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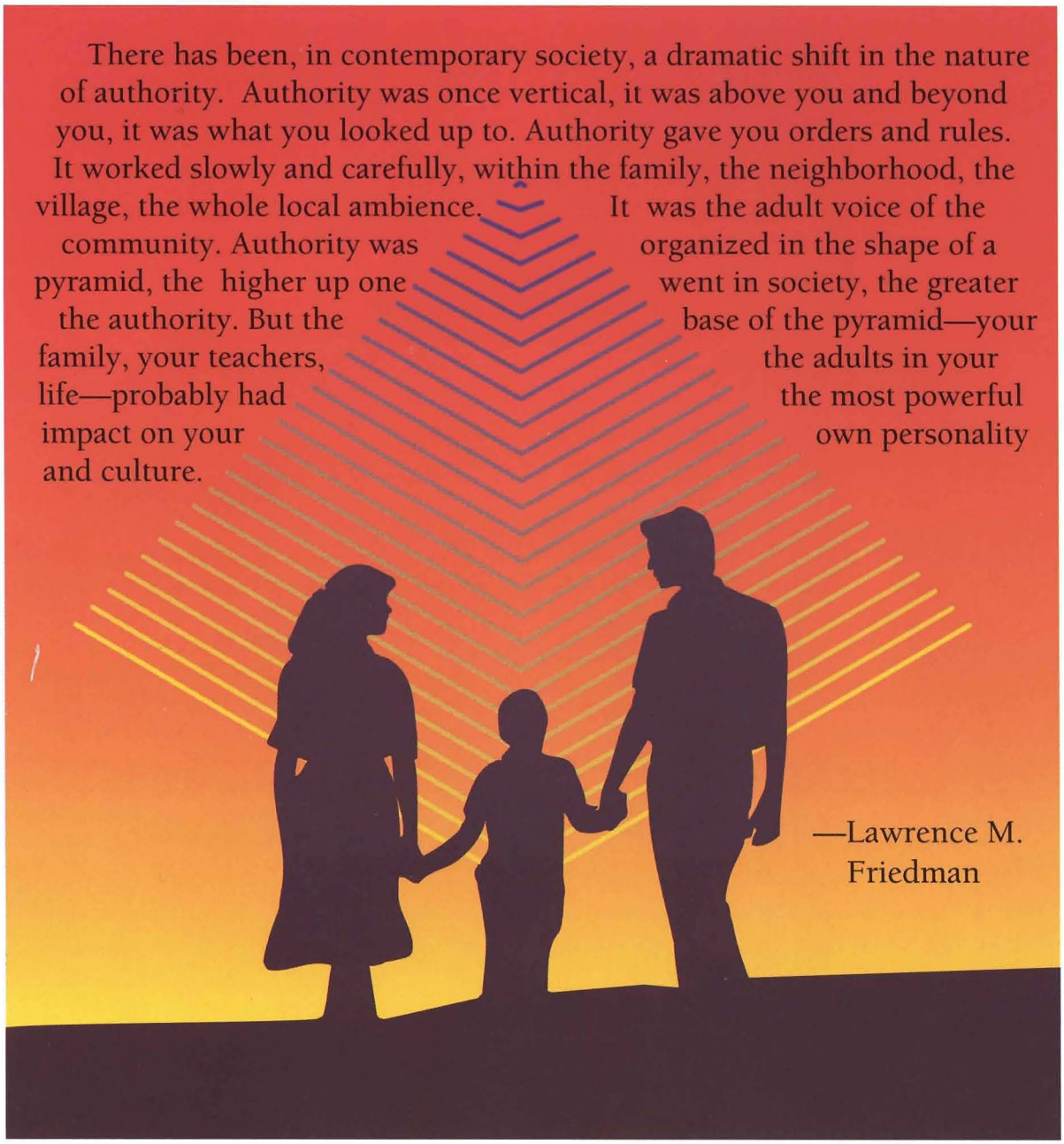
JOURNAL OF DENTISTRY FOR CHILDREN
SEPTEMBER—DECEMBER 1994



THERE CAN BE HOPE FOR A SOCIETY WHICH ACTS AS ONE BIG FAMILY, AND NOT AS MANY SEPARATE ONES.
—Anwar al-SADAT

Handicaps • STEREOTYPE • Sexism
Children in Crisis

ASDC
AMERICAN SOCIETY OF DENTISTRY FOR CHILDREN
JOURNAL OF DENTISTRY FOR CHILDREN
SEPTEMBER-DECEMBER 1994 VOLUME 61 NUMBER 6

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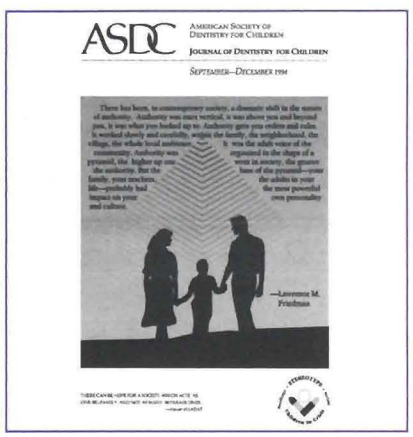

The change in the nature of authority in contemporary society should cause us all to reflect on its impact on our children.

Art and design by Sharlene Nowak-Stellmach

CLINIC

B B TOOTH DECAY

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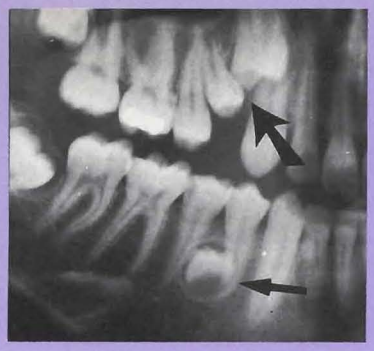

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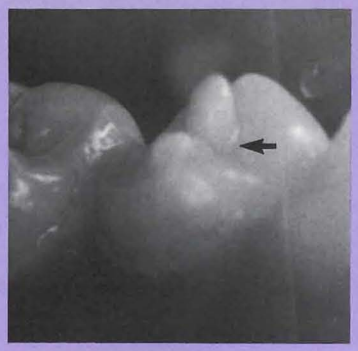

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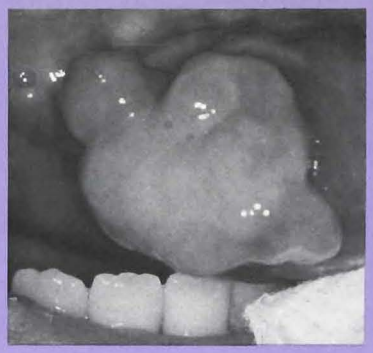

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For the busy reader

An esthetic technique for veneering anterior stainless steel crowns with composite resin—page 321

The authors describe an efficient, chairside, restorative technique that can be used to treat primary anterior teeth, with durable and esthetically pleasing results.

Requests for reprints should be sent to: Dr. Kenneth R. Wiedenfeld, 937 Tall Pine Road, Mt. Pleasant, SC 29464.

Space maintenance: Is it necessary with cuspal interlock?—page 327

The timely eruption of the succedaneous tooth should be assessed, to determine the need for space maintenance in the presence of cuspal interlock. This assessment may be achieved through the use of radiographs, which provide the essential diagnostic tool.

Requests for reprints should be sent to: Dr. Clemens A. Full, University of Iowa, College of Dentistry, Department of Pediatric Dentistry, Iowa City, IA 52241.

Dens evaginatus: Three clinical presentations of dens evaginatus in children—page 330

The purpose of this paper is to present various cases of dens evaginatus with respect to clinical appearance, diagnosis, treatment, and preventive therapy.

Requests for reprints should be sent to: Dr. Maria A. Sgro, 2221 Clifton Avenue, Montreal, Quebec, Canada H4A 2N5.

Hallerman-Streiff syndrome: Case report and recommendations for dental care—page 334

The case of a five-year, eleven-month old white male is discussed, and recommendations for treatment are made.

Requests for reprints should be sent to: Dr. William A. Mueller, Pediatric Dentistry, The Children's Hospital, 1056 E. 19th Avenue, Denver, CO 80218.

Results of a promising open trial to prevent baby bottle tooth decay: A fluoride varnish study—page 338

The disease, usually labeled Baby Bottle Tooth Decay (BBTD), is of epidemic proportions in the third world and in our disadvantaged child population.

Requests for reprints should be sent to: Dr. Philip Weinstein, Professor and Director of Behavioral Research, University of Washington, School of Dentistry, Department of Dental Public Health Sciences, Suite B509, SM-35, Seattle, WA 98195-9950.

White spots caries in Mexican-American toddlers and parental preference for various strategies—page 342

A significant number of white spot lesions, seen in infants and very young children, are regions of decalcification, which, when left untreated, will eventually become carious.

Requests for reprints should be sent to: Dr. Peter Domoto, Associate Professor and Chair, Department of Pediatric Dentistry, School of Dentistry, University of Washington, Seattle, WA 98195-9950.

Teaching parents at WIC clinics to examine their high caries-risk babies—page 347

Feeding and childrearing practices have been identified as the most important etiological factor. While bottle and even breast feeding practices have been shown to be related to BTD, recent work suggests snacking as an important risk factor, especially for Native American children.

Requests for reprints should be sent to: Dr. Peter K. Domoto, University of Washington, School of Dentistry, Department of Pediatric Dentistry, Health Sciences Center, S242, SB-26, Seattle, WA 98195.

Snacking and oral health habits of Washington state WIC children and their caregivers—page 350

Frequently low-income children do not benefit from proven caries preventive measures: systemic and topical fluorides, dental sealants, oral hygiene measures, and dietary modification.

Requests for reprints should be sent to: Ms. Mary P. Faine, Assistant Professor and Director of Nutrition Education, University of Washington, School of Dentistry, Department of Prosthodontics, Health Sciences Center, Room D683, SM-52, Seattle, WA 98195-9950.

Falling between the cracks: Oral health survey of school children ages five to thirteen having limited access to dental services—page 356

There are certain individuals in all segments of society who are caries prone.

Requests for reprints should be sent to: Salwa P. Rizk, 7334 Runningbrook Way, Indianapolis, IN 46254.

Pediatric dentistry and national health insurance: A more than favorable opportunity—page 361

The impact on dentistry will be determined by the extent to which dental care is included within the guaranteed benefits package and the future tax treatment of employer provided health benefits.

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

You are treating children in federal programs for child welfare, foster care, and adoption—page 365

Spending for children is projected to increase by about 75 percent to about $113 billion in 1995 with the largest component growth for the Medicaid program.

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

Computed tomography in the management of impacted teeth in children—page 370

Computed tomography (CT) has become more widely available and is recognized as an important diagnostic tool for complex conditions in the oral region.

Requests for reprints should be sent to: Dr. Lipa Bodner, Department of Oral and Maxillofacial Surgery, Soroka Medical Center, P.O. Box 151, Beer-Sheva 84101, Israel.

Relationship between oral parafunctions and craniomandibular dysfunction in children and adolescents: A review—page 378

In all studies but one, bruxism was constantly correlated with signs and symptoms of craniomandibular dysfunction in children.

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

Supernumerary teeth: Report of three cases and review of the literature—page 382

This paper reviews the literature and reports three cases of bilateral supernumerary teeth impeding the eruption of permanent maxillary central incisors and emphasizes the importance of early diagnosis and management.

Requests for reprints should be sent to: Dr. Faiez N. Hattab, Department of Pediatric Dentistry, Faculty of Dentistry, Jordan University of Science and Technology, P.O. Box 3030, Irbid, Jordan.

Turner's syndrome: Review of the literature and report of a case—page 394

The most frequent oral findings in Turner's syndrome include high palatal vault and hypoplastic mandible.

Requests for reprints should be sent to: Dr. Melinda Madléna, School of Dentistry, University of Medicine, 4012 Debrecen, Nagyerdei krt. 98, Hungary.

Unerupted second primary molars: Report of two cases—page 397

In this paper two cases with an unerupted primary mandibular second molar will be presented. In one of them the second premolar was congenitally missing, in the other it was unerupted and positioned occlusally and lingually to the unerupted primary molar.

Requests for reprints should be sent to: Dr. Seppo H.K. Järvinen, Karjusaarenkatu 67, 15240 Lahti, Finland.

Bilateral pyogenic granuloma of the tongue in graft-versus-host disease: Report of case—page 401

Diagnosis is confirmed by biopsy and histopathological examination. Treatment is by surgical excision and removal of local irritants.

Requests for reprints should be sent to: Dr. Angela Wandera, University of Michigan, School of Dentistry, Ann Arbor, MI 48109-1078.

Orofacial manifestations in the Wiskott-Aldrich syndrome—page 404

The diagnosis is evident in the first year of life. In its severest form, the disease will cause death by six years of age.

Requests for reprints should be sent to: Professor C. Scully, Centre for the Study of Oral Disease, University Department of Oral Medicine, Surgery and Pathology, Bristol Dental Hospital and School, Lower Maudlin Street, Bristol BS1 2LY, U.K.

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CLINIC

An esthetic technique for veneering anterior stainless steel crowns with composite resin

Kenneth R. Wiedenfeld, DDS
Robert A. Draughn, DSc
Joel B. Welford, DMD

Primary anterior teeth that require extensive restorative therapy due to caries, trauma, or developmental defects can present a particularly challenging problem to the dentist. The ideal restorative technique would assure strength, durability, esthetics, and efficiency in placement. Many of the currently available regimens fail to fulfill one or more of the goals mentioned above.[1,2] Couple these concerns with the technical difficulties of operating on children with behavior management problems, and the dentist is often left with few reliable restorative options.

Restorative modalities currently in use to treat primary anterior teeth include bonding with composite resin as in strip crowns, polycarbonate crowns, conventional stainless steel crowns, open-faced stainless steel crowns, and commercially veneered stainless steel crowns. Each of these techniques presents technical, functional, or esthetic compromises that complicate their efficient and effective usage.[1-3]

Bonding with composite resin requires an environment free of salivary or blood contaminants. Even though the results, in a cooperative patient, can be esthetically pleasing, heavy functional loads coupled with inadequate retention often result in unpredictable longevity.[1] Polycarbonate crowns are associated with the common clinical problems of fracture, debonding, and dislodgement.[2,4,5] Conventional stainless steel crowns have excellent longevity and ease of placement when compared to bonding; they often result, however, in poor esthetics.[1-3,6] Open-faced stainless steel crowns result in some metal being exposed, which is an esthetic concern. In addition, facings may become dislodged, and the patient time required for fabrication is significantly greater than that of conventional stainless steel crowns.[1] Finally, commercially veneered stainless steel crowns are often difficult to fit, due to problems with trimming and crimping of the preveneered surfaces.[1] Advances in restorative materials and metal-bonding procedures have made possible, however, new restorative techniques that combine the advantages of stainless steel crowns with the cosmetics of composite restorative materials. This paper describes an efficient, chairside, restorative technique that can be used to treat primary anterior teeth, with durable and esthetically pleasing results.

Dr. Wiedenfeld is a Pediatric Dental Resident; Dr. Draughn is Chairman and Professor of Materials Science and Dr. Welford is Pediatric Dental Resident at the Medical University of South Carolina.

TECHNIQUE

Anterior teeth that could benefit from restoration with stainless steel crowns are selected for treatment. These teeth are prepared in a manner that allows the operator to select slightly under-contoured stainless steel crowns. This step allows for a more naturally contoured veneered crown, because the addition of the composite facing adds to the crown's overall dimensions. Following the selection of the crowns, trimming and crimping are completed as necessary. The crowns are then removed from the teeth, and each crown is clamped on the lingual surface with locking cotton pliers or hemostats, which are labeled with the corresponding tooth numbers. The following technique is used to veneer the crowns at chairside.

- ☐ The esthetic surfaces of the crowns are sandblasted with 50-micron aluminum oxide for two to four seconds, per the manufacturer's instructions, using a sandblaster (Microetcher®, Danville Engineering Inc., San Ramon, CA) (Figures 1,2).
- ☐ A composite resin cement (Panavia OP®, J. Morita USA Inc., Tustin, CA) is applied to the sandblasted surfaces in a thin layer using an appropriate instrument, such as a one-half Hollenback (Figure 3).
- ☐ A thin coat of opaque, light-cured pit-and-fissure sealant (Delton®, Johnson and Johnson, New Brunswick, NJ) is applied by rolling the Panavia bonded surfaces in a drop of sealant that has been placed on a mixing pad (Figure 4).
- ☐ The sealant is light cured for twenty seconds.
- ☐ An amount of composite sufficient to veneer the entire esthetic surface of the crown, approximately 1 mm thick, is applied to the facial surface with a one-half Hollenback or plastic instrument. The composite is tapped down, flattened, and wiped to cover the incisal, gingival, mesial, and distal bonded surfaces (Figure 5).
- ☐ The veneered composite is light cured for forty seconds.
- ☐ A composite finishing bur of choice is used to contour and smooth the veneered surfaces as necessary.
- ☐ The crowns are tried in and cemented in the customary manner.

Danville Engineering, Inc. recommends sandblasting of metal surfaces at an angle of approximately ninety degrees between the Microetcher nozzle and the metal surfaces. A ninety-degree angle gives the best surface-texture for bonding. After sandblasting, the crown surfaces to be veneered should have a dull frosty appearance. In addition, veneering of the esthetic surfaces of the crown should follow within thirty minutes of sandblasting. Bond strengths to sandblasted metals have been found to be affected adversely by a delay between the sandblasting and the bonding of the resin cement.[7]

Panavia cement bonds tenaciously to sandblasted non-precious metals.[8-11] The cement is mixed to a consistency similar to that of zinc phosphate cement. It is then spread thinly on a mixing pad that allows lengthy working times since Panavia cement will not set in an aerobic environment. Next, the cement is spread just thick enough to opaque the metal surfaces that are to be veneered (Figure 3). The Panavia cement sets quickly after the application of the sealant, which creates an anaerobic environment, and bonds to the Panavia. In fact, any non-filled light-cured resin will work in place of the sealant. The opaque sealant enhances, however, the metal opaquing process. Application of the sealant is best accomplished by rolling the bonded surfaces of the crown in a drop of sealant. If a brush is used, the Panavia cement will tend to be displaced, exposing metal. It is important to inspect the veneered surface after curing of the sealant. If an area of Panavia cement remains uncovered, reapplication of sealant in that area is necessary.

Figure 1. Fitted stainless steel crowns are sandblasted with aluminum oxide for two to four seconds.

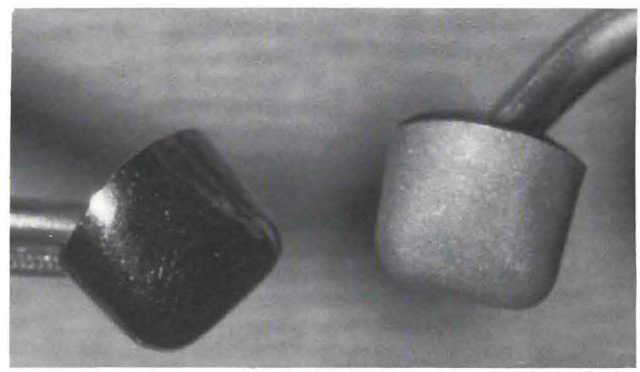


Figure 2. After sandblasting, the crown will lose its shiny finish and will appear frosty.

After the sealant is applied and cured, the surfaces to be veneered are covered with a layer of composite. A light-to-medium shade of a nonsticky hybrid composite (TPH®, Caulk/Dentsply Milford, DE) is applied in one layer over the facial area of the crown. It is technically easier to apply the composite in one application, rather than in layers. During the application and subsequent shaping of the composite, it is important to keep the instrument moistened with a nonfilled resin. This step helps to create a smooth contoured veneer. If deemed necessary, finishing can be accomplished with a twelve-fluted bur or any other desired polishing instrument.

The veneered stainless steel crowns should be tried in before final cementation. It may be necessary to adjust the interproximal widths of the veneered crowns to allow the crowns to seat properly. This is a greater concern in cases where interproximal spacing was inadequate before tooth preparation. Finally, the gingival contours and incisal heights of the crowns are assessed, and the crowns cemented in the customary fashion.

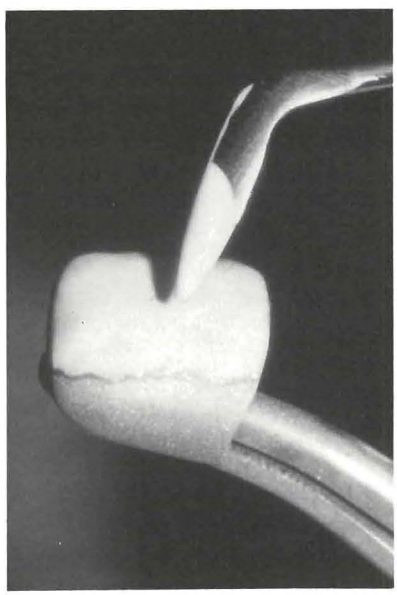


Figure 3. Panavia cement is spread thinly over the sandblasted surfaces that are to be veneered.

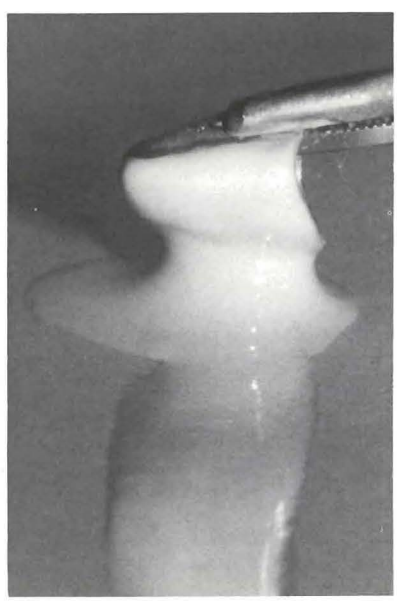


Figure 4. The esthetic crown surfaces that have been bonded with Panavia cement are rolled in a drop of light-cured opaque sealant.

DISCUSSION

The chairside veneering technique described results in a durable and esthetic crown. The actual veneering of each crown takes approximately three to five minutes and can be taught easily to an auxiliary. The armamentarium required consists of materials routinely found in dental offices.

The bond strengths created by this technique are very strong. A study on ten specimens was conducted at the Medical University of South Carolina, in which beads of composite resin were bonded to sandblasted stainless steel crowns in a manner identical to the described technique. The bond strengths of the beads to the crowns were measured by applying shear stresses at a rate of 1mm1min, using a mechanical testing system (MTS system 810, MTS Corp., St. Paul, MN) (Figure 6). The bonding failed at the Panavia cement and metal interface. The results included mean shear bond strengths of 3520 PSI (24.4 MPa) with a standard deviation of 290 PSI (2.0 MPa). Not only does this technique produce esthetic crowns, but also affords one the ability to adapt and trim the crowns accurately, before they are veneered.

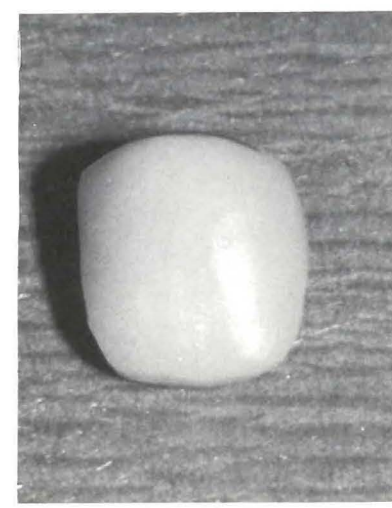


Figure 5. Composite is added to the bonded surfaces completing the veneering process.

The Microetcher easily adapts to most dental units and is available with a quick disconnect feature. A portable dust collector (Microcab®, Danville Engineering Inc., San Ramon, CA) can also be purchased, which evacuates and contains the aluminum oxide spray during sandblasting (Figure 1). These devices allow the convenient use of this technique in an operating room environment. In addition, it is possible to use this veneering technique intraorally on crowns that have fractured veneers or where one would like to add veneers to existing crowns.[9]

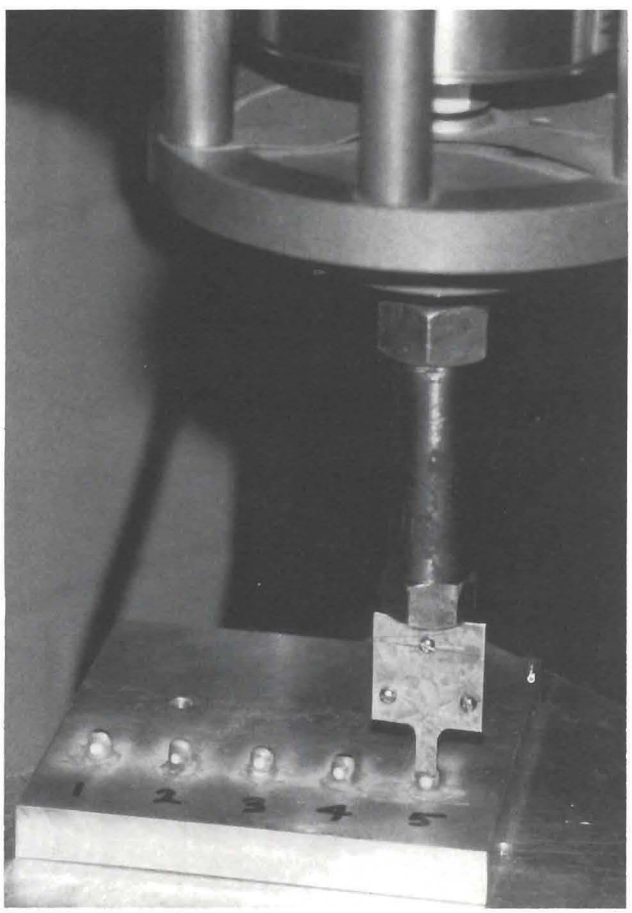


Figure 6. Secured stainless steel crowns were sandblasted, and composite beads were added following the described technique. Composite beads were sheared from the crowns using a mechanical testing system, which resulted in mean shear bond strengths of 3520 PSI (24.4 MPa).

CONCLUSION

The described technique of bonding composite to trimmed and fitted stainless steel crowns offers many advantages over other techniques currently used to restore primary anterior teeth. The patient time required is similar to that of conventional stainless steel crowns. Excellent esthetics and very high bond strengths (24.4 MPa), however, are achieved. In three to five minutes a fitted stainless steel crown can be transformed into a durable and esthetically pleasing bonded crown (Figures 7-12). This procedure will enable the dental team to deliver an esthetic result, similar to composite bonding with the strength and durability of stainless steel.

REFERENCES

1. Waggoner, W.F.: Restorative dentistry for the primary dentition. In *Pediatric Dentistry Infancy through Adolescence*, 2nd Ed., Pinkham, J.R. Editor. Philadelphia: W.B. Saunders Company, 1994, pp 318-324.
2. Braham, R.L.: Restorative procedures for primary anterior teeth with proximoincisal caries. In *Textbook of Pediatric Dentistry*, 2nd Ed. Braham, R.L. and Morris, M.E. EDS. Baltimore: Williams and Wilkins, 1985, pp 549-551.
3. Helpin, M.L.: The open-face steel crown restoration in children. J Dent Child, 50:34-38, January-February 1983.
4. Nitkin, D.A.; Rosenberg, H.M.; Yaari, A.M.: An improved technique for the retention of polycarbonate crowns. J Dent Child, 44: 108-110, March-April 1977.
5. Stewart, R.E.; Luke, L.S.; Pike, A.R.: Preformed polycarbonate crowns for the restoration of anterior teeth. J Am Dent Assoc, 88: 103-107, January 1974.
6. McDonald, R.E. and Avery, D.R.: Restorative dentistry. In *Dentistry for the Child and Adolescent*, 5th Ed. McDonald, R.E. and Avery, D.R., Editors. St. Louis: C.V. Mosby Co, 1987, pp 414-419.
7. Aboush, Y.E.: Cast metal resin-bonded dental restorations: Effect

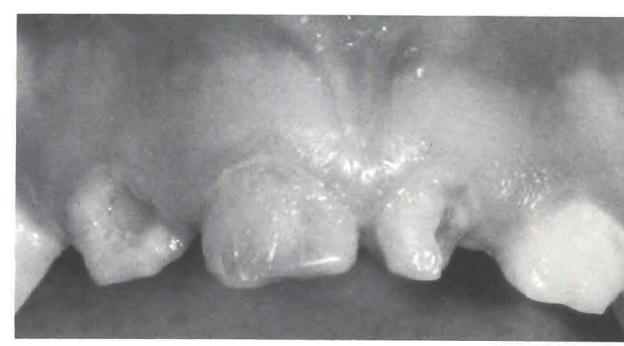
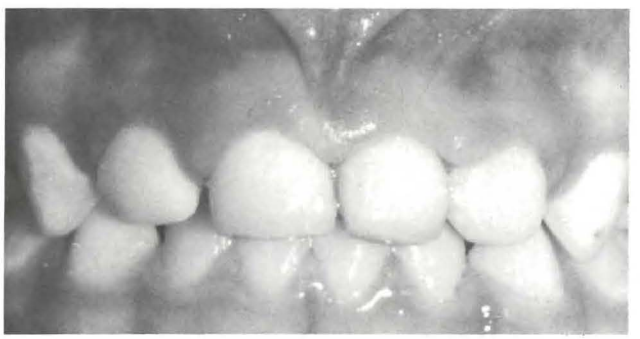
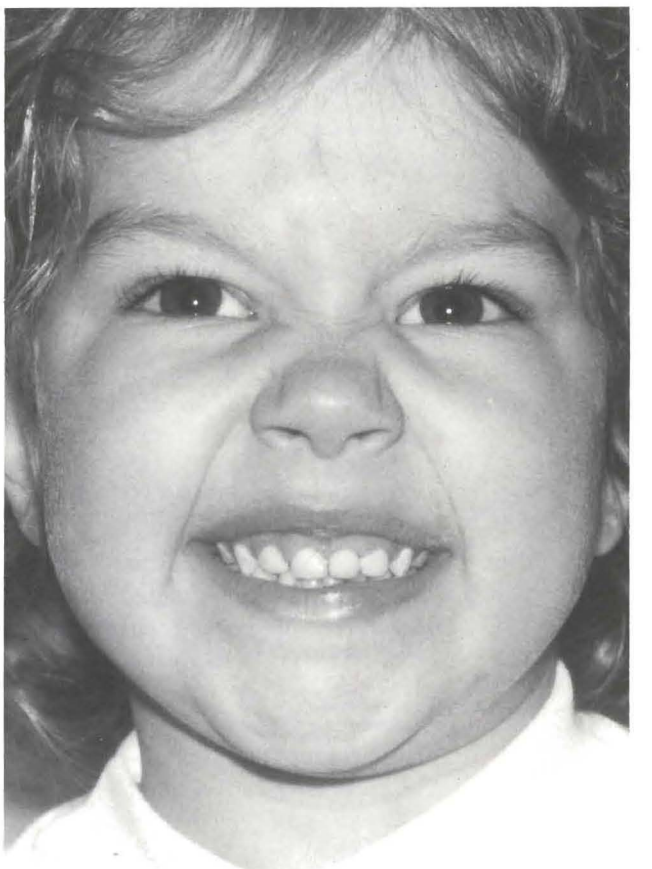
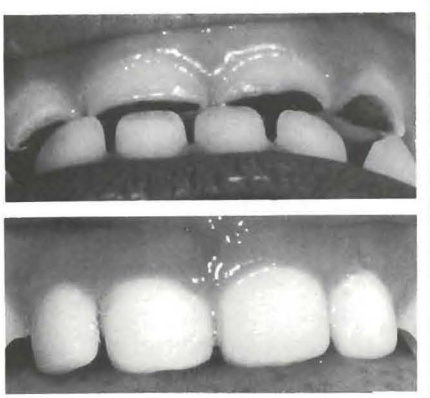


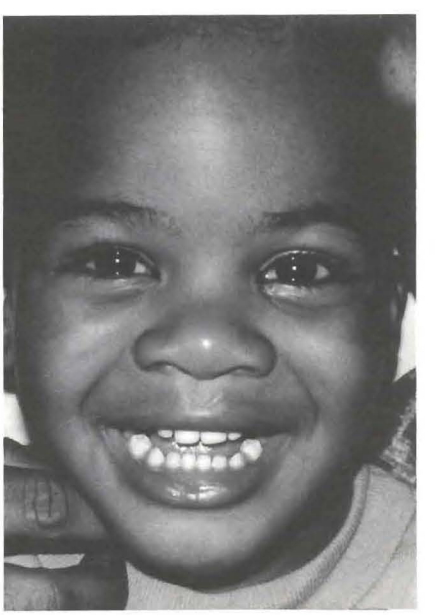
Figure 7. Three-year-old exhibiting defective strip crowns that were placed three months earlier on primary maxillary central and lateral incisors.

Figures 8,9. Defective strip crowns replaced with veneered stainless steel crowns. Note that the mesiodistal width of number F is less than that of number E. A smaller crown was fitted for number F due to severe breakdown of the tooth. The final esthetic results were very satisfactory to the parents.









Figures 10,11,12. Two-year-old with advanced nursing-bottle caries and an edge-to-edge incisor occlusion. Primary maxillary central and lateral incisors restored with veneered stainless steel crowns.

on the resin-to-metal bond of storage conditions before cementation. J Prosthet Dentistry, 67: 293-295, March 1992.
8. Bahannan, S. and Lacefield, W.R.: An evaluation of three methods of bonding resin composite to stainless steel. Intl J Prosthodont, 6:502-505, September-October 1993.
9. Bertolotti, R.L. and Paganetti, C.: Adhesion monomers utilized for fixed partial denture (porcelain/metal) repair. Quintessence Int, 21:579-582, July 1990.
10. Kohli, S.; Levine, W.A.; Grisius, R.J. *et al*: The effect of three different surface treatments on the tensile strength of the resin bond to nickel-chromium-beryllium alloy. J Prosthet Dent, 63:4-8, January 1990.
11. Diaz-Arnold, A.M.; Williams, V.D.; Aquilino, S.A.: Tensile strengths of three luting agents for adhesion fixed partial dentures. Int J Prosthodont, 2:115-122, March-April 1989.

FLUORIDE CHARACTERISTICS OF GLASS IONOMER CEMENTS

Walls has commented that the release of fluoride ions from set glass polyalkenoates is to be expected but the eluted ions seem not to be of structural importance to the set material. The different concentrations of fluoride released by the materials may be linked, however, to the structure of the glass powder used in the construction of the glass ionomer. A glass with a high concentration of fluoride would be expected to release more fluoride than one with a lower concentration.

The present study examined the fluoride release into deionised water. El Mallakh and Sarkar have shown that the release into artificial saliva is less compared with deionised water but, in that study, no account was taken of the presence of plaque or pellicle which may affect diffusion and also concentrate fluoride levels. In addition, the surface area of material exposed will also affect the rate at which fluoride is released.

The results of the second part of this study have shown that all the materials tested were able to take up fluoride and subsequently release it. This uptake increased the ensuing release of fluoride by as much as 43 times, compared with the control, as in the 20 day Chemfil Superior specimens. This dynamic process of uptake and release was maintained throughout the experimental period. The fluoride uptake has been shown to be cumulative and there was a trend towards a progressive increase in fluoride levels with time in the experimental groups in the present study. The clinical implications of this mechanism may be significant. The glass ionomer restoration or liner acts as a continuous sump for fluoride, which is released from the material over a long period. The continued presence of small amounts of fluoride in the aqueous phase around tooth mineral will reduce the effect of local undersaturation conditions during a drop in plaque pH. Replenishment of the fluoride within the material will, therefore, enhance the anticariogenic activity of the glass ionomer.

The clinical significance of the released fluoride is yet to be fully confirmed. Many factors, such as the site into which the fluoride diffuses and the rate of dilution, will influence its anticaries effectiveness. The property of glass ionomers, however, to take up and release extrinsic fluoride would indicate that this mechanism may be able to take place in vivo.

Creanor, S.L. *et al*: Fluoride uptake and release characteristics of glass ionomer cements. Caries Res, 28:322–328, September–October 1994.

Space maintenance: Is it necessary with cuspal interlock?

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Clemens A. Full, BS, DDS, MS

Today, there appears to be concern as to when to use a space maintainer following the early loss of the primary molars. The rise in cost of dental care has prompted the need to consider more accurately the use of unwarranted treatment without compromising dental care.

Space loss due to premature primary tooth loss has been known to cause space loss with the resultant malocclusion. The maintenance of such space may eliminate or reduce the need for prolonged orthodontic treatment. Thus to treat adequately the pediatric population, a clear understanding of tooth eruption patterns, occlusion, and space maintenance is required in addition to knowledge and training in other essential disciplines.

REVIEW OF LITERATURE

As a general rule, when a primary molar tooth is extracted or exfoliated prematurely, the teeth both mesial and distal to the space tend to drift or be forced into it. This movement is achieved mainly by the tipping and rotation of those adjacent to the site of the extraction and may result in the impaction of the succedaneous tooth, a shift of the midline of the dental arch to the affected side, overeruption of the opposing tooth and consequent impairment of function.[1]

Tooth drifting is dependent mainly on the following important factors:

- ☐ The dental age at the time of extraction;
- ☐ Space conditions;
- ☐ Eruption path and time;
- ☐ Intercuspation.[2]

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The drifting pattern (direction, magnitude, and speed of drifting) varies from individual to individual and from tooth type to tooth type.[2] This effect was observed on space and on sagittal occlusion in the primary canine-to-second-primary-molar region after extraction of the first primary molar before eruption of the first permanent molars. It was noted that the space loss in the maxilla is due, predominantly, to mesial drifting of second primary molars, and in the mandible is due, predominantly, to distal drifting of the primary canines. Mesial drifting of second mandibular primary molars and distal drifting of maxillary primary canines take place, but to a much lesser degree.[2]

It was noted further that the permanent molars in both jaws drift mesially; but at different rates. The movement of the maxillary first permanent molar consists, in part, of an uprighting of the path of eruption, and in part of a mesial drift. The movement of the maxillary first permanent molar is frequently combined with a rotation around the palatinal root, whereas tipping of the tooth is seldom seen. The second permanent maxillary molar has the same drifting pattern, but with a greater tendency for tipping. During drifting, the mandibular permanent molars will tip mesially; and in some instances move lingually and rotate. In general, the maxillary permanent molars move a much greater distance than the mandibular molars.[2]

Thus the need for space maintenance is essential in situations where the mesial or distal drifting of adjacent teeth into the extraction site may occur. The drifting effect of unilateral extraction of primary molars in both arches has been observed through a longitudinal study in which the factors listed above were considered.[3]

This study involved two groups with premature unilateral extractions in the maxillary and mandibular arches:

Group 1 Extraction of first primary molar (both arches).

Group 2 Extraction of the second primary molar (both arches).

The cases consisted of extractions in the maxilla and the mandible for skeletal comparisons with the nonextracted sides as the control. The results indicated that the space on the maxillary extraction side in Group 1 was significantly less than on the control side at the age of nine, but not at the age of thirteen. This was due to the fact that the space on the extraction side increased with age, in contrast to the control side, and at thirteen years it was significantly greater than at age nine years in the lateral segment. Other studies have reported this gain in space in the maxilla.[4]

In the mandible the space on the extraction side in the lateral segment and in the total segment was significantly less than on the control side at both nine years and eleven years.[3] In comparing ages, there was no significant difference on the extraction side, but the space decreased on the control side and at thirteen years it was significantly less than at nine years in both the lateral segment and the total.[3]

In Group 2 the differences between the extraction side and the control side were significant in both ages compared (ten years and twelve years).[3] The differences found between Group 1 and Group 2 showed that early extraction of the second primary molar leads to greater loss of space than early extraction of the first primary molar.[3] This indicates that the permanent molar has a greater tendency to drift into the immediately available mesial space. After the eruption of the first permanent molars the primary molars moved into the primary space with continuous migration of the first permanent molars, whenever a space is created anteriorly due to loss of contact.[5]

Thus the longitudinal study indicates that the difference between the space on the extraction side and the space on the control side was significantly less in the premature loss of the first primary molar than in the premature loss of the second primary molar.[3] The difference between the development of the maxilla and of the mandible is due to the fact that the mesiodistal width of the primary molars is greater in the mandible than in the maxilla.[3] The greater space leads to mesial migration of the first and second permanent molars when erupting.

The early extraction spaces in the maxilla close predominantly by the mesial movement of teeth posterior to the extraction space, while in the mandible closure occurs by a combination of the mesial movement of teeth posterior to the extraction space, and a to a lesser extent by the distal movement of those teeth anterior to the extraction space.[1]

Ninety-five percent of all spaces created by the premature extraction of the primary molars close appreciably within six months. It has been stated that there is no method of predicting reliably which spaces will remain stable and which will not, and that there is no method of predicting the eventual ratio of tooth-dimension to available-space relationship.[1] Thus one must consider seriously the provision of an adequate space maintainer.[1]

This inability to predict the ratio is rather contrary to the cast analysis developed by Moyer (1973) for determining the amount of space available for the alignment of the teeth and the amount of space required to align them properly.[6] Another method is the tooth size analysis, sometimes called Bolton analysis, which is accomplished by measuring the mesiodistal width of each permanent tooth.[6] In this analysis, a standard table is used to compare the summed width of the maxillary to the mandibular anterior teeth (excluding second and third molars).[6]

The best method of preventing space-loss is to delay the extraction until (1) the root of the permanent successor is at least half completed, (2) the resorption of the roots of the primary tooth has allowed the crown of the unemerged premolar to erupt as far as the bifurcation, and (3) the extraction of the primary tooth will remove the only obstacle in the path of eruption.[1] Thus in such situations, the need for space maintenance is not necessary.

It appears that there was failure to associate this timing of the extraction of the primary molars for reducing space loss with the fact that the first permanent molars by now may be in intercuspal lock. This may occur because the anticipated times of eruption of the premolars occur around ten to twelve years of age, by which time the first permanent molars are in occlusion and the second permanent molars are in the process of erupting.

In achieving normal occlusion in the typical human pattern, the late mesial shift of the lower permanent molar subsequent to shedding of the primary molars secures the proper intercuspation of the first molars, even after the premature loss of the primary molars.[5]

If the mandibular molars already intercuspate with the opposing teeth, their mesial shift may be minimal when the primary second molar is maintained in one jaw, while its antagonist is lost.[7,8] The occlusion of the first permanent molar must be taken into consideration in evaluating the available leeway space.[8] The leeway space is the space created by the difference in the sizes of the primary molars, which are significantly larger than the

premolars that replace them.[6] The cuspal interlock tends to produce less mesial shifting of the mandibular first permanent molars in utilizing the leeway space. If the primary molars are in end-on relationship, usually seen in the mixed dentition, the entire leeway space may be used during the mesial migration.[5]

DISCUSSION

The literature supports the use of space maintainers as essential in sustaining proper occlusion in accordance with lack of proximal contacts. Certain issues involving their use must be considered very thoroughly, however, without thinking stereotypically. The general thought is that cuspal interlock in which the permanent first molars are in Class 1, neutroclusion, tends to reduce mesial shifting. This shifting may be caused by loss of proximal contact as the result of caries, extraction, or physiological shedding of an anterior tooth. The mesial shifting may be minimal and space maintenance might not be necessary. This determination is dependent on a number of factors.[9] Some of these factors are relevant to determining, as in the case of cuspal interlock, the need for a space maintainer. The factors for consideration are:

- ☐ *The dental age of the patient.* It has been observed that if a primary molar was extracted, space maintenance is indicated, especially if the extraction occurred before age eight.[4] The average eruption dates must not influence decisions regarding the construction of a space maintainer, however, because of the wide variation in eruption times. A tooth erupts when its root has grown to three-quarters of its full length, regardless of the child's age.[10] The age at which the primary tooth was lost can influence the emergence time of the succedaneous tooth. For example, if the primary molar is lost at age four years, the emergence of the premolar could be delayed by as much as one year.[9]
- ☐ *Amount of bone covering the unerupted tooth.* Infection caused by the cariously involved primary tooth may result in loss of bone covering the unerupted tooth. In this situation the emergence of the permanent tooth may be accelerated. The most reliable determination is the amount of root development. The succedaneous tooth may erupt early and thus the space maintainer may be functional only for a short time. A guide for predicting emergence is that erupting premolars usually require four to five months to move through 1 mm of bone as measured on a bite-wing radiograph.[9]
- ☐ *Sequence of the eruption of teeth* The relationship of the developing and erupting teeth adjacent to the space created by the early loss of a tooth is also important. For example, if a second primary molar has been lost prematurely and the second permanent molar is ahead of the second premolar in its eruption, there is a possibility the second permanent molar will exert a strong force on the first permanent molar, causing it to drift. Similarly, in the case of the erupting permanent lateral incisor, the erupting force may cause the primary canine to intrude upon the extracted first primary molar space. The result here is a shift in the midline toward the area of loss of space.

