeks ($P < 0.001$). There was no significant expansion after four weeks. The experimental wires changed significantly ($P < 0.001$), when the expansion due to heat treatment was included in the calculations. Dimensional change in the experimental wires after Baseline 2 measurement was not significant.

The control wires expanded significantly more than heat-treated wires, when expansion due to heat treatment was not taken into account ($P < 0.001$). Conversely, the overall expansion of the heat-treated wires was significantly greater than that of the control wires, when the expansion due to heat treatment was taken into account ($P < 0.001$).

To calculate the overall expansion for the compression tests, the initial widths of the control group wires were subtracted from the eight week width. The experimental wire, Baseline 1 (before heat treatment) was used as the initial arch width for calculating overall expansion. The twenty experimental wires exhibited a mean expansion of 2.97 mm and generated a mean force of 273.4 grams. The control wires underwent a mean overall expansion of 0.66 mm and generated a mean force of 35.0 grams (Table 2). One control wire experienced an overall constriction in its interarch distance and was excluded from the remaining portion of the study. A strong linear relationship existed between the overall amount of expansion and the force generated during the expansion (Figures 5 and 6). The Pearson Correlation Coefficient indicated a correlation of 0.96 for the thirty-nine samples, 0.75 for the nineteen control wires, and 0.76 for the twenty experimental wires. To compensate for the fact that the universal testing machine was unable to generate wires of exact dimensions, the percent of expansion was also evaluated (Table 2). An extremely high

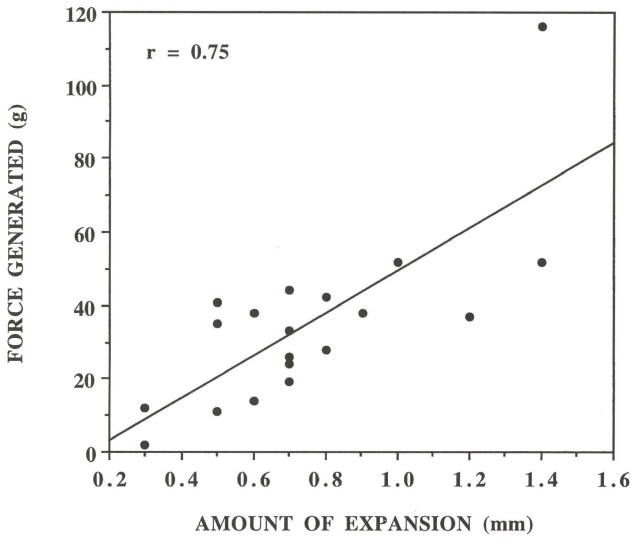

Figure 5. Force generated by expansion of control wires.

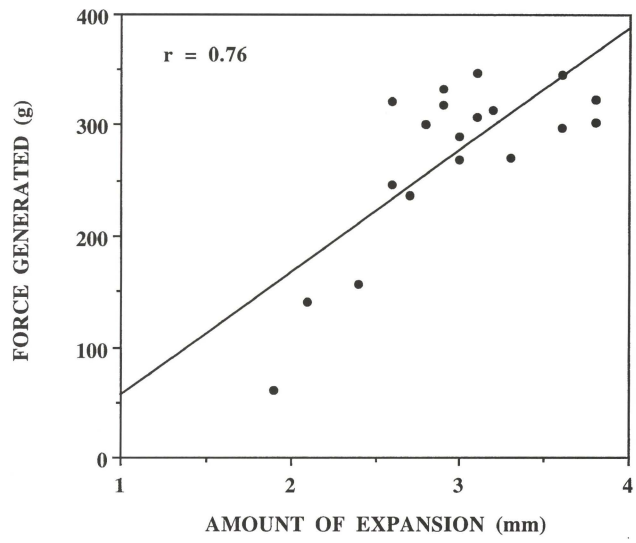

Figure 6. Force generated by expansion of experimental wires.

correlation existed between the amount and the percent of expansion as evidenced by the Pearson Correlation Coefficient of 0.99.

DISCUSSION

Heat-treated wires underwent a significant amount of immediate expansion as a result of heat treatment. After initial expansion, no significant change in dimension occurred, demonstrating that post-initial expansion is achieved. The expansion in the control group, however, continued for a period of four weeks. These results concurred with those of Durr *et al*, who tested 0.036" diameter cobalt-chromium orthodontic wires.[7,8]

The mean forces generated by expansion were 35.0 grams in the control group and 273.4 grams in the experimental group. Although these forces may not be directed toward a single tooth, they are still of sufficient magnitude to produce orthodontic movement.[2] The forces in the current study were far greater than those attained by Durr *et al*.[8]

The current study demonstrated the effect of heat-treating an arch-shaped appliance of 0.036" diameter stainless steel wire. Stress was eliminated within the wire, but the heat-treating process produced a dimensional change that may lead to the orthodontic movement of teeth. Heat treated appliances that fit passively on the working casts and in the mouth can be considered inactive. If an appliance is adjusted and heat-treated, it is essential that it is checked before cementation to ensure that it seats passively on the teeth.

CONCLUSIONS

When 0.036" diameter stainless steel orthodontic wire was bent into an arch shape and subjected to heat treatment, a significant increase in interarch width was seen as an immediate result of the heat treatment. This was followed by a stabilization of the arch dimension. The control wires continued to expand at eight weeks.

The force generated as a result of the expansion of the control and experimental group wires was of a magnitude capable of producing tooth movement.

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Etiology of oral habits

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Salvador Orozco, Lic. Econ
Karla Montoya, BS

Progress in dental care has been evidenced by a reduction in the incidence of the main problems in oral health. We are facing, however, a growing frequency of dental lesions due to bruxism, malocclusion, temporomandibular joint dysfunction, and developmental abnormalities of the maxillae associated with some nonphysiologic activity of the oral cavity.[1-4]

Survival of the newborn depends upon instinctive oral sucking, which allows for nutritional satisfaction. We recognize that lips and tongue, as well as oral mucosae allow for pleasurable satisfaction that nourishes and builds the child's initial psychological and interpersonal functions of the binomial system mother/son; which in turn facilitates an initiation of the exploration of the social environment.[5,6]

Once the biological and psychological functions of the child undergo maturation, he or she can separate from the mother without experiencing significant anxiety (between ages nine and eighteen months); the child will find himself exposed to another type of self-gratifier, adequate to his own new circle of social activities, spontaneously doing away with many oral habits.[7]

The participation of J. Fernando Bayardo, MD is appreciated for his efforts to present this paper in the English language.

Dr. Bayardo is former coordinator of the Master of Science Program in Dentistry; Dr. Mejia is Former Dean of the School of Psychology; Miss Montoya is a student in the School of Chemistry, Universidad de Guadalajara, Mexico. Lic. Orozco is in private practice in Guadalajara City.

The persistence of sucking habits (digital, nursing bottle, tongue, lips, cheeks, objects, atypical deglutition) and the appearance of biting habits (onychophagia, bruxism, biting parts of the body or objects, etc.), according to Freud, have been associated with an arrest in the evolution (fixation) of the psychosexual oral phase, which will repercuss in a short while in a distortion or perversion of oral psychophysiologic processes.[5] The latter may alter the stomatological structures, depending on their duration, intensity and frequency.[8]

Some of the etiological factors considered responsible for oral habits include family conflicts, jealousy, school pressure, the stress of a large city, lack of satisfaction through nourishment, imitation of media activities that are inappropriate for minors, irritations associated with tooth eruption, occlusal interferences, breathing obstruction, etc.[5,9,10]

The treatment of pernicious oral habits on the basis of the utilization of orthodontic appliances or occlusal therapy has brought about unsatisfactory results.[11] Inadequate knowledge about the source of the problem, whether on the part of the dentist, parents or teachers, may encourage poor conduct by the child, when the soothing effects of oral habits are interrupted. This could trigger rebelliousness, sleep and nourishment problems, enuresis, and learning problems, resulting in a persistence of the habit.[4,6,12]

Optimal use of pedodontic charts, and the support of professionals from other disciplines, could offer us valuable guidance in the management of this complex problem.

METHODS

One thousand six hundred (1600) admission case histories (parental questionnaires) were analyzed. They pertained to patients, ages two to fifteen years, who were initially seen in a pedodontic private practice in the City of Guadalajara, Mexico, between the years of 1977 and 1992. The purpose of the analysis was to determine general epidemiologic data of oral habits and some possible factors associated with them.

Oral habits were classified according to type, digital sucking, lip or tongue sucking, bruxism, onychophagia, other oral habits (object biting or sucking) and mouth breathing.

The following observations pertaining to family structure were included: the number of siblings, classifying them as single child, child with one or two siblings, three or four, and five or more. It was also determined whether the father was present in the home, thus recognizing indirectly whether the mother was single, widow or divorced.

As far as the information gathered regarding the biological aspects of the patient, we took into consideration past and present state of health, types of past and present illnesses.

The information obtained from the case histories was collected in dBase III + in an IBM PS I, manipulating the original computer fields to obtain additional auxiliary fields, subsequently transferring them to seek possible statistical relationships through the EPI-5 program, thus establishing associations of the possible attendant or etiological factors of oral habits through inferences of the type chi square.

RESULTS

Fifty-six and eight tenths percent of the analyzed sample demonstrated some type of oral habit, of which 23.7 percent presented with onychophagia, 23.6 percent with bruxism, 11.7 percent with oral breathing, 11.3 percent with digital sucking, 6.9 percent with lip or tongue sucking, and 2.4 percent with other habits (Figure 1).

The prevalence of oral habits is illustrated in Figure 2 showing greater prevalence in the groups, ages five and ten years.

The female group participants showed a greater susceptibility toward this particular health problem, detecting a significant statistical difference as compared with the male group (Figure 3).

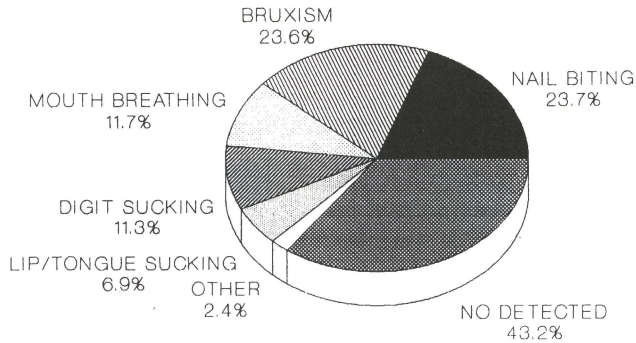

Figure 1. Distribution of oral habits.

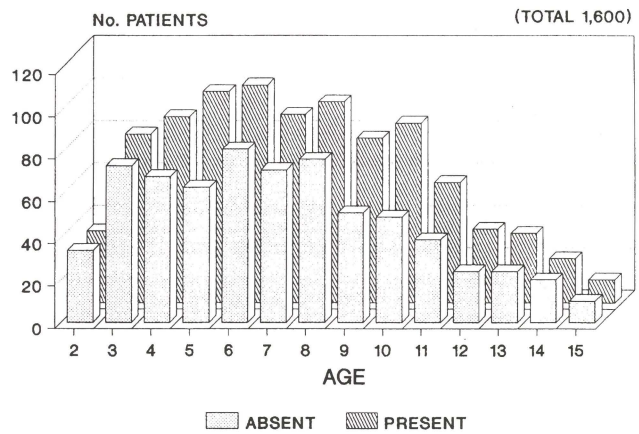

Figure 2. Prevalence of oral habits.