CONCLUSION

Although some dentists may not advocate the use of space maintainers, when the first permanent molars are in occlusion, the literature indicates that cuspal lock only minimizes drifting and does not inhibit it completely. The use of the space maintainer should be considered, if there is concern about the complete use of the leeway space (i.e. in preservation of arch-length) during the drifting.[8]

The timely eruption of the succedaneous tooth should be assessed, to determine the need for space maintenance in the presence of cuspal interlock. This assessment may be achieved through the use of radiographs, which provide the essential diagnostic tool.

REFERENCES

1. Dearing, S.G.: Space loss and malocclusion. New Zealand Dent J, 77:62-67, April 1981.
2. Kisling, E. and Høffding, J.: Premature loss of primary teeth: Part III, Drifting patterns for different types of teeth after loss of adjoining teeth. J Dent Child, 46:34-38, January-February 1987.
3. Rönnerman, A. and Thilander, B.: A longitudinal study on the effect of unilateral extraction of primary molars. Scand J Dent Res, 85:362-372, July 1977.
4. Maclaughlin, J.A.; Fogels, H.R.; Shiere, E.R.: The influence of premature primary molar extraction on bicuspid eruption. J Dent Child, 34:399-405, September 1967.
5. Baume, L.J.: Physiological tooth migration and its significance for the development of occlusion. J Dent Res, 29:336, June 1950.
6. Proffitt, W. R.: *Contemporary orthodontic.* St. Louis: The C.V. Mosby Company, 1986.
7. Moorrees, C.F.A.; Grøn, A.; Lebret, L.M.L. *et al*: Growth studies of the dentition: A review. Am J Orthod, 55:608-609, June 1969.
8. Nance, H. N.: The limitations of orthodontic treatment. Am J Orthod, 33:208, April 1947.
9. McDonald, R.E. and Avery, D.A.: *Dentistry for the child and adolescent.* Managing space problems. St. Louis: The C.V. Mosby Co., pp 722-726, 1987.
10. Grøn, A.M.: Prediction of tooth emergence. J Dent Res,:41:573-585, May-June, 1962.

Dens evaginatus: Three clinical presentations of dens evaginatus in children

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Dens evaginatus is a developmental dental anomaly characterized by a projection of enamel and dentin that usually encloses pulp tissue. This defect is found predominantly in persons of Mongoloid origin, including Chinese, Japanese, Filipinos, American Indians, and Intuits.[1] It is usually present on the occlusal surfaces of premolars, mandibular more often than maxillary. Recent reports show its presence, however, on molars, canines and incisors.[2] This projection or supernumerary cusp may be present unilaterally or bilaterally, and in one or both of the dental arches. This slender cusp often extends above the occlusal plane and is thus readily fractured due to attrition or trauma, leading to pulpal exposure. Pulpal death and periapical infection in otherwise seemingly healthy teeth are common consequences. This scenario presents a challenge in diagnosis and treatment, especially when necrosis occurs before the completion of root formation.[3] In the case of an incomplete root, apexification must be achieved before endodontic therapy. This procedure often leads to a shorter root.

The purpose of this paper is to present various cases of dens evaginatus with respect to clinical appearance, diagnosis, treatment, and preventive therapy.

CASE 1

History

A twelve-year-old Chinese male presented to the emergency dental clinic complaining of a severe toothache in the lower right quadrant. The patient reported having spontaneous pain for the past two days and was able to localize the mandibular right second premolar as the tooth in question.

Clinical examination

A thorough intraoral examination revealed a virgin tooth showing no signs of decay. There was no evidence of swelling or trauma; the tooth was acutely sensitive, however, to percussion and mastication.

Radiographic examination

A periapical radiograph (Figure 1) revealed a normal, incompletely developed root with an open apex of the tooth in question. An additional bitewing radiograph failed to show any morbidity.

Treatment

Although the patient's symptoms clearly indicated that the tooth was undergoing pulpal death, the cause of this degeneration was not at first apparent. Since the patient's parent did not want to commit to endodontic therapy, the patient was treated symptomatically with antibiotics and analgesics. Treatment was sought from his private dentist, who also could not find the cause of the pulpal necrosis. The patient returned to the clinic after a specialist diagnosed a fractured supernumerary cusp located in the center of the occlusal surface on the mandibular premolar. At this time a periapical radiograph revealed a well localized periapical abscess. The tooth was subsequently opened, the necrotic pulpal tissue extirpated and calcium hydroxide treatment started in order to encourage apexogenesis. Endodontic therapy will follow once apexification has been achieved.

Special thanks to Drs. Marie Dagenais and Stephane Schwartz for their advice and guidance.

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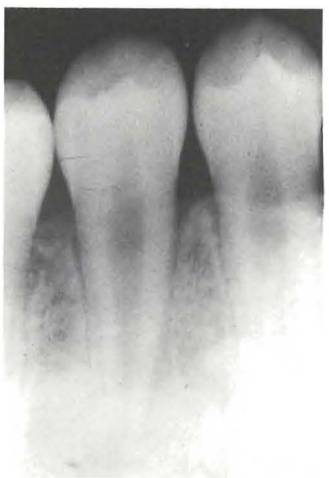


Figure 1. Radiographic presentation of right mandibular second premolar at the time of the initial emergency visit.

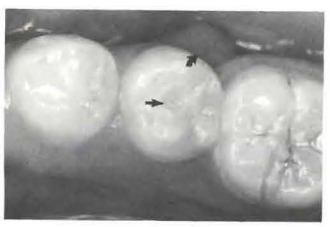


Figure 2. Occlusal view of left mandibular second premolar following the fracture of the supernumerary cusp.

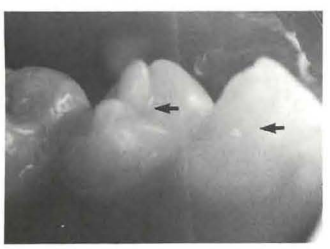


Figure 3. Clinical picture of first and second premolars with dens evaginatus.

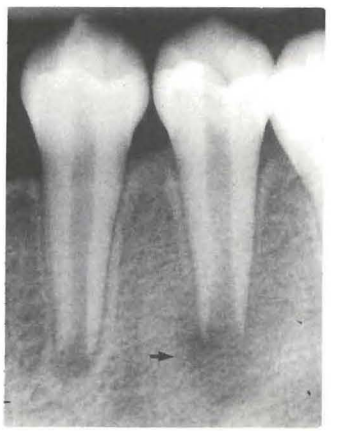
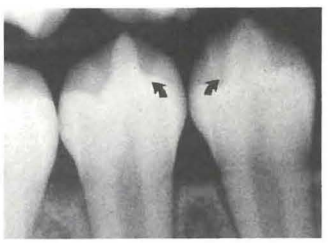
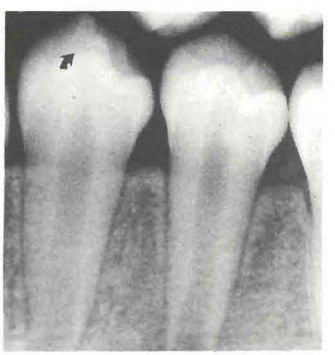


Figure 4. Periapical radiograph revealing a large periapical radiolucency associated with the premolar in question.





Figures 5 and 6. Radiographic presentation of the remaining premolars with dens evaginatus.

CASE 2

History

An eleven-year-old Chinese female presented to the emergency dental clinic with a left buccal space infection. Three months earlier, the patient had a similar infection and swelling, which was treated by her pediatrician with penicillin. The initial swelling resolved, but recurred three weeks afterwards, at which time her family dentist ruled out any dental origin. Penicillin was prescribed to alleviate the infection and the patient referred back to her pediatrician to find a possible cause for the swelling. No cause was found, and the swelling recurred several weeks later.

Clinical examination

Extraoral examination revealed a severe left buccal space infection along with a swollen parotid lymph node. The area was erythematous, tender to palpation, and its temperature elevated. Intraorally an abscess was present on the buccal gingiva adjacent to an apparently virgin caries-free left mandibular second premolar. After careful inspection, a small catch on the occlusal surface was detected with an explorer (Figure 2). The tooth was also slightly mobile. Examination of the remaining dentition revealed that all premolars in the maxilla and mandible possessed the dens evaginatus anomaly to varying de-

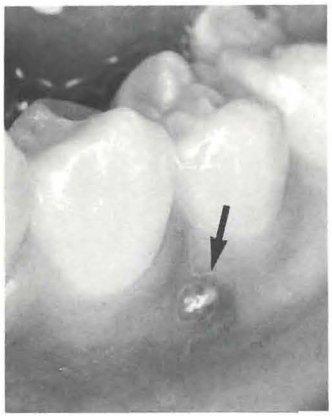


Figure 7. Clinical presentation of the fistula adjacent to the left mandibular premolar.

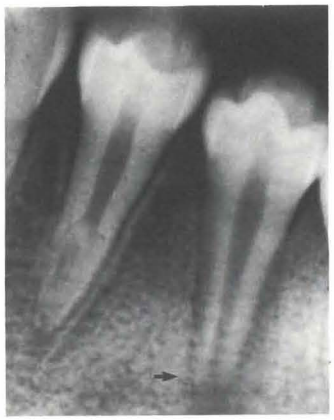


Figure 8. Radiographic view of the left mandibular premolar. Note the incompletely developed root.

grees of severity, with the mandibular premolars more severe than those of the maxilla (Figure 3).

Radiographic examination

A large periapical radiolucency around an incompletely formed premolar was present on the periapical radiograph (Figure 4). There was evidence of severe bone loss along the mesial root surface, and pulpal exposure in the area where the tubercle had fractured off. All remaining premolars demonstrated the typical appearance of dens evaginatus composed of enamel, dentin, and pulpal tissue within (Figures 5, 6).

Treatment

The second premolar was opened and drainage established through the coronal portion. The patient was placed on penicillin, and apexification with calcium hydroxide commenced as soon as the swelling had resolved. Endodontic therapy will follow after apexification is completed.

Preventive treatment

In an effort to avoid abscess formation before complete root closure, the following preventive measures were immediately initiated to diminish the chances of inadvertent fracture of the remaining tubercles.

- ☐ The tubercles were reinforced by bonding composite (Herculite) around their bases to the occlusal surfaces.
- ☐ All occlusal interferences opposing each projection were reduced, so that in all excursive movements there were no contacts with the tubercles.

CASE 3

History

A nine-year-old Chinese male presented for a routine dental checkup. Upon examination a fistula was found on the buccal aspect adjacent to the left mandibular first premolar (Figure 7). The tooth was asymptomatic with no history of pain. The patient did not know when the lesion first appeared, nor was he aware of its presence.

Radiographic examination

The periapical radiograph revealed a large radiolucency associated with the apex of the left mandibular first premolar whose root was not fully developed (Figure 8).

Treatment

Calcium hydroxide apexification was initiated. When the tooth was opened, the pulp was necrotic and the pulp chamber empty. The patient was placed on antibiotics for one week to reduce the swelling. Upon root closure, routine endodontic therapy will be initiated.

DISCUSSION

The above cases demonstrate how dens evaginatus can present a unique problem to the practicing dentist. Due to the possibility of early pulpal necrosis, endodontic therapy can be complicated further by an incomplete root with an open apex. Emphasis should be placed on early detection of this anomaly and on prophylactic treatment. Previous studies have advocated treatment protocols that include grinding of the cusp to encourage

secondary dentin formation, removal of the tubercle followed by direct or indirect pulp capping, and partial pulpotomies.[4,5] Results of these treatments have been unpredictable. With the advent of stronger composite materials a more conservative treatment option is available. By bonding composite to the base of the cusp and occlusal surface, support is provided to resist fracture of the extra cusp. In conjunction with the elimination of opposing occlusal interferences, the risk of fracture and attrition is reduced and root formation can proceed in a normal fashion. Once root development is complete, any fracture resulting in pulpal necrosis can be treated by conventional endodontic therapy. By following this treatment option, early pulpal death can be prevented, thus eliminating the need for apexification, which may compromise root formation.

REFERENCES

1. Goto, T. *et al*: Clinical and radiographic study of dens evaginatus. Dentomaxillofac Radiol, 8:78-83, 1979.
2. Loh, H.S.: A case of an evaginated odontome in a mandibular central incisor: a report and review. Sing Dent J, 26: 21-24, May 1981.
3. Tershakowec, G.A.: Dens evaginatus: a case report. ODJ, 59:20-22, June 1982.
4. Oehlers, F.A.C. *et al*: Dens evaginatus (Evaginated Odontome). Dent Pract Dent Rec,17:239-244, March 1967.
5. Yong, S.L.: Prophylactic treatment of dens evaginatus. J Dent Child, 41:289-292, July-August 1974.

TRENDS IN CARIES PREVALENCE IN UNITED STATES CHILDREN

There have now been four surveys of American schoolchildren which included oral health data from a nationally representative sample. The first two of these were held during the 1960s and the early 1970s, and the caries severity reported by them was much higher than in more recent years.

There have been no national surveys of oral health since 1986–87, and because of the regional variations in the United States the extent of the decline since then is hard to estimate. Surveys from a number of states have been reported since the last national survey, these range from examinations of state-wide representative samples of schoolchildren to more selective samples. Most are one-time surveys with no predecessors in the recent past and so do not provide a clear basis for comparisons, and the regional variations are still evident. Caries experience higher than the national average was recorded in Utah, where fluoride exposure is relatively limited, and in West Virginia, an Appalachian region where caries experience is traditionally high. Overall, however, there is nothing in these surveys which suggests that the caries decline in permanent teeth has been reversed.

Data from the Indian Health Service (IHS), the federal government agency responsible for provision of health services to Native American tribes, show that caries is still more severe among Indian children than among the general population.

Other identifiable populations exhibit a severity of caries which is clearly greater than that seen in the general population. Children in the national preschool programme called Head Start, who by definition are drawn from the lower socio-economic groups, consistently have caries scores well in excess of national averages. The same is true of the children of migrant agricultural workers, and for people who depend on public health agencies for their dental treatment.

Burt, B.: Trends in caries prevalence in North American children. Internat Dent J, 44:403–413, August 1994 (Suppl 1)

Hallerman-Streiff syndrome: Case report and recommendations for dental care

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First reported by Aubry in 1893, Hallerman-Streiff syndrome is a congenital anomaly due to a developmental disturbance of the first branchial arch during the fifth and sixth weeks of intrauterine life.[1-4] Hallerman (1948), Streiff (1950) and Francois (1958) fully described the syndrome that is characterized by dyscephaly, frontal and parietal bossing, open sutures and fontanelles, cataracts, microphthalmia, nystagmus, strabismus, hypotrichosis, skin atrophy, osteoporosis, scoliosis, hyperextensible joints and dental abnormalities.[1-6] Short stature, hypogenitalism, scant axillary and pubic hair, cardiac anomalies, and wormian bones can also be found.[1-6] The patients present with a small, peculiar bird facies, a long, thin nose, and mandibular and maxillary hypoplasia.[1]

The dental abnormalities are present in 80 percent of the cases.[1] The most common findings are malocclusion, open bite, crowding, severe caries, supernumerary and neonatal teeth, enamel hypoplasia, hypodontia, premature eruption of primary teeth, agenesis of permanent teeth and maxillary hypoplasia with poor development of the paranasal sinuses.[1,3,7,8] Other findings include microstomia, periodontal abnormalities, and high arched palate; the condyles may be anteriorly displaced or even absent.[1,9,10] Malerman and Album discussed the difficulties of the orthodontic and restorative treatments.[3] Surgical correction is usually prone to relapse.[10] Ohishi *et al* described a patient with six natal teeth whose extraction seemed to aggravate the maxillomandibular hypoplasia.[7] Honda *et al* observed that the anterior displacement of the temporomandibular joint is a useful diagnostic criterion for this syndrome.[11] The same authors also pointed out that the close proximity of the root apices of molars to the lower border of the mandible reported by Hutchinson is not specific to the condition. Crowding and rotation of the tooth germs within the alveolar bone were described by Slootweg and Huber.[8] They found the enamel to be normal and the periodontal ligament to be absent.

There is no sex predilection and cytogenetic studies are usually normal.[1,4,6] To date the most likely hypothesis is a single mutant gene.[2] Jones pointed out that there are insufficient data regarding the follow-up of patients to analyze their growth defect and mortality.[2] Gestation and birth weight are usually normal.[1,2,4] Affected children may have feeding and respiratory problems, however, because of their unusual upper airway anatomy. Death may occur due to respiratory infections.[1,2] Sclaroff and Eppley presented a good summary of the differential diagnosis from other anomalies with craniofacial features.[10]

According to Gorlin *et al*, all the cases have been sporadic (there are about 150 recorded cases worldwide).[1] Although dental abnormalities are frequently seen in this anomaly, very few cases have been described in the dental literature. The following is a case report of a white male who presented to the dental clinic of The Children's Hospital in Denver, CO, for an examination. The findings and recommendations for treatment are discussed.

CASE REPORT

A five-year-eleven-month-old white male with Hallerman-Streiff syndrome presented for an initial examina-

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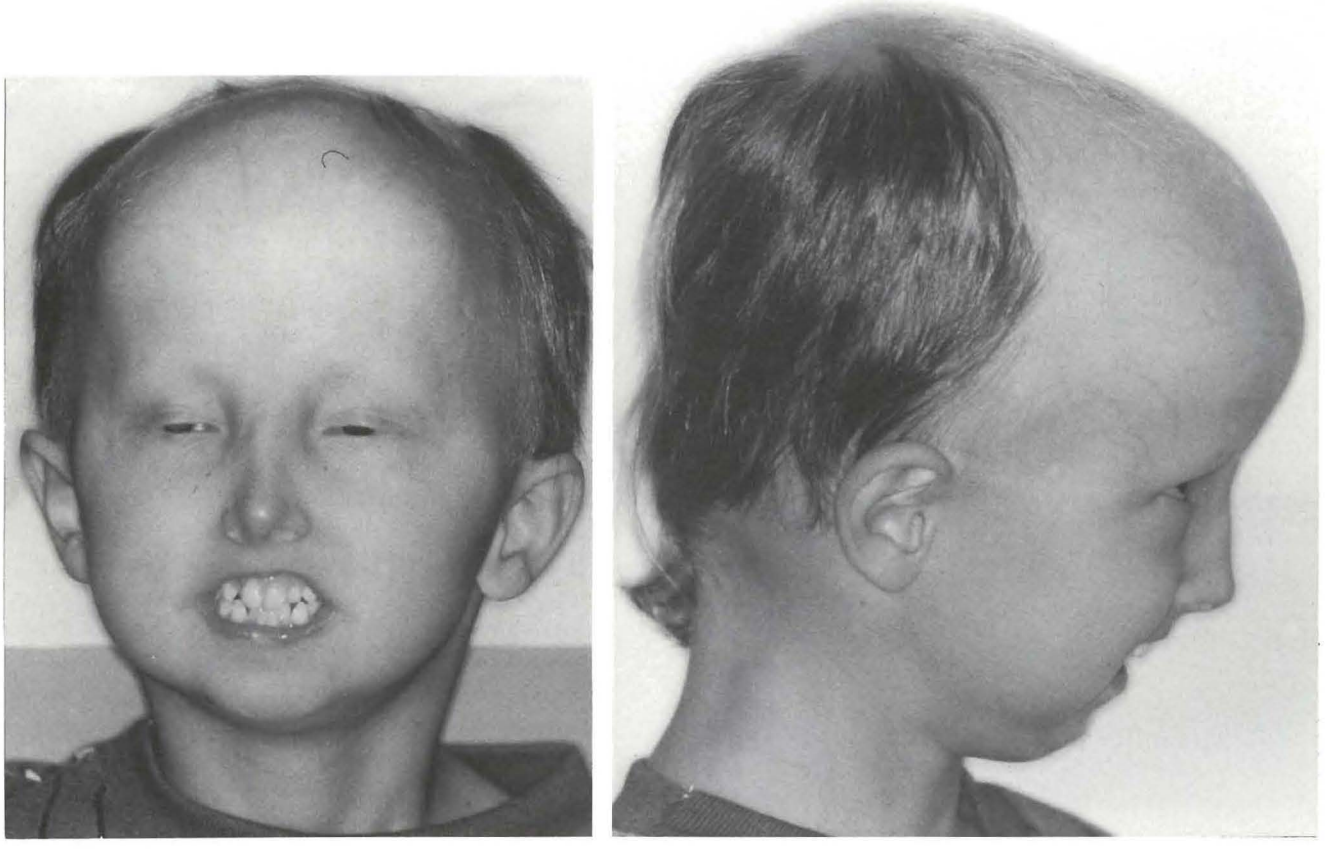

Figures 1 and 2. Characteristic facies of Hallerman-Streiff syndrome (frontal and lateral views).

tion in the dental clinic at The Children's Hospital in Denver, CO. The pregnancy was normal and the delivery was uneventful. The patient was taking no medications, had no known drug allergies and no systemic problems. He had a narrow nasal passage and was somewhat developmentally delayed. There were no complaints regarding his oral cavity.

Several ophthalmologic problems were present from birth. At seven weeks of age, he underwent a cataract removal and an anterior vitrectomy in the right eye. Six months later, the same procedures, plus a sphincterectomy, were performed in the left eye. A pupillary membrane and a secondary cataract were obstructing the right visual pathway and were excised. At age four years nine months, he underwent strabismus repair. The patient wore contact lenses due to aphakia. All the surgeries were performed under general endotracheal anesthesia, with inhalation induction and direct orotracheal intubation. Extubations were done with the patient completely awake and breathing on his own. The recoveries were unremarkable with a patent airway and no history of respiratory distress.

The patient presented the typical bird facies of the syndrome: a long thin, pointed nose with maxillary and mandibular hypoplasia. He was short for his age, brachycephalic with parietal and frontal bossing, microphthalmia, microstomia, low set ears and hypotrichosis of the eyebrows, lashes and scalp where the veins were prominent (Figures 1 and 2). The intraoral examination revealed good oral hygiene, healthy soft tissues, a normal-sized-tongue and a high-arch palate (Figure 3). The primary dentition was intact. Malocclusion was evident with crowding in both arches and the upper lateral incisors in crossbite (Figures 3 and 4). The panoramic radiograph showed agenesis of the permanent dentition except for the first and second molars (Figure 5). The temporomandibular joint was within normal limits.

The dental treatment consisted of prophylaxis, topical fluoride, and sealants in all primary molars. Oral hygiene instructions were given and demonstrated to the patient

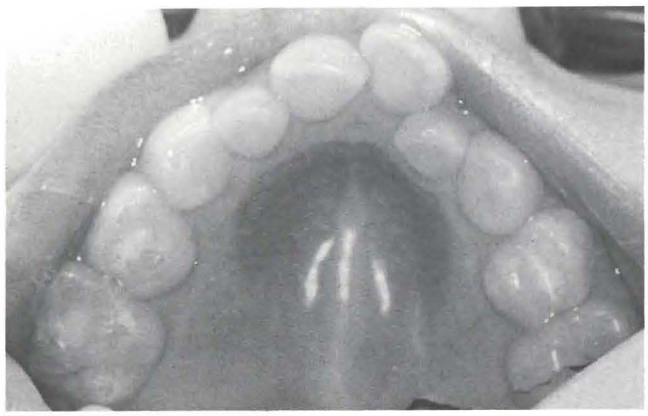

Figure 3. Note the high arched palate and crowding.

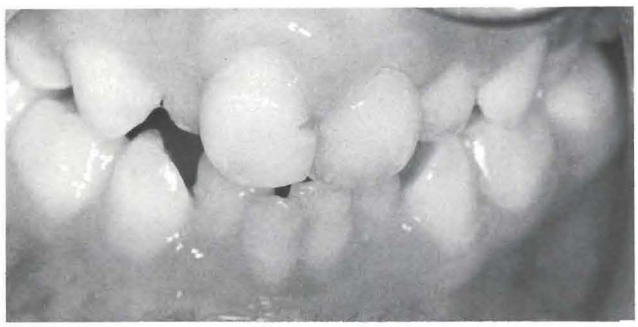

Figure 4. Frontal view of primary dentition.

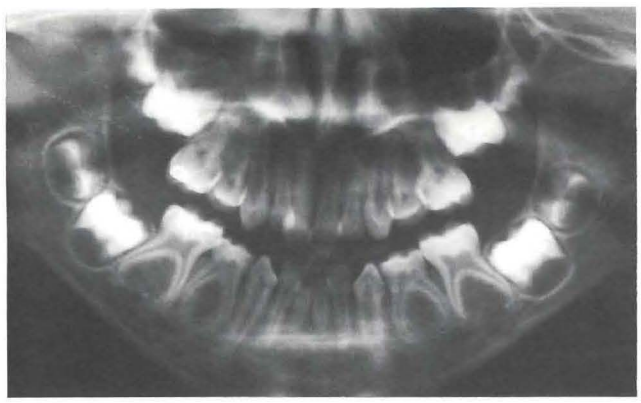

Figure 5. Panoramic radiograph revealed several congenitally missing teeth.

thetic appliances, and dental implants are viable options for the future. He will be observed regularly in our clinic.

DISCUSSION

Most cases of Hallerman-Streiff syndrome are reported in the ophthalmologic literature, due to the high frequency of eye disorders and congenital cataracts that may resorb spontaneously.[2,6] Despite surgery, blindness is usually the final outcome.[2] The visual disorders and the developmental delay interfere with the achievement of optimal oral hygiene. Furthermore, the patients may present enamel hypoplasia, premature eruption of primary teeth, agenesis of permanent teeth, malocclusion and periodontal problems, which contribute to extensive caries. It is imperative that children with Hallerman-Streiff syndrome be started, therefore, in a strong dental prevention program as early as possible with active participation by their caretakers. Because of the agenesis of permanent teeth, a healthy primary dentition is of paramount importance. Regular dental examinations are strongly recommended.

One of the alternatives for the management of severe caries in young children, especially those with mental or developmental delay, is to use general anesthesia. Patients with Hallerman-Streiff syndrome have serious upper airway deformities; intubation is very challenging, therefore, if not impossible.[6] The anterior displacement or absence of condyles, the anterior displacement of the trachea and microstomia with a normal sized tongue make direct visualization very difficult.[6] Nasal intubation is impaired by a high palatal arch, anterior glottis, hypoplastic nose, and deviated septum.[4,6] A fiberoptic laryngoscope is of great help. The patients' short and thick necks make an emergency tracheotomy unusually difficult.[6] A consultation with an otolaryngologist before surgery is advisable. The management of patients with Hallerman-Streiff syndrome using general anesthesia should be conducted, therefore, in centers where an otolaryngology service is available. Furthermore, the hyperextendibility of the joints, osteoporotic vertebrae, and occasional spinal deformities complicate the positioning of the patient on the surgical table. In some cases reported in the literature, the tracheotomy is done before the general anesthetic is administered or before extubation, which should not occur until the patient is completely awake and breathing spontaneously.[4,6] Fortunately the surgeries our patient underwent were uneventful. Oral conscious sedation is not advised because of the risk of respiratory depression, which can eventually lead to a complicated emergency situation. Care should be taken with the patient's positioning in the dental chair. Hyperextension of the neck should be

avoided because of the occasional vertebral osteoporosis and the danger of a collapsed airway.

Our patient did not present with neonatal teeth. Agenesis of most of the permanent teeth was observed in this case (Figure 5). Slootweg and Huber explained the agenesis by the premature disintegration of the dental lamina.[8] The same authors also pointed out that the early eruption of primary teeth, in an immature and partly calcified stage, contributed to the severe caries reported in affected children.

CONCLUSION

The enamel hypoplasia, premature eruption of the primary dentition, and malocclusion often encountered in the Hallerman-Streiff syndrome make the affected patients prone to severe caries. Moreover, the visual impairments and occasional developmental delay contribute to poor oral hygiene. Agenesis of the permanent dentition makes it imperative that the primary teeth be kept as long as possible. The use of general anesthesia and oral conscious sedation is not advised, because their complicated upper airway anatomy make intubation, extubation, and tracheotomy very difficult and unsafe. Hence it is of paramount importance to start a strong dental prevention program as early as possible, with the active participation of the caretakers. Orthodontic, prosthetic, and surgical consultations are of great assistance in formulating a long-term treatment for these patients.

REFERENCES

1. Gorlin, R.J.; Cohen Jr, M.M.; and Levin, L.S.: *Syndromes of the head and neck*, 3rd edition. New York: Oxford Press University, 1990, pp 306-309.
2. Jones K.L.: *Smith's recognizable patterns of human malformation*, 4th edition. Philadelphia: W. B. Saunders Company, 1988, pp 102-103.
3. Malerman, A.J. and Album, M.M.: Hallerman-Streiff syndrome: Report of case. J Dent Child, 53:287-292, July-August 1986.
4. Ravindran R. and Stoops, C.M.: Anesthetic management of a patient with Hallerman-Streiff syndrome. Anesth Analg, 58:254-255, May-June 1979.
5. Gay C.T.; Bondensteiner, J.B.; Barnes, P.D.: Extensive Wormian bones in a patient with Hallerman-Streiff syndrome. J Child Neurol, 5:50-51, January 1990.
6. Sataloff, R.T. and Roberts, B.R.: Airway management in Hallerman-Streiff syndrome. Am J Otolaryng, 5:64-67, January 1984.
7. Ohishi, M.; Murakami, E.; Haita, T. *et al*: Hallerman-Streiff syndrome and its oral implications. J Dent Child, 53:32-37, January-February 1986.
8. Slootweg, P.J. and Huber, J.: Dento-alveolar abnormalities in oculomandibulodyscephaly (Hallerman-Streiff syndrome). J Oral Pathol, 13:147-154, April 1984.
9. Patterson, G.T.; Braun, T.W.; Sotereanos, G.C.: Surgical correction of the dentofacial abnormality in Hallerman-Streiff syndrome. J Oral Maxillofac Surg, 40:380-384, June 1982.
10. Sclaroff, A. and Eppley, B.L.: Evaluation and surgical correction of the facial skeletal deformity in Hallerman-Streiff syndrome. Int J Oral Maxillofac Surg, 16:738-744, December 1987.
11. Honda, E.; Inone, T.; Domon, M. *et al*: Dental radiographic signs characteristic to Hallerman-Streiff syndrome. Oral Surg Oral Med Oral Pathol, 70:121-125, July 1990.

STAIRWAY-RELATED INJURIES IN CHILDREN

When young children fall down stairs, the injury is likely to be superficial and involve only one body region, most commonly the head and neck. Injuries to the extremities and trunk occur occasionally. Significant injuries including concussion, skull fracture, cerebral contusion, intracranial hemorrhage, and cervical spine fracture can occur, and were observed in 22 percent of the patients in this series. Small infants who fall with their caretaker while being carried on the stairs require an especially close evaluation for significant head injury.

Severe head injury is compatible with a stairway-related fall. However, injuries involving mutiple body regions, or severe truncal or extremity injuries should prompt a search for an alternate mechanism, including intentional trauma.

Chiaviello, C.T. *et al*: Stairway-related injuries in children. Pediatr, 94:679–681, November 1994.

B B TOOTH DECAY

Results of a promising open trial to prevent baby bottle tooth decay: A fluoride varnish study

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Babies as young as eleven months are at risk for a distinctive pattern of tooth decay that affects maxillary incisors. Many articles have identified the clinical appearance of the disease, which has a predictable progression. At first, *white spots* appear, usually on more than one of the at-risk teeth. These decalcification lesions may progress to frank caries, usually within six months to a year. The disease, usually labeled Baby Bottle Tooth Decay (BBTD), is of epidemic proportions in the third world and in our disadvantaged child population.[1]

The few studies of BBTD in Hispanic populations indicate the prevalence is high. Hardwick *et al* reported a 21 percent prevalence of BBTD for urban Hispanic children under age five.[2] Barnes *et al* reported a 16 percent prevalence for urban and a 37.7 percent for rural Hispanics.[3] In an initial study of migrant farmworker children, Weinstein *et al* found a 29.2 percent prevalence in babies an average of 27.6 months old.[4] Subsequent work of this research group found in a sample of children with an average age of 17.1 months that 7 percent had at least one maxillary incisor with decay, while over 30 percent had at least one incisor with a white spot lesion.[5] Comparison of data from both studies highlights the importance of decalcification lesions in teeth at risk and the rapid rate of progression of BBTD.

While the literature clearly recommends prevention of BBTD, most traditional stand-alone, health-educational approaches provide general information about BBTD, focus on the bottle as the risk factor, and recommend immediate substitution of the cup at all feedings by twelve months. It is clear that this approach does not work very well. Given the problems of this approach, there is a paucity of studies that do not completely rely on changing dietary risk factors. In ground-breaking work Köhler *et al* using chemotherapeutic counseling, and other interventions reduced mothers' levels of S. mutans, lowered incidence of S. mutans infection, and reduced the caries rate in children.[6-8] Recently, Dasanayake *et al* found the application of an iodine-NaF solution to mothers' dentition after being restored reduced S. mutans and other pathogens, but did not reduce caries in children.[9] There has been very little work attempting to use a chemotherapeutic approach directly with the very young child who is at risk. We learned in a personal communication with R. Berkowitz in 1993 that he has completed promising pilot work with I2-KI solutions. We are not aware of other studies.

There is a definite need for a simple and effective professional intervention of BBTD requiring minimal patient/caretaker cooperation. Preliminary data from our pilot study suggest that high risk Mexican-American mothers in Eastern Washington report acceptance and preference for safe and simple professional application of fluoride.[4] We chose varnish-containing fluoride, Dur-

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aflor, for our initial study. Duraflor, formally called Duraphat has been widely used in Europe and Canada for over twenty years.[10] There have not been any reported clinical trials, however, within the United States.

Duraflor is shown to be effective in preventing dental caries, safe, easy to apply and requires very little patient cooperation. Duraflor has been used on young children and has been shown to be effective in preventing caries in both permanent and primary dentitions. There are many clinical trials showing the effectiveness of Duraflor. The reported caries reduction ranges from 18 percent to 54 percent.[11,12] Hochstein *et al* reported 37 percent reduction in caries after two semiannual applications of Duraflor among three-year olds.[13] Holm also reported 44 percent reduction in defs after four semiannual applications.[14]

Duraflor has a wide margin of safety. It is safer than APF gels, which is the treatment of choice for caries prevention at most dental offices in the United States, because only a very small amount of the material is needed for each treatment. The fluoride concentration of Duraflor is 50mg NaF/ml. Clark *et al* reported that less than 0.5 ml of Duraflor was needed to treat six-to-seven-year olds.[15] They also reported very good patient acceptance of Duraflor. Seppä and Hanhijarvi reported that 0.3 ml varnish was sufficient for preschool children.[16] To treat the BBTD-susceptible four maxillary incisors, less than 0.1 ml (2.0 mg NaF) will be needed. Even if the entire 0.1 ml was accidentally ingested, the amount of fluoride is equivalent to a fluoride tablet for children. Roberts and Longhurst found that the ingestion of fluoride from Duraflor ranged from 5.0 to 5.2 mg for each patient, when 0.5 ml was used.[17]

Application of Duraflor is simple and takes very little time. The material is brushed on dried teeth and the procedure takes less than two minutes. We estimate the application time for four maxillary incisors to be about thirty seconds. For prevention of BBTD, the only compliance required from the parents is to bring the children for semiannual treatment. Semiannual application was found to be as effective as four applications per year.[18]

In all there has been very little work developing new interventions to prevent BBTD in disadvantaged children. This study attempts to build upon pilot research and begin to establish a serious effort to prevent disease in a population of Hispanic children.

If semiannual application of Duraflor is shown to be effective in preventing BBTD among Mexican-Americans, the study can be repeated for other Hispanic and non-Hispanic high-risk populations, such as Native Americans and Pacific Islanders, and many children of the world will benefit. Moreover, future studies may combine application of agents such as Duraflor with other interventions.

METHOD

The Yakima Valley region of Central Washington was selected as a study site, because of the level of organization of the local public dental health program and because the majority of the population, Mexican-Americans, were found in our previous work to have a high prevalence of BBTD. There is a close working relationship between the dental program and the Women Infant Child (WIC) program for promotion of children's dental health in the Yakima Valley.

One hundred thirty-three children between the ages of twelve and twenty-four months and their parents/caretakers participated in the study. The mean age of children participating in the study was seventeen months. Parents/caretakers were interviewed in the language of their choice to assess behavior risk factors. lmmediately afterwards each child and parent/caretaker was taken to a dental operatory, where the child was examined and the fluoride varnish applied. First, all four maxillary incisors were wiped with a 2x2 gauze to remove the plaque and dry the teeth. Second, all surfaces were systematically examined, using a mouth mirror. An explorer was not used. Decay and decalcification lesions were recorded and diagrams of the lesions were drawn on the examination card. Third, the fluoride varnish was painted with a brush on all the surfaces of four maxillary incisors and dried with an air syringe. Finally, the parent/caretaker and child were dismissed after an appointment was made for a six-month follow-up, to reapply the varnish. Parents/caretakers were also instructed not to feed the child for sixty minutes.

There was no attempt to eliminate exposure to other routine forms of caries prevention, i.e., the use of fluoride dentrifices and supplements. Fluoride exposure was estimated, however, by asking questions regarding the drinking of fluoridated water, and the use of fluoride supplements and toothpaste.

At the six-month recall, sixty-two children and their parents/caretakers returned; despite repeated mailing and telephoning, when possible, seventy-one of the 133 remaining children were lost to follow-up. At this visit, the clinical procedure described above was repeated by the same dentist, and the parent/caretaker completed a brief one-page questionnaire assessing caretaker behaviors relative to feeding and sleeping.

The disease rates of the dropouts were not signifi-

Table 1. ☐ Prevalence of decay/decalcification lesions at baseline and six months.

			Baseline			Six Months		
Group	N	Age in months	Decay	Decalcification	Decay or Decalcification	Decay	Decalcification	Decay or Decalcification
All subjects	132	17.1	7%	30%	36%			
Drop-outs	70	17.0	10%	26%	33%			
Follow-ups	62	17.3	3%	35%	39%	16%	21%	31%

cantly different from those who visited at six months. The only significant difference found between dropouts and those who completed the six-month follow-up was the size of the household. Households with one child had a 68 percent follow-up rate, whereas households with two or more children had a 41 percent follow-up rate (p=.011).

RESULTS

Prevalence

The prevalence of decay/decalcification for various subgroups at baseline and six months is presented in Table 1. Disease (decay/decalcification) rates ranged from 33 percent (for dropouts) to 39 percent (for follow-ups). The prevalence of decay/decalcification for follow-ups, six months later was 31 percent, with 16 percent for decay and 21 percent for decalcification. Changes in decay and decalcification rates were significant, using McNemar's test for paired proportions (p = .02 for both).

Table 2 presents an analysis of change of status for both sound and decalcified teeth. Of 130 sound teeth at baseline, 13 percent were decayed or decalcified in six months. Of seventy-three decalcified teeth at baseline, 51 percent were found to be sound in six months. Comparisons were made between central and lateral incisors. While there appears to be more decalcification for teeth E and F, the differences were not statistically significant. On the other hand, differences were found when comparing tooth surfaces. Table 3 shows that buccal surfaces have more decalcification than other surfaces.

Predictors

Analysis of interview and examination variables, using contingency tables, indicated ten variables related to baselines of disease for those who visited at six months. Logistic regression was used to examine the effects of these variables and to determine a predictive model. The results for the final model are presented in Table 4. The dental age of the child was entered first in the model. Dental age was determined by assessing eruption patterns (at least one maxillary anterior tooth was erupted: all four anterior teeth and one cusp on first molar was erupted; all canines and first molars were erupted). No effect of chronological age was observed after adjusting for dental age. Next, the presence of juice vs. other fluids in the bottle was entered. At the next step whether the child slept 0 to 8 hours or 9+ hours, and the education level of the mother were entered.

Questionnaire results collected at the six month follow-up were analyzed to determine whether any questionnaire variable (e.g. change in feeding practices) would be associated with change in disease status. Logistic regression analyses, adjusted for disease at baseline, found no variables to be significant.

DISCUSSION

Results of this uncontrolled study suggest that after six months, fluoride varnish may have had an effect in preventing the vast majority of sound teeth from beginning

Table 2. ☐ Change in tooth status for sound and decalcified teeth at six months.

		Status at six months		
Initial status	Number of teeth	% Sound	% Decalcified	% Decayed
Sound	130	88	5	8
Decalcified	73	51	37	12

Table 3. ☐ Prevalence of decalcification by tooth surface.

Tooth	M	D	B	L
D	7	5	23	5
E	5	2	30	6
F	4	3	29	8
G	5	3	16	6

Table 4. ☐ Logistic regression analysis of baseline decay or decalcification.

Variable	ρ	β	OR	95% CI
Dental age	.0008	1.42	4.05	1.79-9.18
Juice in bottle	.011	1.13	2.96	1.23-7.10
Hours sleep	.043	.88	2.55	1.08-6.00
Mom's education	.031	.88	2.55	1.12-5.81

the progression to caries, and may have contributed to the remineralization of over half the teeth that were decalcified at baseline. While no control group was included, previous work with this age-group in this population revealed a much higher caries rate (30 percent vs 16 percent).[4] We are now conducting a controlled clinical trial in the same population, to understand better the effect of the fluoride varnish.

Subject attrition in this study has been problematic. While we anticipated a forty-percent drop-out rate, the actual rate was fifty percent greater than expected. Despite repeated telephoning and mailed appointment reminders, the vast majority of drop-outs could not be reached. Attempts to enhance follow-up are needed in future studies with migratory workers, especially for families with more than one child. Parents with more than one child may have greater difficulty in managing the logistics necessary to appear at follow-up and/or, because of greater need, are more likely to migrate in order to follow the crops. Strategies such as gathering phone numbers and addresses of family and friends for an intensive effort at contacting potential drop-outs are needed. Such strategies are needed not only for research, but in implementing preventive health measures that require follow-up.

Our study of predictors indicates that risk factors such as dental age, juice in the bottle, etc., influence baseline measures of disease. Our abbreviated set of predictors at six months were not related to outcomes. This suggests that biologic risk factors, such as dental age and behavioral risk factors, such as feeding and sleeping practices, may be more important than changes in feeding practices that occur between one and a half and two years of age. Alternatively, future work must more carefully assess the changes in feeding and other parenting practices that may covary with the implementation of a chemotherapeutic or other intervention. Assessment of feeding practices should occur on an ongoing basis, perhaps monthly; retrospective report may not be reliable. Finally, while promising, these results indicate that additional experimentation with fluoride varnish is needed. Given the importance of dental age, perhaps fluoride varnish interventions should begin earlier and be repeated at least every four months. Such an approach would also probably reduce subject attrition. Moreover, should we attempt to alter feeding, tooth brushing or other parental practices at the same time? Clearly there is much work that must be accomplished before BBTD is effectively prevented in high risk populations.

REFERENCES

1. Ripa, L.W.: Baby bottle tooth decay (nursing caries: a comprehensive review). Pediatr Dent, 10:268-282, December 1988.
2. Hardwick, F.K.; McIlveen, L.M.; Forrester, D.J.: A comparison of nursing caries prevalence in Black and Hispanic children. Abstract, AAPD, 1991.
3. Barnes, G.P.; Parker, W.A.; Lyon, T.C. *et al*: Ethnicity, location, age, and fluoridation factors in Baby Bottle Tooth Decay and caries prevalence in headstart children. Publ Health Reports, 107:168-173, March-April 1992.
4. Weinstein, P.; Domoto, P.; Wohlers, K. *et al*: Mexican-American parents with children at risk for baby bottle tooth decay: pilot study at a migrant farmworkers clinic. J Dent Child, 59, 376-383, September-October 1992.
5. Domoto, P.; Weinstein, P.; Leroux, B. *et al*: White spot caries in Mexican-American toddlers and parental preference for various strategies. J Dent Child, 61:342-346, September-December 1994.
6. Köhler, B.; Andreen, I.; Jonsson, B. *et al*: Effect of caries preventive measures on Streptococcus mutans and lactobacilli in selected mothers. Scand J Dent Res, 90:102-108, April 1982.
7. Köhler, B.; Bratthall, D.; Krasse, B: Preventive measures in mothers influence in the establishment of the bacterium streptococcus mutans in their infants. Arch Oral Biol, 28(3):225-231, 1983.
8. Köhler, B.; Andreen, I.; Jonsson, B.: The effect of caries-preventive measures in mothers on dental caries and the oral presence of the bacteria streptococcus mutans and lactobacilli in their children. Arch Oral Biol, 29(11):879-883, 1984.
9. Dasanayake, A.P.; Caufield, P.W.; Cutter, G.R. *et al*: Transmission of mutans streptococci to infants following short term application of an iodine-NaF solution to mothers' dentition. Community Dent Oral Epidemiol, 21:136-142, June 1993.
10. DeBruyn, H. and Arends, J.: Fluoride varnishes: A review. J Biol Buccale, 15:71-82, June 1987.
11. Hetzer, V.G. and Irmisch, B.: Karies Protection durch Fluorlack (Duraphat)— Klinische Ergebnisse and Erfahrungen. Dtsch Stomatol, 23:917-922, December 1973.
12. Wegner, H.: The clinical effect of application of fluoride varnish. Caries Res, 10:318-320, July-August 1976.
13. Hochstein, H.J. *et al*: Experience with the fluoride lacker Duraphat. ZWR, 84:26-30, January 10, 1975.
14. Holm, A.: Effect of a fluoride varnish (Duraphat) on pre-school children. Community Dent Oral Epidemiol, 7:241-245, October 1979.
15. Clark, D.C.; Stamm, J.W.; Robert, G. *et al*: Results of a 32-month fluoride varnish study in Sherbrooke and LAC-Megantic, Canada. JADA, 111:949-953, December 1985.
16. Seppä, L. and Hanhijarvi, H.: Fluoride concentrations in whole and parotid saliva after application of fluoride varnishes. Caries Res, 17:476-480, September-October 1983.
17. Roberts, J.F. and Longhurst, P.: A clinical estimation of the fluoride used during application of a fluoride varnish. Br Dent J, 162: 463-466, January 20, 1987.
18. Seppä, L. and Tolonen, T.: Caries preventive effect of fluoride varnish applications performed two or four times a year. Scand J Dent Res, 98:102-105, April 1990.

White spots caries in Mexican-American toddlers and parental preference for various strategies

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Baby bottle tooth decay (BBTD), a disease of early childhood, is characterized by a distinctive pattern of tooth decay in the primary dentition. Many articles have identified the clinical appearance and etiology of the disease. At present the disease is of epidemic proportions in the third world and within the disadvantaged child population of the United States.[1]

A problem with most studies of BBTD is that the subjects were children attending Head Start, between the ages of three and five years. This introduces retrospective and recall bias for studying the problem, which manifests itself as early as eleven months of age. Very few studies have been accomplished with children under thirty-six months of age. Hennon *et al* found 8.3 percent of those in the eighteen to twenty-three month-old group had caries, the percentage rising to over 57 percent in children thirty-six to thirty-nine months old.[2] The presence of white spot lesions was not reported in this study. More recently, Graves *et al* found that white spot lesions were associated consistently with caries prevalence.[3] This may indicate that a significant number of white spot lesions, seen in infants and very young children, are regions of decalcification, which, when left untreated, will eventually progress into carious lesions. We believe that the importance of these potentially reversible decalcified lesions has not been systematically recognized in this population. It may be reasonable to hypothesize that this is due to the profession's limited access to children under thirty-six months of age and an intent to document only serious (treatable) disease.

Prevention of BBTD has focused on health education; specifically, parents/caretakers are urged to discontinue bottle feeding at about twelve months, a critical time for the primary prevention of BBTD and what appears to be a naturally occurring time for weaning.[4-6] Unfortunately, few programs have been carefully evaluated, and when they are, results have not been encouraging.[5,7] Most programs have focused on providing all parents/caretakers with the same information.

It may not be effective to insist on weaning at one year, a process that may fly in the face of subcultural or cultural norms and/or may prove to be very difficult for the single parent/caretaker, especially with a difficult (defined by temperament or illness) child.[8] Moreover, given the above difficulties of parents/caretakers in modifying their childrearing practices to lower the risk of BBTD, it is surprising that there is a paucity of professionally applied interventions.[9]

While the Hispanic population may be the largest minority group in the United States by the end of the decade, relatively little is known about their health status, especially of those residing outside the southwest. In a large scale study conducted in 1969-1970, Hispanic children in Texas had more missing teeth and higher DMFT than non-Hispanic white and black children; results from nine other states showed Hispanic children with lower mean DMFT.[10] In a 1978-1981 study of Hispanic children from the fluoridated cities of New York, El Paso,

and Hayward, DMFS scores from El Paso were less than half of those found in Hispanic children from the other cities.[11,12] Sampling children, ages five to seventeen, in five southwestern states, Ismail *et al*, using H Hanes data, found a low overall prevalence of dental caries, similar to that of other children in the region and attributed the finding to naturally fluoridated water supplies.[13] Mexican-American children from low income families had about double the disease experience, however, when compared with higher income families. Pollick *et al* estimated costs of treatment to be higher for Hispanic than for white or black children.[14] While infant data were available from a 1982-1983 H Hanes data set, noncavitated carious lesions were coded as "sound, requiring no treatment". Recently, Hardwick *et al* reported in an abstract that urban Hispanic children under age five had a significantly higher proportion of nursing caries (21 percent) when compared with urban black children (8 percent).[15] Studies of rural Hispanic children are scarce. School-age children of Hispanic migrant farmworkers are reported to have high rates of diseases; recent data indicate twice the decay rates of general populations.[16,17]

Health attitudes and behaviors of Hispanic Americans have been reported to be different from those of non-Hispanics.[18,19] Use of dental services was low compared to non-Hispanic whites and blacks, with symptomatic care when continued pain was experienced.[18] Differences between Hispanics and others were also found for frequency of visits and demands for services. These differences remained after controlling for income and education. It is important to note that there are meaningful distinctions between Hispanic cultures.[20] For example, Mexican-Americans are less likely to have a regular source of medical care than other Hispanic populations.[21]

In all, while much is known about the etiology of BBTD, most epidemiologic studies are retrospective and focus on carious lesions, usually ignoring potentially reversible incipient lesions. Moreover, even though health education interventions that recommend weaning at twelve months, have been ineffective, few alternative strategies have been attempted. We believe targeting preventive measures for children at risk and their parents/custodians may be promising. Such an approach would provide culturally appropriate choices for each family. This study, as part of a long-term commitment to developing effective primary and secondary preventive strategies for BBTD, follows up our pilot work, and attempts to focus attention on white spot lesion rates and on parental understanding and choices of preventive measures in a rural Mexican-American population.[9]

METHOD

Subjects

The Yakima Valley region of central Washington was selected as a study site because the majority of the population, Mexican-Americans, have a high prevalence of BBTD (about 30 percent).[22] There is a close working relationship between the local public dental program and the Women Infant and Children Nutrition (WIC) program for promotion of children's dental health in the Yakima Valley.

Subjects for this pilot study were parents and caretakers who enrolled a child less than three years of age in a WIC or Maternal Child Health (MCH) program for migrant families in the Yakima Valley of central Washington. These farmworkers are of Mexican descent; many of them are recent immigrants. One hundred thirty children, nine to thirty-four months of age, and their parents/caretakers participated; mean age was 17.1 months.

Table 1 ☐ Percentage of decay & white lesions for teeth at risk.

Tooth	Number erupted	Percent decayed	Percent decalcified
D	108	5.6	26.9
E	116	6.9	31.0
F	115	7.0	29.6
G	107	7.5	19.6

Table 2 ☐ Percentage of children with caries or white spot lesions on one, two, three, or four upper incisors (N=129).

Number of lesions	Number of children	Percentage
One	3	2
Two	10	8
Three	6	5
Four	27	20.9
None	83	64.3

Table 3 ☐ Log linear model analyses: maximum likelihood analysis of variance.

Variable	df	χ^2	p
Dental age: lesions	2	5.98	.0503
Chrono age: lesions	2	6.28	.0433
Cariostat: dental age	2	12.56	.0019
Cariostat: chrono age	2	2.75	.2524
Cariostat: lesions	1	.40	.5283

All subjects were interviewed at the Yakima Valley Farmworkers Clinic by one of two trained bilingual interviewers after risks and benefits of the study were explained, and they completed a consent form approved by the university institutional review board. Interviewers were trained in interview skills, using a training videotape made explicitly for this purpose.

The interview instrument attempted to assess demographic variables, child caretaking behaviors relevant to feeding and sleeping, knowledge and belief about BBTD, and willingness to comply with possible BBTD intervention strategies. Self-assessment of the dental health of child and caretaker, self-regulation questions, and items from the Parental Stress Index (Spanish version) were included.[23,24] The interview instrument was translated into Spanish and back-translated in English for accuracy. Interviews were given in the language of the subject's choice. The interview took about thirty minutes to administer. Immediately afterwards, each child was examined by a dentist.

Dental procedures were performed in a dental chair with dental light and took less than five minutes. First, all four maxillary incisors were wiped with 2x2 gauze to dry and remove any plaque present on the teeth. Second, all tooth surfaces were systematically examined, using a mouth mirror. No explorer was used. Findings were drawn on the diagrams of the examination card. The number of decayed and filled surfaces were also recorded. A cotton swab was used to collect plaque for a qualitative caries activity test, the Cariostat.[25,26] Considerable work has been done with this test, though most of the results have been published in Japanese.

RESULTS

Overall results showed that 7 percent of the children had at least one carious maxillary incisor and 30.4 percent had at least one incisor with a white spot lesion. Table 1 presents the percentage of decay and white lesions for each of the four teeth at risk, which ranged from 6 to 8 percent for decay and from 20 to 31 percent for white lesions.

The percentage of children with at least one maxillary incisor decayed or with a white lesion was 35.9 percent. Though there was a trend for older children to have lesions, no significant age differences were found for children with and without lesions ($F=-1.7$, $p=.08$). Table 2 presents the number of decay or decalcification lesions within children. The results indicate that among those who have decay or white lesions, it is likely that most of the teeth at risk are affected; twenty-seven of forty-six (58.6 percent) of these children had decay or white lesions on all four teeth; thirty-three of forty-six (72 percent) of these children had a lesion on three or four teeth.

Using log linear model analyses to establish whether or not measures were independent, chronological age and our classification of dental age (early, late, normal eruption) were found to be related significantly to the presence of dental disease. The Cariostat measure was related to dental age, but not to measures of dental disease. These results are presented in Table 3.

Results of the parental questionnaire focus on awareness of dental disease and preference for specific preventive interventions. When asked whether or not they believed their child had a tooth related problem no differences were found between parents of children with lesions and of those without lesions ($t=1.46$, $p=.15$). Sixty percent of parents of children with lesions indicated they were not aware of a dental problem.

Table 4 presents ratings of likeliness of parents of children with and without lesions to follow a range of recommendations to "help the baby keep his or her front teeth from becoming decayed and toothachy". No differences were manifested between groups. The results

Table 4 ☐ Ratings of unlikely/likely to follow specific recommendation (1=very unlikely, 5=very likely)

Recommendation	Overall percent very likely or likely	$\bar{X}$+SD decay or white spots	$\bar{X}$+SD no decay or white spots
A. Immediately substitute cup for bottle for all feedings.	46%	2.8(1.9)	3.0(1.9)
B. Substitute cup slowly, once a day for first week.	86%	4.4(1.3)	4.4(1.3)
C. Water down milk or juice slowly.	81%	4.5(1.2)	4.1(1.5)
D. Substitute artificial sweetener for sugar.	80%	4.1(1.5)	4.2(1.8)
E. Immediately cut out extra night feedings; even if baby cries.	58%	3.3(2.0)	3.7(1.7)
F. Slowly reduce amount in bottle at extra night feedings.	92%	4.6(1.0)	4.5(1.2)
G. Put fluoride drops in bottle once a day.	94%	4.8(1.1)	4.8(1.0)
H. Bring child to clinic to paint medicine on teeth.	98%	5.0(.8)	5.0(.7)

do indicate preferences, however, for different intervention strategies; while 46 percent indicated they were very likely or likely to substitute immediately a cup for bottle at all feedings, 86 percent preferred to substitute the cup slowly. Similarly, while 58 percent were very likely or likely to eliminate immediately extra night feedings, 92 percent of the mothers indicated they were likely or very likely to reduce slowly the amount in the bottle at extra night feedings. The highest ratings were for putting fluoride drops in the bottle once a day (94 percent) or bringing the child to the clinic to paint medicine on teeth (98 percent).

DISCUSSION

While our results with young children indicate a low caries rate, there is reason for concern. Comparison of cross-sectional data collected from children averaging seventeen months to an older sample averaging thirty-three months highlights the importance of white spot lesions in teeth at risk.[9] While the younger group had a low caries rate (7 percent) and a high rate of white spot lesions (30 percent), the older group had a 30 percent caries rate. Hennon *et al* presented similar findings.[2]

Moreover, our findings show that children with white spots lesions tend to have such lesions on three or four teeth. The probability that many of these lesions are not associated with the etiology of dental caries is low. Conversely, these lesions are a likely early sign of imminent disease; early symptoms of the disease are likely to be observed within twelve to eighteen months. Simply put, we believe these lesions are important and should not be ignored. Ignoring the presence of white spot lesions puts the preventively oriented researcher at a severe disadvantage. How can one demonstrate the efficacy of a primary or secondary preventive intervention, in children less than two years of age, without collecting baseline and follow-up data that include such lesions? The progression, arrest, or reversal of these lesions are the major measures of the efficacy of an effective intervention. Historically, dental research has had limited access to children in this age-group; creative solutions to this access problem are needed. We recommend working closely with other public and private nondental health care providers who regularly interact with very young children and their parents or caretakers. Relying on parents or caretakers to bring very young children to dental offices or clinics is not likely to alter the problem of access in a significant way.

The attempt to identify children who are at great risk for BBTD is an attractive strategy. While the Cariostat measure was not related to the concurrence of dental disease, the measure is intended to predict future disease. Subsequent data will better assess the viability of this technique. Interestingly, both chronological and dental age were related to the presence of dental disease, with dental age showing no distinct advantage over chronological age. This result may be because of the crudeness of the measure of dental age; additional work is needed to specify a better measure. Other studies show the prevalence of mutans streptococci increases with age, or as the number of erupted teeth increases.[27-32]

Results of the parental questionnaire suggest potential intervention strategies. The finding that 60 percent of the parents of children with lesions are not aware of a dental problem, a finding identical to that of Johnsen, points to a strategy to enhance parental awareness of existing lesions.[5] The traditional health education approach provides general information about BBTD. We believe that teaching nondental health providers, such as nurses and WIC workers, to examine the young child for BBTD lesions and also to instruct the parent or caretaker to look into the child's mouth will be a more successful approach than the traditional one, which has not been very successful at all. Such an approach not only allows the nondental health provider to make a referral to an appropriate clinic, if caries is observed, but, in addition, it provides the parent or caretaker with convincing evidence that active oral disease is present and that it could be stopped or reversed. A meaningful discussion of secondary preventive options may then be translated into action.

Other strategies for prevention follow from the findings in Table 4. Recommendation of "immediate substitution of cup for bottle feeding" was likely/very likely to be followed by only 46 percent; slow cup substitution was likely/very likely for almost twice as many parents. Similarly, while only 58 percent endorsed "immediately cut out extra night feedings; even if baby cries", an additional third of the sample endorsed slow reduction in volume at extra night feedings. Some alternatives may be difficult, if not impossible for parents or caretakers to follow. Furthermore, some alternatives may be more culturally appropriate than others.

For at least this population, given this and previous data, "cold turkey" is not a reasonable alternative for many families.[9] Moreover, Ferber, an expert in sleep disorders, recommends gradually decreasing the amount of milk or juice given to a child when falling asleep.

The alternatives that were most highly endorsed were daily fluoride drops (94 percent) and professionally-ap-

plied "medicine" (98 percent). Many parents and caretakers seem willing to do something extra, even on a daily basis, rather than alter their feeding or sleeping practices. In all, parents and caretakers have given us a strong message that they are not likely to follow recommendations that cause familial disruptions.

We believe that the high percentage of parents and caretakers who endorse the chemical agent alternatives indicates that prescriptions work better than proscriptions. We have proposed that topical application of fluoride and other agents have great promise in preventing BBTD for children at high risk.[9] We encourage our colleagues to develop innovative approaches, by providing choices to parents and minimizing, therefore, the need for parent or patient compliance.

REFERENCES

1. Ripa, L.W.: Baby bottle tooth decay (nursing caries: a comprehensive review). Pediatr Dent, 10:268-282, July-August 1988.
2. Hennon, D.K.; Stookey, G.K.; Muhler, J.C.: Prevalence and distribution of dental caries on preschool children. JADA, 79:1405-1414, December 1969.
3. Graves, R.C.; Abernathy, J.R.; Disney, J.A. *et al*: University of North Carolina Caries Risk Assessment Study. III multiple factors in caries prevalence. J Pub Health Dent, 51:134-143, 1991.
4. Goepford, S.J. *et al*: Infant oral health: A protocol. J Dent Child, 53:261-266, July-August 1986.
5. Johnsen, D.C.: Characteristics and backgrounds of children with "nursing caries." Pediatr Dent, 4:218-224, 3rd Quarter 1982.
6. Phillips, M.G. and Stubbs, P.E.: Head start combats baby bottle tooth decay. Child Today, 16:25-28, September-October 1987.
7. Johnsen, D.C.: Baby bottle tooth decay: a preventable health problem in infants. Update in Pediatr Dent, II:1-7, November 1988.
8. Callatin, S. An approach to the resolution of Mexican-American resistance to diagnostic oral remedial pediatric heart care. In: Chrisman, N.J. and Maretzki, T.W. (eds), *Clinically applied anthropology*. Hingham, MA: D. Reidel Publishing Co., 225-350, 1982..
9. Weinstein, P.; Domoto, P.; Wöhlers, K. *et al*: Mexican-American parents with children at risk for baby bottle tooth decay: Pilot study at a migrant farmworkers clinic. J Dent Child, 59:376-383, September-October 1992.
10. C.D.C.: Ten state nutritional survey, 1968-70. Dept. HEW Pub. No. (HSM) 72-8131, Atlanta, GA: Centers for Disease Control, 87-135, 1972.
11. Bell, R.M.; Stamm, J.W.; Graves, R.C.: Results of baseline dental exams in the National Preventive Dentistry Demonstration Program. Pub. No. R-2862-RWJF. Santa Monica: Rand Corp., 1982, pp 1-87.
12. Bohannon, H.M.; Klein, S.P.; Disney, J.A. *et al*: Caries prevalence in the National Preventive Dentistry Demonstration Program, Santa Monica, CA: Rand Corp., 1981, pp 1-8.
13. Ismail, A.L.; Burt, B.A.; Brunella, J.A.: Prevalence of dental caries and periodontal disease on Mexican-American Children aged 5-17 years: Results from southwestern. H. Hanes, 1982-83. Am J Public Health, 77:967-970, August 1987.
14. Pollick, H. *et al*: The estimated cost of treating unmet dental restorative needs of Mexican-American children from southwestern U.S. H. H. Hanes, 1982-83. J Pub Health Dent, 51:195-204, Fall 1991.
15. Hardwick, F.K.; McIlveen, L.M.; Forrester, D.J.: A comparison of nursing caries prevalence in Black and Hispanic children. Abstract, AAPD, 1991.
16. Woofolk, M.; Harmard, M.; Bagramian, R.A.: Oral health of children of migrant farmworkers in Northwest Michigan. J Pub Health Dent, 44:101-105, Summer 1984.
17. Koday, M.; Rosenstein, D.I.; Lopez, G.M.N.: Dental decay rates among children of migrant workers in Yakima, WA. Public Health Rep, 105:530-533, September-October 1990.
18. Trevino, F.M. and Moss, A.J.: Health indicators for Hispanic, black and white Americans. DHS Pub. No. (PHS) 84-1576, Series 10, No. 148. Hyattsville, MD: National Center for Health Statistics, September 1984, pp 1-88.
19. Gracia, J.A. and Juarez, R.Z.: Utilization of dental health service by Chicanos and Anglos. J Health Soc Behav, 19:428-436, December 1978.
20. Ventura, S.J. and Taffel, S.M.: Childbearing characteristics of U.S. and foreign-born Hispanic mothers. Public Health Rep, 100:647-652, November-December 1985.
21. Lewin-Epstein, N.: Determinants of regular source of health care in black, Mexican, Puerto Rican, and non-Hispanic white populations. Med Care, 29:543-537, June 1991.
22. Koday, M.: Yakima Valley Oral Health Status Survey. Unpublished manuscript, 1990B.
23. Abidin, R.R.: *Parenting stress index: short form test manual.* Charlottesville, VA: Pediatric Psychology Press, 1990.
24. Loyd, B.H. and Abidin, R.R.: Revision of the Parenting Stress Index. J Ped Psychol, 10:169-177, June 1985.
25. Matsumura, S.: Bacteriological and epidemiological studies on the Cariostat for dental caries activity test. Jpn J Pedodont, 21:107-130, February 1983.
26. Matsumura, S.; Nishimura, M.; Ohmura, M.: Studies in the evaluation for caries activity on the approximal surfaces. Pediatr Dent J, 1:51-57, January-February 1991.
27. Berkowitz, R.J.; Jordan, H.V.; White, G.: The early establishment of *Streptococcus mutans* in the mouths of infants. Arch Oral Biol, 20:171-174, March 1975.
28. Catalanotto, F.A.; Shklair, I.L.; Keene, H.J.: Prevalence and localization of *Streptococcus mutans* in infants and children. J Am Dent Assoc, 91:606-609, September 1975.
29. Stiles, H.M.; Meyers, R.; Brunelle, J.A. *et al*: Occurrence of *Streptococcus mutans* and *Streptococcus sanguis* in the oral cavity and feces of young children. In: Stiles, H.M.; Loesche, W.J.; O'Brien, T.J., eds. Proceedings, microbial aspects of dental caries. Vol I Sp Supp, Microbiology Abstracts. New York: Information Retrieval, Inc., 187-199, 1976.
30. Masuda, N.; Sobue, S.; Hamada, S.: Longitudinal survey of the distribution of various serotypes of *Streptococcus mutans* in infants. J Clin Microbiol, 10:497-502, October 1979.
31. Köhler, B.; Bratthall, D.; Krasse, B.: Preventive measures in mothers influence the establishment of the bacterium *Streptococcus mutans* in their infants. Arch Oral Biol, 28(3):225-231, 1983.
32. Caufield, P.W.; Cutter, G.R.; Disanayake, A.P.: Initial acquisition of mutans Streptococci by infants: Evidence for a discrete window of infectivity. J Dent Res, 72:37-45, January 1993.
33. Ferber, R.: *Solve your child's sleep problems.* New York: Simon and Schuster, 1985, pp. 81-89.

Teaching parents at WIC clinics to examine their high caries-risk babies

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Babies as young as eleven months are at risk for a distinctive pattern of severe tooth decay. The four maxillary incisors are most affected, while the four mandibular incisors usually remain unaffected. The disease has a predictable progression: at first white spots appear on the teeth; these are usually decalcification lesions, and may progress to frank lesions or caries, usually within six months to a year. Such decalcification lesions will not necessarily progress to cavities, the process may be reversed and the teeth may remineralize.

Results of studies from predominantly western-type cultures found with few exceptions the prevalence of Baby Tooth Decay (BTD) to be approximately 5 percent or less.[1] Disadvantaged children, however, are much more vulnerable.[2,3] The highest prevalence of BTD may be for Native American and immigrant infants.[4-7]

Feeding and childrearing practices have been identified as the most important etiological factor.[1] While bottle and even breast feeding practices have been shown to be related to BTD, recent work suggests snacking as an important risk factor, especially for Native American children.[8,9]

When early BTD goes undetected and unchecked, it causes severe problems for the child. Children may be in considerable pain and they may have difficulty eating and talking. The disease is also a serious threat to the health of other primary teeth and, subsequently, to the health of the permanent dentition. Treatment of frank lesions is expensive and invasive; and has some risks, because general anesthesia, sedation, or physical restraint are almost always required to treat these lesions.

There is consensus that prevention is the only rational approach. Unfortunately, the traditional educational approach provides general information about BTD, focusing on the bottle as the risk factor, and recommends immediate substitution of the cup at all feedings by twelve months. It is clear from research data that such an approach does not work.[10] An alternative strategy would provide specific information about whether or not the child is at risk for BTD or has decalcification or decay. Such an approach includes a screening examination in which the parent plays an active role. When risk is high or results of the examination are positive, culturally appropriate interventions follow. It may not be necessary to insist on weaning at one year, a process that may fly in the face of cultural norms and/or may prove to be very difficult for the parent/caretaker with little social support or with a sick or temperamentally difficult child.[7]

Given that caries is a multifactorial disease caused by pathogenic bacteria, a diet of refined carbohydrates, low

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host resistance, and time for the lesion to develop, interventions may be targeted at all of the above factors. Recent research indicates that an easy to apply fluoride varnish painted on the maxillary incisors of migrant farmworker children less than eighteen months of age decreased disease prevalence when assessed six months later.[11] Other interventions are possible. Bactericidal approaches for lowering the number of cariogenic organisms have been limited; preliminary work with a topical iodine preparation, however, appears promising, according to a recent personal communication with Dr. R. Berkowitz. While, as noted, a very limited approach in promoting early weaning has been unsuccessful, many other culturally-appropriate interventions aimed at influencing diet and time are possible. For example, interventions that focus on limiting time the bottle/breast is available at night or progressively reducing the volume of liquid are possible. In addition, given snacking as a risk factor, attempts to reduce the risk from ad lib snacking (beverages and sweets) seem especially promising in a Native American population.[9]

This study attempted to develop and test materials to train the nondental health worker and parent/caretaker to examine briefly a young child to determine whether or not the upper anterior teeth were decalcified/decayed or healthy. The study was accomplished at WIC programs (The Special Supplemental Food Program for Women, Infants, and Children). These programs, usually administered by local health departments have been suggested as sites for vaccination and smoking cessation programs.[12-14] Johnson and Nowjack-Raymer have already noted that WIC nutritionists may have an opportunity to prevent BTD.[15]

METHOD

Subjects

Seventy-seven parents/caretakers and children at four WIC clinics in King County, Washington participated in the study. Children's ages averaged 24.4 months. Dentists identified disease in 35.1 percent (29/77) of the children.

Procedure

After agreeing to participate in the study, parents/caretakers observed a five-minute videotape. The videotape provides a very brief rationale for lifting the lip and then proceeds 1) to demonstrate positioning for the examination with/without a mirror and 2) to guide parental/caretaker assessments with illustrations of the progression of Baby Tooth Decay in the plaque-accumulating smooth surfaces of the high-risk teeth (Figure 1). A color brochure that reviews the important points from the videotape and focuses attention on the at-risk surfaces in high-risk teeth was created to help the decision-making process of whether or not a tooth problem was identified. Parents/caretakers completed a short questionnaire assessing whether or not they observed any tooth problems, sureness of seeing/not seeing a problem, and problem location and color. Immediately following the parental/caretaker examination a trained dental student or dentist reexamined the young child and completed the same questionnaire.

RESULTS

Parents/caretakers found 29.9 percent (23/77) of their children with tooth problems. Dentists identified disease in 35.1 percent (27/77) of the children. Ninety-five percent of the parents/caretakers and 96 percent of the dentists were sure of their assessments. See Table for a comparison of parent/caretaker and dentist observations. Over 80 percent of the parents/caretakers responded the same as dentists; 11.7 percent of parents/caretakers did not see disease when dentist did, while 6.5 percent saw disease when dentist did not ($K = .59$, $Z = 4.4$, $p < .001$). Reports varied by tooth surface; while parents/caretakers reported 13 percent on lingual surfaces (3/23), dentists noted only 3.7 percent (1/27). For buccal surfaces parents/caretakers observed 39.1 percent (9/23) of the problems, while dentists observed 37 percent (10/27) of the disease. Parents/caretakers reported a 47.9 percent (11/27) rate of problems in both surfaces, dentists a 59.3 percent (16/27) rate. Report of color by parent/caretakers was as follows: 47.9 percent (11/23) perceived light-brown-color problems; 8.7 percent (2/23) perceived chalky white and 43.4 percent (10/23) another color. Dentist reported 29.6 percent (8/27) light brown, 22.2 percent (6/27) chalky white, and 48.2 percent (13/27) other color problems.

Table ☐ Comparison of parent/caretaker vs. dentist observation.

	Dentist did not observe problem	Dentist observed problem.
Parent/Caretaker did not observe problem	45 (58.4%)	9 (11.7%)
Parent/Caretaker observed problem	5 (6.5%)	18 (23.4%)

DISCUSSION

Given the substantial kappa, the results of this study indicate the feasibility of training low-income parents/caretakers to assess their high-risk young children for BTD within a WIC setting. While WIC personnel have access to high-risk children and have been sympathetic to our concerns about early forms of dental disease in their clientele, they have not had the tools to counsel effectively. Proscription of bottle feeding at an early age and distribution of weaning cups have been the state-of-the-art. The materials that have been developed allow the WIC workers and parent/caretakers to conduct a brief dental examination. Subsequent discussion of behavioral risk factors, culturally and personally acceptable strategies to prevent disease and referrals to dental clinics for chemotherapeutic preventive efforts and/or treatment are possible and more likely to be successful than if such efforts occurred without an examination. Subsequent research will probably confirm these assertions.

A brief dental examination in a nondental environment is the first step in intervening to prevent BTD. Brief behavioral risk questionnaires or structured interviews are also necessary, including guidelines and materials that assist the nondental health worker efficiently counsel the parent/caretaker during the critical period that the child is at risk for BTD.

Based on feedback provided by 125 WIC personnel at a presentation during a WIC conference, plans have been made to modify the videotape material and to provide versions of the materials in Spanish. In addition, research will be conducted to identify the most efficient set of behavioral risk factor items and to operationalize and evaluate a brief counseling procedure that can be used in conjunction with chemotherapeutic techniques, such as fluoride varnish.[11]

REFERENCES

1. Ripa, L.W.: Baby bottle tooth decay (nursing caries: a comprehensive review). Pediatr Dent, 10:268-282, July-August 1988.
2. Holt, R.D.; Joels, D.; Winter, G.B. *et al*: Caries in pre-school children: the Camden study. Br Dent J, 153:107-109, August 1982.
3. Johnsen, D.C.; Schultz, D.C.; Schubot, D.B. *et al*: Caries patterns in Head Start children in a fluoridated community. J Pub Health Dent, 44:61-66, Spring 1984.
4. Kelly, M. and Bruerd, B.: The prevalence of baby bottle tooth decay among two Native American populations. J Public Health Dent, 47:94-97, Spring 1987.
5. Broderick, E.; Marby, J.; Robertson, D. *et al*: Baby bottle tooth decay in Native American children in Head Start centers. Pub Health Reports, 104:50-54, 1989.
6. Albert, R.J.; Cantin, R.Y.; Cross, H.G. *et al*: Nursing caries in the Inuit children. Can Dent Assoc J, 54:751-768, October 1988.
7. Weinstein, P.; Domoto, P.; Wöhlers, K. *et al*: Mexican-American parents with children at risk for baby bottle tooth decay: pilot study at a migrant farmworkers clinic. J Dent Child,, 59:376-383, September-October 1992.
8. Heller, K.: Infant dental caries—feeding and social factors in a 12-36 month old Native American population. Unpublished manuscript, 1992.
9. Feinman, S.: An inquiry into the perceptions and correlates of baby bottle tooth decay in Navajoland. Unpublished manuscript, 1993.
10. Johnsen, D.C.: Baby bottle tooth decay: a preventable health problem in infants. Update in Ped Dent, II:1-6, January 1988.
11. Weinstein, P.; Domoto, P.; Koday, M. *et al*: Pilot study of a six month trial of fluoride varnish in prevention of lesions on very young migrant farmworker children. Paper presented at American Association of Public Health Dentistry, San Francisco, November 1993.
12. Hutchins, S.S.; Gindler, J.S.; Atkinson, W.H. *et al*: Preschool children at high risk for measles: opportunities to vaccinate. Am J Public Health, 83:862-867, June 1993.
13. Fleisher, L.; Keintz, M.; Rimer, B. *et al*: Process evaluation of a minimal-contact smoking cessation program in an urban nutritional assistance (WIC) program. Prog Clin Biol Res, 339:95-106, 1990.
14. Mayer, J.P.; Hawkins, B.; Todd, R.A.: Random evaluation of smoking cessation interventions for pregnant women at a WIC clinic. Am J Public Health, 80:76-78, January 1990.
15. Johnson, D. and Nowjack-Raymer, R.: Baby bottle tooth decay (BBTD): Issues, assessment, and an opportunity for the nutritionist. J Am Diet Assoc, 84:1112-1116, August 1989.

EFFECT OF WHEAT STARCH IN FOODS ON DEMINERALIZATION OF ENAMEL

Many microorganisms in plaque are known to be able to ferment maltose, while others, like the currently used mutans strain, are unable to do so. However, very little is known about the inducibility of these systems. One would expect that the frequent consumption of bread, rolls, and other unsweetened or lightly sweetened baked goods would provide sufficient opportunity for microorganisms that are capable of synthesizing maltose-related systems to do so.

Kashket, B. *et al*: Delayed effect of wheat starch in foods on the intraoral demineralization of enamel. Caries Res, 28:291-296, July-August 1994

SNACKING

Snacking and oral health habits of Washington state WIC children and their caregivers

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Although dental caries is declining among young children, by five years of age U.S. children have an average of 3.4 decayed or filled primary teeth.[1] National children's caries surveys show that 75 percent of dental caries is concentrated in 25 percent of the children of minority groups, children of low-income families, and children whose parents have less than a high school education.[2] Frequently these low-income children do not benefit from proven caries preventive measures: systemic and topical fluorides, dental sealants, oral hygiene measures, and dietary modification. This may be attributed to both lack of oral health education and access to professional dental care.

Starting preventive dental health practices early with infants and toddlers is easier than trying to change established routines. Dental health education specifically directed toward the caregivers of these children is one means of lowering their caries risk. In the 1980s in Helsinki, oral health education was directed toward pregnant women and parents of preschool children. The dmft (decayed, missing and filled primary teeth) of five-year-old Finnish children was about one-half the dmft of children in Edinburgh, Scotland, where no dental education program for young families existed.[3] Dietary habits formed early in childhood strongly influence eating patterns during the school years. In addition, children who do not develop caries in the primary dentition have a lower risk of developing caries in the permanent dentition.[4]

Caries is dependent upon the interaction of multiple etiologic agents, including heredity, plaque bacteria, salivary factors, fluoride exposure, oral hygiene practices, and dietary behavior. It has been clearly established that both dietary starches and sugars are important caries-promoting components of foods.[5] A high daily intake of sugars or retentive starch-sugar combinations has been correlated with a high caries experience in preschool and school-age children in three recent studies.[6-8] A child's risk of developing caries is greater when a high intake of fermentable carbohydrate is combined with the presence of high numbers of decay-causing bacteria in the oral cavity.

A statistically significant relationship has also been shown between a high frequency of sugar/starch intake and caries.[9-11] Frequent ingestion of fermentable carbohydrate makes possible a continual formation of enamel-destructive acid. Between-meal ingestion of table sugars, syrups, and sugary desserts was significantly higher in the nine to twenty-nine-year age-group with high DMFT scores, than in the group with low DMFT scores in a national cross-sectional survey.[12] A significant association between frequency of sugar ingestion and caries experience has also been reported in 231 Norwegian preschool children.[13] School-age children in New York State with a high number of decayed, missing and filled tooth surfaces (DMFS) ate more snacks after dinner.[14] Food particles retained in the mouth at the end of the day may provide substrate for bacteria found in plaque for a longer period of time than foods eaten earlier in the day.

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Regular use of fluoride is an important measure for children at high risk for dental caries. Dentifrice with fluoride complements the use of a fluoride supplement or the drinking of fluoridated water. Although toothbrushing may be ineffective in removing all food particles from pits and fissures, toothbrushing with a small amount of fluoride dentifrice is an important means of delivering fluoride to tooth surfaces.[15] The frequency with which children brush their teeth has been reported to be less in children whose mothers are young and possess low occupational skills.[16] The single best predictor of variance in caries experience of a group of Australian preschool children was the mother's education.[17] The caries rate of Norwegian children was reported to be related to the mother's education level, mother's own oral health, and the frequency with which the mother seeks professional dental care for herself.[13] Children of mothers with low educational levels, who made irregular dental visits, and had teeth missing were more likely to have dental caries at thirty-six months of age.

The most realistic means of improving the oral health of high risk children is to incorporate preventive oral health instructions into existing maternal and child health programs. The Special Supplemental Food Program for Women, Infants and Children (WIC) is a federally funded nutrition education program serving low-income, pregnant and breast-feeding women and children up to age five who are at risk of nutritional or medical problems. In addition to health assessments, nutrition counseling, and referral services, WIC in Washington State provides an average of $45.00 per month of WIC checks for prescriptive foods (eggs, cheese, milk, cereal, peanut butter/beans, juice or infant formula). Thus WIC provides a strategic opportunity to reach parents and children when dietary and oral health habits are being formed.[18] The objectives of this study were to assess, by interviewing WIC caregivers:

- ☐ The snack foods most commonly eaten each day by WIC children.
- ☐ How often each snack food was eaten.
- ☐ The number of snacks eaten each day.
- ☐ The dental health habits of caregivers and WIC children.

METHODS

Subjects were participants in three WIC clinics in King County, an urban area of Washington state, which had large case loads of children. In 1993, 75 percent of the children with a nutritional risk enrolled in the Washington WIC program came from families with monthly incomes of $964 or less for three persons.[19] The survey was conducted during two of the busiest clinic days each week over a four-month period in 1993-94. Each English-speaking caretaker with a child one year of age or older was approached in the waiting area of WIC clinics, where they had an appointment, and asked whether they were willing to complete an interview about their child's snacking and dental health habits. Each willing caregiver was given a consent form to read and sign. Two nutritionists and a trained WIC clerk conducted the structured interviews following a standardized interview format and recording procedures. The interview technique has been reported to give more accurate data about preschool children's dietary habits related to dental caries, especially snack intake, when compared with caregivers completing a written questionnaire.[20]

A forty-two-item questionnaire was developed by the authors to assess the oral health and snacking habits of WIC clients, ages one to four years, and their caretakers. The survey instrument was pilot tested with ten WIC caregivers and revised to increase clarity. Demographic information was self-reported. The interviewer asked each WIC caregiver about the usual number of snacks consumed per day, favorite snack foods, and the amount and frequency of intake of fifteen common snack foods.

The caregiver's oral health status was assessed from three questions about the presence of tooth pain and decay within the last year, the frequency of gums bleeding when teeth are brushed, and the time of the last dental visit for nonemergency care. Questions were also asked about the dental habits of the WIC child and the caregiver's attitude toward tooth loss due to dental disease. The survey was approved by the Human Subjects Committee of the University of Washington and Quality Practice and Programs Committee of the Seattle-King County Department of Public Health.

The responses to the survey were numerically coded and analyzed using the Statistical Analysis System (version 6, 1989, SAS Institute, Cary, N.C.). Fisher's exact test (2-tailed) was used to determine statistically significant relationships. Taking into account that a large number of tests were done, .02 level of significance was used.

RESULTS

Eighty-four caregivers of children, ages twelve to forty-eight months (mean = 20.0 months) participated in this survey (Table 1). Children twelve to eighteen months of age represented 41 percent of the sample, while 27 percent were nineteen to twenty-four months, and 19 percent were two to four years of age. Ninety-one percent

of the caregivers (n=76) were the children's mothers; four of the respondents were the grandmothers, and three were the fathers. Sixty-three percent of the caregivers were Caucasian, 10 percent were African American, 10 percent were Hispanic, 8 percent were Asian, and 9 percent were other. A greater number of respondents had high school diplomas or GED (43 percent) or had some college (36 percent), while 21 percent had less than twelve years of education. More than a half of the group (58 percent) were married, 30 percent were single, and 13 percent were divorced. The caregivers who were interviewed ranged in age from sixteen to forty-eight years of age (mean age = twenty-eight years). Two-thirds of the group (66 percent) had participated in the WIC program for one to two years. Compared with the total state WIC caseload, the caregivers in this study were older and had a higher level of education.

Snack food choices and frequency of snacking

The most common number of snacks per day reportedly consumed by the children was three (42 percent) or two (37 percent), although 19 percent of the children reportedly snacked more than three times a day. Most caregivers stated they snacked once or twice daily (78 percent), whereas 17 percent rarely snacked. There was no correlation between the frequency of snacking of caregivers and of children. The most commonly named daily snack foods for WIC children were milk, fruit juice, crackers, bananas, apples, and cheese, which all contribute to obtaining a balanced diet (Table 2). Two WIC foods obtained with WIC checks were reported to be consumed daily as snacks by the children, milk (78 percent) and cheese (33 percent). Raisins have been shown to be highly cariogenic in rat studies. According to caregivers, about 12 percent of WIC study children consumed raisins daily and 26 percent ate them once or twice a week as snacks. A dried fruit roll-up was eaten once or twice a week by 12 percent of the children. Soft drinks and powdered fruit drink were offered as snacks by less than 25 percent of the group. Foods rarely reported as snacks by the WIC toddlers included carrots (63 percent) and broccoli (76 percent).

Eighty-five percent of those interviewed reported that a parent chooses the snacks eaten by the child. Most caregivers had rules about when sweet foods could be eaten. The most frequently stated rules were: *not before meals* (28 percent), *not at night* (15 percent), and *only after meals* (13 percent). These rules were reportedly employed significantly more often by caregivers of children older than eighteen months of age (p=.019). A sweet food was reportedly offered by 22 percent of caregivers *sometimes* to get their children to behave, but *rarely* or *never* by most (78 percent) respondents.

The most frequently stated criteria used in selecting snack foods at the grocery store were because the food was *healthy* (49 percent), or their child's *favorite* (30 percent). There were significant differences (p=.017) between married and single or divorced caregivers, in the stated reason for snack food purchases. Single or divorced caregivers purchased *favorite snack foods* more often than married persons. Twenty-five percent of caregivers reported *always* putting their child to bed with

Table 1 ☐ Demographic characteristics of caregivers and WIC children. N=84

	Number	Percent
Caregiver		
Age		
Less than 20 years	5	6
20-29 years	45	54
30-39 years	27	32
40-48 years	7	8
Marital status		
Married	48	58
Single, divorced	35	42
Education background		
<12 years	17	21
12 years/GED	36	43
Some college	30	36
Race		
White	52	63
Other	31	37
WIC child	**Number**	**Percent**
Gender		
Female	38	45
Male	46	55
Age		
12-18 months	41	49
>18 months	43	51

Table 2 ☐ Most common snack choices of WIC children.

	Eating frequency							
	1-2 X/Day		3-6 X/Wk		1-2 X/Wk		Rarely/Never	
Food	No.	%	No.	%	No.	%	No.	%
Milk	65	78	5	6	1	1	12	14
Fruit juice	57	69	12	15	6	7	8	10
Cracker	37	44	10	12	21	25	16	19
Banana	31	37	17	21	16	19	19	23
Apple	19	33	17	21	29	35	18	22
Cheese	27	33	19	23	18	22	19	23
Graham cracker	14	17	9	11	18	21	43	51
Cookie	16	19	8	10	32	39	27	33
Raisins	10	12	3	4	22	26	49	58
Yogurt	13	16	5	6	19	23	47	56

a bottle, while 12 percent responded *sometimes*. Fifty-six percent put milk or infant formula in the bedtime bottle, whereas, 24 percent put water in the bottle. Only two caregivers put juice in the bedtime bottle.