The number of siblings in the family played a strong role in the occurrence of oral habits, more of a problem for the single child group, and less for those children with three or four siblings (Table 1). The absence of the father in the integrated family also became a significant factor in the occurrence of oral habits (Table 2).

Insofar as biological antecedents, there was a significant correlation of poor current general health and oral habits (Table 3). Allergies were highly influential especially as far as environmental allergies were concerned (Figure 4).

There was a statistically significant difference between past state of health and oral habits (Table 4). In physically frail patients the correlation was positive. There was a high association (P=.03) for chronic illness, with eye, nose, and throat dominating. No significant differences

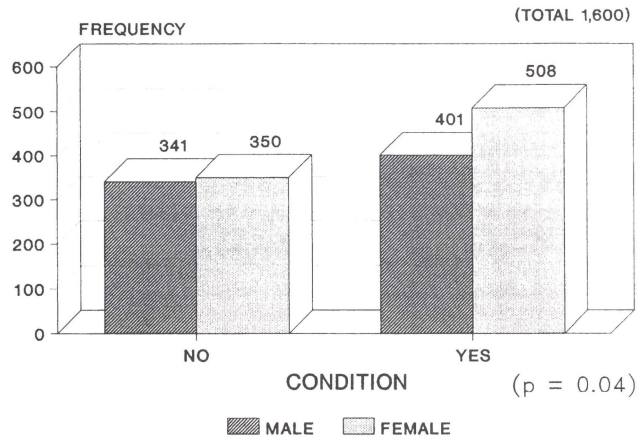

Figure 3. Gender influence on oral habits.

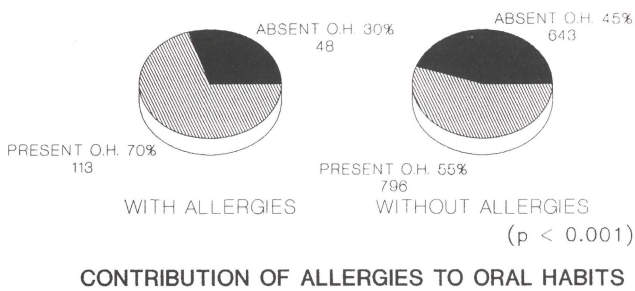

Figure 4. Contribution of allergies to oral habits.

Table 1 ☐ Effect of number of siblings on oral habits incidence.

	Siblings				
O.H.	0	1-2	3-4	5 or +	Totals
No	43	412	210	26	691
Yes	81	562	226	40	909
Totals	124	974	436	66	1,600

(p = 0.03)

Table 2 ☐ Effect of family integrity on oral habits.

O.H.	Father's absence	Father's presence	Totals
No	9	682	691
Yes	27	882	909
Totals	36	1564	1,600

(p = 0.03)

Table 3 ☐ Influence of current health condition on oral habits.

O.H.	Good health condition	Poor health condition	Totals
No	528	163	691
Yes	523	386	909
Totals	1,051	549	1,600

(p < 0.001)

Table 4 ☐ Influence of past health condition on oral habits.

O.H.	Good health condition	Poor health condition	Totals
No	462	229	691
Yes	561	348	909
Totals	1,023	577	1,600

(p = 0.03)

were found among past illnesses, severe or acute, or among congenital or inherited illnesses.

DISCUSSION

The results of this clinical, retrospective investigation should be expected to be representative mainly of children who attend a private pedodontic practice.

The fact that girls show greater susceptibility toward the development of oral habits, is worthy of notice. A general explanation would attempt to explain simple gender difference on the basis of physical build; philosophically, one could argue that greater social and educational pressures could account for the marked difference.

As far as the number of siblings in the family, our statistical method has demonstrated that a single child is more vulnerable to this type of problem. Research has shown the many psychological problems that exist, particularly for the single child: over-protection, loneliness, isolation, and problems of communication.[13] Also to be considered is a father's absence from the family.

On the other hand, studies on socialization, games, communication, and achievement have demonstrated that the family with four children represents the optimum number from the psychological perspective.[13,14] This investigation found that children with three or four siblings were least likely to develop an oral habit.

The statistically significant correlation between oral habits and poor physical health, especially when chronic, only reaffirms that physical conditions are related to psychological problems.

Another aspect of the problem to be considered under this rubric is the high correlation between allergies in general and oral habits, considering the environment as one more influence to be considered as a predisposing factor. Most recent biochemical investigations have demonstrated a very narrow link among chronic stressing situations and biochemical alterations of eosinophils,

lymphocytes and shock organs such as skin, bronchi, and mucosae in general. We do not find it strange that oral habits bring us within the perspective of a psyche problem interrelated with a response of general corporeal hypersensitivity.

The psychological theories of greater impact, as an explanation and solution of the oral habits, are the Pavlovian simplistic theories of the twentieth century, including the stimuli-response theory.[16,17] More complex theories, such as psychoanalysis, have placed much emphasis on the importance of the mouth as the first contact with the external world, and as the first level of pleasurable satisfaction. Attitudes toward the mouth in later life will depend upon the circumstances and vicissitudes of that first stage of life.[18]

Freudian theory argues for an element of "fixation" as the origin of certain frustrations derived from the oral cavity, which in its initial stages, is a partial solution of a state of displeasure, evolving into a habit and then a functional and structural problem.[19,20]

In subsequent publications, we will discuss epidemiological and etiological factors in detail and the resulting effects of other factors that accompany specific oral habits, as well as the etiological differences between biting and sucking habits.

CONCLUSIONS

The parents' reports collected in the admission clinical histories of pedodontic patients assembled valuable information as far as factors affecting the behavior of children, and the integrity of the stomatological system.

The high incidence of oral habits directs us to a restructuring of the hierarchy of current oral health problems. Prevention and treatment of oral habits would have to be socially perceived as a behavioral problem that affects the oral cavity, and which, therefore, shall be accessed from a multidisciplinary perspective in consideration of the fact that this problem is significantly influenced by family, social, environmental and biological factors.

It is obvious that the tackling of the oral habits problem by dentists requires greater attention to correcting the cause of the problems rather than the manifestations of the problem alone.

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DEMOGRAPHICS

Low birth-weight and the relationship to developmental problems

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

"Because of advances in neonatal care systems, many infants who weigh only 750 grams (1 pound, 10 ounces) at birth are now surviving. However, these successful rescue efforts are often associated with significant long-term health and developmental problems among survivors which limit their opportunities to lead full and productive lives."†

In the 1990s, 7.1 percent of the over four million children born annually have low birth-weights (weigh less than 2,500 grams [5 pounds, 8 ounces]).[2]

At school age, children who were born with low birth-weight are more likely than children of normal birth-weight to have mild learning disabilities, attention disorders, developmental impairment, and breathing problems such as asthma. These problems are reflected in much higher proportions of low birth-weight children who are enrolled in special education classes (50 percent more likely than normal birth-weight children). Approximately one half of very low birth-weight children (less than 1,500 grams [3 pounds, 5 ounces]) enroll in special education programs.

"Low birth weight children score significantly lower on intelligence tests than do children of normal birth weight even when sociodemographic risk factors are taken into account."

"The adverse consequences of being born low birth weight are still apparent in adolescence, and experts believe that these abnormalities will be lifelong and will not improve as the children enter adulthood.

Without a thorough medical history, few dentists might consider the association of low birth-weights with difficulties in providing dental services for some youngsters. It is in an effort to provide dental practitioners with a greater awareness of the causes, prevalence and consequences of low birth-weight that the following introduction to the subject is provided.

DEMOGRAPHIC CHARACTERISTICS

By state

In 1992, the percent of births with low birth-weights ranged from 4.9 percent in Arkansas and 5.0 percent in Maine to 9.4 percent in Louisiana, 9.9 percent in Mississippi and 14.3 percent in the District of Columbia.[2]

By race and ethnicity

In the United States, Chinese-American populations experience the smallest rate of low birth-weights, followed by Mexican-Americans, Central and South Americans and the general white population. While only 17 percent

†Unless specified, all material for this presentation was drawn from the extensive series of reports in the publication by the David and Lucile Packard Foundation, The Future of Children—Low Birth Weight.[1]

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of all births are to African-American families, 33 percent of all low birth-weight and 38 percent of all very low birth-weight births are to African-American families.

Underlying the high prevalence of low birth-weight among African-Americans is the strong association of preterm delivery and race. African-American infants are now more than twice as likely as white infants (13.3 percent vs. 5.8 percent) to be born before 37 weeks of gestation. This difference is even greater in very early preterm deliveries. But in terms of specifics, little is known about why African-American babies are at such high risk of adverse birth outcomes (Table 1).

By income and social conditions

Although poverty is strongly and consistently associated with low birth-weight, the precise social and environmental conditions that produce this problem have not been defined. It is thought that poverty with its associated reduced access to health care, poor nutrition, lower educational levels, inadequate housing, greater physical and psychological stress and fewer life satisfactions may be responsible for some of the increased risk for low birth-weight.

A variety of federal programs, including Medicaid (see below) AFDC (Aid to Families with Dependent Children) and the WIC programs (Special Supplemental Food Program for Women, Infants and Children) were developed for low income families at health and nutrition risk. It is thought that prenatal WIC participation may affect the prevalence of low birth-weight through the provision of food supplements and nutritional education during pregnancy.

Minor decreases in the prevalence of low birth-weight have been associated with AFDC participation. It has been suggested that AFDC funds are too limited to have significant direct impact on birth-weights.

In addition, although poorly understood, there are indications that the social and economic conditions in which preterm delivery and low birth-weight thrive are more prevalent in the United States than in other industrialized nations with similar or even fewer economic resources.

For example, the United States ranks 22nd in infant mortality among industrialized nations behind all five Scandinavian nations, most of the rest of Western Europe, Japan, Singapore and Hong Kong. If the white population of the U.S. were considered separately, it would rank 13th in international comparisons. When U.S. and Scandinavian infants of the same birth-weight are compared, it is found that the mortality actually is lower in the U.S. This mortality pattern highlights the fact that we have been effective in the level of treatment, but not at the level of prevention.

By age of mother

There is an increased prevalence of low birth-weights among women who give birth before 15 years of age. The prevalence of low birth-weights is even greater for young teenage mothers who smoke (Table 2).

Introduction of Medicaid

The introduction of the Medicaid program in the mid-1960s and subsequent eligibility expansions in the 1980s brought government funded prenatal and pediatric care to millions of impoverished pregnant women and their children. Reductions in low birth-weight were found for women who were covered in the early stages of the Medicaid program. Later expansions of eligibility to less

Table 1 ☐ Percent of births with low birth-weight: 1992.[2]

	Percent
White	5.8%
African-American	13.3
Native-American	6.2
Asian and Pacific Islander	6.6
Filipino	7.4
Chinese	5.0
Japanese	7.0
Hawaiian	6.9
Hispanic origin*	6.1
Mexican	5.6
Puerto Rican	9.2
Cuban	6.1
Central & South American	5.8
Total	7.1

*May be of any race

Table 2 ☐ Percent low birth-weight, by smoking status, age and race of mother: 1992.[2]

	Age of mother in years					
	<15	15–17	18–19	20–24	25–29	30–34
White						
Smoker	12.6%	11.0%	10.1%	8.8%	9.1%	10.3%
Nonsmoker	9.3	7.8	6.5	5.1	4.4	4.7
African-American						
Smoker	na	16.1	17.0	18.8	22.4	25.7
Nonsmoker	16.0	13.6	12.8	11.3	10.9	11.6
Total*						
Smoker	13.2	11.4	10.8	10.1	11.2	12.9
Nonsmoker	13.4	10.2	8.6	6.6	5.3	5.5

*Includes races not listed separately

severely socioeconomically deprived women, however, were not associated with reductions in the prevalence of low birth-weight.

"Neither the low birth weight nor the preterm delivery rate has improved in the United States in the past quarter century. Most efforts to prevent...(these outcomes) have not proven effective."

Smoking, alcohol and substance abuse

"Up to 20% of all low birth weight births could be prevented if no pregnant woman smoked cigarettes."

Cigarette smoking is the single largest modifiable risk factor for low birth-weight and infant mortality. On average, babies born to smokers weigh about one-half pound less than babies born to nonsmokers. Almost 18 percent of white and 14 percent of African-American women smoke during pregnancy. The prevalence of smoking is greatest for both white and African-American pregnant women who do not complete their highschool education, and smallest for pregnant women with some college education (Table 3).

Because nicotine is such as addictive drug, the success rates of most cessation intervention programs are low. Only 9 percent to 27 percent of women who participate in these programs are able to quite smoking during pregnancy, and an additional 17 percent to 28 percent reduce the amount smoked.

Table 3 ☐ Mothers who smoked during pregnancy, by race and educational attainment: 1992.[2]

Education in years	Percent		
	White	African American	Totals*
0–8	18.3%	11.4%	16.8%
9–11	35.9	19.3	30.6
12	22.1	13.5	20.1
13–15	12.6	10.1	12.0
16+	4.0	4.7	3.9
Not stated	17.5	20.3	17.2
Totals	17.9%	13.8%	16.9%

*Includes other races than white and African American

Table 4 ☐ Profile of substance use among pregnant women: 1987.[1]

	Percent
Abstinence	49.9%
Alcohol only	21.5
Cigarettes only	14.3
Alcohol and cigarettes	10.3
Alcohol, cigarettes and elicit drugs	2.5
Cigarettes and elicit drugs	1.4
Alcohol and elicit drugs	0.6
Elicit drugs only	0.5

And further, more than one in five pregnant women consume alcohol during their pregnancy. An additional 10 percent drink alcohol and smoke cigarettes. Two and one half percent drink alcohol, smoke cigarettes and use illicit drugs (Table 4).

PRENATAL AND POSTNATAL CARE

"Little is done during the standard prenatal care visits that could be expected to reduce low birth weight."

"...most prenatal care programs are designed to detect major complications of pregnancy, not to prevent low birth weight."

There is some doubt as to whether prenatal medical care services are truly effective in preventing low birth-weight. Self selection may have produced the apparent advantage. Women who receive prenatal care are a heterogeneous group, but generally are healthier, more educated, and more advantaged than women who do not receive prenatal care. In the late 1800s the content, timing and frequency of prenatal care were developed to detect the rare but potentially life threatening condition—eclampsia, which results in high blood pressure, convulsions and potentially death. Over time, the enrollment of all pregnant women in a system of prenatal care was promoted as a national policy, to assure the well-being of the mother and fetus and reduce the risk of low birth-weight.

In 1990s, in more than three quarters of births (77.7 percent) prenatal care is begun in the first trimester. In 5.2 percent of births, prenatal care is begun either in the third trimester or there is no prenatal care. Annually there are more than 76,000 live births with no prenatal visits.[2,3]

International comparisons also raise questions regarding the ability of prenatal care to reduce the rate of low birth-weight. American women are more likely than either German or Japanese women to start prenatal care in the first trimester, but rates of low birth-weight and infant mortality are higher in the United States.

THE FINANCIAL COSTS

"Charges for the initial hospitalization for surviving infants weighing 500-600 grams at birth averaged $1 million in one study."

"Analysis of hospital stays suggest that the cost per life-year gained may still be less—perhaps much less—for most infants with birth weights of 501 to 800 grams (1 pound, 2 ounces to 1 pound, 11 ounces) than for many adults."

Of the $11 billion spent annually on health care for infants, more than one third (35 percent or $4 billion) is spent on the added costs of low birth-weight infants beyond those costs associated with normal-weight children.

In 1988, health care, education and child care for the 3.5 to 4 million children ages 0 to 15 born with low birth-weight cost between $5.5 and $6 billion more than they would have, if those children had been born at normal weight.

- ☐ Low birth-weight accounted for 10 percent of all health costs for children. The costs were similar in magnitude to that of unintentional injuries among children and substantially greater than the direct costs of AIDS among Americans of all ages.
- ☐ Low birth-weight children ages three to five years are twice as likely to be hospitalized as normal-weight children, but also spend more time in the hospital once admitted. Increased hospitalization results in a cost of $240 million‡ more than the cost for their normal-weight peers.
- ☐ An additional $50 million‡ is spent for preschool programs for low birth-weight children than for their normal-weight peers.
- ☐ An additional $610 million‡ is spent for health care for six- to ten-year-olds (who were low birth-weight children) than for their normal-weight peers.
- ☐ About 31 percent of low birth-weight children will repeat a school grade by the tenth grade, compared to about 26 percent of normal-weight children. The associated costs for each year that low birth-weight children repeat a grade is approximately $50 million.‡

The addition of various neonatal therapies in the late 1980s has resulted in further survivals, but without changes in the developmental outcomes of the survivors. This may result in a further increase in the numbers of compromised very low birth rate children and associated costs.

"...one unavoidable side effect of the increasing success of neonatal intensive care is a moderate rise in the childhood prevalence of cerebral palsy."

By contrast, there are reports of,

"...no increase in the incidence, complexity, or severity of disability in preterm infants weighing 500 through 1250 g at birth."[4]

But none of these direct dollar figures consider the cost for a family to accommodate the disproportionate care needs of low birth-weight children—ranging from costs of travel for the variety of health services, to the time foregone by parents from other activities, to the limitation in attention to other siblings.

FROM THE PERSPECTIVE OF THE PEDIATRIC DENTIST

The reality is that there are increasing numbers of survivors of preterm deliveries and infants born with low birth-weights—and these youngsters will become the patients of pediatric dentists in a few short years. While the vast majority of low birth-weight children has normal outcomes, as a group they have higher rates of subnormal growth, illnesses and neurodevelopmental problems.

A more comprehensive awareness that cognition, attention and neuromotor functioning may be associated with a history of a low birth-weight could ease the relationships between the practitioner, the youngster and his/her family.

Treating youngsters has never been easy. But understanding the underlying causes of some of the problems with particular children should help. As you complete the medical history of your next pediatric patient remember that,

"Long term follow-up studies...indicate that the adverse consequences of being born low birth weight (are) still apparent in adolescence."

‡In 1988 dollars.

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Poor children remain poor

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

"Children were more likely than other age groups to become poor... Children were much more likely to be poor all 24 months of 1991 and 1992 than (adults) ..."[1]

"Children were more likely to be long-term participants in major assistance programs than elderly persons and non-elderly adults ..."[2]

"Those who are poor or near poor were less likely to have continuous health insurance coverage than others."[3]

Generally we refer to the number of persons or percent of the population living in poverty at a particular time. For example, in 1985, 13.0 million (or 20.7 percent of all) children, compared in 1993, to 15.7 million (or 22.7 percent of all) children lived in poverty.[4] The millions of children living in poverty, however, in 1985 are not necessarily the same children living in poverty in 1993.*

Instead of the usual cross-sectional studies (e.g. the Current Population Survey carried out by the Bureau of the Census) that provide a picture at a particular point in time, more recent longitudinal studies provide an opportunity to measure movement along the income distribution and into and out of poverty for the same persons.[4] The 1995 series of reports *Survey of Income and Program Participation* by the Bureau of the Census provides a thirty-two-month economic review (between October 1990 and August 1993) of a national stratified random sample of households that was queried repeatedly on economic and related factors.[1-3]

OVERALL FINDINGS

While the official poverty estimates of the number of poor persons rose from 35.7 million to 36.9 million between 1991 and 1992, (a "...relatively small...change in the total number and percent of persons in poverty..."), a most significant finding from the study is the frequent transition into and out of poverty.[1] Almost half (48 percent) of the surveyed population spent only two to four months living in poverty and more than two-thirds (68 percent) spent less than nine months living in poverty (Figure 1).

ABOUT FAMILIES

Between 1970 and 1991, the percent of white, single-parent families increased from 10 percent to 23 percent of all white families. African-American, single-parent families increased from 36 percent to almost 63 percent of all African-American families. Between 1980 and 1991, single-parent Hispanic families increased from 26 to 33 percent of all Hispanic families.[7]

In 1993, of families with children and a single mother,

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*For detailed reviews of the numbers of poor children in our nation at particular periods, see previous presentations in the *Journal of Dentistry for Children*.[5,6]

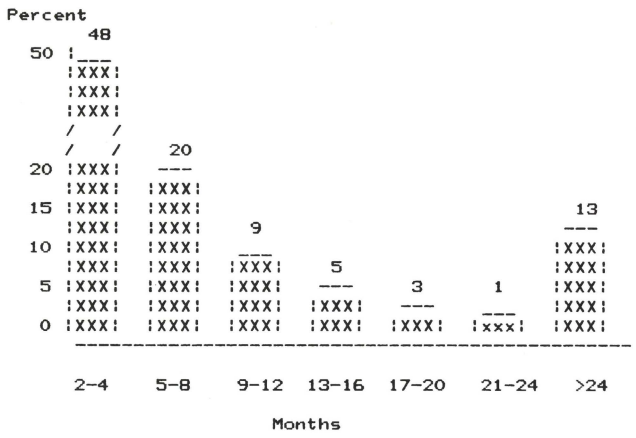

Figure 1. Distribution of poverty spells by spell length: 1991–93.[1]

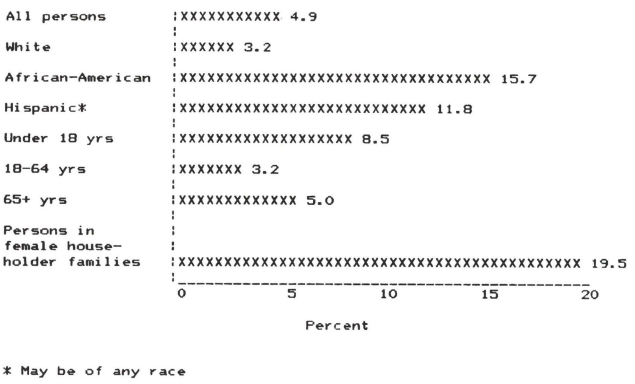

Figure 2. Percent of persons who were poor in all months by demographic characteristics: 1991, 1992.[1]

39.6 percent of white families, 57.7 percent of African-American families, and 60.5 percent of Hispanic families were living in poverty.[4] Almost one person in five (19.5 percent) living in a female-householder family remained in poverty in each month of the twenty-four months of the longitudinal study.**

Almost 16 percent of all African-Americans and 12 percent of all Hispanics, compared to 3 percent of all whites remained below the poverty line in each month of the twenty-four-month study (Figure 2).