Oral health habits

Commonly reported dental problems of caregivers included toothache, cavity, or bleeding gums when brushing. The self-reported dental health status of the caregivers is described in Table 3. One-half of WIC caregivers (51 percent) had not visited a dentist in the last year; whereas, 91 percent of the children had never had an oral examination by a dentist. One-fourth (26 percent) of the children did not have their teeth cleaned by an adult daily. About 83 percent of WIC study children reportedly obtained optimal fluoride from water (57 percent), or were using a fluoride supplement (26 percent). Seventeen percent of the children did not appear, however, to receive adequate fluoride.

Although dental caries is an infectious disease, only 12 percent of caregivers said that bacteria were the main cause of caries. The most frequently stated cause of tooth decay was *not brushing the teeth* (Table 3). *Sugar intake* was considered the primary factor in caries development by caregivers with less than twelve years of education; whereas, *not brushing* was the stated cause of cavities by those with twelve or more years of education. These differences, however, were not significant. Dentists and the media or friends were reported to be the main sources of dental-nutrition information.

A significant difference of opinion existed among caregivers regarding the importance of tooth decay, based on their education level ($p=.013$) and whether they had experienced a toothache or dental decay in the past twelve months ($p=.021$). Most respondents (65 percent) with less than twelve years of education stated it would be a small problem, if their child's tooth had to be extracted because of a cavity; whereas, those caregivers with twelve years of education or more (75 percent) perceived it to be a *big problem*. Caregivers who had experienced a toothache or decay in the past twelve months considered loss of a tooth due to decay by their child would be a *big problem*.

In this study, no association was found between a child's snacking frequency or the types of snack foods eaten and the caregiver's age, education level, ethnicity, or number of years in the WIC program.

DISCUSSION

To provide children a balanced diet low in sugar-rich foods is an oral health standard agreed upon by most health professionals (Table 4). The number of snack times per day reported by the caregivers (two or three) is normal for this age-group and represents a limited insult to oral health for most children, if nonretentive foods are chosen. Fruits, vegetables, nuts, dairy foods without added sugars, and lean meats are snacks with low caries-promoting properties. Two of the most popular snack foods (crackers and bananas), named by the study group, are potentially cariogenic due to their retentiveness. The use of raisins, high in sugar content and retentive in nature, will promote caries. Further public education about the interaction of oral bacteria and retentive carbohydrates in the development of caries is needed. The low intake of soft drinks, cookies, and cakes for snacks reported in this study was also found in chil-

Table 3 ☐ Distribution of caregivers according to dental health behaviors.

Dental behavior	Number	Percent
Gums bleed when brushing		
Always/Sometimes	33	40
Rarely/Never	50	60
Toothache/Cavity in last year	39	47
Dental office visit (nonemergency)		
≤1 year	41	49
>1 year	42	51
Most important cause of cavities		
Bacteria	9	12
Sugar	27	35
Not brushing	40	51
Main source of diet-dental health information		
M.D.	4	5
D.D.S.	29	36
WIC	20	25
TV/friends/magazines	27	34

Table 4 ☐ Oral health standards for children 1-5 years.*

Provide a balanced diet low in sugar-rich foods.
Discontinue bottle feeding and offer liquids exclusively from the cup as soon after twelve months as possible.
Provide systemic fluoride through community water fluoridation or provide dietary fluoride supplements in fluoride-deficient areas.
Encourage the use of fluoride toothpaste for children (a pea-sized amount should be placed on the brush by the parent).
Encourage first dental visit between one and three years of age and routinely thereafter.
Use clinical judgment in applying dental sealants to primary posterior teeth based on tooth morphology, timing of eruption, and patient susceptibility to dental caries.
Provide "basic oral health services" as needed.

*Compilation of guidelines and recommendations from multiple sources (21)

dren participating in an urban New York State WIC program.[22] Milk, the most commonly reported snack, has been correlated positively with a caries-free state in three- and four-year-old New York children. Cheese, a noncariogenic, nutrient-dense food, was a frequently reported snack.

Mothers are more likely to underreport food ingestion of young children than overreport; this is especially true for snacks.[23] Thus, the snack foods reported by caregivers for WIC children and themselves in this study are probably an underestimation of actual between-meal ingestion. Midmorning and midafternoon snacks are usually required by the preschool child in order to obtain adequate calories and essential nutrients, because of insufficient ingestion at mealtime. To recommend no eating between meals to the parents of young children is inappropriate.

The major dental health problem of U.S. children under three years of age, baby bottle tooth decay, is related to feeding practices. The percent of caregivers who reported always or sometimes putting their child to bed with a bottle was high. The number of respondents in the minority ethnic groups was too small to detect any differences in bottle use among the groups. The caries risk associated with extended use of the bedtime bottle is discussed with caregivers by WIC staff in the clinics surveyed. Parents are given a baby cup imprinted with *Stop Baby Bottle Tooth Decay* when each child is six months of age, and are advised to wean the child to the cup by twelve months of age. Cultural beliefs and psychosocial factors may contribute to prolonged use of the bottle.

Statistically significant differences have been reported between caries-free Head Start children and those with smooth-surface lesions, based on the mother's educational level, interaction with grandparents, mother's perceived cause of cavities, and mothers allowing children to eat sweets without restrictions.[24] The use of foods for nonnutritive purposes can have dental consequences; this does not seem to be an issue, however, for the study group. Responses indicate that caregivers control the type of snack foods eaten by their children and when they are consumed. The infrequent use of snack foods to control behavior was also reported in a study of Iowa WIC mothers, where 88 percent did not use food to encourage desired behavior.[25]

In this study, the education level of the WIC caregiver was found to be related to beliefs about the cause of cavities and to caregivers' attitudes toward tooth loss because of dental caries. Oral health habits of children and caregivers were below standards recommended by dental professionals. Only 5 percent of children, eighteen months of age or less and 12 percent of children between seventeen and forty-eight months had seen a dentist. In 1992, 28 percent of King County children on Medicaid received dental care.[26] In comparison, a 1989 national health survey found that a third (32 percent) of children, ages two to four, had visited a dental professional.[27] The national trend toward an increase in the percent of females visiting the dentist and the number of dental visits per year was not observed among the group interviewed. In 1986, 63 percent of U.S. females, ages eighteen to thirty-four, had visited a dentist during the past twelve months.[27] In comparison, 49 percent of WIC caregivers surveyed had visited a dentist in the last year. The low number of children and caregivers seeing a dentist can be attributed to the difficulty encountered by low-income families in obtaining dental care, in their respective geographic areas.

Caregivers may benefit from encouragement to brush their young children's teeth regularly. As compared with a group of Iowa children eighteen months of age, 92 percent of whom had their teeth brushed daily by an adult, 72 percent of Washington WIC toddlers twelve to eighteen months of age, had their teeth cleaned by an adult.[28] Beyond eighteen months of age, 76 percent of Washington caregivers reported cleaning their children's teeth.

CONCLUSIONS

Dietary practices may increase the risk that disadvantaged toddlers will develop caries in newly erupted primary teeth. Further research is necessary to confirm these initial findings about the oral health behaviors and snacking habits of low-income, toddler-aged children.

- ☐ This study revealed that the reported frequency of snacking was reasonable and often healthy snack foods were provided to WIC children. Caregivers need further education, however, about the caries-promoting properties of retentive foods, such as raisins when eaten as snacks.
- ☐ The continued use of the bedtime bottle was a common practice among some WIC caregivers and is a greater threat to oral health than snacking practices. Because caries rates in the permanent dentition are higher in children with *nursing caries*, efforts to prevent inappropriate bottle use by toddlers must be increased.[29]
- ☐ Professional dental care was received by few WIC children. Parent-supervised toothbrushing and optimal fluoride ingestion should be stressed, therefore, in other maternal and child health programs, such as the WIC program.

REFERENCES

1. *Oral health of United States Children. The National Survey of Dental Caries in U.S. School Children: 1986-1987.* National and Regional Findings. Washington D.C: U.S. Department of Health and Human Services; 1989, DHHS(PHS) NIH publication 89-2247, p 6.
2. White, A.B.: Toward improving the oral health of Americans: an overview of oral health status, resources, and care delivery. Pub Health Rep,123:96-106, November-December 1993.
3. Downer, M.C.; Nordling, H.; Blinkhorn, A.S. *et al*: The Edinburgh-Helsinki study: a comparison of dental care for children. Int Dent J, 35:226-231, September 1985.
4. Poulsen, S. and Holm, A-K.: The relation between dental caries in the primary and permanent dentition. J Pub Health Dent, 40: 17-25, Winter 1980.
5. Navia, J.M.: Carbohydrates and dental health. Am J Clin Nutr, 59: 719S-727S, Supplement, March 1994.
6. Sundin, B. and Granath, L.: Sweets and other sugary products tend to be the primary etiologic factors in dental caries. Scand J Dent Res, 100:137-139, June 1992.
7. Burt, B.A.; Eklund, S.A.; Morgan, K.J.: The effect of sugar intake and frequency of ingestion on dental caries increment in a three-year longitudinal study. J Dent Res, 67:14221429, November 1988.
8. Rugg-Gunn, A.J.; Hackett, A.F.; Appleton, D.R. *et al*: Relationship between dietary habits and caries increment assessed over two years in 405 English school children. Arch Oral Biol, 29:983-992, 1984.
9. Holt, R.D.: Food and drinks at four daily time intervals in a group of young children. Br Dent J, 170:137-143, February 23, 1991.
10. Holbrook, W.P.; Kristinsson, M.J.; Gunnardottir, S. *et al*: Caries prevalence, *Streptococcus mutans*, and sugar intake among 4-year-old urban children in Iceland. Community Dent Oral Epidemiol, 17:292-295, December 1989.
11. Palmer, C.A.: Diet and nutrition: crucial factors in the dental health of children. World Rev Nutr Diet, 58:131-159, 1989.
12. Ismail, A.I.: Food cariogenicity in Americans aged 9 to 29 years assessed in a national cross-sectional survey, 1971-74. J Dent Res, 65:1435-1440, November 1986.
13. Grytten, J.; Rossow, I.; Holst, D.; *et al*: Longitudinal study of dental health behaviors and other caries predictors in early childhood. Community Dent Oral Epidemiol, 16:356-359, December 1988.
14. Leverett, D.H.; Featherstone, J.D.B.; Croskin, H.M. *et al*: Caries risk assessment by a cross-sectional discrimination model. J Dent Res, 72:529-537, February 1993.
15. Newbrun, E.: Preventing dental caries: breaking the chain of transmission. J Am Dent Assoc, 123:55-59, June 1992.
16. Paunio, P.; Rautova, P.; Sillanpaa, M. *et al*: Dental health habits of 3-year-old Finnish children. Community Dent Oral Epidemiol, 21:4-7, February 1993.
17. Stacey, M.A. and Wright, F.A.C.: Diet and feeding patterns in high-risk preschool children. Aust Dent J, 36:421-427, December 1991.
18. Steffensen, J.E.M.: Literature and concept review: issues in maternal and child oral health. J Pub Health Dent., 50:S358-S369, Special Issue 1990.
19. Personal communication, Washington Department of Health, May 1994.
20. Schroder, V.; Lindstrom, L.G.; Olsson, L.: Interview or questionnaire? A comparison based on the relationship between caries and dietary habits in preschool children. Community Dent Oral Epidemiol, 9:79-82, April 1981.
21. Wilson, A.A.: Standards in maternal and child oral health. J Pub Health Dent, 50:432S438S, Special Issue 1990.
22. Buffum-Herman, N.: Evaluation of dental caries prevalence in three- and four-year-old children participating and not participating in the WIC program. Utica, NY: Cornell University. 1991 Thesis.
23. Baranowski, T.; Sprague, D.; Baranowski, J.H. *et al*: Accuracy of maternal dietary recall for preschool children. J Am Diet Assoc, 669-674, July 1991.
24. Johnsen, D.C.; Schultz, D.W.; Schubot D.B. *et al*: Caries patterns in Head Start children in a fluoridated community. J Pub Health Dent, 44:61-66, Spring 1984.
25. Seagren, J.S. and Terry, R.D.: WIC female parents' behavior and attitude toward children's food intake-relationship to children's relative weight. J Nutr Educ, 23:223-230, October, 1991.
26. Department of Social & Health Services Medicaid Assistance Administration Draft Report, December 3, 1993.
27. Gift, H.C. and Newman, J.F.: Oral health activities of US Children: results of a national survey. J Am Dent Assoc, 123:96-106, October 1992.
28. Levy, S.M.; Maurice, T.J.; Jakobsen, J.R.: Feeding patterns, water sources of fluoride exposures of infants and 1-year-olds. J Am Dent Assoc, 124:65-69, April 1993.
29. O'Sullivan, D.M. and Tinanoff, N.: Maxillary anterior caries associated with increased caries risk in other primary teeth. J Dent Res, 72:1577-1580, December 1993.

EFFECT OF CHLORHEXIDINE VARNISH

The main problem with antimicrobial treatment is the difficulty in suppressing mutans streptococci for extended periods of time. Factors promoting proliferation of mutans streptococci include sucrose-induced polysaccharide production, pH drops caused by these organisms, and bacteriocinogeny. Due to the ecological pressure induced by these factors, mutans streptococci will eventually overcome the colonization resistance of the established microflora and regrow to their original levels.

Schaeken, M.J.M. *et al*: Effect of chlorhexidine varnish on streptococci in dental plaque from occlusal fissures. Caries Res, 28:269-266, July-August 1994.

EPIDEMIOLOGY

Falling between the cracks: Oral health survey of school children ages five to thirteen having limited access to dental services

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In spite of the recent downward trend in caries prevalence, certain individuals in all segments of American society are caries prone. For a variety of reasons, these individuals are often unable or unwilling to perform appropriate self-care or to seek regular dental services.[1]

A number of studies indicate that children from lower socioeconomic families show a trend toward higher caries prevalence than children from higher socioeconomic families.[2-5]

Samuelson *et al* reported that children of parents with lower educational levels consume more in-between-meal sweets and carbohydrates than children whose parents have higher educational levels.[6] Neglect of tooth brushing and a higher intake of in-between-meal snacks containing carbohydrates and sugar probably accounts for higher caries prevalence rates in children from lower socioeconomic families.[7-8]

Children belonging to higher socioeconomic families have better oral hygiene and less gingivitis than children belonging to lower socioeconomic families.[9,10]

MATERIALS AND METHODS

A total of 367 Indianapolis school children ages five to thirteen were selected for the study from public and private Indianapolis schools. Some children were examined in Marion County clinics and others in the Pediatric Dentistry Department at the Indiana University School of Dentistry. The following selection criteria were used: The children were five to thirteen years of age and healthy, and had parental consent to participate in the study. A socioeconomic medical background questionnaire (Figure 1) and consent form were completed by the parents. Each participant received a toothbrush and oral hygiene instruction pamphlets. When the findings of the examination indicated a need for dental care, the parents were informed of the need.

Examination of the children in a school setting was accomplished using a portable dental chair and light, mouth mirror and explorer. The examinations were performed by one dentist (the senior author), in a systematic manner, starting on the maxillary right posterior quadrant and ending in the mandibular left posterior quadrant. In order to assure intra-examiner reliability and validity, before the study, the senior author (the sole examiner), received intensive training in dental caries and gingival health diagnostic methods by a well-known, long-time oral health investigator (Dr. Mark E. Mallatt, Oral Health Research Institute, Indiana University School of Dentistry). The criteria for diagnosis of dental caries were those developed by Radike, which included the diagnosis of opacities in enamel, softness at the base of the questionable area, and retention of the explorer tine.[11]

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The Papillary-Marginal-Gingival-Index (PMGI), a combination of the Gingival Index by Löe and Silness and the Papillary-Margin-Attached-Index (PMAI) by Schour and Massler, was employed for scoring gingivitis in children.[12-14] The gingival examination was limited to those tissues immediately surrounding erupted permanent and primary teeth. Each papilla and each margin on every erupted tooth were graded.

Table 1 ☐ Range of total scores in each class on the two factor index.

	Class	Range of total scores
High	I	11–18
	II	19–38
Middle	III	34–51
	IV	52–68
Low	V	67–84

SOCIOECONOMIC EVALUATION

The Hollingshead Two Factor Index of Social Ranking was used in this study.[15,16] The Two Factor Index utilizes occupational and educational scales as follows:

Rankings - Occupational Scale (Constant factor = 7)

- ☐ Higher executives of large concerns, proprietors and major professionals.
- ☐ Business managers, proprietors of medium-size businesses, and lesser professionals.
- ☐ Administrative personnel, owners of small businesses, and minor professionals.
- ☐ Clerical and sales workers, technicians, and owners of little businesses.
- ☐ Skilled manual employees (Plumber).
- ☐ Machine operators and semi-skilled employees.
- ☐ Unskilled employees.
- ☐ Unemployed.

Rankings - Educational Scale (Constant factor = 4)

- ☐ Professional (MA, MS, ME, MD, PhD, LLB, DDS, etc.).
- ☐ Four-year college graduate (AB, BS, BM).
- ☐ One to three years of college (Plumber).
- ☐ High school graduate.
- ☐ Ten to eleven years of school.
- ☐ Seven to nine years of school.
- ☐ Less than seven years of school.

The score that each family head received on each scale was multiplied by an approximate constant-factor for each scale. To illustrate: a plumber who attended trade school for two years received a "5" on the occupational scale ranking and a "3" on the educational scale ranking. The "5" was multiplied by the constant occupational factor of 7, resulting in a partial score of 35. The "3" was multiplied by the constant educational factor of 4, resulting in a partial score of 12. These partial scores totaled 47, which fell into the Class III range (34-51), representing an index of middle socioeconomic status. The range of total scores in each class on the Two Factor Index is represented in Table 1.

An effort was made to select a sample population for this study that adequately represented the socioeconomic levels found in Marion County (Indiana) Census. The Newman-Keuls t tests were used to compare the oral health of children in the various classifications.

RESULTS

The age-range of the children examined was five to thirteen years, with a mean age of 8.13 years. Among the 367 children examined, 181 were males and 186 were females. There was a relatively equal distribution of males and females in each of the five socioeconomic groups in this study.

Table 2 presents a comparison of oral health conditions of children among the five socioeconomic groups. The caries prevalence, as measured by the DMFT+deft and the DMFS+defs, was almost doubled in children who belonged to the lowest socioeconomic families (groups 4 and 5) as compared with the highest socioeconomic families (groups 1 and 2). The presence of untreated dental caries in these children (as measured by DS+ds) showed a dramatic increase from groups 1 to 5. The rate of gingival inflammation was doubled in the lowest socioeconomic groups as compared with the highest groups.

Table 3 presents a comparison in the oral health conditions among the children in regard to whether they have assistance (insurance or Medicaid coverage) or whether they take free lunch at school. Accordingly, the children were classified into four categories as follows:

Category 1

Includes those children who fall below the poverty level and thus, are eligible for the free lunch program and receive benefits from Medicaid coverage or other dental insurance.

Category 2

Includes those children whose socioeconomic level allows them free lunch at school, but who are not classified as poor enough to receive assistance (Medicaid or dental insurance coverage).

Table 2 ☐ Comparison of oral disease parameters of children by socioeconomic groups (caries experience and gingival condition).

Group number	DMFT + deft	DMFT + defs	DS + ds	MS + es	FS + fs	GI
1	0.660	0.981	0.038	0.000	0.925	0.367
2	1.159	1.984	0.111	0.000	1.873	0.229
3	1.450	2.270	0.330	0.000	1.940	0.360
4	2.852	4.500	2.511	0.398	1.591	0.607
5	2.762	4.159	2.984	0.397	0.921	0.701

Table 3 ☐ Observed means by lunch program status versus availability of assistance.

Categories	Number of Children	DMFT + deft	DRMF + defs	DS + ds	MS + es	FS + fs	GI
1	49	2.143	3.265	1.857	0.102	1.306	0.585
2	71	3.211	4.802	3.437	0.493	1.000	0.740
3	175	1.251	1.954	0.183	0.000	1.771	0.330
4	72	1.750	3.042	1.167	0.278	1.583	0.397

Category 3

Includes those children who do not get free lunch at school and who have dental insurance coverage.

Category 4

Includes those children who have no free lunch at school and have no dental insurance coverage.

There was a significant difference in the caries experience (measured by the combined DMFT and deft), using the Newman-Keuls sequential ranking at P=0.05 between categories 1 and 2, and 1 and 3, and at P=0.01 between categories 2 and 3, and 2 and 4. The highest scores were observed in category 2 showing DMFT+deft=3.211.

There was a significant difference in the presence of carious lesions measured by DS+ds (at P=0.05) between categories 1 and 2, and 1 and 3, and between categories 2 and 3, 2 and 4, and 3 and 4 (P=0.01), with the highest scores in category 2.

The gingival condition (as measured by the GI) showed a significant difference at P=0.01 between categories 1 and 3, 1 and 4, 2 and 3, and 2 and 4. The highest scores were observed in category 2.

A major finding of this study was the significant difference occurring between categories 1 and 2 in their caries experience and gingival health conditions. Children classified in category 2 are those children who take free lunch at school, but come from families that are not poor enough to be covered by Medicaid. There was a significant difference in their DMFT+deft=3.211, as compared with DMFT+deft=2.143 in children classified in category 1, i.e., children who take free lunch and have assistance.

The clinical presence of carious lesions was almost twice as many in category 2 as in category 1. This finding indicates that those children who fall in category 2 had the most untreated dental caries. They not only lived in poverty, but also had no assistance, compared with those individuals in category 1.

DISCUSSION

The results of this investigation agree with other published studies that show children from lower socioeconomic families to have a trend toward higher caries prevalence rates than children from higher socioeconomic families.[2-5] In this study, the caries prevalence was almost doubled in children from the lowest socioeconomic families (groups 4 and 5) as compared with the highest socioeconomic families (groups 1 and 2).

Likewise this study agrees with others that show that children from lower socioeconomic families experience more gingival disease than children from higher socioeconomic families.[2,9,10] In this study, the rate of gingival inflammation was doubled in the lowest socioeconomic groups as compared with the highest groups.

There was an expected significant difference in the oral health condition occurring between children who have assistance (Medicaid coverage or dental insurance) as opposed to children who have no assistance. The presence of untreated dental caries was more than twice as

Figure 1. Information questionnaire.

Child's name _____ Sex ____ Age ____ Date of Birth _____
(month-day)
Child's address _____ No. years lived there _____
In your home do you have city or well water? _____
Has your child ever resided outside of Indianapolis? _____
If yes, where? How long? _____
Please describe in detail the occupation (job) of head of your household ____
_____
_____
Indicate Industry _____
Circle highest level of education of head-of-household.
1 2 3 4 5 6 7 8 9 10 11 12 College 1 2 3 4 5 6 7 8 Other_
Is anyone else in your household employed? _____ Yes ____ No____
If yes, please describe in detail their job and their relationship to you. ____
_____
Circle highest level of education of spouse.
1 2 3 4 5 6 7 8 9 10 11 12 College 1 2 3 4 5 6 7 8 Other_
Does your child receive dental insurance benefits? _____ yes_____ no
Does your child receive Medicaid benefits? ____ yes____ no
Is your child eligible for free lunch at school? ____ yes____ no
Has your child ever had a serious illness that required hospitalization or a physician's care? If yes, explain what and when _____
_____
Does your child presently have a serious health problem? If yes, explain.____
_____
Has your child taken drugs/medication during the past year? If yes, explain why and when? _____
Has your child ever had any of the following? If yes, please check the appropriate space and explain below:

____Heart trouble	____Allergies	____Breathing problems
____Asthma	____Epilepsy	
____Anemia	____Fainting problems	____Diabetes
____Nervousness	____Jaundice	____Hepatitis
____Kidney or liver disease	____Rheumatic fever	____Tuberculosis
	____Bleeding disorders	____Tumors or growth

Explanation_____
_____

prevalent in children with no assistance as compared with those who had assistance.

A significant difference was observed in the caries prevalence among children from the lowest socioeconomic families that were eligible for free lunch at school and were classified as belonging to categories 1 and 2. Children falling into category 1 are eligible for free lunch at school and are covered by Medicaid or insurance, whereas category 2 children are eligible for free lunch, but their family income is not low enough to allow them to receive Medicaid benefits. Category 2 children had more untreated dental caries, fewer teeth filled and the least accessibility to dental care than the rest of the examined children because they not only live in poverty but also their families have no financial assistance.

SUMMARY AND CONCLUSIONS

A study of 367 Indianapolis school children ages five to thirteen revealed that there are a significant number of children in our society who suffer from untreated dental caries and gingivitis, chiefly because of insurmountable financial barriers. Their poor socioeconomic status makes them eligible for free lunch at school, but their family incomes are not low enough to permit them to receive the benefits of Medicaid coverage. The following conclusions can be made.

- ☐ Children falling into category 2 (i.e., those who get free lunch at school, but do not receive financial aid) are "falling between the cracks." These children have the highest caries and gingivitis scores as compared with the rest of the examined children.
- ☐ Category 2 children have more caries and gingivitis, and fewer filled teeth than children in category 1 whose parents' incomes are even lower, thereby allowing them to receive Medicaid coverage.
- ☐ These findings provide justification for including category 2 children with Medicaid coverage.
- ☐ There is a great need for organized oral health preventive and educational programs targeted at category 2 children.

REFERENCES

1. Johnson, S.: Advanced in dental research. Dental caries continues downward trend in children. J Am Dent Assoc, 117:625, October 1988.
2. Ismail, A.I.; Burt, B.A.; Brunelle, J.A. *et al*: Dental caries and periodontal disease among Mexican-American children from five southwestern states, 1982-83. MMWRCDC Surveill Summ, 37(SS-3):33-45, July 1988.
3. Weddell, T.A.: A socioeconomic correlation of oral disease in six to thirty-six month old children [Thesis]. Indianapolis: Indiana University School of Dentistry, 1980, pp 36-41.
4. Antoft, P.E.; Gadegaard, E.; Jepsen, P.J.: Caries experience dental health behaviour and social status: a comparative study among Danish military recruits in 1972 and 1982. Community Dent Health, 5:255-264, September 1988.
5. Bjarnason, S. and Koch, G.: Dental health in Icelandic urban children aged 11 and 12 years. Community Dent Oral Epidemiol, 15: 289-292, October 1987.
6. Samuelson, G.; Blomquist, H.K.; Crossner, C.G. *et al*: An epidemiological study of child health and nutrition in a northern Swedish country. VII. A comparative study of general and dental health, food habits and socioeconomic conditions in four-year-old children. Acta Paediatr Scand, 64:241-247, March 1975.
7. Köhler, L. and Holst, K.: Dental health of four-year old children. Acta Paediatr Scand, 62:269-278, May 1973.
8. Sreebny, L.M.: Sugar availability, sugar consumption and dental caries. Community Dent Oral Epidemiol, 10:1 -7, February 1982.
9. Dummer, P.M.H.; Addy, M.; Hicks, R. *et al*: The effect of social class on the prevalence of caries, plaque, gingivitis and pocketing in 11-12 year-old children in South Wales. J Dent, 15:185-190, October 1987.
10. Rosenblatt, G.M.; Alongi, J.A.; Deasy, M.J.: Periodontal disease in children. Clin Prev Dent, 5:17-20, May-June 1983.
11. Radike, A.W.: Criteria for diagnosis of dental caries. In: Proceedings of the conference on the clinical testing of cariostatic agents, October 14-16, 1968. Chicago: American Dental Association Council on Dental Research and Council on Therapeutics, pp 87-88.
12. DeLaRosa, M. and Sturzenberger, O.P.: Clinical reduction of gingivitis through the use of a mouthwash containing two quaternary ammonium compounds. J Periodontol, 47:535-537, September 1976.
13. Löe, H. and Silness, J.: Periodontal disease in pregnancy. Acta Odontol Scand, 21:533-551, December 1963.
14. Sehour, I. and Massler, M.: Prevalence of gingivitis in young adults. J Dent Res, 27:733-734, December 1948.
15. Hollingshead, A.B. and Redlieh, F.C.: *Social class and mental illness.* New York: Wiley, 1958, pp 387-397.
16. Hollingshead, A.B.: Two factor index of social position. Yale Station. New Haven, Connecticut, 1957.

DEMOGRAPHICS

Pediatric dentistry and national health insurance: A more than favorable opportunity

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

We must guarantee all Americans access to a comprehensive package of (health) benefits. ..."—Hillary Clinton[1]

"The number of people without health insurance reached 38.9 million last year (1992), up 2.3 million from 1991...."[2]

"Health system reform could have a profound effect on dental care in terms of how the care is financed and delivered... The impact on dentistry will be determined by the extent to which dental care is included within the guaranteed benefits package and the future tax treatment of employer provided health benefits."[3]

More than one trillion dollars will be spent for health care in 1994 (including $47.5 billion for dental services). About 15 percent of the gross domestic product will be consumed by health costs (compared to 10.4 percent in 1982 and about 12.2 percent in 1990).[4] The combination of the spiraling rise in health expenditures and the increasing numbers of uninsured in our communities (e.g. in each of four states, Louisiana, Nevada, Oklahoma and Texas, and the District of Columbia, more than one fourth of the residents are uninsured) is sparking the cry to overhaul the nation's health care system.[2] The health expenditure and uninsured data, however, "...do not necessarily support any specific proposal for extending health insurance."[2] For example, the American Academy of Family Physicians, the American Academy of Pediatrics and the American College of Physicians support the Clinton Administration's program to require employers to provide coverage for their workers. By contrast, the American Medical Association is urging Congress to consider alternatives to such an employer mandate.[2]

The American Dental Association advocates the view, "...that all employers should be required to provide basic preventive medical care and catastrophic hospital-medical-surgical care at 100 percent of cost after a family's out-of-pocket health care spending reaches 10 percent of the family's gross income. Other health coverage, the ADA says, should continue to be employment-based or purchased privately."[3]

While the eventual configuration of a national health plan is far from certain, a significant step for the future of pediatric dentistry was taken by the Clinton Administration when it included a wide range of dental services for children (including emergency, diagnostic and preventive services) in the proposed benefit package for immediate health insurance coverage (Table 1). The significance of this proposal can be considered best in terms of a review of the 27.9 million children without private dental insurance (Table 2) and the consequences of this lack of coverage.

SOURCE OF INFORMATION

The data for this presentation, for the most part, were drawn from the 1989 National Health Interview (NHI) Survey.[6*] The NHI Survey is a continuous cross-sectional national survey conducted by household interview. Each week a probability sample of households is interviewed by personnel of the U.S. Bureau of the Census to obtain information on the health and other characteristics of each member of the household. Information available from the 1989 survey of the civilian noninstitutionalized population represents data from approxi-

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*See a previous presentation in the *Journal of Dentistry for Children* for a detailed review of dental insurance coverage (between the late 1970s and the mid-1980s) and the related use of dental services by children.[8]

mately 49,000 households, including 32,357 children less than eighteen years of age.[9**]

CHILDREN WITHOUT DENTAL INSURANCE

Although there was a decrease between 1986 and 1989 in the percent of children who had no private dental insurance, in 1989:

- ☐ Six million children (54.3 percent) between two and four years of age had no private dental insurance.
- ☐ Somewhat less than a third (32.8 percent) of the more than ten million children between two and four years of age had comprehensive private dental insurance.
- ☐ Half of the children between five and eleven years of age had no private dental insurance.
- ☐ Almost 22 million children between five and seventeen years had no private dental insurance.
- ☐ Approximately 35 to 37 percent of the more than 42 million children between five and seventeen years had comprehensive dental insurance (Table 2).

Children without dental insurance were not distributed uniformly throughout the general population of youngsters. The absence of dental insurance was reported more often for:

- ☐ Younger children than older children.
- ☐ Minority children than nonminority children.
- ☐ Hispanic children than non-Hispanic children.
- ☐ Mexican-American children than other Hispanic children.
- ☐ Children residing in central sections of metropolitan areas and rural areas than children residing in the noncentral sections of metropolitan areas.
- ☐ Children residing in the South than children residing in other regions of the country.
- ☐ Poor children than nonpoor children (Table 3).

**See Gift and Newman for an extensive summary of the NHI Survey with particular emphasis on the preventive care and dental visit pattern of U.S. children.[9]

Table 1 ☐ Summary of Clinton Health Security Act dental benefits for children.[5]

1. Emergency treatment, (including simple extractions) for acute infections, bleeding and injuries to natural teeth and oral structures for conditions requiring immediate attention to prevent risks to life or significant complications, as specified by National Health Board.
2. Prevention and diagnosis of dental disease, including oral dental examinations, radiographs, dental sealants, fluoride applications, and dental prophylaxis.
3. Treatment of dental disease, including routine fillings, prosthetics for genetic defects, periodontal maintenance and endodontic services.
4. Space maintenance procedures to prevent orthodontic complications (for children between three and twelve years). Procedures limited to posterior teeth (with numerous other qualifications)
5. Interceptive orthodontic treatment to prevent severe malocclusion (covered after January 2001 and only for children between six and eleven years).

MEDICAID DENTISTRY

The review of private dental insurance coverage for children does not include Medicaid fund sources. No review of third party support for dental services would be complete without some mention of Medicaid support for the 14.6 million children (21.9 percent of all children under eighteen years of age) who in 1992 lived in poverty; including 8.9 million (16.9 percent) white children; 4.9 million (46.6 percent) African-American children; 3.1 million (39.9 percent) Hispanic children. Note: the poverty threshold for a family of four in 1992 was $14,335.[10]

Government spending for dentistry constitutes a minor component of total dental expenditures. In 1991, $1.1 billion (2.9 percent of the $37.1 billion spent for dental services) was from federal and state sources.

Table 2 ☐ Dental insurance status of children: 1986, 1989.[6,7]

	No private dental insurance			1989				
	1986	1989		Dental insurance				
Age	Percent	Percent	Number (in millions)	Comprehensive	Single service	Both	Unknown	Total
2-4 yrs	59.8%	54.3%	6.0	32.8%	6.6%	0.6%	5.8%	100%
5-11 yrs	57.2	49.7 --	21.9	35.2	8.2	1.0	6.0	100%
12-17 yrs	56.7	46.8 --		36.9	8.5	1.3	6.5	100%

Table 3 ☐ Children without dental insurance by various demographic characteristics: 1989.[6]

	Age		
	2-4	5-11	12-17
Race			
White	51.6%	47.0%	44.7%
Black	66.1	61.4	57.1
Other	61.5	57.6	48.7
Ethnicity			
NonHispanic	52.1	47.5	44.7
Hispanic	68.5	65.8	62.8
Mexican-American	73.5	67.5	66.0
Other	61.0	62.8	57.8
Place of residence			
Metropolitan Statistical Area	52.9	47.7	43.7
Central	59.6	56.2	52.8
Noncentral	48.6	42.4	38.3
Nonmetropolitan	59.2	56.5	56.6
Region			
Northeast	51.4	44.5	43.0
Midwest	49.8	47.0	43.6
South	60.1	56.2	52.7
West	52.5	46.4	44.1
Poverty level			
Below poverty threshold	88.6	86.4	84.6
At or above poverty threshold	44.8	40.6	38.9
Income			
< $10,000	91.2	89.3	87.0
$10,000–$19,999	72.2	68.4	65.9
$20,000–$34,999	49.1	45.0	45.3
$35,000+	33.4	31.1	28.5

Table 4 ☐ Federal and state expenditures for Medicaid dental services: selected fiscal years 1975–1991.[11,12]

Fiscal year	Total expenditures (in millions)	Number of recipients (in thousands)	Expenditures per recipient	Dental service index (1982-84=100)	Constant dollars: Total expenditure (in millions)	Constant dollars: Expenditure per recipient
1975	$339	3,944	$85.95	53.2	$637	$161.56
1980	462	4,652	99.31	78.9	585	125.86
1985	458	4,634	98.83	114.2	401	86.54
1991	710	5,209	136.30	167.4	424	81.42

While the $710 million dollars spent through the Medicaid program in 1991 represented about 80 percent of all government expenditures for dental services, it must be emphasized that these funds may provide the only access to dentistry for the poor.[12]

This "dental safety net" has been eroded significantly, however, since the mid 1970s. Despite the more than doubling in the expenditures for Medicaid dentistry, (from $339 million in 1975 to $710 million in 1991), in terms of constant dollar (i.e. removing the effects of inflation) total expenditures decreased by a third and spending per recipient decreased by approximately 50 percent (Table 4).

"Impact of the Reagan administration on Medicaid dentistry has been both real and subtle. At times, specific efforts to limit federal expenditures has resulted in real cutbacks... But more significant has been the setting of a climate which encourages and permits reductions of dental services for the poor, for whom dental services traditionally have been limited and for whom there are few if any alternatives."[13†]

USE OF DENTAL SERVICES AND INSURANCE COVERAGE

In 1989 (as in previous years) for all age-groups, dental insurance was a significant factor in the use of dental services. A greater percent of children with private dental insurance had reported dental visits, than their uninsured counterparts. Insured children, as compared to uninsured children, had more visits per child, a greater percent with one or more visits, and a greater percent with three or more visits. For one child in five without dental insurance, "cost" was given as the reason for no dental visit in the past year. By contrast, less than 8 percent of insured children did not visit dentists because of economic factors (Table 5).

Table 5 ☐ Children's dental insurance status by dental visit pattern: 1986.[6]

	Age: 2-4 years		5-11 years		12-17 years	
	Insurance	No Insurance	Insurance	No Insurance	Insurance	No Insurance
Number of visits per child per year	1.2	0.8	2.8	1.7	3.8	2.0
Percent with 1 or more visits in past year	43.0%	31.8%	81.8%	64.3%	82.4%	60.8%
Percent with 3 or more visits in past year	2.9%	2.4%	14.9%	10.7%	24.9%	15.6%
	2-17 years					
Reason for no dental visit in past year						
Cost	7.8%	20.5%				
No dental problem	61.4%	60.1%				

PEDIATRIC DENTISTRY AND NATIONAL HEALTH INSURANCE: A COMMENTARY

In the past, I frequently was asked by dental students whether there was a favorable future in pediatric dentistry. The query has now been extended to the thought, "What's the future for pediatric dentistry in a national health insurance setting?"

For more than a decade in scores of writings in the *Journal of Dentistry for Children* I have chronicled and stressed repeatedly the continuing and improving potential for dental practices that provide services to the increasingly diversified population of children in our communities. But my past enthusiasm pales in the light of the potential of extending services to almost 28 million children without private dental insurance (many of whom are the youngsters most in need of services[‡]) under a system of national health insurance.

[†]For a detailed presentation on the economics of Medicaid dentistry, see a previous presentation in the *Journal of Dentistry for Children*.[1,13]

[‡]For a detailed presentation on the limited availability of dental services for minority group children, see a previous presentation in the *Journal of Dentistry for Children*.[14]

More than a quarter of a century of the Medicare and Medicaid programs has taught health providers, however, the very serious consequences of government sponsored and financed health programs (and for that matter, the complexities of any of the nongovernment third-party insurance company systems). But it is this very experience that has prepared the profession to present the necessary arguments to ensure the establishment of a system that meets the needs of the profession and the children we serve.

Just imagine the prospect of millions of children who currently do not have dental insurance, but who would be eligible for care under a national health insurance program, including "(k)ey population subgroups (that) neither received optimal preventive care nor visited a dentist regularly."[9]

How would you respond to current dental students who ask about the future potential of pediatric practice in a national health insurance setting?

REFERENCES

1. Palmer, C. and Weissman, D.: Health system reform. J Amer Dent Assoc, 124:36-41, September 1993.
2. Pear, R.: Fewer now have health insurance. New York Times, December 15, 1993, p A24.
3. *Questions and Answers: Health System Reform.* Chicago: American Dental Association, 1993.
4. Pear, R.: $1 trillion in health costs is predicted. New York Times, December 29, 1993, p A12.
5. Data sheet. *Summary of Clinton Health Security Act Dental Benefit Component.* Washington, D.C.: American Association of Dental Schools, October 28, 1993.
6. Bloom, B.; Gift, H.C.; Jack, S.S.: *Dental Services and Oral Health: United States, 1989.* Vital and Health Statistics, Series 10, Data from the National Health Survey, No. 183. DHHS Pub. No. (PHS) 93-1511. Hyattsville, MD: National Center for Health Statistics, 1992.
7. Jack, S.S. and Bloom, B.: *Use of Dental Services and Dental Health: United States, 1986.* Vital and Health Statistics, Series 10, Data from the National Health Survey, No. 165. DHHS Pub. No. (PHS) 88-1593. Hyattsville, MD, National Center for Health Statistics, 1988.
8. Waldman, H.B.: Dental insurance coverage and the use of dental services by children. J Dent Child,, 56:125-128, March-April 1989.
9. Gift, H.C. and Newman, J.F.: Oral health activities of U.S. children: results of a national health interview survey. J Amer Dent Assoc, 123:96-106, October 1992.
10. Department of Commerce, Bureau of the Census; in America's Poor Showing, Newsweek, October 18, 1993, p 44.
11. Bureau of Economic and Behavioral Research. *The Consumer Price Index and Dental Services.* Chicago: American Dental Association, 1992.
12. *1992 Annual Statistical Supplement to the Soc Sec Bull.* Washington, D.C.: Government Printing Office, 1993.
13. Waldman, H.B.: Medicaid and Medicaid dentistry in the Reagan years. J Dent Child, 55:409-417, November-December 1988.
14. Waldman, H.B.: Are minority children getting their fair share of dental services? J Dent Child, 57:380-384, September-October 1990.

ADIPOSITY AND EATING STYLE

... our data provide direct support for the hypothesis that parents' dieting and child-feeding strategies influence children's eating patterns and that these parental influences mediate similarities between parents' and children's weight outcomes. Genetics may predispose an individual toward obesity, but the family environment, particularly child-feeding practices and other environmental variables such as diet composition and physical activity patterns, serves as the catalyst for expression of obesity.

Many parents assume that children are incapable of regulating their food intake. They believe that, in addition to their parental responsibility to provide healthy choices, they must control how much food their children consume. Unfortunately, our results reveal that the controlling strategies adopted by parents to meet these goals appear to be counterproductive to the development of the child's ability to self-regulate food intake.

Results from this study and from previous investigations indicate that in noncontingent, noncoercive environments, children can regulate energy intake. These results suggest that coercive child-feeding strategies commonly used to ensure adequate intake may be unnecessary and harmful. We emphasize that it is important for parents to create an optimal environment for their children's growth and health by providing a variety of nutritious foods. It should remain within the child's domain, however, to maintain control over how much of these foods are eaten.

Johnson, S.L. and Birch, L.L.: Parents' and children's adiposity and eating style. Pediatri, 94:653-661, November, 1994.

You are treating children in federal programs for child welfare, foster care, and adoption

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

Social Security...is a relatively unknown but crucial component in the public sector effort to aid children."[1]

A previous presentation in the *Journal of Dentistry for Children* reviewed the assistance provided to children under the national Old-Age, Survivors and Disability Insurance (OASDI) (generally referred to as Social Security) and the Supplemental Security Insurance (SSI) programs.[2] The OASDI and SSI expenditures of almost three billion dollars per year are used to aid more than 3.5 million children who are the survivors of retired, deceased or disabled workers or for children who themselves are blind or disabled.

The Social Security Act also contains provisions for funds that are available to states for child welfare, foster care, and adoption activities. (Note: at the federal level, there are additional programs that directly fund child welfare services, including Health Start, and programs for child abuse, homelessness, runaways and teenage pregnancy.*)

In federal fiscal year (FY) 1990, $80 billion were spent by the federal government in a variety of programs that provided benefits to children and their families; $66 billion were specifically for children—or about five percent of the total federal budget. (Note: these estimates exclude administrative costs.) The largest federal cash income support for families with children was provided under the Aid to Families with Dependent Children (AFDC) and Social Security programs. The "in-kind" transfer programs spending the most money for children and their families in 1990 were food stamps and Medicaid activities. Spending for children is projected to increase by about 75 percent to about $113 billion in 1995 with the largest component growth for the Medicaid program.