ABOUT CHILDREN

A greater percent of children than adults (followed in the study):

- ☐ Were poor in an average month.
- ☐ Were poor in two or more months.
- ☐ Were poor in all twenty-four months (Table 1).

2.2 million children (4.2 percent of all children) who were above the poverty level in 1991, were below the poverty level in 1992.

- ☐ The percent of children entering the poverty level was greater than that of adults.
- ☐ 9.6 million children (81.2 percent of all children) who were below the poverty level in 1991 remained in poverty in 1992; by comparison, 1.9 million older adults in poverty in 1991, continued in poverty in 1992 (Table 2).

Table 1 ☐ Poverty status by age: 1991, 1992.[1]

	Less than 18 yrs	18-64 yrs	65+ yrs
Poor in average month (1992)			
Number	14.8	17.8	2.8
Percent	21.9%	11.5%	9.2%
Poor in 2 or more months (1992)			
Number	20.3	26.6	3.5
Percent	30.1%	17.3%	12.3%
Poor in all 24 months (1991-92)			
Number	5.6	4.8	1.3
Percent	8.5%	3.2%	5.0%

Note: all numbers are in millions

Table 2 ☐ Percent of persons entering and exiting poverty by age: 1991-1992.[1]

	Less than 18 yrs	18-64 yrs	65+ yrs
Above poverty in 1991	52.1	137.8	25.4
Entered poverty level in 1992			
Number	2.2	3.7	.6
Percent	4.2%	2.7%	2.2%
Below poverty level in 1991	11.8	12.6	2.2
Remained below poverty level in 1992			
Number	9.6	9.3	1.9
Percent	81.2%	74.0%	88.2%

Note: all numbers are in millions

- ☐ Between 1991 and 1993, the median duration of poverty for children less than eighteen years of age was 4.7 months; four months for adults eighteen to sixty-four years and seven months for persons sixty-five years and older. Note: the longer periods of poverty for persons sixty-five years and older reflect the extreme levels of poverty for the com-

**There are approximately 4.5 million families maintained by women with no spouse present, compared to approximately .4 million families maintained by men with no spouse present.[8]

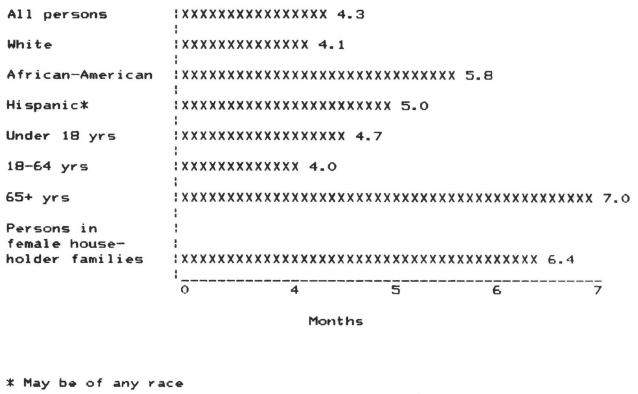

Figure 3. Median durations of poverty spells by various demographic characteristics: 1991–1993.[1]

paratively smaller number of older persons (Figure 3).

PUBLIC ASSISTANCE

15.2 million children (22.5 percent) received some form of public assistance between 1991 and 1992; compared to 9.5 percent of adults eighteen to sixty-four years and 13 percent of senior citizens.

- ☐ 9.0 million children received benefits in each of the twenty-four months reviewed, compared to 8.3 million adults and 2.9 million senior citizens.
- ☐ The median monthly benefits to youngsters (in terms of constant 1992 dollars, i.e. removing the effects of inflation) decreased from $570 in 1991 to $531 in 1992. Benefits for adults remained virtually unchanged, $418 to $417 for adults and $198 for senior citizens (Table 3).
- ☐ The impact of public assistance programs was most pronounced for senior citizens. Government transfers (e.g. Social Security, Aid to Families with Dependent Children, and Supplemental Security Income, etc.) reduced to a limited extent the percent of nonelderly living in poverty. The emphasis on support for the older population, however, reduced the level of poverty below that of children and adults between eighteen and sixty-four years (Table 4).

Table 3 ☐ Receipt of public assistance by age: 1991, 1992.[2]

	Less than 18 yrs	18-64 yrs	65+ yrs
Average monthly participation (1992)			
Number	15.2	14.8	4.0
Percent	22.5%	9.5%	13.0%
Persons ever participating (1992)			
Number	18.8	19.4	4.2
Percent	27.8%	12.6%	14.6%
Persons participating all in 24 months (1991, 1992)			
Number	9.0	8.3	2.9
Percent	13.4%	5.6%	11.1%
Median duration (months) (1991)	7.8	7.8	na
Median monthly benefits (1992 dollars)			
1991	$570	$418	$198
1992	$531	$417	$198

Notes: ☐ All numbers, except median monthly benefits, are in millions

☐ Public assistance includes, Aid to Families with Dependent Children, General Assistance, Supplemental Security Income, Food Stamps, Medicaid and Housing Assistance.

Table 4 ☐ Percent of persons below poverty level, with and without government transfer payments, by race ethnicity and age: 1992.[9]

	Base on Income*	Income without government transfers **
White		
< 18 yrs	16.9%	20.0%
65+ yrs	10.9	48.4
Total pop.	11.6	19.7
African-American		
< 18 yrs	46.6%	51.1%
65+ yrs	33.3	69.8
Total pop.	33.3	41.9
Hispanic***		
< 18 yrs	39.9%	45.6%
65+ yrs	22.0	59.1
Total pop.	29.3	36.6

*Excludes capital gains income

**Includes, Social Security, Employment Compensation, Government Education Assistance, Aid to Families With Dependent Children and Supplemental Security Income.

***May be of any race

HEALTH INSURANCE

Persons living above the poverty line (with no low income months during the study) were much more likely to have continuous health insurance coverage than persons who experienced one or more low income months. Only 14 percent of those above the poverty line spent any time without health insurance, compared with over one-half (52 percent) of those with one or more low income months. Individuals with longer periods of low income were more apt to have Medicaid coverage than those who experienced low income for shorter periods of time.[3]

BUT WE KNEW THAT ALL ALONG

Yes, we all know (or at least we should know) that one out of four children less than six years of age lives in poverty. On an annual basis, almost sixteen million children live in poverty.[9] But what most of us do not realize (as we read the anecdotal tales of welfare abuse†) is the frequent transition into and out of poverty by most public assistance recipients.

Yet the longitudinal study by the Bureau of Census presents the sobering realities that:

- ☐ The individual children remain on the poverty rolls for longer periods of time than adults.
- ☐ The poor or near poor are less likely to have continuous health insurance coverage.
- ☐ All too often, "poor children remain poor."

†For example, George Will's column in Newsweek told the story of a Puerto Rican family of seventeen children in a Boston Housing project which now has seventy-four grandchildren, "virtually all of whom have come of age in the welfare system and many of whom are beginning to apply for welfare themselves... The Rivera family of about 100 may be costing taxpayers from $750,000 to $1 million a year... When (a daughter was asked about) taxpayers' anger, she said, 'Just tell them to keep paying...'"[10]

By contrast, the House Ways and Means Committee issued a report on the typical mother on public assistance. "(She) is under thirty, neither white nor black, has two children and has not finished highschool. More than a third (37 percent) of women on welfare work their way off the rolls in two years."[11] During their lifetime, 19 percent will be on welfare 2-4 years, 20 percent will be on the 4.1 to 8 years, and 25 percent will be on welfare for more than 8 years.[11]

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THE SAFETY OF N_2O

As the debate about exactly what level of exposure is safe wages on, clinicians are wise to limit their exposure as much as possible. In addition to the steps outlined by NIOSH, others offer more advice about curbing N_2O levels.

Lieblich says that when he performs a procedure requiring N_2O, he makes sure the patient breathes 100% oxygen for five minutes at the end of the treatment before removing the mask. This ensures that the patient won't be exhaling N_2O when the mask is off. Using a rubber dam and minimizing conversation are also effective.

But most important of all, never operate the N_2O delivery without a properly functioning scavenging system. Yagiela comments, "I think every office should have scavenging equipment. It is not an expensive proposition and it seems like an easy fix."

Anderson, Karen: N_2O How safe is it? Review, 89:12–22, October 1996.

REPORTS

Neonatal sublingual traumatic ulceration (Riga-Fede disease): Reports of cases

Curt Goho, DDS

Erupted teeth at or immediately after birth is a relatively isolated phenomenon, occurring in about one in 3000 births. These teeth are known as "natal" teeth if present at birth, or "neonatal" teeth if present during the first thirty days of life. They usually represent premature eruption of a primary tooth. Mandibular primary central incisors are most often involved, although maxillary primary incisors, canines, and molars have been reported.[1,2] Neonatal teeth often present with hypoplastic enamel and underdeveloped roots, with resultant mobility.[3] Neither ingestion nor aspiration of a neonatal tooth has been reported, however, in the literature.

One complication from neonatal teeth is an ulceration on the ventral surface of the tongue caused by the tooth's sharp incisal edge. Constant trauma may create ulceration sufficient to interfere with proper suckling and feeding, and put the neonate at risk for nutritional deficiencies. In such instances, dental intervention may be required.

Ulceration of the sublingual area in infants was first described in 1857 by Cardarelli, and was associated with general wasting, leading to "exitus lethalis".[4] In 1881 and 1890 Riga and Fede described this lesion histologically. It has subsequently been known as "Riga-Fede disease". Other authors have referred to this lesion as "Riga's disease", "sublingual growth in infants", "sublingual ulcer", "sublingual granuloma", "reparative lesion of the tongue", and "traumatic ulcerative granuloma with stromal eosinophilia".[4-9] A more appropriate, descriptive term is "neonatal sublingual traumatic ulceration".

Treatment of this lesion has varied over the years. Early treatment consisted of excision of the lesion.[4,5,7,10] Due to the erroneous diagnosis of the etiology, resolution of the lesion occurred only upon weaning of the child. Allwright advocated maintaining the neonatal tooth by smoothing the incisal edge with an abrasive instrument.[11] In cases of mild to moderate irritation to the tongue, such treatment may suffice. If the ulcerated area is large and denuded, however, even a reduced incisal edge may still contact and traumatize the tongue during suckling, enough to delay healing.