The following presentation will review the nature and extent of Social Security Act programs (other than OASDI and SSI), with emphasis on child welfare, foster care and adoption activities.** The intent of this effort is to provide dental practitioners with a general awareness of the "safety net" system that exists for many youngsters, who are reared in troubled family settings. The reality is that many of the youngsters, who receive assistance under the programs, are patients in our practices.

BACKGROUND

"It is generally agreed that it is in the best interest of children to live with their parents."[3]

Child welfare services focus on improving the conditions of children and their families and improving or providing substitution for functions the parents have difficulty in performing. While many private nonprofit and government entities provide a range of child welfare services, the primary responsibility in the governmental sector rests with the individual states. Numerous federal programs provide substantial support for a broad range of activities, however, including: health, child protection, care of homeless and neglected children, child social and

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*Except where noted, all data for this presentation were drawn from the voluminous 1993 background report prepared for the Committee on Ways and Means of the U.S. House of Representatives.[3]

**See previous reviews for discussions of other federal assistance programs for children, including Medicaid and Medicaid dentistry, foodstamps, supplemental food programs for women, infants and children (WIC), etc.[4,5]

nutritional development, and child out-of-home care.

Federal child welfare, foster care and adoption activity programs are categorized as:

- ☐ Nonentitlement authorizations: specific appropriations are made for a program, either for a fixed period of time or on a permanent basis.
- ☐ Authorized entitlements: the federal government has a binding obligation to make payments to any person or unit of government that meets the eligibility established by law.
- ☐ Block grants: services relating to child welfare may be provided at the discretion of individual states under the social services designation. Under the block grant system states have the discretion over what portions of their federal allocations they will spend on various aspects of assistance.

CHILD WELFARE SERVICE PROGRAMS

The programs permanently authorize 75 percent federal matching grants to states for services that protect children from problems that result from neglect, abuse, exploitation or delinquency. In addition, the charge to these programs is to prevent the unnecessary separation of children from their families, place children in foster homes or adoptive families. In fiscal year 1993, the federal government allocated almost $300 million for this effort.

Impact of crack cocaine on the child welfare system

The availability of crack has been linked to the abuse of children of all ages. New York City officials attribute the introduction of crack for a tripling (between 1986 and 1988) in the city's child abuse and neglect cases involving parental substance abuse. The biggest impact of crack on the child welfare system has been the large increases in very young infants entering the foster care system at birth as a result of prenatal drug usage, drug toxicity at birth, and abandonment at the time of birth in the hospital. It has been estimated that 11 percent of all pregnant women use illegal drugs.

Although the actual number of drug-exposed infants born each year is unknown, estimates range from 100,000 to 375,000. A 1989 survey of twelve cities found that 30 to 50 percent of drug-exposed infants enter foster care. New York City reported a 268 percent increase, from 1986 to 1989, in the referrals of drug-exposed infants to the child welfare system. Between 1985 and 1988, New York State foster infant care (for children less than one year of age) admissions to the welfare system increased by 89 percent. In the same period, Illinois had a 50 percent increase.

Table 1 ☐ Summary of AFDC program elements: 1970, 1992[3]

	1970	1992
Average monthly numbers (in millions)		
Families	1.9	4.8
Recipients	7.4	13.6
Children	5.5	9.2
Average family size	4.0	1.9
Benefit expenditures (in billions)	$4.1	$22.2
(in 1992 dollars)*	$14.8	$22.2
Federal share of expenditures (in billions)	$2.2	$12.2
(in 1992 dollars)*	$7.9	$12.2
Average monthly family benefit	$178	$388
(in 1992 dollars)*	$644	$388

*Adjusted for inflation
Note: Does not include foster care children or payments for foster care.

Aid to families with dependent children

Aid to Families with Dependent Children (AFDC) was established by the Social Security Act of 1935 as a cash grant program for needy children (and certain others in the household) who have been deprived of parental support or care because their father or mother is absent from the home continuously, is incapacitated, is deceased or is unemployed. The states define "need", set their own benefit levels, establish (within federal guidelines) income and resource limits and administer the program or supervise its administration. States must provide Medicaid to families receiving AFDC support. Recent changes in the federal Medicaid legislation require states to include:

- ☐ Pregnant women and children up to age six, with family incomes up to 133 percent of the poverty level (at their option, the states may extend income levels up to 185 percent of the federal poverty level).
- ☐ Children born on or after October 1, 1983, with family incomes below the federal poverty level (this provision is phased in to cover all children up to age nineteen by the year 2002). (Note: the use of the federal poverty level in determining eligibility is a critical factor. A number of states use poverty levels for determining Medicaid eligibility that are below the federal determined level. Thus, under this revised approach, increased numbers of children will become eligible for Medicaid services, including Medicaid dentistry.)

Table 2 ☐ AFDC maximum benefits (4 person family) and average monthly number of children receiving AFDC benefits: 1992, 1993[3]

	Maximum benefits	Number of children (in thousands)
	Jan. 1993	FY 1992
Alabama	$ 194	100.7
Alaska	1,025	20.7
Arizona	418	125.9
Arkansas	247	53.4
California	743	1,601.8
Colorado	432	81.5
Connecticut	792	105.1
Delaware	407	17.9
Dist. of Columbia	499	42.2
Florida	364	416.9
Georgia	330	268.3
Guam	417	na
Hawaii	835	33.7
Idaho	357	13.3
Illinois	414	471.8
Indiana	346	133.0
Iowa	495	67.1
Kansas	497	56.7
Kentucky	285	147.1
Louisiana	234	195.5
Maine	569	42.3
Maryland	432	148.8
Massachusetts	628	208.0
Michigan		440.9
Washtenaw County	593	na
Wayne County	563	na
Minnesota	621	125.1
Mississippi	144	127.7
Missouri	342	164.3
Montana	470	20.7
Nebraska	435	32.6
Nevada	407	22.3
New Hampshire	575	17.9
New Jersey	488	240.0
New Mexico	389	57.0
New York		742.6
Suffolk County	824	na
New York City	687	na
North Carolina	297	209.5
North Dakota	491	11.9
Ohio	421	489.0
Oklahoma	402	92.0
Oregon	565	76.2
Pennsylvania	514	396.6
Puerto Rico	204	na
Rhode Island	632	38.9
South Carolina	240	99.6
South Dakota	450	14.4
Tennessee	226	179.5
Texas	221	528.3
Utah	470	34.5
Vermont	740	17.7
Virginia	410	129.0
Virgin Islands	300	na
Washington	642	175.9
West Virginia	312	73.4
Wisconsin	617	164.6
Wyoming	390	12.6
Median maximum benefits for 50 states & DC	$435	
Total number of children		9,086.9

Table 3 ☐ State foster care populations and federal expenditures: selected years FYs 1988–1992,[3].

	Number of children	Federal expenditures (in millions)	Percent growth in expenditures
	FY 1988	FY 1992	FYs 1988-1992
Alabama	4,417	4.5	78%
Alaska	1,232*	4.7	na
Arizona	3,008	15.3	503
Arkansas	1,077	5.8	820
California	66,763**	418.8	246
Colorado	3,100*	18.9	649
Connecticut	3,631	15.1	492
Delaware	698	1.3	153
Dist. of Columbia	2,210	6.6	−10
Florida	7,544**	40.8	1,118
Georgia	13,325**	24.8	218
Guam	39	na	na
Hawaii	1,400	2.0	na
Idaho	700	1.3	570
Illinois	19,296**	94.9	598
Indiana	6,043	21.2	1,787
Iowa	4,012	12.3	528
Kansas	4,443	16.7	367
Kentucky	3,232	35.2	673
Louisiana	6,097	25.9	127
Maine	1,815	6.9	188
Maryland	5,868	45.5	491
Massachusetts	10,284**	45.2	853
Michigan	11,302	103.5	168
Minnesota	5,900	28.8	188
Mississippi	2,702	3.5	227
Missouri	6,902	39.7	222
Montana	na	2.9	109
Nebraska	2,296	8.7	200
Nevada	1,590	2.2	489
New Hampshire	1,445	6.5	441
New Jersey	8,798**	21.3	120
New Mexico	2,195	6.9	169
New York	52,189	675.2	362
North Carolina	6,126	11.1	451
North Dakota	589	3.8	372
Ohio	14,200**	75.2	367
Oklahoma	2,217	5.9	65
Oregon	3,885	17.9	214
Pennsylvania	15,416**	143.7	221
Puerto Rico	2,252*	na	na
Rhode Island	2,569	6.4	646
South Carolina	3,583	11.1	418
South Dakota	446	1.9	227
Tennessee	5,077	17.4	923
Texas	5,449	66.4	590
Utah	1,118	4.9	434
Vermont	1,025	6.3	186
Virginia	6,011	11.9	227
Washington	5,725	na	469
West Virginia	1,955***	4.9	−39
Wisconsin	5,018	36.1	219
Wyoming	762	1.3	788
Total U.S.		2,209.9	305%

*FY 1986 data
** FY 1989 data
*** FY 1987 data

Between 1970 and 1992:

- ☐ The number of AFDC recipients increased by 83 percent, from 7.4 million to 13.6 million.
- ☐ The number of AFDC families increased by 150 percent from 1.9 million to 4.8 million.
- ☐ The number of child recipients increased by 67 percent from 5.5 million to 9.2 million.
- ☐ The average AFDC family size decreased from 4.0 to 1.9 persons.
- ☐ AFDC benefit expenditures increased 51 percent, (after adjusting for inflation) from $14.8 billion to $22.2 billion.
- ☐ The average monthly AFDC benefit per family de-

creased 40 percent, (after adjustment for inflation) from $644 to $388 (Table 1).

In January 1993, the median state maximum benefits for an AFDC family of four was $435, ranging from $1,025 in Alaska and $835 in Hawaii, to $221 in Texas, $204 in Puerto Rico, and $144 in Mississippi (Table 2). The median state maximum benefit for an AFDC family of two was $310; $367 for a family of three; $506 for a family of five; $577 for a family of six.

In FY 1992, 9.1 million children received AFDC benefits, ranging from 1.6 million in California and 743 thousand in New York, to 12.6 thousand in Wyoming and 12 thousand in North Dakota (Table 2).

AFDC foster care

This program permanently authorizes funds to states for the maintenance payments for AFDC children in foster care family homes, private nonprofit child care facilities or public child care facilities with less than twenty-five children. State expenditures are matched by the federal government at between 50 percent to 83 percent, depending upon state per capita income. Between FY 1982 and FY 1992 the estimated number of children enrolled in the AFDC foster care program more than doubled from 93,000 to 222,315. (Note: in 1991, approximately 200,000 additional children who were not from AFDC eligible homes were in foster care arrangements. At the end of 1991, it is estimated that there was a total of 429,000 children in foster care settings, with a total of 636,000 children having received foster care some time during the year.)

While the states of California and New York led the country in the late 1980s with the greatest number of children in foster care, (67 thousand and 52 thousand, respectively) foster care is provided to children in all states (Table 3).

States are permitted to set the basic monthly rate. In FY 1992, the average basic monthly rate for a sixteen-year-old foster child was $385, ranging from $621 in Alaska and $572 in Connecticut to $234 in Alabama. California and New York accounted for almost a half of the estimated expenditures. The nationwide monthly average rate for two-year-old foster children was $311, and $332 for nine-year-old children. The total federal expenditure for the program was $2.2 billion, ranging from $675 million in New York and $418 million in California to $1.3 million in each of three states, Delaware, Idaho, and Wyoming (Table 3).

Between FY 1985 and FY 1992 federal expenditures increased by 305 percent, ranging from an increase of 1,787 percent in Indiana and 1,118 percent in Florida to decreases of 10 percent in the District of Columbia and 39 percent in West Virginia (Table 3).

Independent living program

The program was established to assist adolescent youths who are to be emancipated from the foster care program. Funds are used to assist sixteen-year-olds to make a successful transformation from foster care to independent adult living, when they become ineligible for foster care maintenance payment at eighteen years of age. In FY 1992, $70 million in federal funds were appropriated for 90,000 eligible youths.

Adoption assistance program

"Only 2 percent of unmarried mothers placed their children for adoption in the 1980s, down from 9 percent some fifteen years earlier."

Table 4 ☐ Finalized adoptions and children awaiting adoptive placement: FY 1989.[3]

Age	Finalized adoptions	Awaiting adoption
0-1 yr.	3.6%*	3.1%*
1-5 yrs.	48.0	32.7
6-12 yrs.	36.6	42.5
13-18 yrs.	9.1	20.5
19+ yrs.	.2	1.1
unknown	.1	.1
Race/ethnicity		
White	54.9**	47.0***
Black	28.9	42.2
Hispanic	10.2	6.6
Other	3.8	2.5
Unknown	2.2	1.7
Special needs status		
1 or more special needs	63.7	63.6
No special needs	35.7	26.1
Unknown	.6	10.3
Time awaiting adoptive placement****		
0-6 months		22.7
6-12 months		14.2
1-2 yrs.		16.3
2 or more yrs.		45.3
Unknown		1.5

*Data provided by 23 states
**Data provided by 28 states
***Data provided by 27 states
****Data provided by 19 states

Table 5 ☐ Proportion of special needs children in foster care, awaiting adoption and adopted: 1984, 1989.[3]

	1984	1989
Number of children in foster care	276,000	383,000
Percent with special needs	22%	17%
Number of foster care children awaiting adoption	17,000	20,000
Percent with special needs	43%	64%
Number of foster children adopted	20,000	16,000
Percent with special needs	57%	64%

"Only 50,000 U.S. children become available each year for nonrelatives to adopt; half of these are healthy infants."

"In 1991, 36,000 babies were born to unmarried girls age fifteen and under."

"In June 1992, 442,000 children were in foster care, a 68 percent jump from 1982."

"Prospective parents have to wait at least two years on average to adopt. Some pay fees of $50,000 to more than $100,000 to get babies through private adoption."[6]

The adoption assistance effort is an open-ended entitlement program required of states that participate in AFDC (all states participate). The states develop assistance agreements with parents who adopt eligible children with special needs.†

In FY 1992, $220 million in federal funds for adoption assistance were requested. There was an average of 66,300 children per month under the program, who required some form of substitute care.‡

The composition of children awaiting adoption is somewhat different from children whose adoptions have been finalized. Children awaiting adoption generally are older and include a greater percent of black children. In addition, of the children awaiting adoption, 45 percent had been waiting for two or more years (Table 4).

Of the adoptions that were finalized in FY 1989, the two largest age-groups of children were between one and five years of age (48 percent) and between six and twelve years of age (37 percent). The majority of children (55 percent) were white, while 29 percent were black. Sixty-four percent of these children had one or more special needs that could have posed barriers to adoption (Table 5).

FROM THE PERSPECTIVE OF THE DENTIST

In the early 1990s, there are approximately 66 million children:

- ☐ 14.3 million children (21.8 percent of all children) are living in poverty.
- ☐ 5.7 million children (24.6 percent of the children less than six years of age) are living in poverty.
- ☐ 8.6 million children (20.2 percent of children between six and seventeen years) are living in poverty.
- ☐ 4.8 million African-American children (46 percent of all African-American children) are living in poverty.
- ☐ 3.1 Hispanic children (40 percent of all Hispanic children) are living in poverty.
- ☐ More than nine million children live in AFDC families.
- ☐ During any single year more than 635,000 children live in foster home settings that receive federal support funds.

The vast array of federal programs under the umbrella of the Social Security Act provide a safety-net for the evolving landscape of family settings in which our children are being reared. Given the scope of the problem, few dental practices would not come in contact with at least some of these children.

As dentists treat increasing diverse populations of children, they are confronted by the reality that "there is no such thing as a typical family."[7] By now most practitioners are aware of the need to extend their initial medical and dental patient history reviews to consideration of the general setting within which their pediatric patients are being reared. A general understanding of government programs to assist millions of children in need provides a further dimension to an appreciation of the continuing complex world of today's children (who happen also to be our patients).

†A special need child is defined as one whom the state determines is in a special condition or situation, such as age, membership in a minority or sibling group, or a mental, emotional or physical handicap that prevents placement without special assistance.

‡Substitute care is defined as a living arrangement in which children are residing outside of their own homes under the management and responsibility of the state child welfare agency. Living arrangements may include foster care or adoptive foster care, group homes, child care facilities, emergency shelter, supervised independent living, etc.

REFERENCES

1. Security for America's Children: a report from the Annual Conference of the National Academy of Social Insurance. Soc Sec Bull, 55:57-62, Spring 1992.
2. Waldman, H.B.: Social Security and providing for our children. J Dent Child, 61:57-61, January-February 1994.
3. Overview of Entitlement Programs: 1993 Green Book. Committee on Ways and Means, U.S. House of Representatives, 103 Congress. Washington, D.C.: Government Printing Office, 1993.
4. Waldman, H.B.: Is Medicaid dentistry worth the effort? Compend of Cont Educ in Dent, 12:515-518, July 1991.
5. Waldman, H.B.: Public assistance—general information for the dental practitioner. Annals of Dent, 43:34-40, Spring 1984.
6. Where the kids are. Newsweek, March 21, 1994, p 65.
7. Waldman, H.B.: There is no such thing as a typical family. J Dent Child, 58:310-313, July-August 1991.

TOMOGRAPHY

Computed tomography in the management of impacted teeth in children

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Impaction is a common developmental anomaly of the mixed and permanent dentitions.[1,2] Although any tooth may be impacted, the maxillary and mandibular third molars and maxillary canines are the most frequent ones, followed by the premolars and supernumerary teeth.[3,4] Impaction of primary teeth is less common.[5] Radiography is the only accurate procedure in a clinical examination for detection and diagnosis of impacted teeth. The necessity and timing for surgical intervention are also based on radiographic findings. Radiographs not only make it possible to detect impacted teeth, but also provide information regarding morphology, inclination, and relation of the impacted tooth to adjacent structures. In addition, the existence of complications or predictions of risk of their later appearance can also be made using radiographs.

Since conventional (plain film) radiographs (periapical, occlusal, and panoramic) are a two-dimensional image of a three-dimensional object, their use is limited for diagnostic purposes. When plain film radiographs are used for a three-dimensional analysis, at least one additional radiograph perpendicular to the plane of the first radiograph, or two different angulated views are necessary.[6] The use of such radiographs cannot always precisely determine the true morphology and relationship of an object in all dimensions, or overcome the misleading effect of superimposition.

Computed tomography (CT) has become more widely available and is recognized as an important diagnostic tool for complex conditions in the oral region.[7] Little has been written, however, regarding its value in diagnosis and treatment planning of impacted teeth in children.

The purpose of this article is to evaluate, retrospectively, the contribution of CT in the diagnosis and management of impacted teeth in children.

MATERIALS AND METHODS

During 1991 to 1992, nine children, ages ten to sixteen, with eighteen impacted teeth were referred for consultation regarding impacted teeth. All patients underwent panoramic or intraoral radiographic examination, as well as CT examination before the consultations. CT scans were conducted, using an Elite 2400 Scanner (Elscint Co., Haifa, Israel) with a Denta Scan software.[8] As the panoramic, intraoral, and CT scans were already available, it was interesting to compare them. The following factors were evaluated: the morphology of the impacted tooth, the inclination and proximity of the tooth to adjacent teeth, the nasal cavity, the maxillary sinus or mandibular canal were estimated on both conventional radiographs and on CT scans by the same investigator, and recorded with an accuracy of 1 mm. Diagnostic quality of the two types of images was scored as optimal visualization (+++), adequate (++), poorly diagnostic (+), or unacceptably poor (−) for all impacted teeth, as previously described by Kashima *et al.*[9]

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Table 1 □ Impacted teeth diagnosed by CT examination.

Age	Gender	Jaw	Tooth type
11	M	Maxilla	Central incisor mesiodens
10	F	Maxilla	1st premolar 2nd premolar
15	M	Maxilla	1st premolar
11	F	Maxilla	Central incisor mesiodens odontoma
16	M	Maxilla	1st premolar 2nd premolar
		Mandible	1st premolar
14	F	Maxilla	1st premolar 2nd premolar
12	F	Maxilla	Canine 1st premolar
14	M	Mandible	Canine
11	F	Maxilla	Central incisor mesiodens

RESULTS

The age and gender of the children, and the impacted teeth evaluated are shown in Table 1.

Morphology of impacted tooth

The morphology of crown and root, stage of root development, number of roots and their divergences were poorly shown on plain film radiographs but were clearly demonstrated on CT scans (Figures 1A-C, 2A-C, 3A-C).

Inclination of impacted tooth

The buccolingual inclination of the long axis of the tooth, dilaceration, an angulation between the root and crown, were undetected on plain film radiographs, whereas CT scans clearly showed those anomalies (Figures 1B, 3A).

Position and relation to adjacent structures

The proximity of the impacted tooth to neighboring teeth, maxillary sinus or mandibular canal should be assessed for decision-making regarding treatment planning for the impacted tooth. These vital structures are often superimposed on the impacted tooth and cannot, therefore, be always clearly demonstrated on plain film radiographs. CT scans, however, by reconstructing the image in three-dimension, were found to be very accurate for this purpose (Figures 2C, 3C, 4A-C, 5A-B).

The scores assigned to the diagnostic qualities of the radiographic features of impacted teeth interpreted from plain film radiographs as compared with CT scans are shown in Table 2. Both plain film radiographs and CT scans allowed identification of the presence of impacted teeth in the maxilla or mandible. The morphology, inclination, and the relation of the impacted tooth to adjacent structures were poorly-delineated, however, by plain film radiographs, whereas CT provided this information with greater clarity.

DISCUSSION

Several complications, such as dentigerous cysts, retarded eruption of other teeth, and resorption of adjacent teeth may result from teeth that have been impacted for a long period. Tumors may also be observed in association with impacted teeth.[6,10-12] Radiographs of the impacted teeth are of utmost importance, therefore, for early recognition of such complications. As superimpositions of anatomic structures frequently oc-

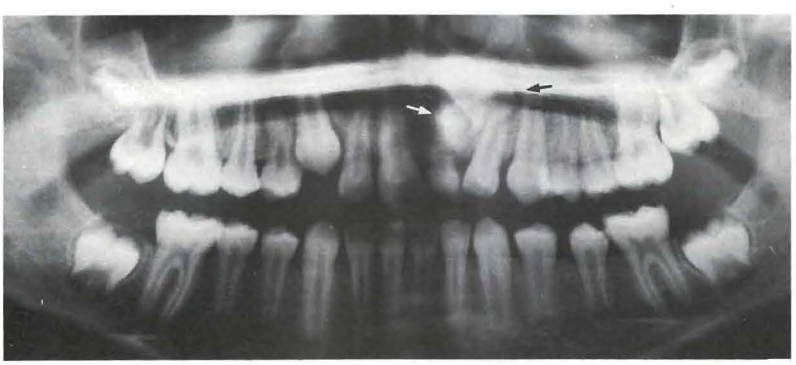

Figure 1A. An eleven-year-old girl with a retained tooth #61 was referred for surgical exposure of impacted tooth #21. Panoramic radiograph shows that apically of tooth #61 there is an amorphous radiopaque mass partially superimposed on the crown of tooth #21. The morphology of tooth #21 is unclear.

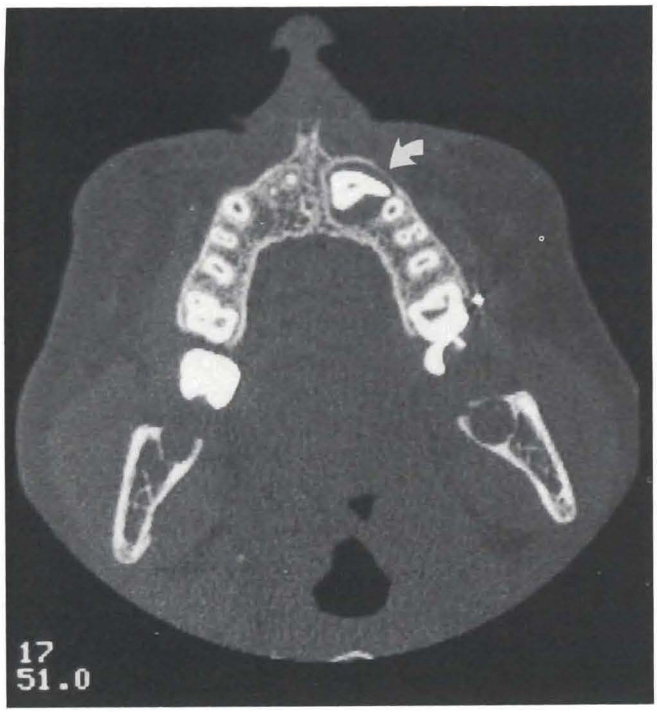

Figure 1B. Axial CT scan demonstrates the distal inclination of the crown of tooth #21. No root can be seen.

cur, it is difficult or impossible to obtain adequate diagnostic images through conventional radiography. In these instances CT scans should be used to obtain a valid diagnosis.

The treatment strategy for impacted teeth can be a conservative approach with periodic radiographic follow-up, surgical exposure followed by guided eruption, or surgical extraction.

Asymptomatic impacted teeth without signs of complication and with minor risk for their later appearance may be left in the jaws. Periodic radiographic follow-up is mandatory to detect changes early.[13] In cases with a good prognosis for guided eruption and orthodontic treatment, surgical exposure should be performed. CT analysis to evaluate crown and root morphology, buccolingual inclination, and the relation to adjacent structures may provide important information for treatment planning. Once the decision to remove the impacted tooth has been made, the surgical procedure should be carefully planned. CT analysis can contribute important and accurate information and can reduce the frequency and severity of postoperative complications.[6,13,14]

The optimal time to remove surgically an impacted tooth is highly controversial. There are two schools of

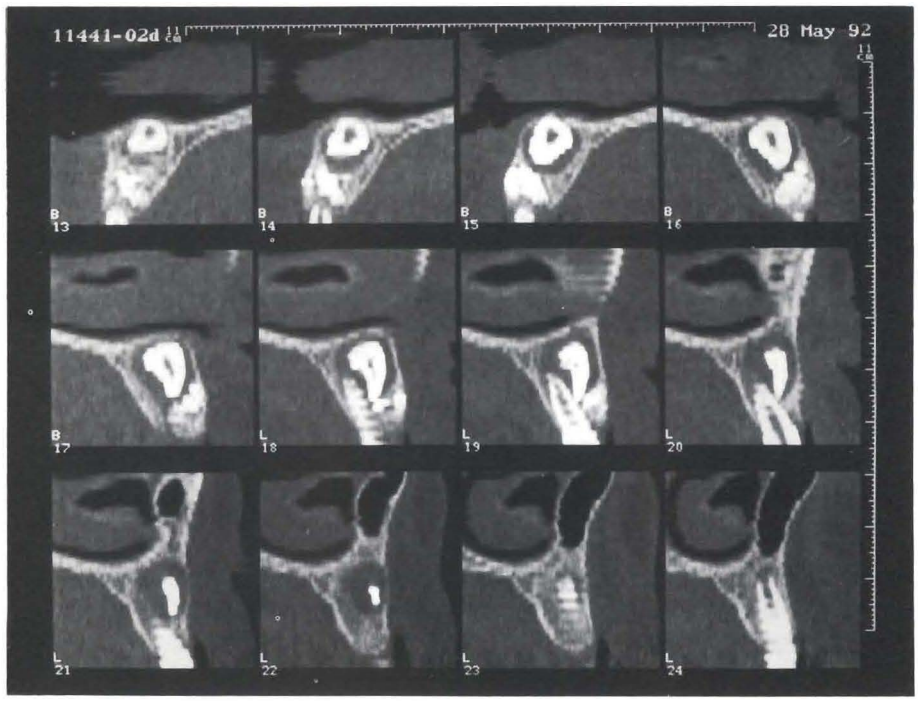

Figure 1C. CT reconstruction of the maxilla at a buccopalatal plane demonstrates in serial cuts that tooth #21 has only crown and no root.

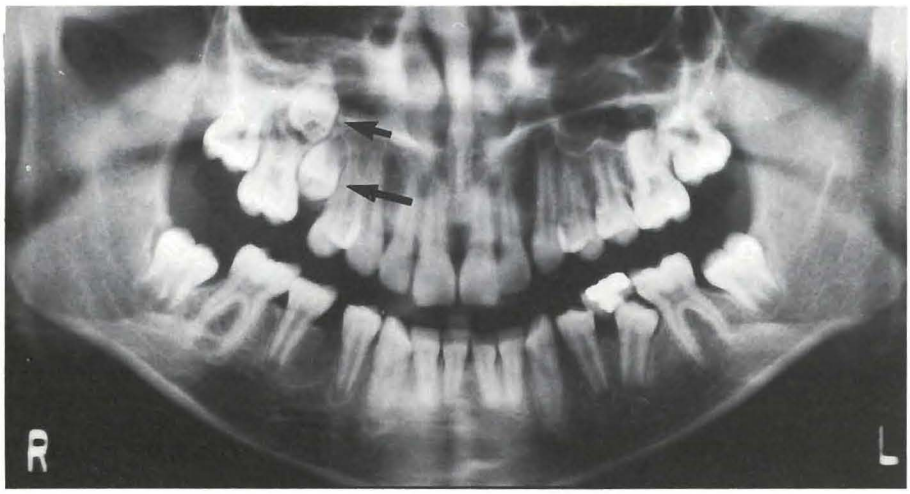

Figure 2A. Unerupted tooth #15 in a fourteen-year-old girl. Panoramic radiograph shows two impacted permanent second premolars one above the other. The precise morphology of their crowns and roots is unclear.

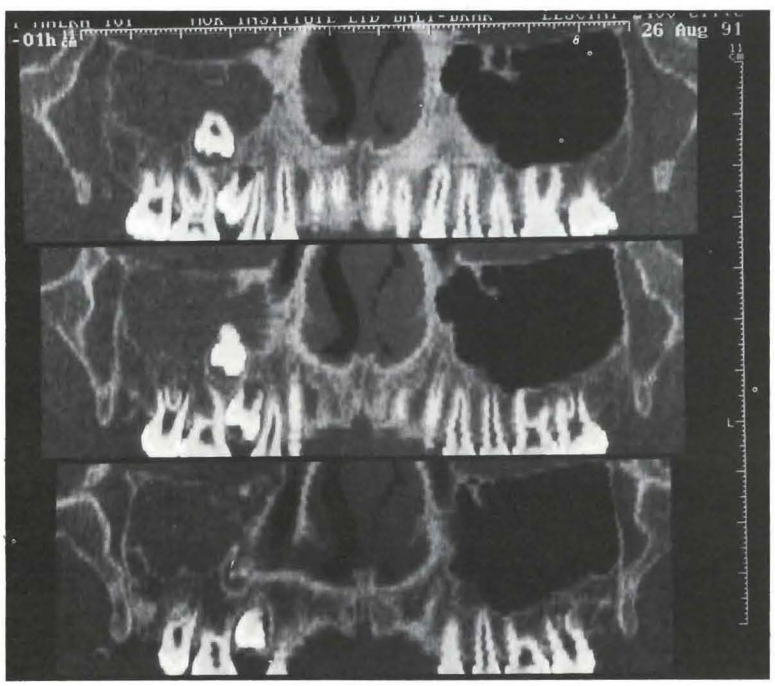

Figure 2B. Panoramic view scans demonstrate that the lower impacted tooth is in a stage of root development. The upper tooth has a closed apex adjacent to the crowns and no further roots formation can be anticipated.

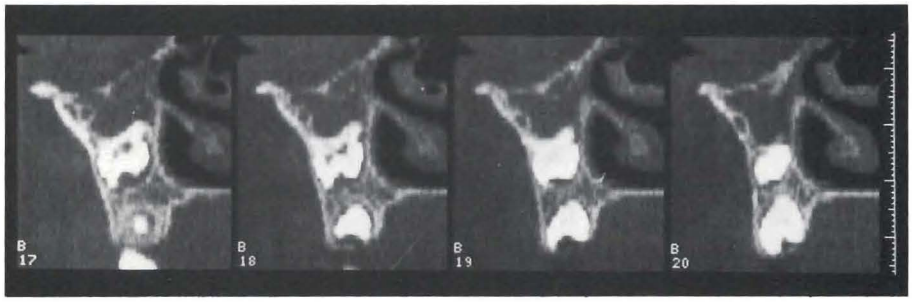

Figure 2C. CT reconstruction of the maxilla at a buccopalatal plane, further demonstrates that the upper tooth has no root and is close to the buccal cortical bone plate, allowing safe surgery from a buccal approach.

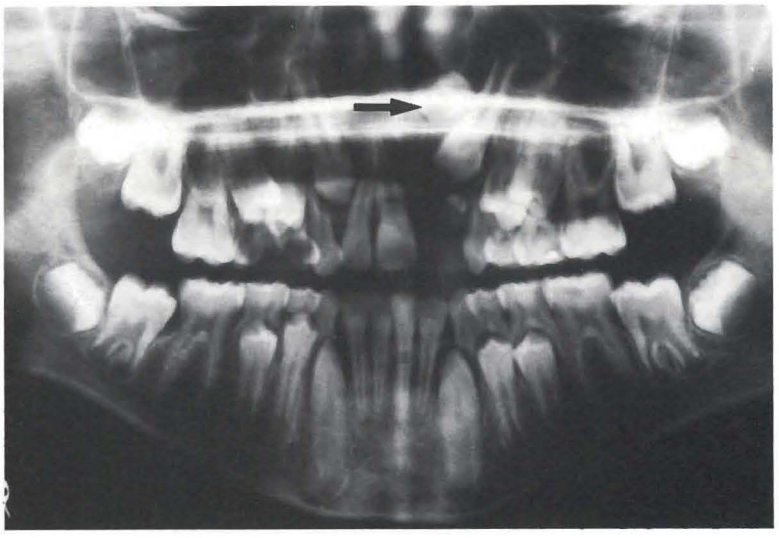

Figure 3A. Unerupted teeth #21 and #22 in an eleven-year-old boy. Teeth #61 and #62 were traumatically avulsed at age five. Panoramic radiograph demonstrates a tooth fragment mesial to tooth #63. Tooth #23 is inclined mesially, and at 90° to it, there is another radiopaque structure (arrow).

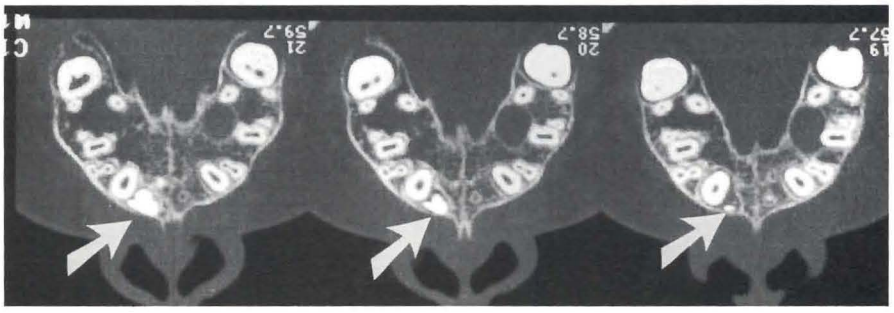

Figure 3B. Axial CT scans demonstrate that mesial to tooth #23 a crown-like structure (perhaps of tooth #21) inclined distally can be observed (arrows).

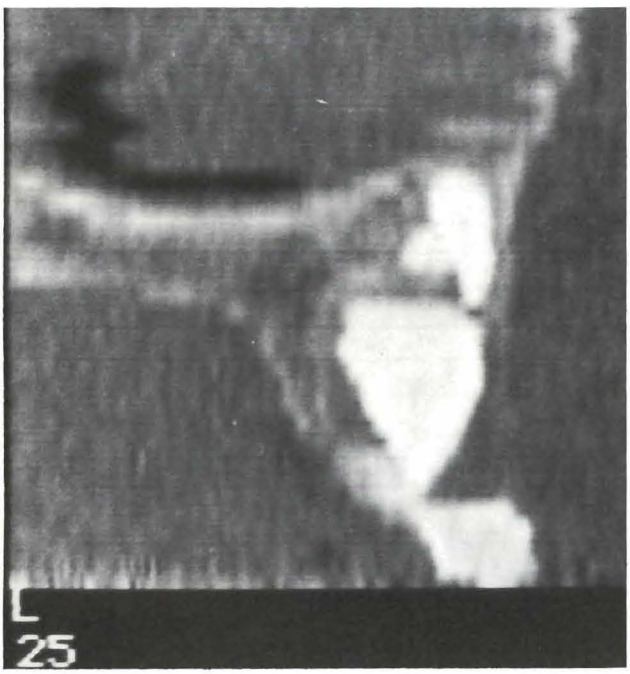

Figure 3C. CT reconstruction of the maxilla at the buccopalatal plane shows that tooth #21 has only a crown and no root. Palatal to the crown there is another round tooth-like structure. The crown is adjacent to, but separated from the nasal cavity and close to the buccal bone plate, allowing safe surgery from a buccal approach.

thought: in the delayed approach, intervention is recommended upon apical maturation of the adjacent teeth in the immediate approach, teeth are removed shortly after initial diagnosis of their presence.[4,15] With either approach, CT scan can be useful since it determines apical maturation more accurately and allows detection of potential surgical hazards with early intervention.

Similar to our results on impacted teeth in children, CT was found superior to plain film radiographs in adults in:

- ☐ The demonstration of the extent of benign tumors of the jaws;
- ☐ Evaluation of the postoperative maxillary cysts.
- ☐ The diagnosis of pseudocysts of the mandibular condyle;
- ☐ The differential diagnosis of submandibular lesions;
- ☐ Planning endosseous implant surgery;
- ☐ Diagnosis and treatment planning of cystic jaw lesions;
- ☐ The early detection of root resorption of the lateral maxillary incisor in children due to ectopically erupting maxillary canines.[8,16-22]

In the cases examined in this retrospective report, CT scans provided information, over and above that available from plain film radiographs, that was relevant and

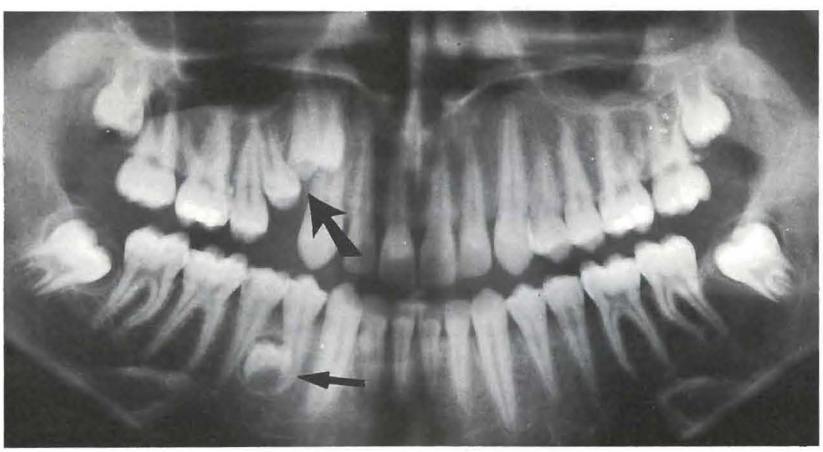

Figure 4A. Unerupted tooth #14 in a fifteen-year-old boy. Panoramic radiograph shows two impacted first premolars in the right maxilla. The mesial tooth is planned for extraction, following determination of its proximity to the nasal cavity, maxillary sinus, and cortical bone plate. Note additional anomaly in the right mandible.

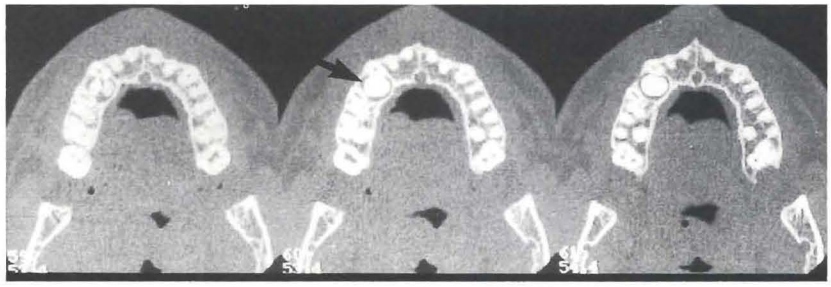

Figure 4B. Axial CT scans show that the crown of the mesial tooth is between the buccal and palatal bone plates (arrow).

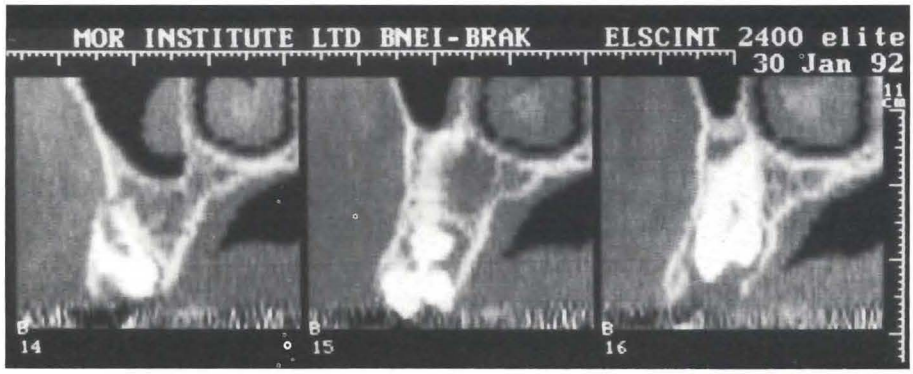

Figure 4C. CT reconstruction of the maxilla at the buccopalatal plane clearly demonstrates that the mesial tooth is adjacent to, but separated from the nasal cavity and maxillary sinus, and is close to the buccal cortical bone plate, allowing safe surgery from a buccal approach.

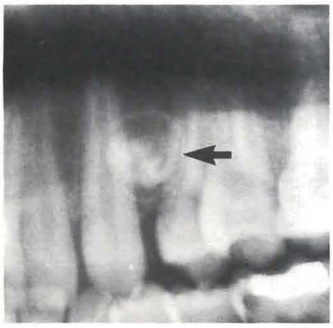

Figure 5A. Supernumerary tooth affecting the left maxilla of a fifteen-year-old boy. Panoramic radiograph shows a supernumerary tooth or odontoma distal to tooth #23. The buccopalatal localization is unclear.

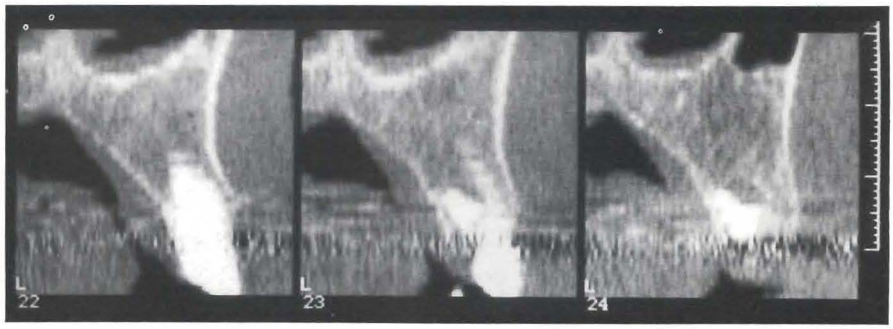

Figure 5B. CT reconstruction of the maxilla at the buccopalatal plane clearly demonstrates that the supernumerary tooth is located palatally, indicating that the surgery should be by a palatal approach.

Table 2 ☐ Radiographic features of impacted teeth interpreted from plain film radiographs and CT scans.

Features of impacted teeth	Plain film radiography	Computed tomography
Morphology	+	+++
Inclination	++	+++
Relation to adjacent teeth	+	++
Relation to nasal cavity	−	+++
Relation to maxillary sinus	+	+++
Relation to mandibular canal	+	+++
Relation to cortical jawbone	++	+++

Scale: +++ = Optimal visualization
++ = Adequate
+ = Poorly diagnostic
− = Unacceptably poor

important for the management of the patients. CT scans were beneficial for comprehensive evaluation and for treatment planning of impacted teeth, especially in the crowded field of the mixed dentition. Correct surgical approach and precise performance were able to shorten the surgical period, which was advantageous to the child.

Radiation risk from panoramic or intraoral radiographs is lower than from CT scans.[23,24] Thus, in prospective cases the radiation risk should be weighed against the diagnosis and surgical benefits of precise preoperative management of the impacted tooth. CT scans in children should be limited and used only where additional three-dimensional demonstration is essential for diagnosis and treatment planning. Patient selection criteria for the use of CT in children are very important and they should be established for the benefit and safety of the child.

REFERENCES

1. Kramer, R.M. and Williams, A.C.: The incidence of impacted teeth. Oral Surg, 29:237-241, February 1970.
2. Little, J.J.: Indications and contra-indications for removal of the impacted tooth. Dent Clin North Am, 23:333-346, January-February 1979.
3. Grover, P.S. and Lorton, L.: The incidence of unerupted permanent teeth and related clinical cases. Oral Surg, 59:420-425, April 1985.
4. Primosch, R.E.: Anterior supernumerary teeth-assessment and surgical intervention in children. Pediatr Dent, 3:204-214, April 1980.
5. Bodner, L. and Horowitz, I.: Impacted primary incisor: Report of a case. J Dent Child, 54:363-364, September-October 1987.
6. Sewerin, I.P.: Radiology in management of impacted teeth. Int Dent J, 37:25-30, March 1987.
7. Gibbs, S.J.: Comparative imaging of the jaws. Oral Maxillofac Infect, 2:55-63, January-February 1992.
8. Schwartz, M.S.; Rothman, S.L.G.; Rhodes, M.L. *et al*: Computed tomograph. Part I: Preoperative assessment of the mandible for endosseous implant surgery. Int J Oral Maxillofac Implant, 2:137-141, March-April 1987.
9. Kashima, I.; Tajima, K.; Nishimura, K.: Diagnostic imaging of disease affecting the mandible with the use of computed panoramic radiography. Oral Surg Oral Med Oral Pathol, 70:110-116, July 1990.
10. Franklin, T.; Pange, A.; Yuen, S.: Unerupted maxillary anterior supernumerary teeth: Report of 204 cases. J Dent Child, 51:289-294, July-August 1984.
11. Lustman, J. and Bodner, L.: Dentigerous cysts associated with supernumerary teeth. Int J Oral Maxillofac Surg, 17:100-102, April 1988.
12. Eliasson, S.; Heimdahl, A.; Horderman, A.: Pathological changes related to long-term impaction of third molars. Int J Oral Maxillofac Surg, 18:210-212, August 1989.
13. Koch, H.; Schwartz, O.; Klausen, B.: Indications for surgical removal of supernumerary teeth in the premaxilla. Int J Oral Maxillofac Surg, 15:273-281, June 1986.
14. Van Gool, A.V.; Ten Bosch, J.J.; Boering, G.: Clinical consequences of complaints and complications after removal of mandibular third molars. Int J Oral Surg, 6:29-37, February 1977.
15. Barren, M.V.: Surgical treatment of an unerupted supernumerary tooth attached to an unerupted permanent incisor. Pediatr Dent, 5:83-84, February 1983.
16. Cohen, M.A. and Mendelsohn, D.B.: CT and MR imaging of myxofibroma of the jaws. J Comput Assist Tomogra, 14:281-285, May-June 1990.
17. Pe, M.B.; Sano, K.; Kitamura, A. *et al*: Computed tomography in evaluation of postoperative maxillary cysts. J Oral Maxillofac Surg, 48:679-684, July 1990.
18. Friedlander, A.H.; Manson, M.L.; Friedlander, M.D. *et al*: Pseudocysts of the mandibular condyle. J Oral Maxillofac Surg, 50:821-824, August 1992.
19. Kurabayashi, T.; Ida, M.; Sasaki, T.: Differential diagnosis of submandibular cystic lesions by computed tomography. Dentomaxillofac Radiol, 20:30-34, January 1991.
20. Bodner, L.; Bar-Ziv, J.; Kaffe, I.: Computed tomography of cystic jaw lesions. J Comput Assist Tomogra, 18:22-26, January, 1994.
21. Ericson, S. and Kurol, J.: CT diagnosis of ectopically erupting maxillary canines: A case report. Eur J Orthodont, 10:115-120, May 1988.
22. Peene, P.; Lamoral, Y.; Plas, H. *et al*: Resorption of the lateral maxillary incisor: Assessment by CT. J Comp Assist Tomogra, 14: 427-429, May-June 1990.
23. Huda, W. and Sandison, G.A.: The use of the effective dose equivalent as a risk parameter in computed tomography. Br J Radiol 59:1236-1238, December 1986.
24. Gibbs, S.J.; Pujol, A.; McDavid, W.D. *et al*: Patient risk from rotational panoramic radiography. Dentomaxillofac Radiol, 17:25-32, January 1988.

REVIEW

Relationship between oral parafunctions and craniomandibular dysfunction in children and adolescents: A review

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The etiology of craniomandibular dysfunction (CMD) in children and adolescents is considered multifactorial.[1,2] Trauma, emotional states, malocclusion, and oral parafunctions are the known etiologic factors. In a previous review study, it was reported that malocclusion cannot be either "necessary" or "sufficient" cause of signs and symptoms of CMD.[3] It would be useful to know, therefore, whether such a relationship exists between oral parafunctions and CMD.

The purpose of this paper is to review the literature with respect to the relationship between oral parafunctions and signs/symptoms of CMD and to discuss their causal relationship by applying the epidemiologic approach of "necessary" and/or "sufficient" etiologic factor.

LITERATURE REVIEW

Table 1 presents most of the relevant information provided by each study. Of the reviewed studies, two were longitudinal while the others were cross-sectional.[4,5] CMD was defined in all studies by the presence or absence of one or more signs or symptoms recorded in each investigation. Although the criteria used to identify CMD signs were similar in the reviewed studies, differences in the examination technique are expected. Hardison and Okeson reported that different techniques for recording joint sounds reveal different findings even in the same patient.[6]

The investigations by Egermark-Eriksson *et al*, Lindqvist, and Kampe and Hannerz were conducted by one examiner.[5,7,8] Wigdorowicz-Makowerowa *et al*, did not report the number of the examiners, while two or more examiners participated in the rest of the studies.[9] The information regarding signs/symptoms of CMD was collected by a clinical examination in the studies by Lindqvist, Wigdorowicz-Makowerowa *et al*, and Goho and Jones, while Nilner used a clinical examination and an interview.[8-12] Egermark-Eriksson *et al*, Magnusson *et al*, Gazit *et al*, Kritsineli and Shim, and Kampe and Hannerz obtained the information by a clinical examination and a questionnaire.[4-6,13,14] Reliability tests were performed by Egermark-Eriksson *et al*, Nilner, Goho and Jones and reported acceptable inter- or intraexaminer variability.[7,10-12] In two studies the examiners had trained on patients before the beginning of the investigation.[11,12]

The oral parafunctions recorded in the reviewed studies were bruxism (grinding and/or clenching), lip/cheek biting, tongue biting, nail biting, biting on foreign objects, and thumbsucking. Bruxism was recorded in all studies, while the rest of oral parafunctions were included in the questionnaires of the investigations by Egermark-Eriksson *et al*, Nilner, Kritsineli and Shim, Kampe and Hannerz, and WigdorowiczMakowerowa *et al*. Bruxism was recorded by interview in the studies by Nilner, while a questionnaire was used in the investigations by Egermark-Eriksson *et al*, Kritsineli and Shim, Kampe and Hannerz, and Magnusson *et al*.[4,5,7,11,12,14] Lindqvist diagnosed bruxism clinically and on plaster models by recording bruxofacets of the permanent teeth. The diagnosis of bruxism in the rest of the studies was performed clinically using wear facets or occlusal wear as criteria of the presence or absence of this parafunction.

The studies by Lindqvist, Gazit *et al*, Goho and Jones, Kritsineli and Shim, and Wigdorowicz-Makowerowa *et*

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al recorded the nocturnal bruxism. Kampe and Hannerz measured the diurnal one while Egermark-Eriksson *et al*, Nilner, Magnusson *et al* measured both diurnal and nocturnal bruxism. Because nocturnal bruxism occurs during the night, individuals have no awareness of this parafunction.[15] It is likely that the recorded frequency of bruxism by an interview or a questionnaire, therefore, is underreported. Bruxofacets or occlusal wear has been considered as an objective indicator of measuring bruxism.[8,10] Although this variable can be measured objectively, it may not indicate the subject's current level of bruxism.[16] Subjects to bruxism may exhibit facets, even if the habit no longer exists, while subjects who have recently begun the practice may not show signs of occlusal wear. Also, dental wear can be caused by many factors other than bruxism.[17] Bruxofacets or occlusal wear is not necessarily synonymous, therefore, with current bruxism status and current symptoms not correlated with bruxofacets may be associated with bruxism. Contrary to bruxism, the other oral parafunctions recorded in the reviewed studies hit consciousness immediately, and it is most likely that the reported frequency reflects the true prevalence of these activities. Of the reviewed studies only two recorded how frequently oral parafunctions occurred in the subjects.[4,7] The results were reported, however, without taking into consideration this factor. In addition none of the studies recorded the duration and intensity of these parafunctional activities.

The correlations between oral parafunctions and CMD were carried out between each sign/symptom and each type of oral parafunction, the Helkimo's clinical and anamnestic dysfunction indices and each type of oral parafunction, and finally one study tested the difference in the frequency of signs/symptoms of CMD between children with and without bruxism.[4,5,7-14,18] Kritsineli *et al* and Wigdorowicz-Makowerowa *et al* did not report the significance level, while the 95 percent probability was used in all other studies.[9,14]

Table shows the correlations between signs or symptoms of CMD and oral parafunctions. All studies but one found highly significant correlations between signs and/or symptoms and different types of oral parafunctions, irrespective of the method used to record parafunctions.[10] More specifically, bruxism was correlated with clinical dysfunction index, the anamnestic dysfunction index, muscle tenderness, pain in temple region, joint sounds, recurrent headaches, and condylar displacement.[4,5,7,8,11-14] Lip/cheek/tongue biting was correlated with recurrent headaches, pain in temple region, anamnestic dysfunction index, and subjective symptoms,

Table ☐ Studies on the relationship between oral parafunctions and signs and symptoms of craniomandibular dysfunction.

Investigator	Sample size M	Sample size F	Age (yrs)	Correlations: Type of parafunction	Correlations: Signs/symptoms	Correlations: P-value
Egermark-Eriksson et al 1981	136 131 135		7 11 15	bruxism nail/lip/cheek/tongue biting biting on foreign object	Helkimo's index signs/symptoms	<.01 NR
Lindgvist 1974	117 pairs of twins		10.8–14.1	bruxism	muscle tenderness	<.001
Wigdorowicz-Makowerowa et al 1979	2100		10–15	statistically significant difference in the frequency of CMD between children with and w/out bruxism		NR
				tooth-foreign body tooth-mucous membrane	signs/symptoms	NS
Nilner 1983a	222	218	7–14	bruxism	pain in temple region muscle tenderness	.01 < p <.05 .01 < p <.05
				nail/lip/cheek biting	pain in temple region recurrent headaches	<.001 <.001
Nilner 1983b	147	162	15–18	bruxism	pain in temple region recurrent headaches	<.001 <.001
				nail/lip/cheek biting	signs/symptoms	>.05
Gazit et al 1984	188	181	10–18	bruxism	joint sounds	.03
Magnusson et al 1985	66 53		11 15	bruxism nail/lip/cheek/tongue biting biting on foreign object	subjective symptoms	<.01
Goho and Jones 1991	100		3–6	bruxism	signs/symptoms	>.05
Kampe and Hannerz 1991	29	35	19–21	bruxism tongue/cheek biting	headache anamnestic dysfunction index	<.001 <.001
Kritsineli and Shim 1992	48	32	primary & mixed dentition patients	bruxism	clicking condylar displacement	NR
				thumb-sucking	clicking condylar displacement	NR

Notes: NS = Not Significant NR = Not reported

while nail biting was correlated with recurrent headaches, pain in temple region, and subjective symptoms.[4,5,7,11,12] Finally, one study reported correlation between thumb sucking and clicking and condylar displacement.[14]

DISCUSSION

The reviewed epidemiologic studies investigated the causal relationship between different types of oral parafunctions and signs/symptoms of CMD. As it has been pointed out, the clinical examination methods as well as an interview or a questionnaire are not as accurate, however, as the experimental methods.[7,11,12,19] The majority of the reviewed studies did not report a reliability test, while those that did perform one reported acceptable inter- or intraexaminer variability. In addition, muscular hyperactivity was recorded by interview, questionnaire, or bruxofacets; and the reviewed studies, although suggestive, are limited in that they did not measure actual levels of muscle activity. Nevertheless, objective methods of recording bruxism are not available yet and the detection of this parafunction is dependent on the history and/or the presence of bruxofacets or occlusal wear. Taking into consideration the methodological limitations of the reviewed studies, an attempt was made to interpret the results by applying the epidemiologic approach of "necessary" and/or "sufficient", which implies that there must be a one-to-one relationship between the etiologic factor and the disease.[20]

Regarding bruxism, in all studies but one, this parafunction was constantly correlated with signs and/or symptoms of CMD, which suggests that a one-to-one relationship exists between bruxism and the dysfunction of the masticatory system.[10] The reported correlations were highly significant, irrespective of the methods used to record bruxism. The strength and consistency of reported correlations support causality. One would argue, however, that bruxism was not constantly correlated with the same signs and/or symptoms. A plausible explanation is that the susceptibility of the masticatory system may differ from individual to individual and the same etiologic factor can cause, therefore, different signs of CMD in different individuals.

Although the reviewed studies indicated a one-to-one relationship between bruxism and CMD, a central issue is whether this parafunction is necessary, and/or sufficient cause for the dysfunction to occur. Necessary refers to the fact that the factor must be present for the disease to occur, while sufficient indicates that if the factor is present, the disease can occur (but the factor's presence does not always result in the disease's occurrence). In the reviewed studies, the presence of subjects with a history of bruxism or bruxofacets, but without any sign and/or symptom, suggests that bruxism is a sufficient cause for CMD to occur. It is likely that these subjects overreported this parafunction, however, or the wear facets did not indicate the current level of bruxism, which implies that bruxism cannot be a sufficient cause of CMD. On the other hand, the development of signs/symptoms of dysfunction is dependent on the frequency, duration, and intensity of this parafunction. Rugh and Solberg found that bruxist behavior varies not only from individual to individual, but also from night to night for the same individual.[21] In an adult study, Clarke observed an average of five nonfunctional clenches per night.[22] The duration of each episode was an average of 7.8 seconds and total average bruxing time per night was forty seconds. In an experimental study, Vestergaard-Christensen demonstrated that in healthy adults who ground their teeth for thirty minutes, muscular pain reached a maximum about two hours after bruxism session.[23] Also, in electromyographic study, Clark *et al* reported that the greater the level of muscular activity the greater the possibility of developing signs and symptoms of dysfunction.[24] The presence of a group of individuals with a history of bruxism or bruxofacets, but without signs/symptoms of CMD can be attributed, therefore, to methodological limitation in recording this parafunction and/or to low frequency, intensity, and duration of bruxism. Both possibilities can be true and they are not mutually exclusive. The second one suggests, however, that even if the methodological limitation were eliminated, subjects with a history of bruxism or bruxofacets but without signs/symptoms of CMD, could be present in the studied groups. On the basis of this information bruxism can be considered, therefore, as a sufficient cause of CMD.

Another important issue that comes up in dealing with the causal relationship between bruxism and CMD is whether this parafunction is a sufficient cause for all individuals or for a group of them. The presence of subjects with signs/symptoms of CMD, but without bruxism, in the reviewed studies, may indicate that a number of individuals were not aware of this parafunction, or bruxofacets were not developed yet at the time of examination; and/or subjects with other etiologic factors that produce signs and symptoms of dysfunction were included in the samples. Again both possibilities may exist and they are not mutually exclusive. The first one suggests, however, that bruxism can be a sufficient cause for all individuals, while the second possibility ap-

plies only to a group of them. Other studies have shown that subjects with trauma or emotional states have significantly higher frequency of signs of dysfunction than those without these factors.[25-27] Since a differential diagnosis of the subjects according to the known etiologic factors was not applied in the reviewed studies, it is most likely that individuals with other etiologic factors were included in the samples. Even if the methodological limitation in recording bruxism were eliminated, subjects with signs and symptoms of CMD, but without a history of bruxism or bruxofacets, could be present, therefore, in the samples. Based on this information, bruxism can be considered as a sufficient cause for a group of individuals. In the framework of the multifactorial etiology of CMD, it seems that for a number of patients bruxism acts independently to produce signs and/or symptoms of dysfunction.

Concerning the rest of oral parafunctions, three studies showed highly significant correlations between lip/cheek/nail/tongue biting and symptoms, while two did not find any correlation.[4,5,9,11,12] One study did not report whether the correlation between these parafunctions and CMD was significant, while another one showed significant correlation between thumb sucking and clicking and/or condylar displacement.[7,14] The role of these parafunctions in the development of CMD cannot be clearly established, however, by the reviewed studies, and more research is necessary, therefore, to investigate their causal relationship.

CONCLUSION

Based on the reviewed studies, it can be concluded that bruxism cannot be considered as necessary, but a sufficient cause of craniomandibular dysfunction for a certain group of individuals.

REFERENCES

1. Egermark-Eriksson, I.; Carlsson, G.E.; Magnusson, T.: A long term epidemiologic study of the relationship between occlusal factors and mandibular dysfunction in children and adolescents. J Dent Res, 66:67-71, January 1987.
2. Vanderas, A.P.: An epidemiologic approach to the etiologic factors of craniomandibular dysfunction in children and adolescents: The host-agent model. J Craniomand Pract, 6:172-178, April 1988.
3. Vanderas, A.P.: Relationship between malocclusion and craniomandibular dysfunction in children and adolescents: a review. Ped Dent, 15:317-322, September-October 1993.
4. Magnusson, T.; Egermark-Eriksson, I.; Carlsson, G.E.: Four-year longitudinal study of mandibular dysfunction in children. Community Dent Oral Epidemiol, 13:117-120, 1985.
5. Kampe, T. and Hannerz, H.: Five-year longitudinal study of adolescents with intact and restored dentitions: signs and symptoms of temporomandibular dysfunction and functional recordings. J Oral Rehab, 18:387-398, 1991.
6. Hardison, J.D. and Okeson, J.P.: Comparison of three clinical techniques that evaluate joint sounds. J Craniomand Pract, 8:307-311, October 1990.
7. Egermark-Eriksson, I.; Carlsson, G.E.; Ingervall, B.: Prevalence of mandibular dysfunction and orofacial parafunction in 7-, 11- and 15 year-old Swedish children. Eur J Orthod, 3:163-172, 1981.
8. Lindqvist, B.: Bruxism in twins. Acta Odont Scand, 32:177-187, 1974.
9. Wigdorowicz-Makowerowa, N.; Grodzki, C.; Panek, H. *et al*: Epidemiologic studies on prevalence and etiology of functional disturbances of the masticatory system. J Prosth Dent, 41:76-82, January 1979.
10. Goho, C. and Jones, H.L.: Association between primary dentition wear and clinical temporomandibular dysfunction signs. Ped Dent, 13:263-266, September-October 1991.
11. Nilner, M.: Relationships between oral parafunctions and functional disturbances and diseases of the stomatognathic system among children aged 7-14 years. Acta Odontol Scand, 41:167-172, 1983a.
12. Nilner, M.: Relationships between oral parafunctions and functional disturbances in the stomatognathic system among 15- to 18-year-olds. Acta Odontol Scand, 41:197-201, 1983b.
13. Gazit, E.; Lieberman, M.; Hirsch, N. *et al*: Prevalence of mandibular dysfunction in 10-18 year-old Israeli school children. J Oral Rehabil, 11:307-317, 1983.
14. Kritsineli, M. and Shim, Y.S.: Malocclusion, body posture, and temporomandibular disorder in children with primary and mixed dentition. J Clin Ped Dent, 16:86-93, 1992.
15. Okeson, J.P.: Temporomandibular disorders in children. Ped Dent, 11:325-329, December 1989.
16. Allen, J.D.; Rivera-Morales, W.C.; Zwemer, J.D.: The occurrence of temporomandibular disorder symptoms in healthy young adults with and without evidence of bruxism. J Craniomand Pract, 8:312-318, October 1990.
17. Carlsson, G.E.; Johansson, A.; Lindqvist, S.: Occlusal wear. A follow-up study of 18 subjects with extensively worn dentitions. Acta Odontol Scand, 43:83-90, 1985.
18. Helkimo, M.: Studies on function and dysfunction of the masticatory system. Swed Dent J, 67:101-121, 1974.
19. Vanderas, A.P.: Prevalence of craniomandibular dysfunction in children and adolescents: a review. Ped Dent, 9:312-316, December 1987.
20. Lilienfeld, A.M. and Lilienfeld, D.E.: *Foundations of epidemiology*. 2nd ed. New York: Oxford University Press, 1980, pp.289-321.
21. Rugh, J.D. and Solberg, W.K.: Electromyographic studies of bruxist behavior. Calif Dent J, 43:56-59, 1975.
22. Clarke, N.G. and Townsend, G.C.: Distribution of nocturnal bruxing patterns in man. J Oral Rehabil, 11:529-534, 1984.
23. Vestergaard-Christensen, L.: Facial pain and internal pressure of masseter muscle in experimental bruxism in man. Arch Oral Biol, 16:1021-1031, 1975.
24. Clark, G.T.; Beemsterboer, P.L.; Rugh, J.D.: Nocturnal masseter muscle activity and the symptoms of masticatory dysfunction. J Oral Rehabil, 8:279-286, 1981.
25. Vanderas, A.P.: Prevalence of temporomandibular dysfunction in white children six to ten years of age in a dental school population. A cross-sectional study. Thesis, University of Pittsburgh, 1987.
26. Chunn, D.S. and Koskinen-Moffett, L.: Distress, jaw habits, and connective tissue laxity as predisposing factors to TMJ sounds in adolescents. J Craniomand Disord Facial Oral Pain, 4:165-176, 1990.
27. Vanderas, A.P.: Prevalence of craniomandibular dysfunction in white children with different emotional states. Part III. A comparative study. J Dent Child, 59:23-27, January-February 1992.

REPORTS

Supernumerary teeth: Report of three cases and review of the literature

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In dentistry, one encounters numerous anomalies in the size, shape, number, structure, and eruption of teeth. These anomalies occur because the developing teeth are influenced by a complex interaction of genetic and environmental variables. Identification of anomalies of odontogenesis are important in evaluating etiological factors, dental and medical histories, present states of oral health, and control and management.

Tooth development is a continuous process in which a number of physiologic growth processes and various morphologic stages interplay to achieve the tooth's final form and structure. The physiological processes involved in tooth development are: initiation, proliferation, histodifferentiation, morphodifferentiation, apposition, and calcification.[1] Initiation represents the beginning of formation of the dental lamina and tooth bud from the oral epithelium. Interference with the stage of initiation, a momentary event, may result in single or multiple missing teeth (anodontia, oligodontia, or hypodontia) or supernumerary teeth (also called hyperdontia). Supernumeraries denote teeth formed in a number in excess of that found in the normal series, varied in their form and location, and occurring in both the primary and permanent dentitions. Supernumerary teeth may be single or multiple, unilateral or bilateral, malformed morphologically or normal in size and shape, and erupted or impacted.[2-6] Supernumerary teeth, impacted or erupted, may remain in position for many years without clinical manifestations, either pathologic or orthodontic. Many complications can be associated with supernumeraries, however, including impaction, delayed eruption, or ectopic eruption of adjacent teeth, crowding, development of a median diastema, eruption into the floor of the nasal cavity, formation of primordial or follicular cysts with significant bone destruction, and root resorption of adjacent teeth.[3,4,7-9]

This paper reviews the literature and reports three cases of bilateral supernumerary teeth impeding the eruption of permanent maxillary central incisors and emphasizes the importance of early diagnosis and management.

REVIEW OF THE LITERATURE

The etiology of supernumerary teeth is not well understood, but several theories have been advanced to explain the anomaly, based on developmental interference and heredity. Histological evidence indicates that after initiation of tooth germ, the dental lamina begins to degenerate at that location. Remnants of the dental lamina can persist, however, as epithelial pearls or islands, "rests of Serres", within the jaw. If the epithelial remnants are subjected to initiation by induction factors, an extra

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tooth bud is formed resulting in the development of either a supernumerary tooth, or an odontoma. A supernumerary tooth may also arise separately (as an offshoot) from continued activity of the dental lamina after the formation of the normal number of tooth buds, or result from complete division (dichotomy) of an early developing bud. Support for this theory came from clinical findings that showed a tendency for the development of permanent supernumerary teeth in cases with higher incidences of primary supernumeraries.[10,11]

Heredity plays an important role in the occurrence of supernumerary teeth. The proposed modes of inheritance follow simple Mendelian principles, recessive, autosomal, or sex-linked inheritance. Even atavism (phylogenetic reversion or evolution throw back) was believed a cause for the occurrence of a distomolar (fourth molar). In a survey of 200 patients with supernumerary teeth, Stafne found that in 90 percent of his cases there was a definite genetic influence. A familial tendency has been demonstrated in ten out of twenty-three cases.[13] In a more comprehensive familial study, Brook reported much higher frequencies of supernumerary teeth among first-degree relatives than in the general population, suggesting a significant genetic component in the etiology.[14] Foster advanced the genetic aspect to conclude that supernumerary teeth are a purely inherited feature, excluding environmental factors from any responsibility for their occurrence.[15,16] The observation that males are affected more than females (2:1) led Bruning *et al* to conclude that hyperdontia is a sex-linked inheritance.[17] Sedano and Gorlin, on the basis of scanty information, suggested the possibility of an autosomal dominant inheritance with lack of penetrance in some generations.[18] The available data confirmed that supernumerary traits have a strong hereditary component, but do not appear to conform with a simple Mendelian pattern. Environmental factors may also play a part. It is appropriate to consider that hyperdontia as a multifactorial inheritance disorder originates from hyperactivity of the dental lamina.

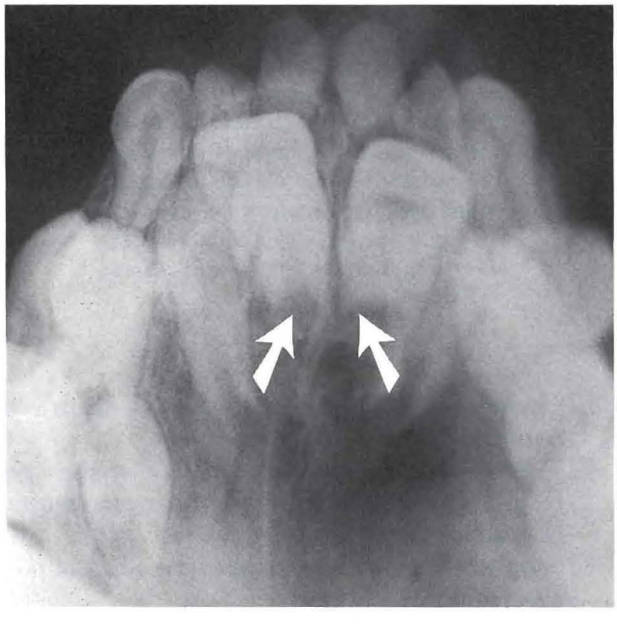

Figure 1. Occlusal radiograph of twelve-year-old male with cleidocranial dysplasia. Note the overretained maxillary incisors, the unerupted permanent incisors, and two supernumerary teeth superimposed on the central incisors.

Supernumerary teeth can be parts of genetic syndromes such as cleidocranial dysplasia and Gardner's disease. Cleidocranial dysplasia is a disease of unknown etiology, characterized by absence of clavicles, pathognomic face and head, underdeveloped maxilla, prolonged retention of the primary teeth and subsequent delay in the eruption of succedaneous teeth, and the characteristic presence of numerous unerupted supernumerary teeth (Figure 1). Gardner's syndrome can also present multiple impacted supernumerary teeth along with its usual complex of multiple polyposis of the large intestine, osteomas (usually arising from bones), multiple epidermoid or sebaceous cysts of the skin. Supernumerary teeth can be associated with cleft lip and palate as a result of fragmentation of dental lamina during cleft formation.[15] As high as 28 percent of patients with cleft lip and palate had at least one supernumerary tooth.[19]

The prevalence of supernumerary teeth ranges from 0.15 to 3.8 percent in different populations and appears to be on the increase.[5,6,12,13,20] Besides racial variations, differences in the ages of the subjects and examination methods may significantly account for this wide range. Clinical surveys without the use of radiographs result in underestimates of the incidence, because unerupted teeth escape detection. McPhee, in one such study, found an incidence of erupted supernumerary teeth of about 1 in 333.[21] Stafne conducted the most comprehensive survey, in which full-mouth periapical radiographs of 48,550 dental hospital patients were examined.[12] He found that 0.9 percent had at least one supernumerary tooth, but only 12 percent of these had two supernumeraries and 1 percent had three. Approximately 90 percent of all supernumeraries occur in the maxilla and half of these were found in the incisor regions. In Britain, a survey of nearly 2,000 school children revealed supernumerary teeth of all kinds in 0.8 percent of primary dentitions and 2.1 percent of per-

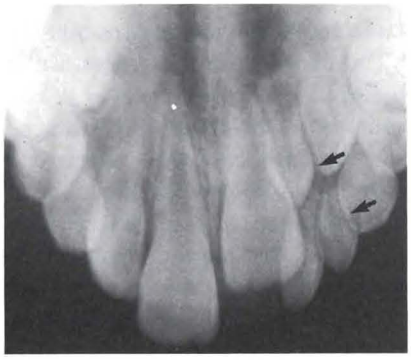

Figure 2. Occlusal radiograph of eight-year-old male showing a (supplemental type) in the right lateral incisor region superseded by extra tooth in the permanent dentition. The primary supernumerary tooth caused delay in the eruption of the permanent central and lateral incisors.

manent dentitions.[5] Several other studies confirmed that multiple supernumerary teeth occur in about 14 percent of the subjects examined and supernumeraries are found infrequently in the primary dentition with a prevalence ranging from 0.03 to 1.9 percent.[6,13,22,23] While there is no significant sex distribution of primary supernumerary teeth, in the permanent dentition males are affected approximately twice as frequently as females.[5,24-26] Humerfell *et al* stated that hyperdontia in the primary dentition is often overlooked, because the supernumerary teeth are often of normal shape (supplemental type), erupt normally, and appear to be in proper alignment; and can be mistaken for gemination and fusion anomalies.[23] An interesting finding is that in 35-50 percent of cases, supernumerary teeth in the primary dentition are superseded by extra teeth in the same location in the permanent dentition (Figure 2).[25] The review of literature showed that maxillary midline supernumeraries are the most common type, followed by maxillary lateral incisors, mandibular third premolars, mandibular fourth molars and maxillary paramolars.[9,13,22-24] Other reports indicated that the maxillary distomolar (fourth molar) is the second most common supernumerary tooth.[12,26]

Supernumerary teeth may be classified according to their form (size and shape) and location.[3-5] Supernumeraries in the primary dentition are usually normal or conical in shape. In the permanent dentition, they have a greater variety of forms and may be classified as follows:

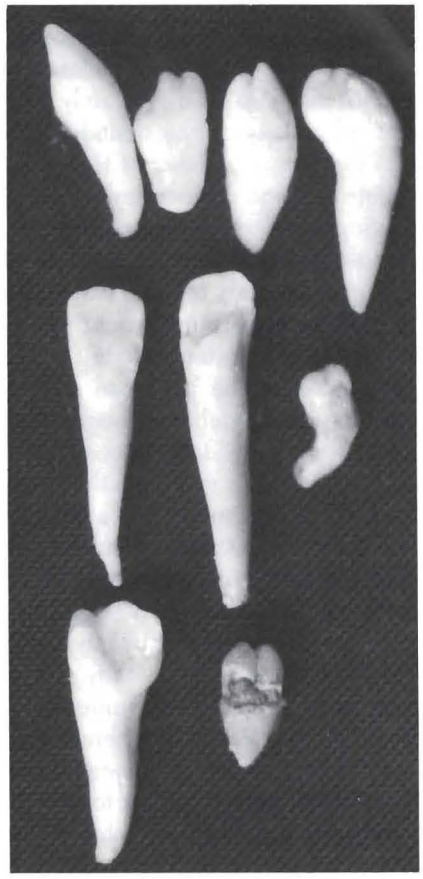

Figure 3a. Extracted maxillary supernumerary teeth of various forms. The upper row from left (a) Conical, (b and c) Tuberculate with incomplete (stunted) root formation, (d) Tuberculate with the crown consisting of multiple tubercules. The middle row from the left: (e) Supplemental type, (f) Supplemental with coronal invagination, (g) Odontoma. The lower row from the left: (h) Molariform, (i) Paramolar.

- ☐ Conical: Small, peg-shaped (coniform) teeth with normal root.
- ☐ Tuberculate (multicusped): Short, barrel-shaped teeth with normal appearing crown, or invaginated but rudimentary root.
- ☐ Supplemental: Teeth resembling the adjacent non-affected teeth.
- ☐ Odontomas: Having no regular shape (Figure 3 a,b).

Primosch classified supernumerary teeth into two types according to the shape: supplemental and rudi-

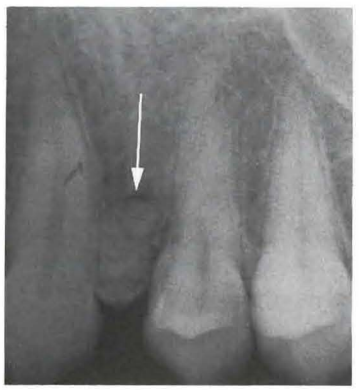

Figure 3b. Radiographic appearance of odontoma.

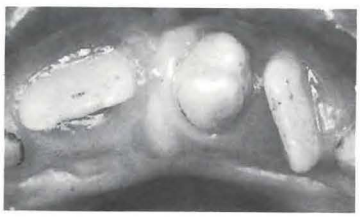

Figure 4a. Clinical appearance of eight-year-old male showing a rare molariform supernumerary tooth (resembling the morphology of a premolar) between the maxillary central incisors caused rotation of the left central incisor and wide median diastema.

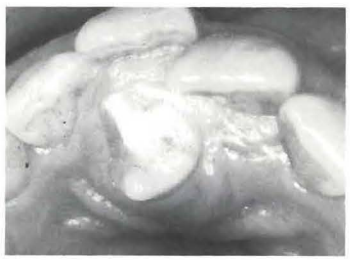

Figure 4b. Molariform supernumerary tooth in a twelve-year-old male had created displacement of the right permanent central incisor and crowding. Its root was completely formed.

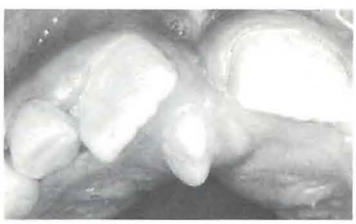

Figure 5a. Clinical appearance of eight-year-old male disclosing a conical-shaped mesiodens erupted palatally in the midline causing rotation of the right permanent central incisor and abnormal median diastema.

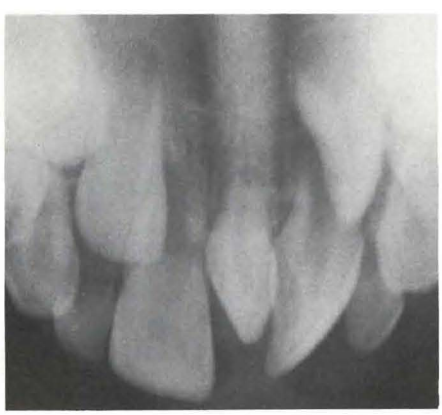

Figure 5b. Radiographic appearance of the mesiodens.

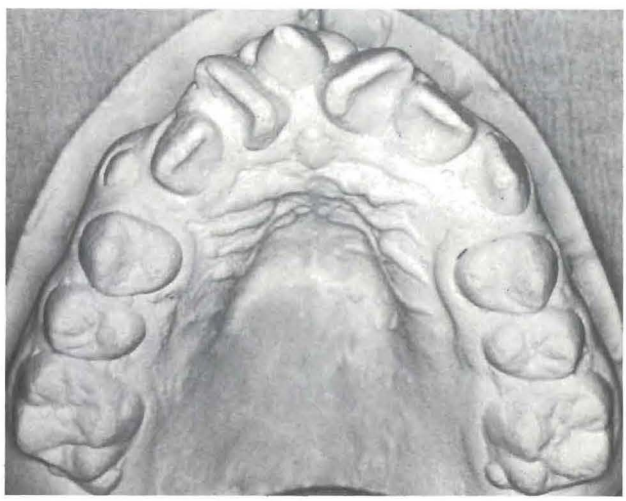

Figure 5c. A case of uncommon labially located mesiodens causing rotation and displacement of the maxillary central incisors.

mentary.[6] Supplemental (or eumorphic) refers to supernumerary teeth of normal shape and size, and may also be termed incisiform. Rudimentary (or dysmorphic) defines teeth of abnormal shape and smaller size, including conical, tuberculate, and molariform types. Koch *et al* registered 56 percent conical, 12 percent tuberculate, 11 percent supplemental, and 12 percent other configurations.[27] The molariform type has been only rarely reported (Figure 4).

Conical-shaped supernumerary teeth have certain specific characteristics that distinguish them from the tuberculate-shaped teeth.

- ☐ They usually are located between the permanent maxillary central incisors, but rarely erupt labially.
- ☐ They erupt during childhood.
- ☐ They have a complete root formation, ahead of or as early as that of the adjacent teeth.
- ☐ They rarely delay the eruption of adjacent central incisors, but may cause displacement of the incisors (Figure 5).

The tuberculate supernumerary tooth also occurs in

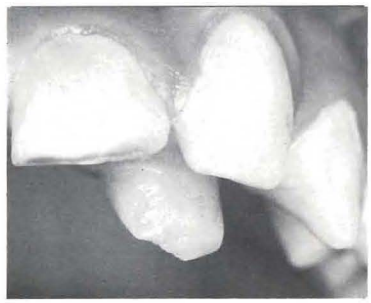

Figure 6a. Photograph of eleven-year-old male revealing palatally erupted supplemental lateral incisor.

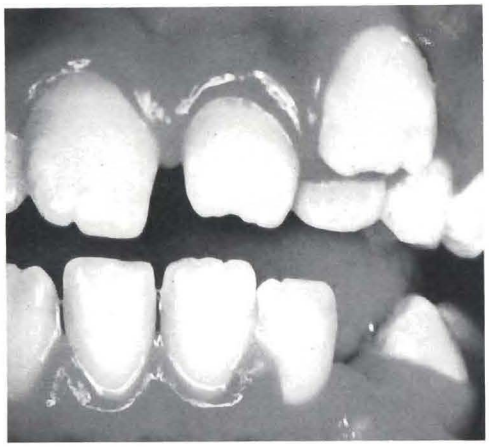

Figure 6b. Supplemental central incisor, in ten-year-old male, erupted labially. Note these supernumeraries have created crowding and esthetic problems.

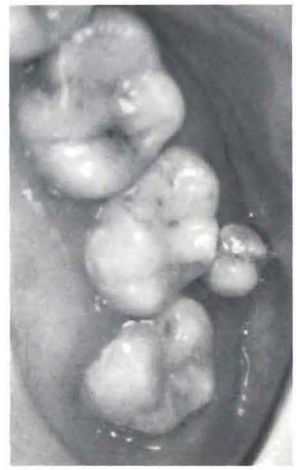

Figure 7. Upper occlusal view of a twenty-four-year-old female illustrating a paramolar supernumerary tooth erupted buccally in the interproximal space between the second and third molars. The tooth was small and rudimentary, resembling the distomolar (Figure 8).

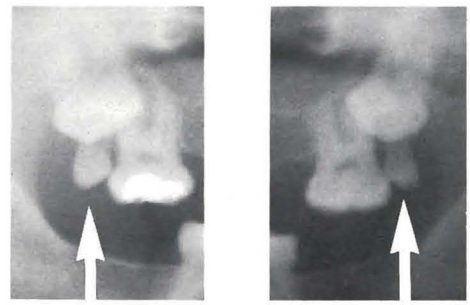

Figure 8. Panoramic radiograph of a twenty two-year-old male showing bilateral maxillary distomolars (fourth molar). This case is unusual because the supernumeraries interfered with the eruption of the third molars.

the premaxilla. It differs from the conical type in the following set:

- ☐ The tuberculate tooth develops later than the conical tooth, with incomplete (stunted) or totally absent root formation.
- ☐ It appears, characteristically, on the palatal aspect of the permanent upper central incisor.
- ☐ It rarely erupts in childhood.
- ☐ It may be unilateral or bilateral, and is uncommonly associated with supernumerary teeth of other types.
- ☐ It delays the eruption of the permanent maxillary central incisors.
- ☐ It is usually larger in size than the conical type (Figure 3 and Case 1).

Supplemental teeth, as the name implies, refers to duplication of teeth in the normal series (Figure 6). In the permanent dentition they appear most commonly as extra maxillary and mandibular lateral incisors, whereas in the primary dentition, they are most likely to appear as upper central incisors. It is less common to find supplemental premolars and molars (see Case 1), except in African and Asian populations.[15] It may be difficult to distinguish the normal tooth from its supplemental "twin". A supplemental tooth may exhibit a deep cingulum pit and have a coronal invagination (Figure 3).

Supernumerary teeth can be categorized into three types according to their location:

- ☐ Mesiodens: A typical conical supernumerary tooth situated between the maxillary central incisors. Mesiodens may be single or multiple; unilateral or bilateral; erupted or impacted; vertical, horizontal or inverted (Figure 5, see Case 1).
- ☐ Paramolar: A supernumerary molar, usually small and rudimentary situated buccally or lingually to

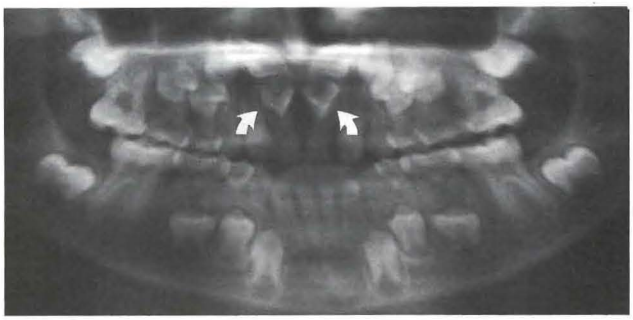

Figure 9A. (Case 1). This patient was first seen at the age of nine years. Baseline panoramic radiograph showing overretention of the maxillary primary central incisors and the presence of bilateral unerupted mesiodentes.

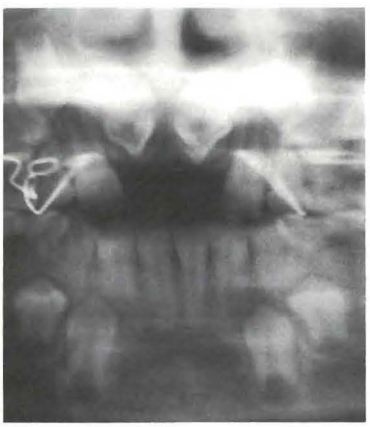

Figure 9b. The primary central incisors were extracted straight away and space maintainer was constructed. Three years later, the supernumeraries were surgically removed.

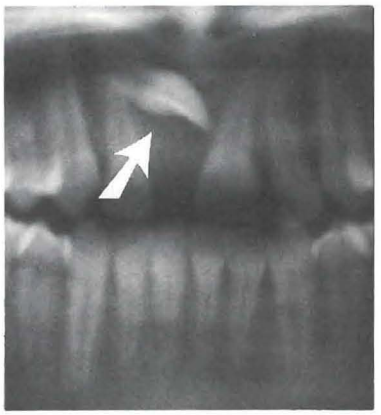

Figure 9c. After 18 months, the left central incisor was fully erupted while the right central was still highly placed in a horizontal position. Surgical exposure was performed and an orthodontic appliance with elastic ligature was placed.