Rococz placed a smooth, rounded composite restoration over the offending tooth and healing occurred in two weeks.[12] This patient, however, was ten months old, had normal, nonmobile primary incisors, and a chronic lesion that was painless, due to the insensitivity to pain associated with familial dysautonomia. Immediate resolution of the lesion was not as critical as with an acute ulceration in a neonate, nor was the extreme mobility of neonatal teeth an issue in this case. Extraction of the traumatizing tooth is another treatment option and may be required for rapid resolution of a lesion.[13,14] Vitamin K supplementation should be confirmed with the infant's pediatrician before extraction during the first ten days of life.

The following cases document the treatment of neonatal teeth, to resolve neonatal sublingual traumatic ulcerations.

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The opinions expressed in this paper are those of the author and not necessarily those of the U.S. Army Dental Corps.

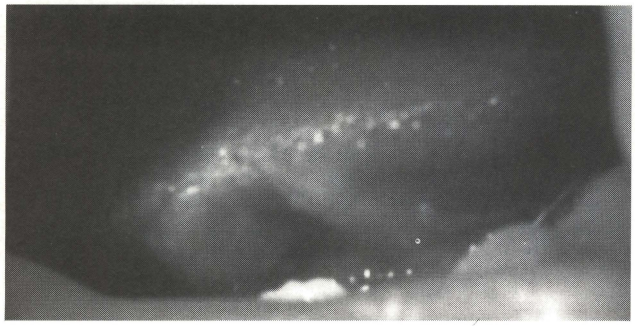

Figure 1. "Riga-Fede Disease". Sublingual ulceration caused by neonatal tooth.

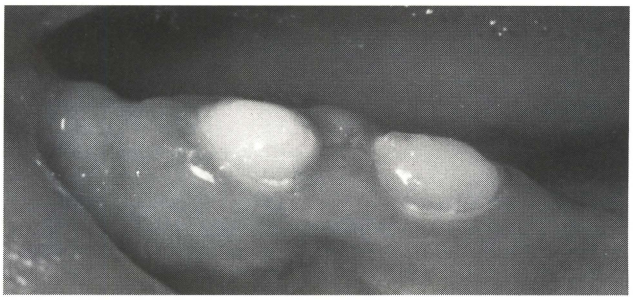

Figure 2. Smooth composite coverage of rough enamel on neonatal teeth to resolve sublingual ulcerations.

CASE 1

A twenty-one-day-old female was referred by a pediatrician for evaluation of an ulcerated area on the ventral surface of the tongue. The mother reported that the child exhibited pain during suckling, and would not nurse. The attending pediatrician attributed the infant's refusal to nurse to the tongue ulceration, and had significant concerns over the infants lack of weight gain and the potential for infection of the ulcerated area.

Dental examination revealed a tooth projecting 4 mm from the mandibular alveolar ridge in the left central incisor position. The natal tooth showed Class II mobility. The ventral surface of the tongue exhibited a 7 mm × 12 mm ulceration that extended from the anterior border of the tongue to the lingual frenum (Figure 1). Any palpation of the area elicited a pain response from the patient. Radiographic examination revealed a natal tooth, probably a primary incisor, with a well-formed crown but minimal root development.

The pediatrician's concern over the infant's failure to gain weight due to the ulceration's interference with suckling dictated the need for rapid resolution of the lesion. Extraction of the natal tooth was chosen over more conservative, but slower healing treatments. The area was anesthetized with 0.1 cc 2 percent lidocaine and the tooth was extracted with forceps. At a follow-up appointment five days later, the lesion was fully resolved, and the infant was feeding normally. The parent was advised that the tooth probably was a primary incisor, and that evaluation of eruption and space maintenance would be required.

CASE 2

A ten-day-old female was referred by the pediatrics clinic for evaluation of natal teeth. Two natal teeth were present in the primary central incisor position. The enamel was thin, and the incisal edges were rough and abraded. The teeth exhibited Class I mobility. The sublingual area was slightly irritated, and was attributed to trauma from the lower incisors during suckling. A radiograph confirmed that these teeth were primary central incisors. Root development was minimal.

Because of the minimal ulceration on the tongue, treatment consisted of reducing the rough incisal edge, etching the enamel, and placing a smooth, rounded composite coverage over the incisal portion of the tooth (Figure 2). At the one-week follow-up, the sublingual abrasions were resolved.

SUMMARY

Neonatal sublingual traumatic ulcerations (Riga-Fede disease) can present as mild to severe ulcerations. The associated pain can interfere with infant nursing and nutrition. The preferred, conservative treatment consists of smoothing rough incisal edges, or placing round smooth composites over the incisal edges. Acute ulcerations, and a need for definite, rapid healing, may require extraction of the neonatal teeth.

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THE IMPORTANCE OF PARENTS

In addition to understanding your children, it is also vital for you to understand that you, the parent, are a participant in their journey. By placing so much emphasis on the technology of behavior, we have drawn our experts into the spotlight and seem to relegate parents to the role of spear carriers or walk-ons. This will never play well to the children. You, the parent, are the most important person in their lives and you alone are their best teacher. Experts may provide technical advice or directions but they will never replace you. This is why I have placed such an emphasis on parents as the teachers of their children.

Williamson, Peter: *Good kids, Bad behavior.*
New York: Simon and Shuster, 1990, p 242.

Transposition and fusion in the primary dentition: Report of case

Karen Duncan, BDS, LDSRCS, MSc
Peter J.M. Crawford, BDS, MScD, FDS RCSEd, MCPCH

Transposition of maxillary teeth is a well recognized phenomenon in the permanent dentition: canines and first premolars are the teeth most commonly involved; the lateral to central incisor displacement is very uncommon (3 percent).[1] An extensive search of the literature offered no reports of transposition in the primary dentition.

Fusion is defined as the partial or complete union of two adjacent tooth germs, resulting in a bifid crown and two root canals. By contrast, gemination arises from a single tooth germ and results in a bifid crown with a single root or root canal.[2] The term "double teeth" is sometimes used to describe fusions and geminations, a failure by authors to differentiate between the two phenomena.[3]

The reported prevalence of fused teeth in the primary dentition is low, varying from 0.14 percent to 5 percent, with no gender predilection.[4,5] They have been reported only in the anterior region, more often in the mandible than in the maxilla.[2] Approximately 50 percent of cases of fusion in the primary dentition experience further problems in the permanent dentition, including missing incisors, supernumerary teeth, double teeth, and delayed eruption.[5,6]

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CASE REPORT

A five-year-old girl was referred to the Dental Hospital by her general dental practitioner, who expressed concern at her high caries rate. On examination, the twenty primary teeth were present. Widespread carious lesions were apparent.

In addition, the maxillary right incisors were transposed and fused. The clinical crowns of the teeth typified a primary central incisor and a primary lateral incisor, respectively, and corresponded to the appearances of the contralateral teeth (Figures 1, 2). The vertical groove between the two fused crowns was carious on its labial aspect.

Radiographs showed that the fused primary crowns had separate root formations (Figures 3, 4). They also revealed the probable fusion of the underlying permanent central and lateral incisors at this site. These teeth did not appear, however, to be transposed.

On the left, the permanent central incisor appeared to be larger than expected. The lateral incisor was present, with its image apparently superimposed on the enlarged left central incisor. The permanent canine tooth appeared rotated. All other teeth appeared to be developing normally.

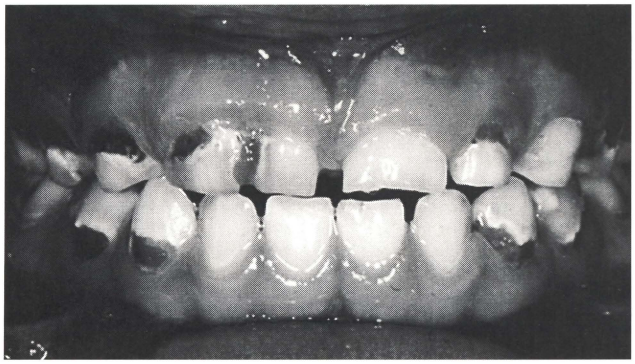

Figure 1. Labial view of patient A. S. showing widespread caries and "double tooth" in site 51, 52 (Maxillary right primary incisors).

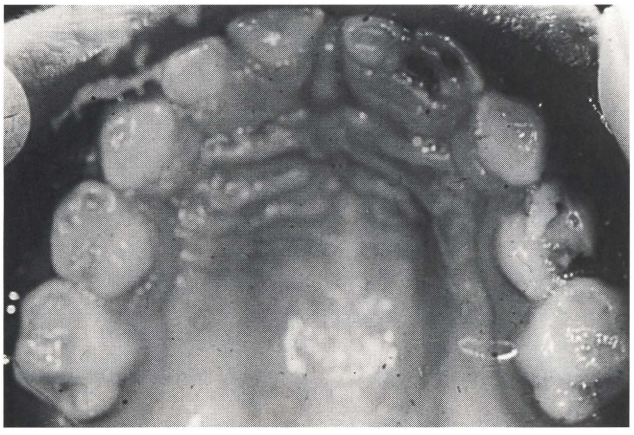

Figure 2. Palatal (mirror) view of patient A. S. showing "double tooth" in site 51, 52 with dental appearances suggestive of transposition of these teeth.

DISCUSSION

The present case appears unique to the literature because it concerns the transposition of primary teeth.

Peck and Peck, in their authoritative review of permanent maxillary tooth transpositions, noted that four of the five sites of transposition seen in the literature involved a canine tooth and that the central–lateral incisor transposition was the only exception.[1] They suggested three possible etiological factors in cases of transposition: genetic influences; the early loss of primary teeth; and the effects of trauma, either directly upon the forming permanent teeth or indirectly, by creating space into which a forming tooth might migrate.

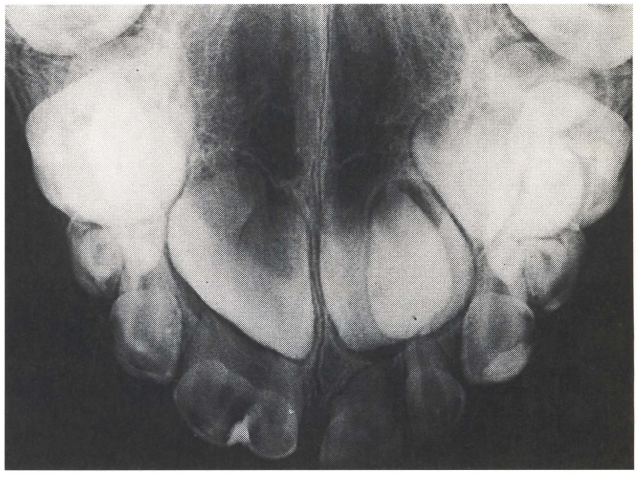

Figure 3. Nasoclusal radiograph of patient A. S. showing two-crown, two-rooted appearance of "double tooth" in site 51, 52 and probable fusion of the underlying permanent central and lateral incisors at this site. The left permanent central incisor appears to be larger than expected.