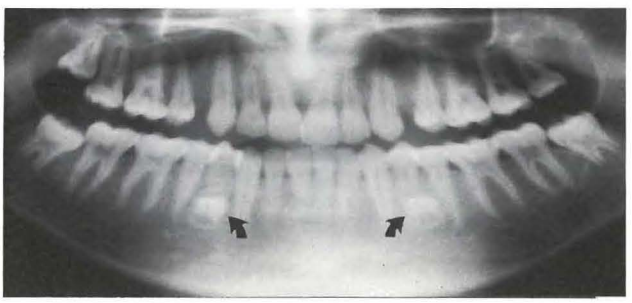

Figure 9d. Posttreatment radiograph (at age of seventeen years) shows the central incisor in position and supernumerary premolar crowns developed beneath the right and left mandibular premolars.

one of the maxillary molars or in the interproximal space buccal to the second and third molars (Figure 7).

- ☐ A distomolar is located distal to the third molar. Like the paramolar, the distomolar is usually small and rudimentary, but rarely delays or impedes the eruption of the normal tooth (Figure 8).

Supernumeraries are frequently discovered when a normal tooth is either delayed in its eruption or displaced. Delayed eruption of maxillary anterior permanent teeth due to the presence of a supernumerary tooth may result in an arch-length inadequacy. A labially positioned supernumerary tooth may cause lingual deflection of an incisor that may erupt in a rotated or cross-bite relationship. Sometimes the unerupted supernumerary tooth causes no occlusal problems. In general, supernumerary teeth, particularly in the maxillary anterior region, may cause the following clinical problems:

- ☐ Delayed or prevented eruption of permanent succedaneous teeth (26-52 percent of cases).
- ☐ Displacement or rotation of permanent teeth (28-63 percent of cases).
- ☐ Crowding of the affected region.
- ☐ Abnormal diastema or premature space closure.
- ☐ Dilaceration or abnormal root development of permanent teeth.
- ☐ Cystic formation; in 4-9 percent of the cases.
- ☐ Eruption into the nasal cavity.[3-7,28,29]

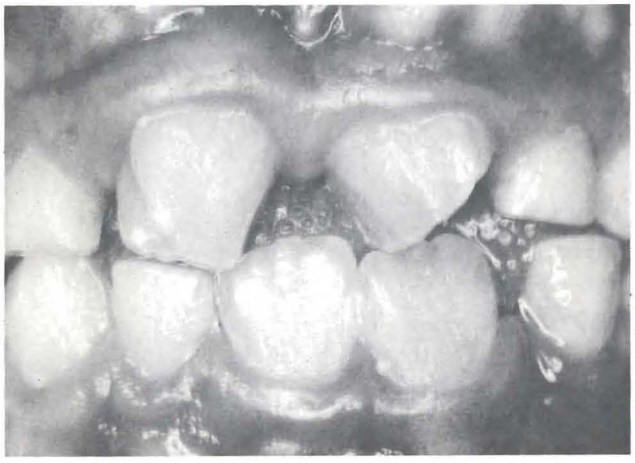

Figure 10a. (Case 2) Frontal view of six-year-old patient showing erupted, malposed maxillary "permanent" central incisors.

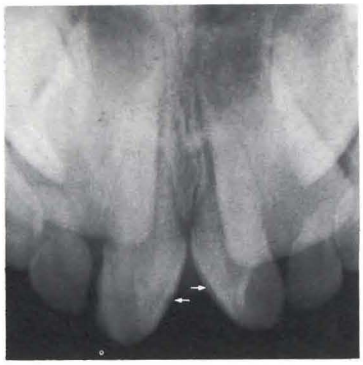

Figure 10b. Radiograph shows the presence of two tuberculate-shaped supernumeraries causing impaction and displacement of the maxillary permanent central incisors.

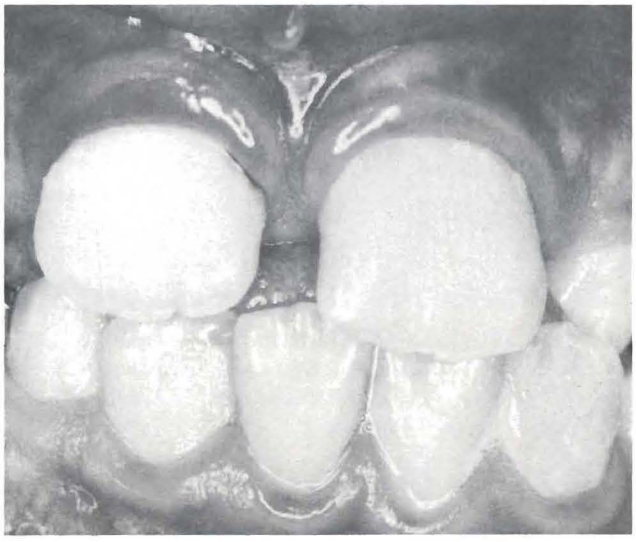

Figure 10c. Six to eight months after the removal of supernumeraries, the permanent central incisors have spontaneously erupted into proper position and alignment.

The types of supernumerary teeth are associated with their respective effects on the dentition.[3,5] The supernumeraries that cause the greatest problems in orthodontics are those lying close to the midline in the maxillary arch, namely the mesiodens. The most common clinical complication of mesiodens is displacement of the central incisors (Figure 5), while the tuberculate type prevents or causes delay in the eruption of the adjacent teeth (see Case 2). Delayed eruption of the central incisors contributes to the following problems:

- ☐ Medial movement of the lateral incisors and reduction in the arch space.
- ☐ Diminished development of the dentoalveolar height, resulting in failure of the central incisors to erupt fully.

Supplemental supernumerary teeth increase the crowding potential and may cause an aesthetic problem (Figure 6). Difficulty may be encountered in distinguishing the normal tooth from its supplemental 'twin'. If both teeth are equally well formed the correct extraction is the tooth that is most displaced (Figure 6).

Supernumeraries may also develop in the premolar or third molar region (see Case 1 and Figure 8). These usually develop after most permanent teeth have erupted, thus their effect upon the occlusion is limited. Unlike other supernumerary teeth, extra premolars are more likely to develop in the mandible than in the maxilla.[11] Severe dilacerated permanent teeth caused by supernumeraries may make orthodontic movement of the affected teeth very difficult. Often the portion of the root apical to the irregularity is resorbed during tooth movement. A mildly dilacerated tooth can be moved, however, in the normal fashion. Occurrence of a supernumerary canine is rare.[12,13]

Detection of supernumeraries is best achieved by thorough clinical and radiographic examination. The frequency of erupted primary supernumerary teeth is much higher than that of erupted permanent supernumeraries (73 percent versus 25 percent).[12,13,23,28] Thus, the majority of permanent supernumeraries can be diagnosed only by means of radiography. Anterior maxillary supernumerary teeth can be disclosed radiographically from the newborn period to adulthood. Before attempting surgi-

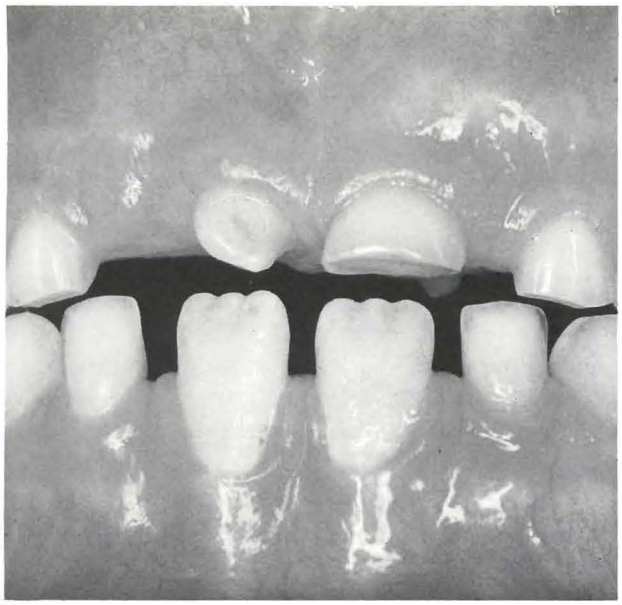

Figure 11a (Case 3) Frontal view of eight-year-old patient showing overretained maxillary left primary central incisor and small, partially erupted maxillary right "permanent" central incisor.

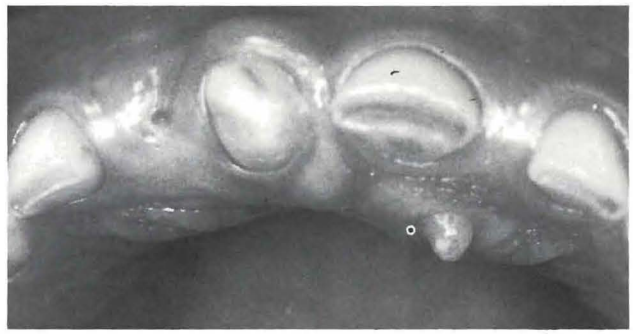

Figure 11b. The tip of the maxillary left "permanent" central incisor was emerged palatally.

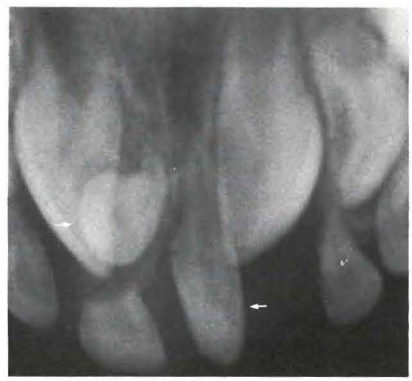

Figure 11c. Anterior occlusal radiograph revealed the presence of bilateral mesiodens angled in different directions, vertical and horizontal.

cal removal, the position of the supernumerary tooth must be located. One method of localizing impacted supernumerary teeth uses the parallax technique (also called the buccal object rule or the horizontal shift technique).[30] The technique involves taking two radiographs of the same object from two horizontal (lateral) angles, but maintaining the vertical direction. A lingually impacted tooth should move in the same direction as a moving X-ray source, or in the opposite direction if the impacted tooth is located labially to the reference tooth. This phenomenon is referred to as the S.L.O.B. rule (Same on the Lingual, Opposite on the Buccal).

The purpose of this paper is to report three cases of delayed eruption of maxillary permanent central incisors caused by the presence of bilateral supernumerary teeth. The patients presented in the Department of Pediatric Dentistry in Jordan University of Science and Technology.

Case 1

M.S., a nine-year-old male (born on December 25th, 1976) presented at the dental clinic in January 1985. His father was concerned by the uneruption of his maxillary anterior front teeth. The family medical and dental histories were noncontributory and the patient had no record of orofacial trauma.

Clinical examination revealed a mixed dentition comprising the first permanent molars, maxillary lateral incisors, and mandibular incisors. The maxillary primary incisors were sound and no sign of mobility was evident. Radiographic (panoramic and occlusal views) examination showed slight root resorption of the overretained maxillary primary central incisors. Two supernumerary teeth of mesiodens type were positioned on a level with the apices of the primary central incisors. Above the supernumeraries, crowns of the permanent central incisors were observed, positioned close to the nasal cavity (Figure 9a). Radiographic inspection denoted a delay in the development of the succedaneous teeth and inhibition of the resorption of the roots of the primary teeth. Because the unerupted central incisors present the only problem in an otherwise normal occlusion, the overretained primary central incisors were extracted, using local anesthesia. A maxillary removable appliance was fitted to maintain the space in the incisor region (Figure 9B). Thirty-six months elapsed before the patient returned to the dental clinic, reporting the loss of the space maintainer the previous year. Radiographic examination disclosed no appreciable movements of the supernumeraries and the impacted central incisors. The supernumerary teeth were surgically removed using local anesthesia and sedation with meperidine and promethazine hydrochloride. Eighteen months later (May

1989) at the age of thirteen and half years, the left central incisor was fully erupted, while the right central incisor was still highly placed in a horizontal position (Figure 9c). Surgical exposure of the right central incisor was made and an elastic ligature was placed from an arch wire to a bracket directly bonded to the tooth. The patient was scheduled for extraction of the maxillary first premolars, but he failed to appear. Nine months later, the right central incisor still was not fully erupted, due to insufficient space. Extraction of the maxillary first premolars was performed and orthodontic treatment was continued.

The patient was seen on a recall in August 1993, at the age of seventeen years and eight months. A panoramic radiograph revealed the presence of newly-developed, asymptomatic supernumerary premolar crowns located at the apical third of the roots of the left and right mandibular first premolars (Figure 9d). Parallax technique indicates that the supernumerary premolars are lingually positioned with no sign of root resorption of the original premolars. The crowns of the supernumeraries were almost completed, resembling a chronological age of five to six years.

Case 2

S.O., a six-year-old male presented to the dental clinic for routine dental care. The mother expressed concern of "unesthetic appearance of upper front teeth" and gave a history of exfoliation of the predecessors. The medical and dental histories were unremarkable.

Intraoral examination revealed an early mixed dentition. The erupted permanent teeth were as follows: mandibular first molar and central incisors and maxillary "central incisors". Both maxillary "central incisors" were malposed and almost completely erupted with abnormal central diastema. The left central incisor was rotated and interfered with the occlusion by forming a premature contact; while the one on the right was labially displaced (Figure 10a). The color of the maxillary central incisors was more yellowish and less translucent than the opposing teeth. The mesiodistal crown diameter of the maxillary central incisors was approximately 5 mm. Unlike the normal succedaneous tooth, no mammelons were seen on the incisal edge and the marginal ridges were ill defined. The patient presented with a Class I occlusion, with minimal spacing and anterior edge-to-edge relationship.

Radiographic examination disclosed bilateral impacted maxillary permanent central incisors superimposed upon two supernumerary teeth of tuberculate type (Figure 10b). The supernumerary teeth were the main cause of impaction of the central incisors. The roots of the impacted central incisors were incompletely developed, while the roots of the supernumerary teeth were short. The impacted central incisors were close to their normal eruption path. Their crowns measured, on the radiograph, approximately 10 mm wide at their greatest mesiodistal diameter. Sufficient space was available for the unerupted central incisors.

Spontaneous eruption of the impacted maxillary permanent central incisors was anticipated, following the removal of the supernumerary teeth. Six to eight months postoperatively, both central incisors were completely erupted and well aligned (Figure 10c).

Case 3

M.S., an eight-year-old male visited the dental clinic with a chief complaint of a "funny looking upper front tooth". His medical and dental histories were noncontributory. A history of orofacial trauma was denied by the mother.

Clinical examination revealed a mixed dentition consisting of fully erupted permanent mandibular central incisors and first molars. The remaining dentition consisted of primary teeth, including lateral incisors, canines, first and second molars. The maxillary left primary central incisor was overretained, while the antimere had exfoliated and been replaced by a small, slightly rotated and partially erupted succedaneous tooth (Figure 11a). A cusp-like projection was emerging palatally to the predecessor, the left primary central incisor (Figure 11b). Periapical and occlusal radiographs showed the presence of impacted maxillary central incisors and two mesiodens located between them (Figure 11c). The roots of the permanent central incisors were almost completely developed, while the roots of the supernumeraries were short and malformed.

The treatment was extraction of the primary left central incisor and removal of both supernumeraries. Extraction was performed using local anesthesia without difficulty, and special care was taken not to traumatize the central incisors. Two months later, both central incisors were partially erupted. Eight months after the removal of the supernumerary teeth, the central incisors were fully erupted into normal position.

DISCUSSION

Supernumerary teeth are considered to be one of the most significant dental anomalies affecting the primary

and early mixed dentition. Anterior maxillary supernumeraries in young patients are of great concern to both dentist and parents because of the delayed eruption, occlusal and esthetic problems they can create. Early diagnosis of supernumeraries is crucial, if these complications are to be avoided or minimized. Clinically the presence of supernumeraries should be suspected, if there is a significant delay in the eruption of the maxillary permanent central incisors. Usually the problem is not noticed until the maxillary lateral incisors start to erupt or have erupted and when one, or both, central incisors are missing and a primary predecessor(s) is present (Figure 10A). Once the unerupted permanent incisor is close to its normal eruption path, the adjacent teeth may tilt; but when the unerupted tooth is highly placed, the neighboring teeth may close the space by bodily movement. Palpation is another valuable adjunct in final location of the unerupted tooth. Radiographic examination is essential to confirm diagnosis, the position of the supernumerary and its relation with the adjacent teeth, and the distance of the unerupted permanent teeth from the occlusal plane.

Supernumerary teeth should be extracted immediately, if any of the above-cited complications is present. Munns stated that the earlier the offending supernumerary is removed, the better the prognosis.[31] Surgical removal of a supernumerary in the primary dentition is usually not recommended, because of the risk of displacing the permanent tooth during the operation.[6,23] Moreover, most primary supernumeraries erupt normally because the presence of the interdental spaces allows for uneventful eruption of the extra tooth. It is likely many such teeth exfoliate without being recognized as supernumeraries.[6]

The optimal time for surgical removal of an unerupted maxillary anterior supernumerary is controversial. Some authors advocate immediate removal of the supernumerary teeth following initial diagnosis of their presence, while others favor postponement of surgical intervention until the age of eight to ten years, when the root development of the central and lateral incisors is nearly completed.[6,22,32-36] The possible disadvantages of immediate intervention are:

- ☐ Damage to the adjacent teeth, resulting in the loss of vitality and root malformation.
- ☐ Inability of a young child to tolerate psychologically the surgical procedure.

The possible disadvantages of delayed intervention are:

- ☐ Diminished eruptive forces of the adjacent teeth.
- ☐ Loss of anterior arch-space.
- ☐ Midline shift.

Several reports show that the majority of unerupted teeth (approximately 75 per cent) will erupt spontaneously, once the supernumerary tooth is removed.[4,13] The time taken by the unerupted tooth to appear, following removal of the supernumerary, ranged between sixteen months and three years.[4,31,33] Four factors were found to have an effect on the time taken for the unerupted tooth to appear:

- ☐ The type of the supernumerary tooth.
- ☐ The degree of displacement and inclination of the unerupted tooth.
- ☐ The space available for eruption.
- ☐ Time of diagnosis and surgical intervention.[3,4]

The patient's chronological age, as well as the degree of root maturity, inclination and curvature, appear to have little influence on the eruption rate.[4] In cases where spontaneous eruption fails to occur, surgical exposure of the unerupted permanent incisor is indicated by the following:

- ☐ Lack of eruptive movement following removal of the supernumerary and after six-month observation period.
- ☐ Existence of sufficient arch-space for eruption.

A less conservative approach advocated by some authorities entails removal of the supernumerary and exposure of the unerupted tooth at the same time, with or without placement of a bonded attachment or ligature for orthodontic traction.[32,34] This approach, however, may result in a poor gingival margin and a discrepancy of the gingival level between the exposed tooth and its neighboring teeth. It has been suggested that forces (about 1 to 2 oz) are more effective than heavy forces in moving impacted and unerupted teeth and providing good gingival position and contour.[35,36] To provide adequate gingival tissue attachment, an apically repositioned flap rather than a window technique is recommended for exposing the crown of the unerupted tooth.[6,37]

In the present cases, the complications associated with the presence of supernumerary teeth were: delayed eruption of the permanent incisors, displacement, rotation, impaction, overretained central incisors, and diastema formation. This report indicates that early removal of the supernumeraries results in spontaneous eruption and alignment of the permanent central incisors within six to eight months, provided sufficient arch-space was available and the impacted tooth in a favorable (vertical) position (Case 2 and Case 3). In both cases, the supernumeraries were extracted before the eruption of the lateral incisors. In case 1, the delay in removing the unerupted supernumerary until the age of twelve years, due to patient unattendance, prolonged the period for spon-

taneous eruption of the left maxillary central incisor. The antimere was horizontally placed and required, therefore, surgical exposure and orthodontic treatment to bring the tooth into position. The orthodontically tracked tooth had erupted, however, to within 1-mm short of the occlusal plane. This phenomenon was attributed to the presence of a matured gingival fiber system.[6,36]

Case 1 is unusual for two reasons: (1) the concurrent occurrence of two types of supernumerary teeth (anterior conical-shaped and posterior supplemental type); (2) the supplemental supernumeraries in the premolar region (postpermanent dentition) were formed late and detected on a routine posttreatment radiograph. The prevalence of supernumerary premolars is reported to be one in 10,000 individuals.[38] Grahnen and Lindahl reported a higher incidence (0.29 percent) of supernumerary premolars in Swedish dental students, representing 9.1 per cent of all supernumerary teeth.[39] In an orthodontic population, the frequency was as high as 0.64 per cent and all supernumerary premolars were detected between the ages of eleven and sixteen years.[40] The formation of normal premolar teeth is not expected after the dental age of eight years.[41] In case 1, the formation of supernumerary premolars seems to have begun after the age of fourteen years and the crowns were almost completed at the age of seventeen years and eight months. It has been demonstrated that supernumeraries in the lower premolar region were first visible at the ages of nine, twelve to twelve and a half, thirteen and a half or fourteen in various cases.[7,42-44] In the present case it is difficult to predict whether the roots of the supernumerary premolars will develop after this age; the case is kept, however, under observation. Orchler reported a continued root growth of supernumerary premolars in a twenty-three-year-old man.[45] On the other hand, Mills believed that the roots of these supernumeraries frequently do not form and the teeth remain stationary.[36] The present finding and recently reported cases highlight the need for follow-up radiographs for orthodontic patients, because 75 percent of supernumerary premolars are unerupted and the majority appear to be asymptomatic.[42-44]

In cases 2 and 3, the following features were found in common: (1) the unerupted permanent central incisors were nearly in vertical position and placed superficially, (2) sufficient space for the unerupted permanent central incisors to erupt was present, (3) the roots of the unerupted incisors were incompletely formed.

The frequency with which supernumerary teeth occur and their deleterious effects on normal occlusal development justify the radiographic examination of preschool children. Early diagnosis allows early intervention, more favorable prognosis and minimal complications.

REFERENCES

1. Bhaskar, S.N.: *Orban's oral histology and embryology.* St. Louis: C.V. Mosby Co., 1986.
2. Bazan, M.T.: Anomalous dental development with medical and genetic implications. Pediatr Ann, 14:108-116, February 1985.
3. Foster, T.D. and Taylor, G.S.: Characteristics of supernumerary teeth in the upper central incisor region. Dent Practit, 20:8-12, September 1969.
4. DiBiase, D.: The effects of variations in tooth morphology and position on eruption. Dent Practit, 22:95-108, 1971.
5. Brook, A.H.: Dental anomalies of number, form and size : their prevalence in British schoolchildren. J Int Assoc Dent Child, 5:37-53, December 1974.
6. Primosch, R.E. : Anterior supernumerary teeth-assessment and surgical intervention in children. Pediatr Dent, 3:204-215, June 1981.
7. Mitchell, L.: Supernumerary teeth. Dent Update, 16:65-69, March 1989.
8. Thoma, K.H.: *Oral surgery*, 3rd ed. St. Louis: C.V. Mosby Co., 1958, pp 395-398.
9. Shafer, W.G.; Hine, M.K; Levy, B.M.: *A textbook of oral pathology*, 4th ed. Philadelphia: W.B. Saunders Co., 1983, p 47.
10. Munro, D.: Supernumerary teeth of the permanent and deciduous dentitions. Br Dent J, 93:321-322, 1952.
11. Grahnen, H. and Granath, L.: Numerical variations in primary dentition and their correlation with the permanent dentition. Odontol Revy, 12:348-357, 1961.
12. Stafne, E.C.: Supernumerary teeth. Dental Cosmos, 74:653-659, July, 1932.
13. Mckibben, D.R. and Brearly, L.J.: Radiographic determination of the prevalence of selected dental anomalies in children. J Dent Child, 28:390-398, November-December 1971.
14. Brook, A.H.: A unifying aetiological explanation for abnormalities of human tooth number and size. Arch Oral Biol, 29:373-378, 1984.
15. Foster, T.D. : *A textbook of orthodontics.* Oxford: Blackwell Scientific Publications, 1982, pp 145-149.
16. Witkop, C.J.: *Genetics and dental health.* New York: McGraw-Hill Book Company, Inc., 1962, p 203.
17. Bruning, L.J.; Dunlop, L.; Mergele, M.E. : Report of supernumerary teeth in Houston, Texas School Children. J Dent Child, 24:98-105, Second Quarter, 1957.
18. Sedano, H.O. and Gorlin, R.J. : Familial occurrence of mesiodens. Oral Surg, 27:360-361, March 1969.
19. Millhon, J.A. and Stafne, E.C.: Incidence of supernumerary and congenitally missing lateral incisor teeth in 81 cases of harelip and cleft palate. Am J Orthod, 37:559-604, November 1941.
20. Brabant, H.: Comparison of the characteristics and anomalies of the deciduous and the permanent dentition. J Dent Res, 49:897-902, 1967.
21. McPhee, G.G.: The incidence of supernumerary teeth in consecutive series of 4,000 School Children. Br Dent J, 58:59-60, 1935.
22. Nazif, M.M.; Buffalo, R.C.; Zullo, T.G.: Impacted supernumerary teeth : A survey of 50 cases. J Am Dent Assoc, 106:201-204, February 1983.
23. Humerfelt, D.; Hurlen, B.; Humerfelt, S.: Hyperdontia in children below four years of age: A radiographic study. J Dent Child, 52: 121-124, March-April 1985.
24. Bergstrom, K.: An orthopantomographic study of hypodontia, supernumeraries and other anomalies in school children between the

ages of 8-9 years—An epidemiological study. Swed Dent J, 1: 145-157, 1977.
25. Winter, G.B. and Brook, A.H : Tooth abnormalities. In : *A Companion to Dental Studies*: Vol 3. Clinical Dentistry. Rowe, A.H.R., ed. Oxford: Blackwell Scientific Publications, 1986, pp 55-103.
26. Shah, R.M.; Boyd, M.A.; Vakil, T.F.: Studies of permanent tooth anomalies in 7,886 Canadian individuals. J Canad Dent Assoc, 44: 265-276, 1978.
27. Koch, H.; Schwartz, O.; Klausen, B.: Indications for surgical removal of supernumerary teeth in the premaxilla. Int J Maxillofac Surg, 15:273-281, June 1986.
28. Tay, F.; Pang, A.; Yuen, S.: Unerupted maxillary anterior supernumerary teeth: report of 204 cases. J Dent Child, 51: 289-294, July-August 1984.
29. Ziberman, Y.; Malron, M.; Shteyer, A.: Assessment of 100 children in Jerusalem with supernumerary teeth in the premaxillary region. J Dent Child, 59:44-47, January-February 1992.
30. Langlais, R.P; Lang Land, O.E.; Morris, C.R.: Radiographic localization technics. Dent Radiog Photog, 52:69-77, 1979.
31. Munns, D.: Unerupted incisors. Br J Orthod, 8:39-42, January 1981.
32. McDonald, R.E. and Avery, D.R.: *Dentistry for the child and adolescent*. St.Louis: The C.V. Mosby Co., 1987, p 786.
33. Mitchell, L. and Bennett, T.G.: Supernumerary teeth causing delayed eruption—A retrospective study. Br J Orthod, 19:4146, February 1992.
34. Linder-Aronson, S. and Rolling, S.: *Pedodontics*. Magnusson, B.O. (ed). Copenhagen: Munksgaard, 1981, p 265.
35. Cangialosl, T.J.: Management of a maxillary central incisor impacted by a supernumerary tooth. J Am Dent Assoc, 105:812-814, November 1982.
36. Mills, J.R.E.: *Principles and practice of Orthodontics*. Edinburgh: Churchill Livingstone, 1987, pp 49-50, 147-154.
37. Howe, G.L.: *Minor oral surgery*. Bristol: Wright, 1985, pp 161-162.
38. Poyton, G.H.; Morgan, G.A.; Crouch, S.A: Recurring supernumerary mandibular premolars. Oral Surg, 13:964-966, August 1960.
39. Grahnen, H. and Lindahl, B.: Supernumerary teeth in the permanent dentition : A frequency study. Odont Revy, 12: 290-294, 1961.
40. Rubenstein, L.K; Lindauer, S.J.; Isaacson, R.J. *et al*: Development of supernumerary premolars in an orthodontic population. Oral Surg Oral Med Oral Path, 71:392-395, March 1991.
41. Proffit, W.R.: *Contemporary orthodontics*. St. Louis: C.V. Mosby Co., 1986.
42. Turner, C. and Hill, C.J.: Supernumerary mandibular premolar: The importance of radiographic interpretation. J Dent Child, 53: 375-377, September-October 1986.
43. Treasure, P. and O'Neill, N.M.: Late discovery and location of unerupted supplemental premolars—At what age to screen ? Dent Update, 17:431-433, December 1990.
44. Breckon, J.J.W. and Jones, S.P.: Late forming supernumeraries in the mandibular premolar region. Br J Orthod, 18:329-331, November 1991.
45. Ochlers, F.A.C.: Postpermanent premolars. Br Dent J, 93:157-158, 1952.

INTERCEPTION OF IMPACTION

Ericson and Kurol suggested that extraction of the deciduous canine is the treatment of choice to correct palatally erupting maxillary canines in individuals aged 10-13 years, provided that normal space conditions are present. In 36 of the 46 canines they investigated the initially palatal eruption path changed to normal and the canines finished in a clinically correct position. All 36 canines which improved did so within 12 months and were in clinically good positions at 18 months. Ninety-one percent of the canines which overlapped the adjacent lateral incisor root by less than half in the orthopantomogram (OPG) normalized. Sixty-four percent of the canines that overlapped the lateral incisor by more than half of the root initially, normalized. The degree of palatal position relative to the dental arch at the start of treatment also influenced the result. More canines with a moderate palatal path of eruption normalized than canines in a true palatal position (90 percent and 65 percent, respectively).

Jacobs, S.G.: Palatally impacted canines: aetiology of impaction and the scope of interception. Report of cases outside the guidelines for interception. Australian Dent J, 39:206-211, August 1994.

Turner's syndrome: Review of the literature and report of a case

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Zoltán Szilágyi, DDS
Gusztáv Keszthelyi, MD, LDS, MSc, PhD

A chromosome disorder is a microscopically visible chromosome abnormality. One child in every 200 live births has a chromosomal problem.[1] The incidence is considerably higher in stillbirths and spontaneous abortions. Factors that seem to predispose to chromosome aberrations include:

- ☐ Advanced maternal age.
- ☐ Radiation and cytotoxic drugs.
- ☐ Virus infections of the mother.
- ☐ Certain autoimmune diseases.

There are structural, numerical, and/or sex-chromosome abnormalities. Turner's syndrome (Turner-Albright syndrome; gonadal dysgenesis or agenesis; ovarian short-stature syndrome; genital dwarfism) was described in 1938.[2] This is a chromosomal abnormality with an additional deficiency in gonadal and adrenocorticotropic secretions. In 1959 Ford *et al* demonstrated that patients with this condition had only forty-five chromosomes, forty-four autosomes and one X chromosome.[3] The other sex chromosome is absent. A variant with an additional fragment has been described.[4] The characteristics of Turner's syndrome have also been found in many mosaics (chromosome complement of 45X in some cells and 46 XX in others).[1] Twenty-five per cent of patients with Turner's syndrome (45XO) are mosaic.[5] Kariotype (photomicroscopic chromosome analysis) is usually determined from blood lymphocytes.

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Turner's syndrome occurs in about 1 out of 5000 live births, and the frequency has been found to be extremely high in spontaneous abortions. It has been demonstrated that about 25 percent of abortuses with chromosomal aberrations are due to 45X chromosomal constitutions.[6] The most characteristic features are short stature, webbed neck (pterygium colli), cubitus valgus, sexual infantilism, and primary amenorrhea in females, broad shield-like chest with widely spaced nipples. Low posterior hairline recessing into the middle of the neck.[6] The axillary, pubic, and body hair is scanty or absent.[7] Lymphedema of extremities, coarctation of aorta and renal anomalies occasionally occur.[6,8] IQ is normal in the majority of patients.

Other changes often include lymphangiectatic edema of the hands and feet, digital anomalies, deformation of nails and ears (protruding auricles), ptosis of upper eyelids, epicanthi, cataracts, strabism and corneal clouding.[6] The most frequent oral findings in Turner's syndrome include high palatal vault and hypoplastic mandible.[3,9-12] Laine and Alvesalo in 1986 determined length and width of the alveolar arch of the mandible and maxilla from hard stone casts of 45X females.[13] Mandibular arch width was larger in relation to the maxillary arch. The total absolute length of the mandibular arch was slightly reduced in 45X females. The broader and shorter alveolar arch of the mandible in relation to the narrower but normal length maxillary arch reflects imbalanced facial growth in subjects with one X chromosome, instead of the normal two. Primary teeth of eleven Finnish 45X

females were examined and their sizes were found to be smaller than the corresponding teeth of control females. This finding might be explained by the decreased number of products of growth-promoting factors located in the X and Y chromosomes.[14]

The mouth also was found to be small with the corners pulled down by the pterygium colli producing the characteristic "sphinxlike" visage.[11]

Early tooth eruption has been reported.[1] Twins, one normal and the other with Turner's syndrome, have been studied. Patients with Turner's syndrome differ in their tooth-eruption patterns and in their craniofacial development, compared with the normal. In Turner's syndrome teeth have been found to be prematurely erupted, the first permanent molars erupting between one and one-half and four years of age with abnormally short roots[8,11]

Otherwise the teeth are small and may be somewhat widely spaced, because of a diminished number of teeth caused by aplasia or failure of eruption of certain permanent teeth. In some cases the primary teeth are retained because the permanent teeth fail to erupt. Takala *et al* in 1985 studied the prevalence of dental caries in permanent teeth (DFS) in fifty patients with Turner's syndrome and forty-one normal first-degree female relatives.

Caries prevalence was lower in 45X women than in the controls, more pronounced in the incisor region than in the regions of the posterior teeth.

The tongue may become furrowed, because of the size of the mouth in relation to the size of the dental arch.[7]

CASE REPORT

A six-year-old female presented at our department with her dental esthetic problem. At the first visit her facial dysmorphia was conspicuous: vertical cleft of the nose, and a nevus pigmentosus on its left side. Her ears were abnormally large and deformed (Figure). She had pterygium colli and epicanthus.

Micrognathia, especially the mandibular arch, resulted in an extremely high palate and an abnormal dental status in respect to her age, including a very early eruption of the incisor teeth (two to three years earlier than average). None of the teeth was carious. A pediatrician, on the basis of a chromosome analysis, determined the diagnose: Turner's syndrome, including facial dysmorphia, shield-like chest, and lymphedema of the foot. Because of a developmental defect she had frequent respiratory infections. Somatic development is otherwise

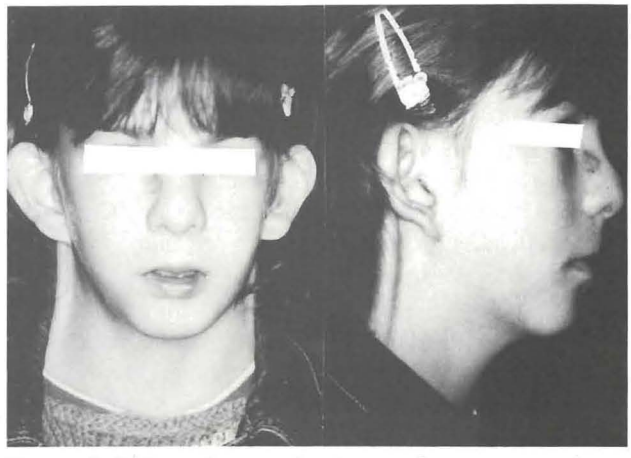

Figure. (left) Frontal view of patient with Turner's syndrome depicting "sphinxlike" face; (right) lateral view of the same patient.

appropriate. Her dyslalia was diagnosed by the otolaryngologist. According to the gynecological examination, the ultrasound showed sexual infantilism (in place of the uterus there is only a small mass of tissue and the ovaries are not separated). Estrogen was prescribed at the age of ten to eleven years.

After the clinical examination, radiographs and photographs were taken. Two primary teeth (67, 73) were removed because of considerable narrowing in the lower arch. The patient is recalled regularly for observation.

In order to treat the narrow of arches, upper and lower removable activated acrylic appliances were fabricated. Activating and checking of appliances are performed monthly. New appliances will be necessary in accordance with developmental changes (dentition, eruption). Obviously, not only the orthodontic abnormality, but the presence of periodontal disease and carious lesions should be detected and treated. The patient should be observed on a regular basis.

CONCLUSIONS

Turner's syndrome is a chromosomal abnormality with additional deficiency in gonadal and adrenocorticotropic hormones. Among the craniofacial anomalies the two main oral findings are a high palatal vault and a hypoplastic mandible. Caries prevalence is not very high, and teeth erupt early. Facial development is abnormal. The early enrollment of patients with Turner's syndrome into regular dental and orthodontic recall systems is a basic part of their therapies. Because of the developmental problems associated with the syndrome, orthodontic

anomalies frequently occur. The teeth of these patients are not more vulnerable to caries than those of the healthy population. Narrowing of dental arches and the subsequent malocclusion favor the accumulation of plaque and development of periodontal disease and dental decay. The orthodontic anomalies can promote the development of temporomandibular joint dysfunction. The child patient must be well motivated to wear the appliances and to practice good oral hygiene. Poor cooperation either delays the successful completion of the treatment or destines our efforts to total failure. The well-designed treatment plan, the scheduled extractions, and appropriate orthodontic appliances help to prevent more serious anomalies (tooth crowding, ectopy, torsion).

Because of these problems, the dentist plays a significant role in managing patients with Turner's syndrome.

REFERENCES

1. Loevy, H.T.: *Dental management of the child patient.* Chicago: Quintessence, 1981, pp 81-86.
2. Turner, H.H.: A syndrome of infantilism, congenital webbed neck, cubitus valgus. Endocrinology, 23:566-574, November 1938.
3. Ford, C.E.; Jones, K.W.; Polani, P.E. *et al*: A sex chromosomal anomaly in a case of gonadal dysgenesis (Turner 's Syndrome). Lancet, 1:711-713, April 1959.
4. Vaharu, T.: Gonadal dysplasia and enlarged phallus in a girl with 45 chromosomes plus "fragment". Lancet, 1:1351, June 1961.
5. Pinkham, J.R.: *Pediatric dentistry.* Philadelphia: W.B. Saunders Co., 1988, pp 187-188.
6. Stewart, R.E. and Prescott, G.H.: *Oral facial genetics.* Saint Louis: C.V. Mosby Co., 1976, pp 448-454.
7. Lyons, D.C.: *Oral and facial signs and symptoms of systemic diseases.* Springfield: Charles Thomas Publisher, 1968, pp 139-140.
8. Gorlin, R.J.: Chromosomal abnormalities and oral anomalies. J Dent Res, 42:Suppl.2, 1297-1306, 1963.
9. Wilkins, L. and Fleischmann, W.: Ovarian agenesis. J Clin Endocr, 4:357-375, August 1944.
10. Boue, D.C.; Boue, C.P.; Naves, R.J. *et al*: Turner's syndrome: controlling facial growth: Report of a case. J Int Assoc Dent Child, 14:21-30, June 1938.
11. Silver, H.K. and Kempe, C.H.: Ovarian agenesis (Congenital aplastic ovaries) in child. Amer J Dis Child, 85:523-530, May 1953.
12. Turner, E.K.: A case of probable ovarian agenesis with Turner syndrome in a female child aged seven months. Med J Aust, 1:39-42, January 1954.
13. Laine, T. and Alvesalo, L.: Size of alveolar arch of the mandible in relation to that of the maxilla in 45X females. J Dent Res, 65: 1432-1434, December 1986.
14. Kari, M.yesalo, L.; Manninen, K.: Sizes of deciduous teeth in 45X females. J Dent Res, 59:1382-1385, August 1980.
15. Takala, I.; Alvesalo, l.; Palin-Palikas, T. *et al*: Caries prevalence in Turner's syndrome (45X females). J Dent Res, 64:126-128, February 1985.

ANALYSIS OF DENTAL ADHESIVE SYSTEMS

With the exception of Gluma 2000, all of the dentine bonding systems evaluated appear to adapt better to visibly moist dentine.

Resin-infiltrated dentine layers are evident on the scanning electron micrographs. Although these are the primary mechanism by which adhesives bond to dentine, their actual clinical significance has not been determined. If the etching agent penetrates deeper than the primers, there is a zone of partially demineralised dentine that may be susceptible to microleakage phenomena. The performance of these systems may depend primarily on the depth of etching and subsequent infiltration of the monomers into etched dentine.

Transmission electron microscopy (TEM) could be used to further evaluate etching depth and monomer penetration.

Perdigao, J. and Swift, E.J.: Analysis of dental adhesive systems using scanning electron microscopy. Intern Dent J, 44:349-359, August 1994.

Unerupted second primary molars: Report of two cases

Seppo H.K. Järvinen, Dr Odont

The noneruption of a primary tooth is a rarity.[1] A review of the literature reveals only a few case reports of unerupted primary molars in children, teenagers or young adults.[2-9] Most of the unerupted teeth were mandibular second molars and discovered during routine radiographic examinations. In some cases an unerupted primary mandibular second molar has been positioned inferiorly to its permanent successor, the second premolar.[7-9]

In this paper two cases with an unerupted primary mandibular second molar will be presented. In one of them the second premolar was congenitally missing, in the other it was unerupted and positioned occlusally and lingually to the unerupted primary molar.

CASE REPORTS

Case 1

An eight-year-old Finnish girl was referred for orthodontic treatment because of mesial migration of the permanent mandibular left first molar. According to the clinical records, the primary mandibular left second molar was missing.

An orthopantomogram (Figure 1a) showed that the primary left mandibular second molar had not erupted. The well developed tooth was located near the lower border of the mandible. The radiograph revealed also an unerupted mandibular second premolar. The almost fully developed and mesially inclined crown of the tooth was positioned occlusally to the unerupted primary molar. The tooth was covered with alveolar bone, and the follicles of the two unerupted teeth seemed continuous. The clinical examination showed a firm enlargement on the lingual side of the alveolar bone in the area of the crown of the second premolar.

The medical history of the patient was unremarkable. No history of intraoral trauma or infection was recorded by the dentist or reported by the parents.

The unerupted primary molar was extracted surgically. Two years after the operation the developmental stage of the second premolar was nearly normal but the tooth was still mesially inclined (Figure 1b). Also the neighboring first permanent molar was tilted mesially and the first premolar had migrated distally. Ten months later, after orthodontically opening the space and surgically exposing the crown, the second premolar was erupted (Figure 1c). The crown of the tooth was well developed, but the root seemed to be late in development. The tooth was rotated about 30° mesiolingually, but the occlusal relationships were satisfactory.

Case 2

The patient, a seven-year-old Finnish boy, was referred for orthodontic treatment because of a slight mandibular prognathism and a tendency to an open bite. The clinical examination revealed that the primary left mandibular

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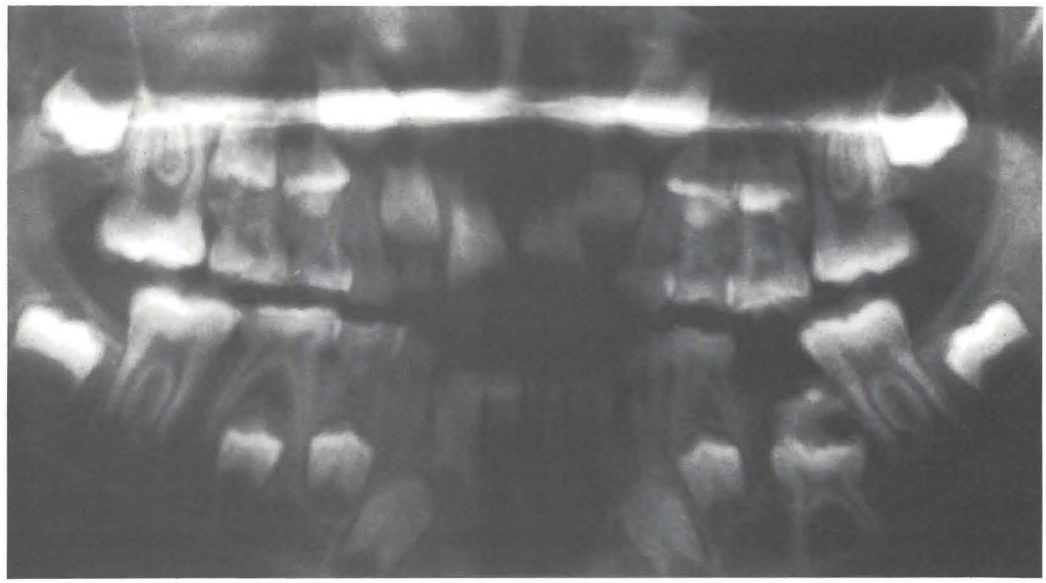

Figure 1a. The orthopantomogram taken at the age of eight shows the unerupted primary left mandibular second molar. The developing second premolar is positioned occlusally to the crown of the unerupted primary tooth.