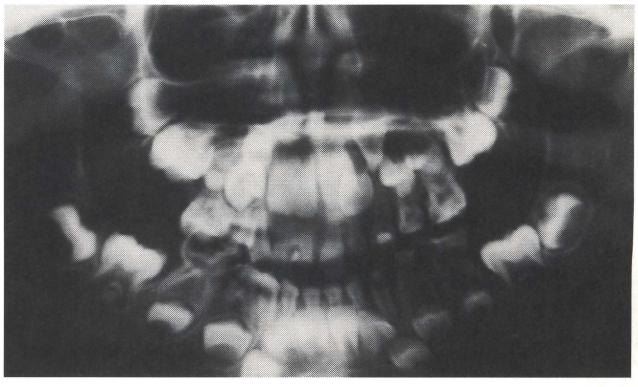

Figure 4. Orthopantomogram showing widespread dental caries, the presence of all forming permanent teeth with the exception of third molars, separation of the crowns of 22 and the enlarged 21, and a rotated 23.

There was no history of injury in the present case, or of a preeruption disturbance or other local abnormality.

Peck and Peck proposed a further category of *pseudotranspositions*, possibly related to the presence of a supernumerary tooth in the (apparently) transposed site. The present case shows no evidence of additional tooth-structure in the primary dentition, mesial to the maxillary right canine, which might have supported the view that the structure presented was a fusion between the

primary central incisor and a mesiodens. We are at a loss to suggest any alternative etiology for this finding and suggest that the diagnosis is one of true transposition, allied to fusion of the affected teeth.

"Double teeth" in the primary dentition have been reported to be associated with disturbances in the permanent teeth varying from macrodontia, to enlargement of the contralateral tooth, to supernumerary teeth or even missing teeth.[5] In the present case it is interesting to note that there are further disorders of tooth development affecting the permanent teeth in the upper anterior region. It is likely, therefore, that there is an underlying genetic predisposition to tooth malformation, acting either generally or locally.

The management of the case as a whole has involved the introduction of strict preventive measures in view of the high caries rate and poor patient compliance. The management of the fused teeth involves preventive measures, backed up with clinical intervention as deemed appropriate.

Prolonged retention of the fused teeth will have to be addressed, when it occurs.[5] The esthetics, alignment and condition of the two macrodont permanent incisors will undoubtedly influence the future management and treatment of the maxillary labial segment.

SUMMARY

A case is reported of the fusion and transposition of the maxillary right central and lateral primary incisors. The unerupted permanent successors exhibit anomalies as detailed previously in this paper.

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WHAT ABOUT TODAY'S CHILDREN?

What about today's children? Are they really so much more difficult? They are, of course, healthier than they have been in the past. That is, they are less susceptible to the types of infectious diseases that would regularly hew down a good proportion of children in generations past. Now we are made aware of disorders that have only recently been discovered—or invented. We are told of children having "attention deficit disorders." We have to worry about our children's cholesterol levels and need to worry about their level of stress. We are told there are epidemics of teenage suicides in various communities. Of course, there is the glassy-eyed spector of drug abuse nodding off in any child's room of the future.

Williamson, P.: *Good kids, bad behavior.* New York: Simon and Shuster, 1990 p 238–239.

Talon cusp in permanent dentition associated with other dental anomalies: Review of literature and reports of seven cases

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Kazem S. Al-Nimri, BDS

The talon cusp is an uncommon dental anomaly referring to an accessory cusplike structure projecting from the cingulum area or cementoenamel junction of the maxillary or mandibular anterior teeth in both the primary and permanent dentitions. This anomalous structure is composed of normal enamel and dentin and varying extensions of pulp tissue. The first recorded case of talon cusp was in 1892, when W.H. Mitchell described an accessory cusp on the lingual surface of an upper central incisor as "a process of hornlike shape, curving from the base downward to the cutting edge".[1] Since then the anomaly has received very little attention and was only briefly addressed in some textbooks of oral pathology.[2,3] It was not until 1970 that the talon cusp was again mentioned in the literature. Mellor and Ripa named the accessory cusp as talon cusp because of its resemblance in shape to an eagle's talon.[4] Gorlin and Goldman defined talon cusp as a very high accessory cusp, which may connect with the incisal edge to produce a T-form or, if lower in level, a Y-shaped crown contour.[5] Shafer *et al* reported that the talon cusp occurs in permanent incisors.[6] Henderson in 1977 first described a case of talon cusp in a primary tooth, involving the maxillary central incisor of a four-year-old Filipino girl.[7] Recently, Chen registered six cases of talon cusp in primary incisors of Chinese children.[8]

The etiology of the condition remains unknown. Similar to other defects in tooth form, talon cusp originates during the morphodifferentiation stage of tooth development. It may occur as a result of outward folding of inner enamel epithelial cells (precursors of ameloblast) and transient focal hyperplasia of the peripheral cells of mesenchymal dental papilla (precursors of odontoblast). Control of the complex processes of dental development appears to be multifactorial: primary polygenetic with some environmental influence. The majority of cases reported in the literature indicate that talon cusp is an isolated anomaly rather than an integral part of any disorder. Mader suggested that talon cusp may be associated with other somatic and odontogenic anomalies, such as peg-shaped lateral incisor and impacted mesiodens and canine.[9] Natkin *et al* reported a case of talon cusp accompanied with complex odontoma and impaction.[10] Davis and Brook found that talon cusp was associated with supernumerary teeth, megadont, and dens evaginatus.[11] The anomaly also appears to be more prevalent in patients with Rubinstein-Taybi syndrome, Mohr syndrome (oral-facial-digital II syndrome), and Sturge-Weber syndrome (encephalo-trigeminal angiomatosis).[8,12,13]

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The talon cusp affects both sexes and may be unilateral or bilateral. This anomaly varies widely in shape, size, structure, location, and site of origin. Some reports have presented talon cusp as a markedly enlarged or exaggerated cingulum on the maxillary incisors.[13,14] Others described talon cusp as a "projection of a millimeter or more ..." or "extends at least half the distance from the cementoenamel junction to the incisal edge".[9,15,16] Some talon cusps are quite sharp and spike-like, while others have rounded and smooth tips. Talon cusp can be horn-like, conical, or pyramidal in shape. The tip of the cusp may stand away from the rest of the crown or it may be in close approximation to the lingual surface of the tooth. An extension of pulp tissue into the cusp may or may not be present.[4,8-10]

Radiographically, the talon cusp is visible as a radiopaque structure, in which the enamel, dentin, and occasionally the pulp can be seen. Typically, the cusp looks like a V-shaped structure superimposed over the normal image of the crown. A talon cusp on an unerupted maxillary incisor may present diagnostic problems, because its radiographic image can mimic a mesiodens or compound odontoma and lead to an unnecessary surgical procedure in an attempt to remove these "supernumeraries".

The present report describes seven cases of talon cusp found on the palatal surfaces of permanent maxillary incisors. All cases were Jordanian Arabs, presented to the Dental Center at the Faculty of Dentistry for routine examination or seeking various kinds of treatment during the period of September 1992 to February 1993.

REPORTS OF CASES

Case 1

K. M., a healthy seventeen-year-old male reported to the dental clinic for the replacement of an accidentally missing maxillary left central incisor.

A clinical examination revealed no soft tissue abnormalities. The occlusion was Class I molar relationship with excessive overbite, approximately 4 mm, and spacing in the anterior segments of the maxilla and mandible. Examination disclosed a prominent, cusplike structure on the palatal surface of the maxillary right lateral incisor. The talon cusp was pyramidal in shape and extended from the cementoenamel junction to the incisal edge. It was attached to the palatal surface and extended perpendicularly to the mesiodistal surface of the tooth crown, forming with the incisal edge a T-shaped outline (Figure 1). The tip of the anomalous cusp was rounded and not irritating. The cusp measured 7 mm in length (incisocervically), 4.5 mm in width (mesiodistally), and 2 mm in thickness (labiolingually). Deep developmental grooves were present on the lateral aspects of the cusp where it joined the palatal surface of the tooth. These grooves were packed with dental plaque. A carious lesion was detected, clinically and radiographically, in the distal groove. Electric pulp testing showed that the sensitivity of the taloned tooth was about one-third that of the neighboring teeth. The talon cusp was found to interfere with the occlusion and wear-facets were present on the cusp and the incisal edge of the opposing tooth. A periapical radiograph showed a typical V-shaped radiopaque structure superimposed over the image of the affected crown, with the point of the "V" toward the incisal edge (Figure 2). The cusp was demarcated by two distinct white lines converging from the cervical area of the affected tooth toward the incisal edge. Pulp extension could be traced radiographically to the middle of the cusp.

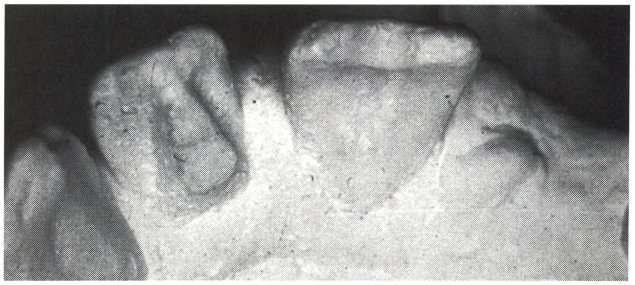

Figure 1. Case 1. Close-up of cast of maxilla showing talon cusp, pyramidal in shape, forming with the incisal edge T-shaped outline on the palatal surface of the maxillary right lateral incisor.

Other dental abnormalities detected were: (1) a shallow groove "enamel cleft" on the labial surface of the taloned tooth extending from the incisal edge to the cervical third of the crown; (2) bifid cingulum on the maxillary right central incisor.

Case 2

I.N., a nine-year-old female with a complaint of "unesthetic appearance" of her teeth was examined.

The oral inspection showed a Class II occlusion. The right maxillary lateral incisor exhibited a pronounced, well-demarcated anomalous cusp connected to the palatal surface of the crown. The cusp, conical in shape, projected from the cementoenamel junction, occupied the mesial half of the palatal surface, and extended al-

Figure 2. Radiograph showing talon cusp with pulp horn extended to the middle of the cusp. Note the carious lesion in distal developmental groove (Case 1).

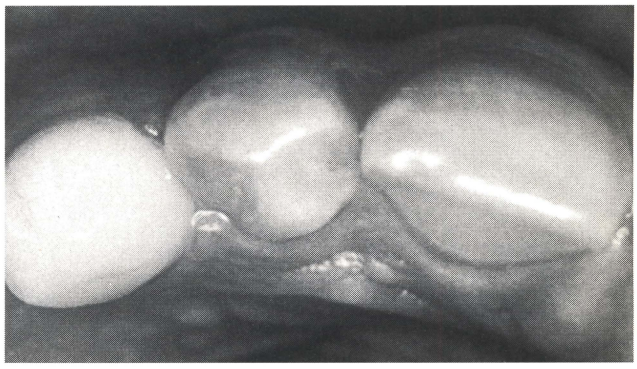

Figure 3. Case 2. Occlusal view showing talon cusp, conical in shape, on the maxillary right lateral incisor. The cusp extended almost to the incisal edge, forming a Y-shape crown outline.

most to the incisal edge, forming a Y-shape crown outline (Figure 3). The tip of the cusp was somewhat sharp and coincided with the midline of the long axis of the tooth. The cusp measured 7 mm in length, 4.5 mm in width and 2.5 mm in thickness. A noncarious developmental groove was found on the mesial aspect of the talon cusp. The taloned tooth responded normally to electric pulp testing. The cusp did not irritate the tongue during speech and mastication nor interfere with occlusion, perhaps due to the increased overjet. Radiographically, separate enamel and dentin were seen in the V-shape structure, but pulp extension could not be traced (Figure 4).

Other dental abnormalities recorded were: (1) a shallow groove running vertically over the entire labial surface of the tooth; (2) the maxillary primary canines had accentuated palatal ridges; (3) panoramic radiograph revealed agenesis of the maxillary permanent left canine.

Figure 4. Radiograph of tooth depicted in Figure 3 showing talon cusp outlined by two thin white lines representing the enamel, but pulp extension could not be traced.

Case 3

N.H., a sixteen-year-old male, was referred by a private dentist for evaluation of an overretained maxillary right primary central incisor and an impacted succedaneous tooth. His medical and dental history was noncontributory.

Intraoral examination revealed a full complement of permanent teeth, except for the "missing" maxillary right central incisor, and a Class I molar relationship with reduction in the overjet. Inspection showed that the maxillary left lateral incisor had a prominent, well-defined talon cusp on the palatal aspect, projecting from the cervical margin and extending almost to the incisal edge.

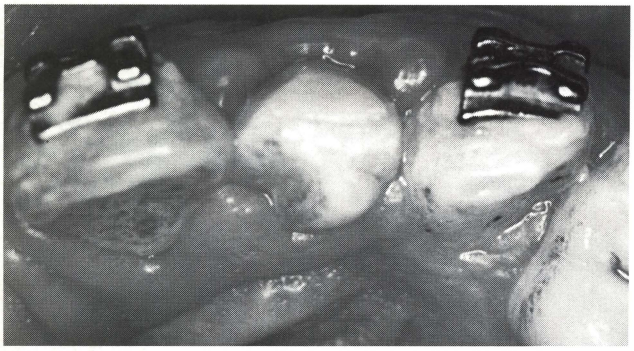

Figure 5. Case 3. Palatal view revealing talon cusp on the left lateral incisor. The tip of the cusp is sharp and irritating.

The cusp occupied the distal half of the palatal surface with its tip deviated to the middle of the incisal edge (Figure 5). The tip was pointed and sharp, causing irritation to the tongue during speech and mastication. The anomalous cusp measured 6 mm in length, 4 mm in width, and about 2 mm in thickness. Noncarious, shallow developmental grooves were present at the junction of talon cusp with the palatal surface of the tooth. Responses to electrical pulp testing were within normal limits. The cusp interfered with occlusion and attrition marks were present on both sides of the cuspal tip. The talon cusp was composed of enamel, dentin and a horn of pulp tissue (Figure 6).

Other odontogenic aberrations detected were as follows: (1) shovel-shaped contralateral maxillary lateral incisor associated with dens invaginatus (type 1); (2) accessory cusps on the distal side of the palatal surface of the maxillary right second premolar and first molars. These cusps resemble the talon cusp in the anterior teeth. They were pyramidal in shape and extended from the cementoenamel junction approximately half way to the occlusal surface.

Figure 6. Radiographic appearance showing talon cusp composed of enamel, dentin and a horn of pulp tissue (Case 3).

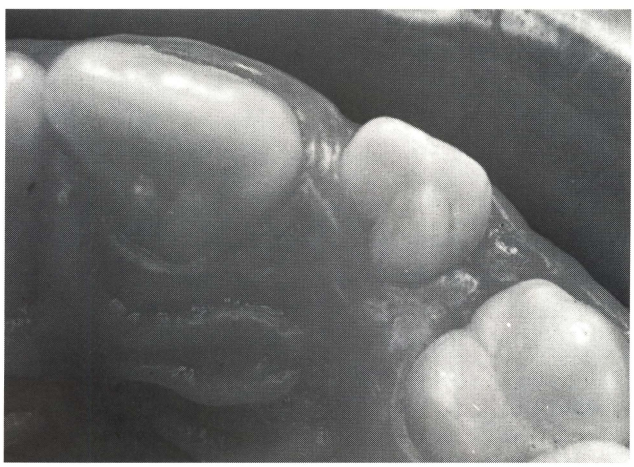

Figure 7. Case 4. Talon cusp on the maxillary left lateral incisor, forming Y-shaped crown outline. The talon cusp resulted in proclination of the affected tooth. Observe the shovel-shape central incisor with enlarged bifid cingulum (Trace talon).

Case 4

M.A., a nine-year-old male presented with an abnormally shaped maxillary left lateral incisor and excessive overbite.

Oral examination disclosed a talon cusp on the palatal surface of the maxillary left lateral incisor. The cusp, pyramidal in shape, was projecting from the cingulum area, occupied the distal half of the crown and extended to within 0.5 mm of the incisal edge, forming a Y-shaped crown outline (Figure 7). The tip of the talon cusp was in close proximity to the crown surface. The cusp measured 5 mm in length, 3.5 mm in width, and 2 mm in thickness.

Noncarious developmental grooves were observed at the lateral sides of the cusp. The involved tooth was not fully erupted and drifted labially. Otherwise, the tooth appeared normal and it responded normally to electrical pulp testing. Radiographic examination of the affected tooth revealed an inverted V-like radiopaque structure, composed of enamel and dentin of normal radiodensity.

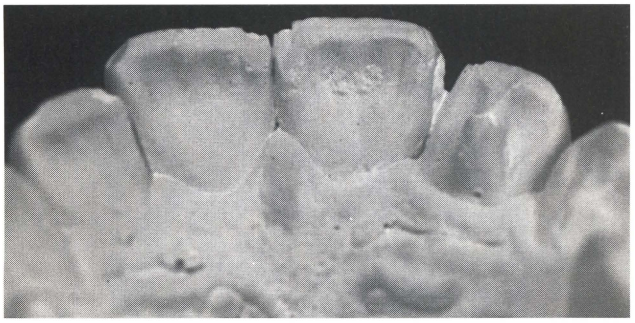

Figure 8. Case 5. Cast model of the maxilla showing talon cusp on the left lateral incisor extended to within 1 mm of the incisal edge, forming Y-shaped crown contour.

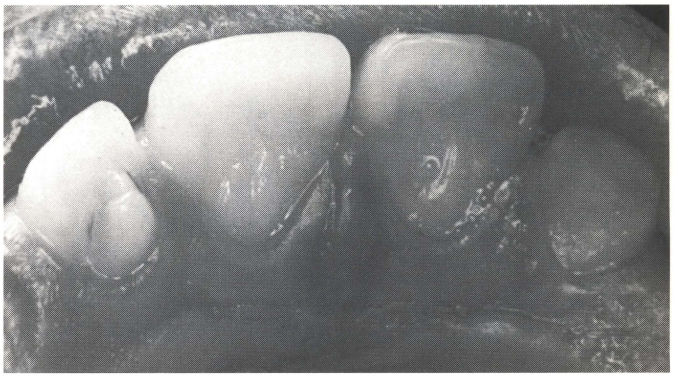

Figure 9. Case 6. Photograph showing talon cusp on the maxillary right lateral incisor extending the halfway to the incisal edge and also disclosing carious lesion of the distal aspect of the cusp. Note the shovel-shaped central incisors: The right central incisor had bifid cingulum and the left central incisor exhibited tubercle-like cingulum.

No distinct pulpal horn entering the anomalous cusp could be traced radiographically. The cusp did not interfere with occlusion and the patient had no symptoms related to it.

Other dental abnormalities observed were: shovel-shaped central incisors with a bifid cingulum on the left central incisor and tubercle-like cingulum on the right central incisor.

Case 5

T.R., a twelve-year-old female reported to the dental clinic for routine check-up.

During the clinical examination, a talon cusp was noted on the maxillary left lateral incisor. The cusp, pyramidal in shape, was projected from the cementoenamel junction, occupied the central portion of the palatal surface and extended to within 1 mm of the incisal edge. The anomalous cusp was attached to the palatal surface and the tip of the cusp was connected to the incisal edge by two divergent enamel ridges, giving rise to a Y-shaped crown contour (Figure 8). The cusp measured 5 mm in length, 4 mm in width and 2 mm in thickness. Shallow fissures were present along the mesial and distal aspects of the cusp where it joined the palatal surface of the tooth. None of the fissures however was carious. The involved tooth responded normally to electric pulp testing. Attrition marks were seen on the tip of the talon cusp and the incisal edge of the opposing tooth. Radiographic examination disclosed an abnormal cusplike structure composed of enamel and dentin; the pulp extension, however, was not discernible.

Other dental variations were: (1) accentuated marginal ridges on the contralateral incisor, (2) large cusps of Carabelli were present on the maxillary first molars.

Case 6

B.A., an eleven-year-old female was seen for a routine dental examination. Inspection showed that the maxillary right lateral incisor exhibited a talon cusp on the palatal surface, pyramidal in shape and extending from the cementoenamel junction halfway to the incisal edge. The tip of the cusp was pointed and coincided with the midline of the long axis of the tooth (Figure 9). The cusp measured 4 mm in length, 3 mm in width and 3 mm in thickness. Deep developmental grooves were present on the lateral aspects of the cusp. These grooves harbored dental plaque, and the distal groove was carious. Response to electrical pulp testing of the affected tooth was within normal limits. The talon cusp did not interfere with the occlusion, because the patient had an anterior open bite. Pulp extension could be traced radiographically to the base of the talon cusp (Figure 10).

The talon cusp was accompanied by shovel-shaped central incisors. The right central incisor had a bifid cingulum and the left central incisor exhibited a tubercle-like cingulum.

Case 7

A.R., an eighteen-year-old male presented with a periodontal problem. His medical and dental history was uneventful.

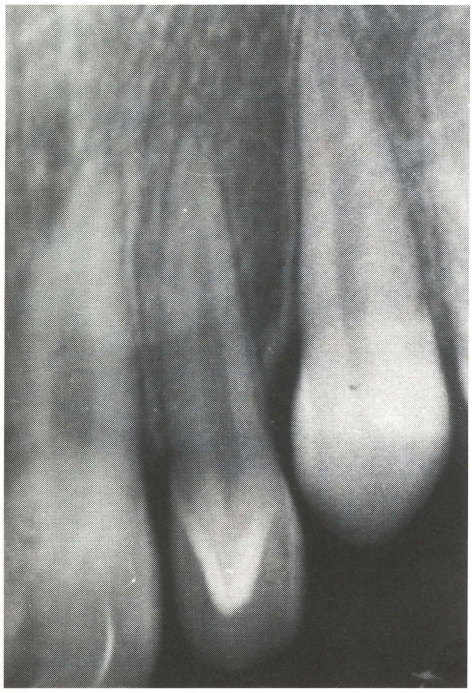

Figure 10. Radiograph of Case 6 indicating pulp extension limited to the base of the cusp.

Figure 11. Case 7. Close-up of cast of maxilla revealing different forms of talon cusps affecting the anterior teeth. The anomalous cusps on the canines and left lateral incisor are teatlike and extending one-third the distance from the cementoenamel junction to the incisal edge. They are categorized as a variation of enlarged cingula and classified as trace talon (see Discussion). The talon cusp on the right lateral incisor, cylindrical in shape, extends less than half the distance from the cementoenamel junction to the incisal edge: classified as semitalon (see Discussion). The central incisors shows bifid cingula (Trace talon).

Clinical examination revealed a large tongue (macroglossia) with flaring of the anterior teeth. Four small prominent cusps were observed on the palatal surfaces of the maxillary lateral incisors and canines. The cusps on the canines and left lateral incisors were teatlike, extended one-third the distance from the cementoenamel junction to the incisal edge. These anomalous cusps represent a variation of enlarged cingula and were classified as trace talon (see Discussion). The accessory cusp on the right lateral incisor, cylindrical in shape, was projected from the cementoenamel junction and extended less than halfway to the incisal edge (Figure 11). This type of accessory cusp was classified as semitalon (see Discussion). The developmental groove on the distal side of the semitalon cusp was dark stained.

A periapical radiograph revealed a tubercle-like radiopaque structure composed of enamel and dentin; the pulp extension, however, was not visible (Figure 12). The enamel component of these anomalous cusps appeared as two white lines emerging from the cervical third of the root and extended toward the tip of the cusp forming a continuous outline.

Other dental abnormalities noted were: (1) shovel-shaped maxillary right lateral incisor; (2) bifid cingulum on the maxillary right incisor; (3) additional tubercle on the maxillary left central incisor; (4) exaggerated cusps of Carabelli on the maxillary first molars (Figure 13).

DISCUSSION

Talon cusp is a relatively rare odontogenic anomaly arising during the morphodifferentiation phase of tooth development. It is not an entirely innocuous defect, however, because it may give problems to the patient and clinician. There are insufficient data on the prevalence of this anomaly, criteria for categorization, association with other dental abnormalities, and management. Since talon cusp was first described in 1892, about seventy-three cases (with ninety-six affected teeth) have been reported in the primary and permanent dentitions. A review of the literature, including the present work, showed that fifty-five (75 percent) of the cases exhibited talon cusp in their permanent dentition and eighteen (25 percent) of the cases in the primary dentition. Males showed a higher frequency than females. Of the seventy-three cases, the male to female ratio was 47:26 (64 percent vs 36 percent). Talon cusp showed a striking predilection for the maxilla over the mandible. In 92 percent of the cases, the cusp was found in the maxillary anterior teeth. One-fifth of the cases showed bilateral distribution of talon cusp. All reported talon cusps in primary teeth affected the maxillary central incisors, while in the permanent dentition the maxillary lateral incisor was most frequently involved (67 percent) followed by the central incisors (24 percent) and canines

Figure 12. Radiographically, the accessory cusp in Case 7 deviates in appearance from the typical V-shape talon cusp. It is mainly composed of enamel and emerging from the cervical part of the root.

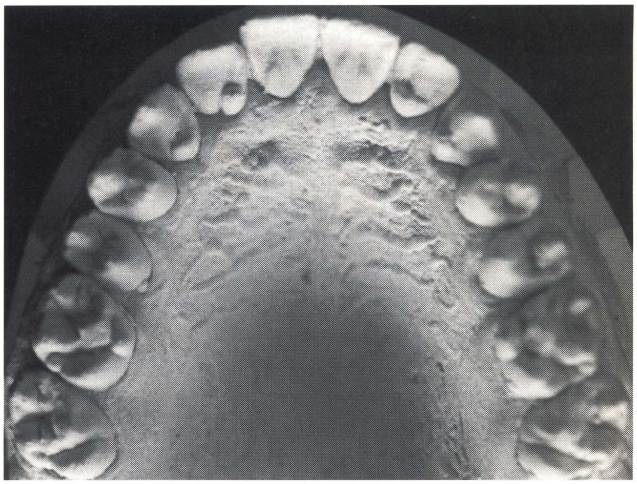

Figure 13. Full-arch view of Case 7. Large cusps of Carabelli are seen in the maxillary first molars.

(9 percent). Recently, a "facial" talon cusp was observed on the maxillary right central incisor of an eight-year-old black girl.[17]

The present series of cases showed an association of talon cusp with other dental anomalies and variations. Out of the seven cases, there were four with shovel-shaped maxillary incisors, three cases of bifid (divided) cingula, two cases with additional tubercles on the palatal surfaces of the central incisors, two cases of exaggerated cusps of Carabelli, and two cases with dens invaginatus (type 1; coronal) in the lateral incisor (Table 1). A shovel-shaped incisor is a polygenic inheritable trait characterized by accentuated marginal ridges that surround a deep lingual fossa.[18] Dens invaginatus or "dens in dente" is a developmental defect resulting from invagination in the surface of a tooth crown before calcification has occurred, i.e. exaggeration in the development of the palatal pit.[19] As in the case of talon cusp, the maxillary lateral incisors are the most commonly af-

Table ☐ Cases of talon cusp reported by the present study.

Case No.	Sex	Age (years)	Taloned teeth	Manifestations	Associated abnormalities & teeth involved
1	M	17	22	Caries, interfered with occlusion, attrition	Labial groove 12, bifid cingulum 12, agenesis, 18, 28
2	F	9	12	None	Labial groove 12, agenesis 23
3	M	16	22	Interfered occlusion, irritating to tongue, attrition	Dens invaginatus 12, accessory cusp 16, 15, 26
4	M	9	22	None	Labial drifting 22, additional tubercle 11, 21, shovel incisors 11, 21
5	F	12	22	Interfered with occlusion, attrition	Prominent marginal ridge 12, exaggerated cusp of Carabelli 16, 26
6	F	11	12	Caries	Shovel-shaped 11, 21
7	M	18	13,12, 22,23	None	Shovel-shaped 12, bifid cingulum 11,21 exaggerated cusp of Carabelli 26

fected teeth with shoveling and dens invaginatus than central incisors and canines. The susceptibility of the lateral incisors to abnormalities could partly be related to compression of the tooth germ of the lateral incisor by the adjacent central incisor and canine, which develops about seven months earlier than the lateral incisor. Increased localized external pressure on a tooth germ during the morphodifferentiation stage may result in either outfolding of the dental lamina as in the case of talon cusp and shoveling or infolding of the dental lamina as in dens invaginatus. Family histories revealed that four of the cases reported here have consanguineous parents.

Talon cusp has been reported as a rare or quite uncommon condition. The term talon cusp has been applied, however, somewhat loosely to an accessory cusp-like structure that varies considerably in size, ranging from an enlarged cingulum to a well-delineated anomalous cusp extending at least to the half crown height.[9,11,13,14] Davis and Brook stated that talon cusp may represent the extreme of a continuous variation progressing from a normal cingulum to an enlarged cingulum to a small accessory cusp to a talon cusp.[11] In a clinical and radiographic survey on American children, Buenviaja and Rapp reported that the prevalence of talon cusp was 0.17 percent, but did not indicate the criteria used for diagnosis of talon cusp, nor whether the anomaly occurred in the primary or permanent dentition.[20] Chawla *et al* used wider criteria for categorization of an accessory cusp as a talon cusp and found a prevalence of 7.7 percent in the North Indian children.[15] Natkin *et al* questioned the general assertion that talon cusp is a rare anomaly.[10] Support for this view came from Poyton who stated that enlarged cingula and additional cusps "are seen not infrequently in maxillary anterior teeth".[21] The incidence of talon cusp in the Chinese population is much higher than previously considered.[8,11] Our observation on the occurrence of talon cusp in Jordanian-Arabs indicates that this anomaly is not a rarity.

It is essential to have precise criteria for categorization of an accessory cusp as a talon cusp. Without standardization of terminology and firm diagnostic criteria, the prevalence of the talon cusp and its clinical significance cannot be reliably estimated and evaluated. We suggest a classification system of these anomalous cusps based on the degree of their formation and extension. These are:

Type 1. Talon. A morphologically well-delineated additional cusp that prominently projects from the palatal surface of a primary or permanent anterior tooth and extends at least half the distance from the cementoenamel junction to the incisal edge (Figures 1 to 10).

Type 2. Semitalon. An additional cusp of a millimeter or more but extending less than half the distance from the cementoenamel junction to the incisal edge. It may blend with the palatal surface or stand away from the rest of the crown (Figure 11).

Type 3. Trace talon. Enlarged or prominent cingula and their variations, i.e. conical, bifid, or tubercle-like (Figure 11). Radiographically it may appear typically as a V-shaped radiopaque structure, as for true talon and semitalon, or tubercle-like, originating from the cervical third of the root (Figure 12).

Several reports indicated that talon cusps usually contain an extension of the pulp tissue.[4,6,7] Radiographic examination is inherently difficult in tracing pulpal configuration inside the talon cusp, because the cusp is superimposed over the affected tooth crown. Microscopic and radiographic examinations of ground sections of extracted or exfoliated teeth with talon cusp failed to reveal a pulpal horn in the anomalous cusp.[10,16,22] Pitts and Hall reduced a talon cusp by 3 mm in one visit without pulp exposure.[23] We have removed 1 to 1.5 mm from six talon cusps without exposing the pulp. It has been suggested that large talon cusps, especially those that are separated or stand away from the tooth crown are more likely to contain pulp tissue.[22]

Early diagnosis and management of talon cusp is important because it may present a number of problems both to the patients and clinicians as follows: compromised esthetics; occlusal interference; displacement of the affected tooth; carious developmental grooves and pulpal necrosis; periodontal problems due to excessive occlusal forces; advanced attrition leading to pulpal exposure and periapical pathosis; irritation of the tongue during speech and mastication; interference with tongue space; and an unerupted taloned tooth may be mistaken radiographically for a supernumerary tooth, leading to unnecessary surgical intervention.[4,23-25]

Most cases of talon cusp need definitive treatment. The developmental grooves at the lateral aspects of the anomalous cusps are susceptible to caries. Deep, noncarious grooves and fissures should be cleaned of debris and plaque and prophylactically sealed with fissure sealant. If the grooves are carious the lesion should be eradicated and the cavity filled with glass-ionomer restorative material.[26] In case of premature contact and occlusal interference, the anomalous cusp should be reduced. If the treatment needs the removal of a substantial portion of the cusp, then reduction should be gradual and on consecutive visits at 6 to 8 week intervals, to allow deposition of reparative dentin for pulpal protection. Following each grinding procedure, the tooth surface

should be treated with a desensitizing agent, preferably fluoride varnish (Duraphat™).[27]

It is hoped that the large number of cases we reported here and elsewhere will increase the awareness of clinicians to the clinical significance of talon cusp and its association with other dental and somatic abnormalities. The classification of talon cusp proposed in the present study may provide a basis for future prevalence surveys. Guidelines for treating teeth with talon cusp were discussed.

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