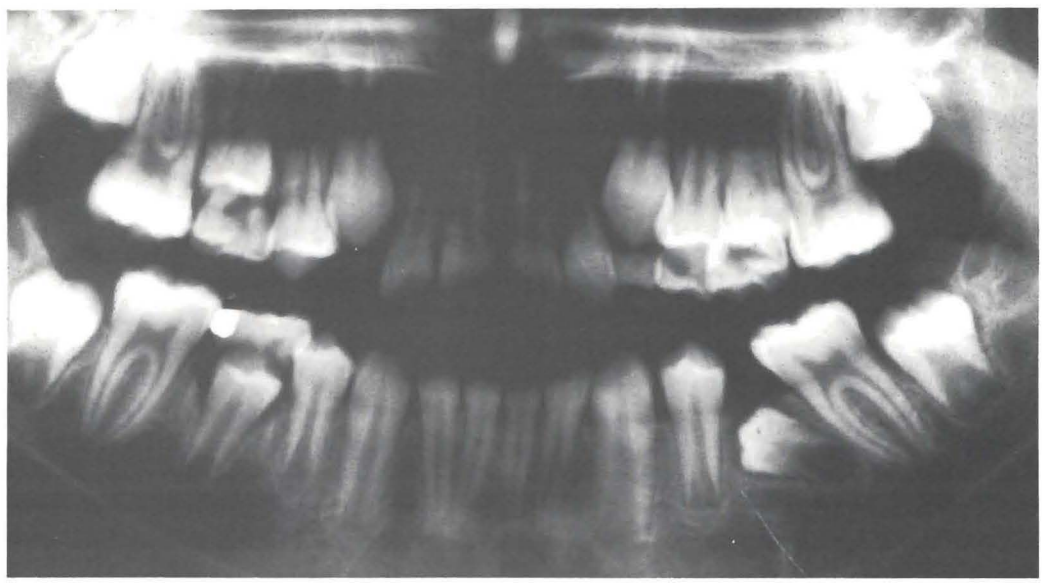

Figure 1b. At the age of ten, after surgical extraction of the unerupted primary molar, the developmental stage of the second premolar is nearly normal, but the crown of the tooth is still mesially inclined. The permanent first molar is also mesially inclined.

second molar was apparently missing. According to the earlier clinical records, the tooth had been found to be "missing" for the first time at the age of three years.

The radiographic examination revealed that the primary left mandibular second molar was unerupted (Figure 2). The distally inclined crown of the tooth was well developed, but the roots were remarkably short. The tooth had been covered with alveolar bone, but the bone

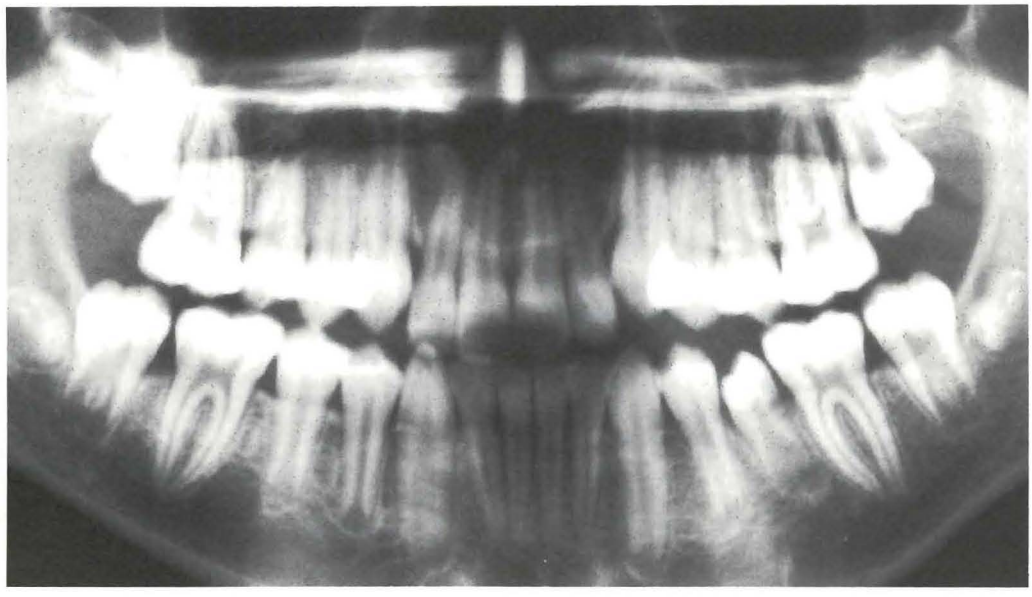

Figure 1c. At the age of eleven the mandibular left second premolar has erupted after orthodontic space opening and surgical exposure of the crown. The tooth is obviously late in development.

above the crown was fractured mesially. The mandibular left second premolar was congenitally missing.

The case history revealed no hereditary, environmental or developmental factors that could have been linked with the disturbance in dental development.

The unerupted primary molar was extracted surgically because of the obvious risk of pericoronal infection. A space maintainer was placed to prevent mesial tilting of the first permanent molar. The slight Class III occlusion was treated with an elastic open activator.

DISCUSSION

Primary molars can be found with their occlusal surfaces below the occlusal planes of the neighboring primary or permanent teeth. In the literature this condition has been most commonly called ankylosis, submergence or infraclusion. According to Kurol, a good description of the clinical appearance of the condition is given by the term "infraclusion".[10]

The degree of infraclusion can vary from a minor deviation below the occlusal plane to complete disappearance of the tooth within the alveolar bone. The infracluded tooth is one that was fully erupted and in occlusion, but correlation was lacking between its vertical movement and the growth of the alveolar process.[10] Infraclusion of primary molars has been found to be age-dependent. Its prevalence rises to a maximum (about 14 percent) in eight to nine-year-old children. The mandibular first molars are most commonly involved, almost twice as often as the mandibular second molars.[10]

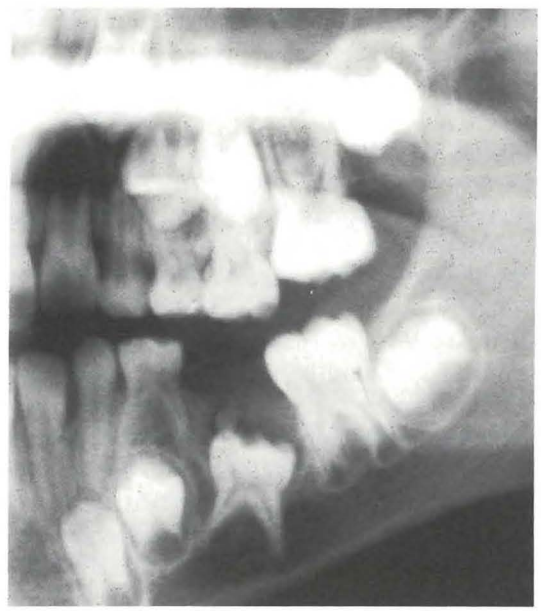

Figure 2. The orthopantomogram taken at the age of seven reveals the unerupted primary left mandibular second molar. The alveolar bone is fractured occlusally of the crown. The mandibular left second premolar is congenitally missing.

A tooth becomes embedded, if the alveolar bone grows and covers it.[11] This condition is the result of a disturbance in the continuous eruptive process (vertical drift) of the tooth during the childhood period.[12] The direct cause of infraclusion is ankylosis, but the origin of ankylosis is not clearly understood.[13]

Also an unerupted primary molar can be ankylosed.[6] It is to be expected that an ankylosed unerupted primary tooth will become more deeply covered with the alveolar bone during the growth period of the child. Noneruption cannot be caused by ankylosis, however, because ankylosis can occur only after root formation. It was suggested that noneruption of second primary molars could result from abnormal development of the primary molar germ or malposition of the second premolar anlage before one year of age.[9] The present Case 1 gives support to the latter theory.

According to Kurol and Koch, extractions of infracluded primary molars should be avoided, because the resorption of the roots and the exfoliation of the teeth will most often occur within the normal time.[13] One should distinguish, however, between infraclusion and noneruption of a primary molar, especially if there are other developmental anomalies involved. In Case 1 with the unerupted primary molar positioned below the second premolar, the normal resorption of the primary tooth was inhibited by the anomaly itself. In Case 2, the permanent successor of the unerupted primary molar was congenitally missing. Bruszt described a case of an unerupted primary maxillary second molar.[5] The tooth was resorbed almost completely during a nineteen-year observation period. No normal resorption of the unerupted teeth could be expected in the present cases, and hence the teeth were extracted, as was done in some of the cases reported in the literature.[3,6,9] Extraction of an unerupted primary molar is recommended, if it seems that normal eruption of the tooth cannot occur, or that the tooth is interfering with the development of the premolar, or if a risk of cyst formation exists.[6] The risk for pericoronal infection is also an indication for extraction.

Becker and Karnei-R'em found that infraclusion of primary molars was related to severe tilting of the two adjacent teeth toward the affected tooth, with minimal space loss in the dental arch at the occlusal level, but a wide separation of the root apices.[14] They also found that the degree of tilting was in relation to the degree of infraclusion. The patterns of tilting can be different in individual cases. Becker and Shochat reported a case of an eleven-year-old boy with an embedded primary left mandibular second molar. The tooth was covered with alveolar bone, and the permanent first molar had tilted mesially. After surgical removal of the embedded tooth, a complete space closure occurred as a result of mesial tilting of the first permanent molar and distal tilting of the first premolar. A similar loss of space could be seen in Case 1, but the situation could be corrected orthodontically, after which the second premolar erupted following surgical exposure of its crown.

Most of the unerupted primary mandibular second molars reported in the literature were found during routine radiographic examination.[6] As demonstrated earlier and in this report, an apparently "missing" primary molar can be unerupted. An unerupted primary molar should be extracted, but the time of extraction should be defined carefully by taking into consideration the development of the succedaneous premolar. Extractions made too early should be avoided, because a malformation of the succedaneous tooth-bud may result from surgical trauma. If the permanent successor is missing, the removal of the primary tooth can be delayed until the late mixed-dentition stage.

REFERENCES

1. Pindborg, J.J.: *Pathology of the dental hard tissues*. Philadelphia: W. B. Saunders Co, 1970, p.241.
2. Black, S.L. and Zallen, R.D.: An unusual case of deciduous impacted molar. Oral Surg, 43:160, January 1977.
3. Perri de Carvalho, A.C. and Sanches, M.G.: A rare case of impacted deciduous second molar. Oral Surg, 43:647, April 1977.
4. Brady, F.A. and Blum, M.: Unerupted deciduous mandibular molar. Oral Surg, 47:201, February 1979.
5. Bruszt, P.: Complete impacted upper second deciduous molar, a 19 year follow-up. Cited by Amir, E. and Duperon, D.F. Unerupted second primary molar. J Dent Child, 49:365-368, September-October 1982.
6. Amir, E. and Duperon, D.F.: Unerupted second primary molar. J Dent Child, 49:365-368, September-October 1982.
7. Bateman, R.C. and Emmering, T.E.: Deciduous impaction. Oral Surg, 42:852-853, December 1976.
8. Park, J.K.: Submerged impacted primary molar. Oral Surg, 48:383, October 1979.
9. Tsukamoto, S. and Braham, R.L.: Unerupted second primary molar positioned inferior to the second premolar: Clinical report. J Dent Child, 53:67-69, January-February 1986.
10. Kurol, J.: Infraclusion of primary molars: an epidemiologic and familial study. Community Dent Oral Epidemiol, 9:94-102, April 1981.
11. Stafne, E.C. and Gibilisco, J.A.: *Oral roentgenographic diagnosis*. Philadelphia: W.B. Saunders Co, 1975, p 50.
12. Enlow, D.H.: *Handbook of facial growth*. Philadelphia: W.B. Saunders Co, 1982, p 158.
13. Kurol, J. and Koch, G.: The effect of extraction of infraoccluded deciduous molars: A longitudinal study. Am J Orthod, 87:46-55, January 1985.
14. Becker, A. and Karnei-R'em, R.M.: The effects of infraocclusion: Part 1. Tilting of the adjacent teeth and local space loss. Am J Orthod Dentofac Orthop, 102:256-264, September 1992.
15. Becker, A. and Shochat, S.: Submergence of a deciduous tooth: Its ramifications on the dentition and treatment of the resulting malocclusion. Am J Orthod, 81:240-244, March 1982.

Bilateral pyogenic granuloma of the tongue in graft-versus-host disease: Report of case

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Pyogenic granuloma is a benign, well circumscribed, inflammatory lesion commonly found on the gingiva, lips, tongue, and buccal mucosa.[1-3] Though there is no significant age and gender predilection, some workers report predominant occurrences in the eleven-year-to-forty-year age-group and in females.[1-4] In the pediatric age-group, it occurs most frequently in early childhood, rarely in adolescents, and affects males more frequently than females.[5] Etiological factors are microbial, hormonal changes, trauma, poor oral hygiene, foreign bodies, vasculitis, and hypertension.[3,4,6,7] Typically, it grows to approximately 1x1 cm to 3x6 cm in several days or weeks, but a report of a 10x5x4 cm lesion has been made.[1,2,8] Clinical presentation is a pedunculated or sessile, exophytic growth with a lobulated or smooth surface, and may be accompanied by ulceration, spontaneous bleeding, mucinous exudate, and suppuration.[1,2,7,8] It is nontender and soft, and the color ranges from pink to deep red or purple to brown.[1,2,7] There may be evidence of alveolar bone resorption in longstanding lesions.[1] Differential diagnosis includes squamous cell carcinoma, verrucous carcinoma, fibrosarcoma, and peripheral giant cell granuloma.[9] Histologically the tissue comprises vascular connective tissue within an edematous matrix and occasional infiltrate of plasma cells and lymphocytes.[2,3,5] There is evidence of connective tissue synthesis and glycosaminoglycan, fibroblast and pericyte production. The rough endoplasmic reticulum is well developed and immature extracellular collagen fibers are present.[5] Diagnosis is confirmed by biopsy and histopathological examination.[1] Treatment is by surgical excision and removal of local irritants.[2,9] Recurrence is rare in nonpregnant individuals.[9]

Fanconi's anemia (FA) is the most common of the rare bone marrow failures.[10] It is inherited as an autosomal recessive disorder and is characterized by marrow hypoplasia and may be accompanied by skeletal, cardiac, and renal abnormalities, microcephaly, strabismus, genital hypoplasia, skin pigmentation, and mental retardation. Allogenic bone marrow transplant (BMT) is the only form of treatment for FA that carries a good prognosis and is available to less than 25 percent of these patients.[10,11]

Graft-versus-host disease (GVHD) is an immunological reaction following BMT. In a review of oral mucous membrane lesions in thirty-four BMT patients, ulceration, mucositis, angular cheilitis, candidiasis, herpes simplex viral infection, and gingival overgrowth were reported. The oral lesions are direct effects of chemotherapy and radiotherapy, myelosuppression and GVDH. The eight who had GVHD had lichenoid, reticular and papular mucosae, and atrophic or plaque-like tongue lesions. Histology of GVHD oral mucosa exhibits saw-toothed epithelium, vacuolar degeneration, focal keratinocyte necrosis, and lymphocytic infiltration.[12]

A search of the literature for pyogenic granuloma in Fanconi anemia, BMT recipients, GVHD patients or the

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immune suppressed yielded no documented reports. This report illustrates the occurrence of an unusual lesion in an individual with compromised immunity.

REPORT OF CASE

A fourteen-year-old Caucasian female with a history of FA treated by allogenic BMT with subsequent development of GVHD presented in the University of Minnesota Hospital Dental Clinic with complaints of growths on the tongue. The swellings had been present for three weeks and were painless. The BMT was performed four months before presentation in the clinic.

She had a history of asthma and was on Bactrim, cephalosporin, cyclosporin, cimetidine, Tylox, and prednisone.

Oral examination revealed pedunculated, firm, lobulated, nontender, yellow/buff/red-colored swellings on the right (3x3 cm) and left (2x1 cm) lateral borders of the tongue, midway anteroposteriorly (Figures 1, 2 and 3). The differential diagnosis was pyogenic granuloma, traumatic fibroma, and lymphoma. Excisional biopsy with a Luxar LX-20D CO_2 laser (Luxar Corporation, Bothel, WA) was performed under local anesthesia and submitted for histopathology and microscopic examination for organisms.

Histopathological examination reported pyogenic granulomas of the right and left tongue evidenced by reactive, subacute inflammatory response, featuring large numbers of capillaries within a loose, edematous connective tissue framework. An abundance of acute and chronic inflammatory cells was seen. The mucosal aspects were ulcerated. Neither specific etiologic organisms nor pathogenic fungi were observed.

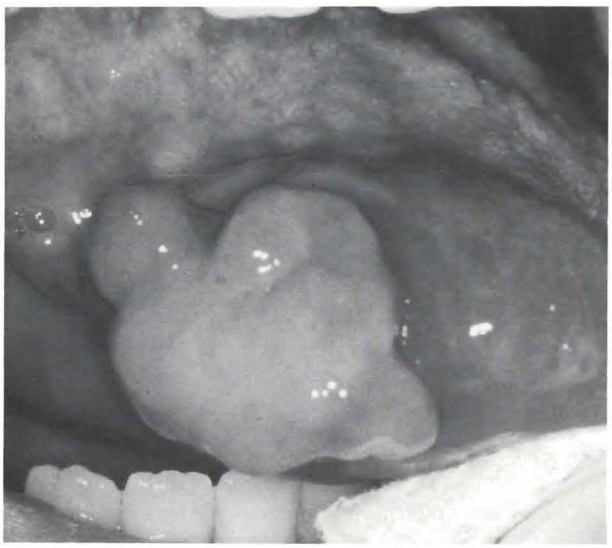

Figure 1. Pyogenic granuloma measuring 3x3 on right lateral border of tongue.

DISCUSSION

The purpose of this report is to sensitize pediatric dentists and other practitioners to unusual oral lesions in pediatric patients who are immune-suppressed, due to illness, medication, or viral infection.

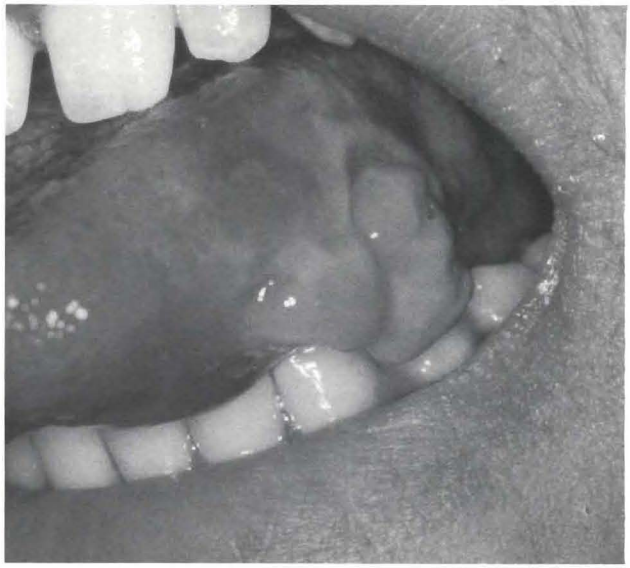

Figure 2. Similar but smaller (2x1 cm) lesion on left lateral border of tongue.

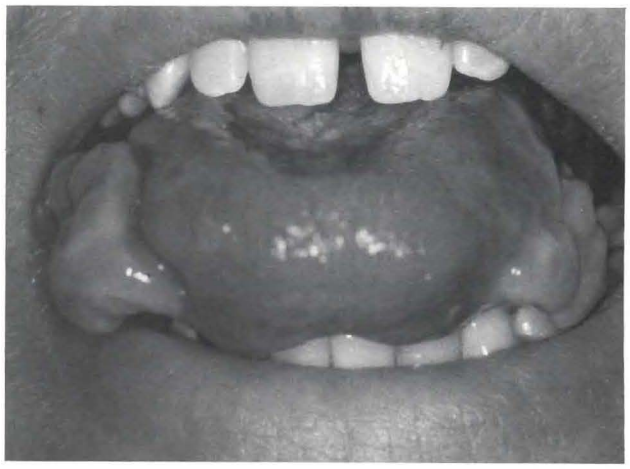

Figure 3. Left and right granulomas in direct occlusal relationship with mandibular teeth.

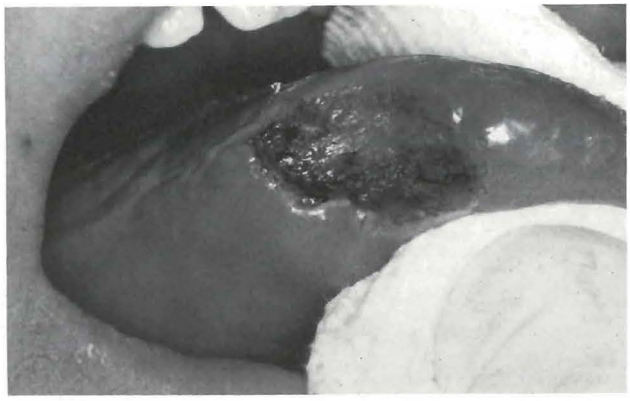

Figure 4. Right lateral border of tongue immediately postoperative, after treatment with CO_2 laser.

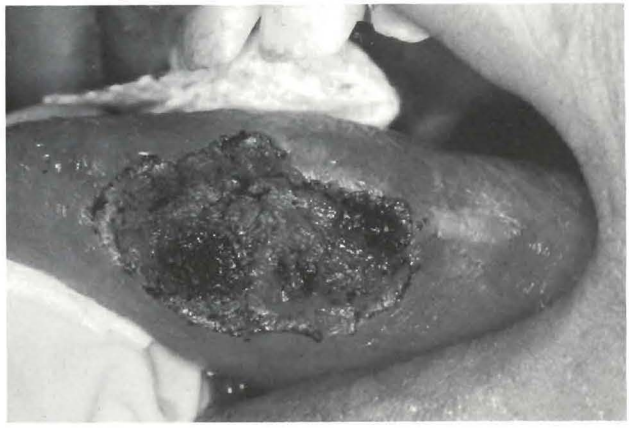

Figure 5. Postoperative CO_2 laser therapy wound on left lateral border of tongue.

Reports of pyogenic granuloma in young children and adolescents have failed to establish etiology, but strongly suggest hormonal influences and trauma.[5-7] The site of the lesions in this case is suggestive of traumatic injury from tongue biting. As the patient was in the pubertal stage, the possibility of hormonal influence exists. Patrice *et al* in their review of 178 pediatric cases, report, however, a linear decline in frequency with age and a 3:2 male to female ratio in conflict with the hormonal theory.[5] Davies *et al* in an ultrastructural study, found inclusion bodies in fibroblasts suggestive of gene derepression, possibly due to viral infection.[13] Superimposition of these etiologic factors on GVDH may have contributed to this rare occurrence.

As more patients survive BMT and GVDH, atypical lesions of the oral cavity are likely to be seen. Prompt recognition and appropriate management by the dental practitioner reduces the physical and psychological suffering these children experience.

REFERENCES

1. Angelopoulos A.P.: Pyogenic granuloma of the oral cavity: statistical analysis of its clinical features. J Oral Surg, 29:840-847, December 1971.
2. Shafer, W.G.; Hine ,M.K.; Levy, B.M.: *A Textbook of Oral Pathology*. Ed. 4. Philadelphia: W.B. Saunders Co., 1984, pp 359-361.
3. Baskhar, S.N.: Pyogenic granuloma, clinical features, incidence, histology and result of treatment: report of 242 cases. J Oral Surg, 24:391-398, September 1966.
4. Vilmann, A.; Vilmann, P.; Villman, H.: Pyogenic granuloma: evaluation of oral conditions. Br J Oral Maxillofac Surg, 24:376-382, October 1986.
5. Patrice, S.J.; Wiss, K.; Mulliken, J.B.: Pyogenic granuloma (lobular capillary hemangioma): a clinicopathological study of 178 cases. Pediatr Dermatol, 8:267-276, December 1991.
6. Papergeorge, M.B. and Doku, C.H.: An exaggerated response of intra-oral pyogenic granuloma during puberty. J Clin Pediatr Dent, 16:213-216, Spring 1992.
7. Mills, S.E.; Cooper, P.H.; Fechner, R.E.: Lobular capillary hemangioma: the underlying lesion of pyogenic granuloma. Am J Surg Pathol, 4:471-479, October 1980.
8. Chiong, A.M.; Chiong, C.M.; Dy, A.Y. *et al*: A massive pyogenic granuloma in the gingiva. Auris Nasus Larynx, 16: 227-231, 4, 1990.
9. Butler, E.J. and Macintyre, D.R.: Oral pyogenic granulomas. Dent Update, 19:194-195, June 1991.
10. Alter, B.P.: Fanconi's anemia. Am J Pediatr Hematol Oncol, 14: 170-176, May 1992.
11. Di Bartolomeo, P.; Di Girolamo, G.; Olioso, P. *et al*: Allogenic bone marrow transplantation for Fanconi anemia. Bone Marrow Transplant, 10:53-56, July 1992.
12. Dahllof, G.; Heimdahl, A.; Modeer, T. *et al*: Oral mucous membrane lesions in children treated with bone marrow transplantation. Scand J Dent Res, 97:268-277, June 1989.
13. Davies, M.G.; Barton, S.P.; Atai, F. *et al*: The abnormal dermis in pyogenic granuloma: histochemical and ultrastructural observations. J Am Acad Dermatol, 2:132-142, February 1980.

Orofacial manifestations in the Wiskott-Aldrich syndrome

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The Wiskott-Aldrich syndrome (WAS) is a rare x-linked recessive disorder principally characterized by immunodeficiency, eczema and thrombocytopenia.[1,2]

In the fully expressed form, the diagnosis is evident within the first year of life, and most patients succumb from infections or severe hemorrhage by about six years of age, unless bone marrow transplantation is successful.[3,4] There is a spectrum of disease severity and patients with milder forms of WAS may survive into adult life.[5,6]

There appear to have been few detailed reports of this syndrome in the dental literature and there are no reported associations between chronic bullous disease of childhood and Wiskott-Aldrich syndrome.

CASE REPORT

A four-month-old boy presented with a six-week history of an abdominal rash that had spread to his head, face, and limbs. His mother also reported that the child had a tendency to bruise easily. There was no family history of atopy or bleeding disorders and no suggestion of child abuse.

On initial examination the child was lively, alert, and apyrexial. There was a petechial rash over his trunk, face, arms, and legs. Hematological investigations confirmed the patient was thrombocytopenic with a platelet count of 49 x 10^9 /1. A bone marrow aspirate showed normal granulopoiesis and erythropoiesis and large numbers of megakaryocytes at all stages of maturation, indicating that the thrombocytopenia was due to peripheral destruction of platelets. No platelet autoantibodies were detectable at this time. The child remained thrombocytopenic and by two years of age had developed atopic eczema. He was found to have a low serum IgM level and a diagnosis of Wiskott-Aldrich syndrome was confirmed by the absence of the lymphocyte surface glycoprotein gpL 115 (Table 1).

Shortly after the definitive diagnosis of Wiskott-Aldrich syndrome had been made, the child developed a pruritic hemorrhagic vesiculobullous rash affecting the perineum, the limbs and trunk, and ulcers both on the dorsum of the tongue and in the buccal mucosa. The skin lesions resembled chronic bullous dermatosis of childhood, but the mother was reluctant for a biopsy to

Table 1 ☐ Investigations of Wiskott-Aldrich patient.

Bone marrow aspirate	Normal
Platelet antibodies	Negative
Serum autoantibodies	Reticulum positive
Serum immunoglobulins	IgG borderline low
	IgM low
	IgA normal
	IgE elevated
Lymphocyte surface protein gpL 115	Absent

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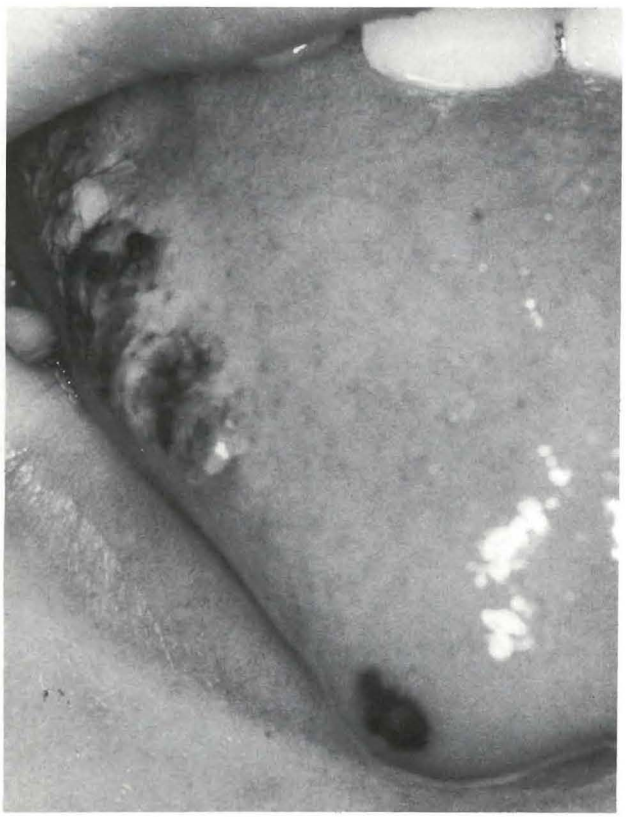

Figure 1. Purpuric lesions on the right lateral border of tongue.

be undertaken and a diagnosis, therefore, could not be confirmed histologically. Both the skin and the oral lesions rapidly responded to dapsone therapy (20 mg/daily); suggesting the diagnosis of chronic bullous disease of childhood, however, was probably appropriate. Skin and oral bullae have not recurred since dapsone was discontinued eighteen months ago.

In the two years following diagnosis of Wiskott-Aldrich syndrome the child has remained well, although his consistently low platelet count has caused frequent episodes of epistaxis, purpura, and ecchymosis, which have been treated by platelet transfusions. He has also had one episode of oral thrombocytopenic purpura that affected the tongue (Figure 1), floor of mouth, and buccal mucosa (Figure 2).

At present the child is in good health, having only had mild upper respiratory tract and middle ear infections and awaits a suitable bone marrow donation.

DISCUSSION

Wiskott-Aldrich syndrome (WAS) is a rare primary immunodeficiency, the cardinal clinical and immunological

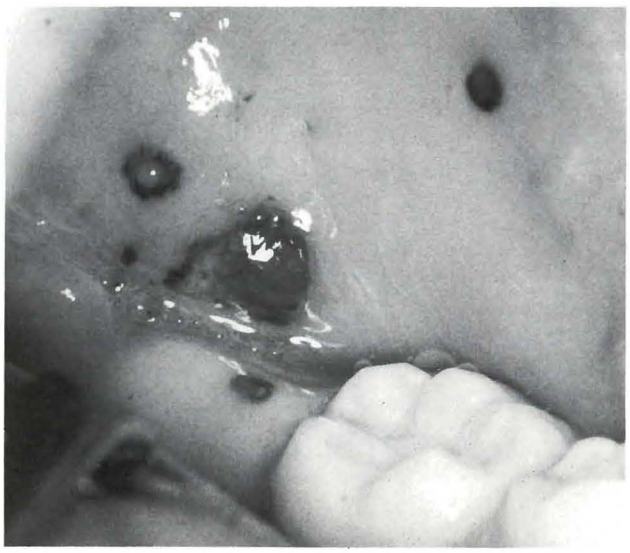

Figure 2. Purpuric lesions on the left buccal mucosa.

features of which are discussed below.

The present patient has had mucocutaneous features of both Wiskott-Aldrich syndrome and chronic bullous disease of childhood. To our knowledge this is the first report of the possible concurrence of these disorders and it is one of the very few reports of oral aspects of Wiskott-Aldrich syndrome.

Chronic bullous disease of childhood may precede adult linear IgA disease and affects the perioral and perineal surfaces, the trunk, and limbs.[7-9] There are linear deposits of IgA at the epitheliomesenchymal junction and circulating autoantibodies to epitheliomesenchymal junctional components can be found in some patients.[10-12] Although biopsy tissue was not available in the present case, the nature of the cutaneous lesions, presence of anti-reticulin antibodies, and good response to dapsone therapy strongly suggest that the diagnosis of chronic bullous disease of childhood was correct. A more detailed consideration of IgA bullous diseases is given elsewhere in the dental literature.[13,14]

Wiskott-Aldrich syndrome (WAS) has an incidence of approximately four per million live male births, the ef-

Table 2 ☐ Occasional clinical associations with Wiskott-Aldrich syndrome.

Nephropathy
Recurrent nonseptic arthritis
Asthma
Acute leukemia
Neutropenia
Lymphoid malignancy*
Monoclonal gammopathy without any obvious malignancy
Hemolytic anemia
Intracranial neoplasia

*Up to 12%

Table 3 ☐ Immune and platelet defects in Wiskott-Aldrich syndrome.

A. *Abnormalities in cellular immunity*
Progressive age-related T cell lymphopenia
Impaired *in-vivo* cell mediated immune function
Diminished mitogen and antigen specific responsiveness
Impaired production of migration inhibitor factor (MIF)
Lymphocytes with short, blunted or absent microvilli
Reduced CD4 cell number and CD4:CD8 ratio
B. *Abnormalities in humoral immunity*
Normal or raised IgE
Low serum IgM, levels of IgA and IgG
Diminished antibody responses to many antigens especially polysaccharides
Absence of isohemagglutinins
Hypercatabolism of immunoglobulins
Transient paraproteinemias
C. *Platelet abnormalities*
Shortened platelet half-life
Poor adhesiveness
Diminished aggregation in response to adrenaline, thrombin, collagen and ristocetin
Mean platelet volume reduced
Absence of the storage pool of adenosine nucleotides
Impaired mitochondrial ATP regeneration

fective gene lying on the short arm of the X chromosome.[4,14,15]

Clinical features of WAS typically appear in the first few months of life, characteristically with flexural eczema, which may be indistinguishable from typical atopic eczema.[15] Immunodeficiency gives rise to recurrent or persistent viral, fungal, protozoal, and bacterial infections.[4] Patients may suffer from skin abscesses, otitis media, and chronic sinusitis interspersed with life-threatening episodes of pneumonia, meningitis, and septicemia.[3] Infections with herpes varicella zoster virus and herpes simplex virus may occur and can be fatal.[16,17] Candidosis can be severe and intractable. A variety of other rare clinical problems can also occasionally arise (Table 2).

The bleeding tendency of WAS predisposes to life-threatening intracranial, pulmonary or gastrointestinal hemorrhage.[3] Bloody diarrhea is frequent and may result in anemia.[18] Thrombocytopenia is predominantly due to increased platelet destruction, although there may also be reduced platelet production.[19-21] There is an increased susceptibility to lymphoid malignancy in WAS, which may be due to the immune deficiency or altered expression of an oncogene in the vicinity of the WAS gene.[22]

Cell mediated dysfunction is the principal immunodeficiency in WAS although humoral abnormalities possibly reflecting underlying T cell dysfunction also occur (Table 3).[3,5,16,19,21-31] The associated impaired antibody response to capsular polysaccharide antigens may be thymus independent and indicative of a humoral defect.[16,32,33] Lymphocytes of WAS patients contain reduced amounts of the heavily sialiated cell surface glycoprotein gp1 115 (sialophorin) and platelets have reduced amounts of glycoproteins GP1a and 1b—important in platelet adhesion.[22,34-36] The reduction in sialophorin levels, loss of its rod-like structure or incomplete sialiation may impair cell-to-cell interactions, antigen recognition and processing, or regulation of cell senescence.[3,22,36] Sialophorin has carbohydrate side chains that are structurally similar to those of the platelet surface glycoprotein GP1b. It has thus been speculated that a defect in the metabolism of these common carbohydrate components underlies the lymphocyte and platelet defects of WAS, but this remains to be confirmed.[23,36]

The immune defects in WAS can be corrected by bone marrow transplantation after ablation of the recipient marrow by busulfan or total body irradiation.[37] In the absence of a suitable bone marrow donor, a more conventional treatment approach is required: infections can be managed with gammaglobulin therapy and prophylactic antibodies and the bleeding tendency may be corrected by corticosteroid therapy, splenectomy or platelet infusions.[5,20,38-40] Since the eczema may have an allergic basis, allergens, such as milk and eggs should be avoided and corticosteroids may be of some benefit.[3,41] Malignancy is treated with radiation and cytotoxic drugs but these predispose to fulminant infection.[42]

The exact frequency of oral lesions in Wiskott Aldrich syndrome is not known but palatal petechiae are common even when the skin is unaffected and there can be spontaneous gingival bleeding.[19-21,32,43,44] Oral ulceration has recently been observed in Wiskott—Aldrich syndrome.[45] This is the first report of which we are aware of buccal and lingual purpura in a patient with WAS.

In planning dental treatment, consideration should be given to anemia with regard to general anesthesia, the need for antibiotic and/or steroid cover, the risk of bleeding with oral surgical procedures, and rapid aggressive treatment of orofacial infections. It is also important not to administer drugs that impair platelet function or may produce an allergic response in these atopic patients.

Periodic oral examination of WAS patients is required not only for preventive dental measures, which are of paramount importance, but also for surveillance of oral manifestations that may indicate increasing disease severity and need for supportive care.

REFERENCES

1. Wiskott, A. Familiarer, angeborener Morbus Werlhofii? Mschr Kinderheilk, 68: 212-216, June 1937.
2. Aldrich, R.A.; Steinberg, A.G.; Campbell, D.C.:Pedigree demonstrating a sexlinked recessive condition characterized by draining ears, eczemoid dermatitis and bloody diarrhoea. Pediatric, 13:133-139, February 1954.

3. Standen, G.R.: Wiskott-Aldrich syndrome: new perspectives in pathogenesis and management. J Coll Physicians Lond, 22:80-83, April 1988.
4. Perry, G.S. III; Spector, B.D.; Schuman, L.M., *et al*: The Wiskott-Aldrich syndrome in the United States and Canada (1892-1979). J Pediatr, 97:72-78, July 1980.
5. Asherson, G.L.; Webster, A.D.B.: *Diagnosis and treatment of immunodeficiency diseases.* Oxford: Blackwell Scientific Publications, 1980, pp 240-249.
6. Mandl, M.A.J.; Watson, J.I.; Rose, B.: The Wiskott-Aldrich syndrome: Immunopathologic mechanisms and a long term survival. Ann Intern Med, 68:1050-1059, May 1968.
7. Marsden, R.A.; McKee, P.H.; Bhogal, B. *et al*: A study of benign chronic bullous dermatosis of childhood and comparison with dermatitis herpetiformis and bullous pemphigoid occurring in childhood. Clin Exp Dermatol, 5:159-176, June 1980.
8. Wojnarowska, F.; Marsden, R.A.; Bhogel, B.; Black, M.M.: Chronic bullous disease of childhood, childhood cicatricial pemphigoid and linear IgA disease of adults. A comparative study demonstrating clinical and immunopathologic overlap. J Am Acad Dermatol, 19:792-804, November 1988.
9. Chorzelski, T.; Jablonska, S.: Evolving concept of IgA linear dermatosis. Seminars in Dermatology, 7:225-232, September 1988.
10. Dabrowski, J.; Chorzelski, T.P.; Jablonska, S. *et al*: The ultrastructural localization of IgA deposits in chronic bullous disease of childhood (CBDC). J Invest Dermatol, 72: 291-295, June 1979.
11. Horiguchi, Y.; Toda, K.; Okamoto, H. *et al*: Immunoelectron-microscopic observations in a case of linear IgA bullous dermatosis of childhood. J Am Acad Dermatol, 14:593-599, April 1986.
12. Roberts, S.L.J.; Sontheimer, R.D.: Chronic bullous dermatosis of childhood. Immunopathologic studies. Pediatr Dermatol, 4:10-16, May 1987.
13. Porter, S.R.; Scully, C.; Midda, M.; Eveson, J.W.: Adult linear IgA disease manifesting as desquamative gingivitis. Oral Surg Oral Med Oral Pathol, 70:450-453, November 1990.
14. Porter, S.R.; Bain, S.E.; Scully, C.: Desquamative gingivitis secondary to adult linear IgA disease. Oral Surg Oral Med Oral Pathol, 74:179-182, August 1992.
15. Peacocke, M. and Siminovitch, K.A.: Linkage of the Wiskott-Aldrich syndrome with polymorphic DNA sequences from the human X chromosome. Proc Natl Acad Sci USA, 84:3430-3433, May 1987.
16. Cooper, M.D.; Chase, H.P.; Lowman, J.T. *et al*: Wiskott-Aldrich syndrome. An immunologic deficiency disease involving the afferent limb of immunity. Am J Med, 44:499-513, April 1968.
17. St. Geme, J.W. JR; Prince, J.T.; Burke, B.A. *et al*: Impaired cellular resistance to herpes simplex virus in Wiskott-Aldrich syndrome. N Engl J Med, 273:229-234, July 1965.
18. Spitler, L.E.; Levin, A.S.; Stites, D.P. *et al*: The Wiskott-Aldrich syndrome. Immunologic studies in nine patients and selected family members. Cell Immunol, 19: 201-218, October 1975.
19. Murphy, S.; Oski, F.A.; Naiman, J.L. *et al*: Platelet size and kinetics in hereditary and acquired thrombocytopenia. N Engl J Med, 286:499-504, March 1972.
20. Krivit, W.; Good, R.A.: Aldrich's syndrome (thrombocytopenia, eczema and infection in infants). Am J Dis Child, 97:137-153, February 1959.
21. Ochs, H.D.; Slichter, S.J.; Harker, L.A. *et al*: The Wiskott-Aldrich syndrome: studies of lymphocytes, granulocytes and platelets. Blood, 55:243-252, February 1980.
22. Remold-O'Donnell, E.; Zimmerman, C.; Kenney, D.M. *et al*: Expression on blood cells of sialophorin, the surface glycoprotein that is defective in Wiskott-Aldrich syndrome. Blood, 70:104-109, July 1987.
23. Pidard, D.; Didry, D.; Le Deist, F. *et al*: Analysis of the membrane glycoproteins of platelets in the Wiskott-Aldrich syndrome. Br J Haematol, 69:529-535, August 1988.
24. Oppenheim, J.J.; Blaese, R.M.; Waldmann, T.A.: Defective lymphocyte transformation and delayed hypersensitivity in Wiskott-Aldrich syndrome. J Immunol, 104:835-844, April 1970.
25. Wybran, J.; Levin, A.S.; Spitler, L.E. *et al*: Rosette-forming cells, immunologic deficiency diseases and transfer factor. N Engl J Med, 288:710-713, April 1973.
26. Kenney, D.; Cairns, L.; Remold-O'Donnell, E. *et al*: Morphological abnormalities in the lymphocytes of patients with the Wiskott-Aldrich syndrome. Blood, 68:1329-1332, October 1986.
27. Wade, N.A.; Lepow, M.L.; Veazey, J. *et al*: Progressive varicella in three patients with Wiskott-Aldrich syndrome: treatment with adenine arabinoside. Pediatrics, 75:672-675, August 1985.
28. Radl, J.; Dooran, L.J.; Morell, A. *et al*: Immunoglobulins and transient paraproteins in sera of patients with the Wiskott-Aldrich syndrome: a follow-up study. Clin Exp Dermatol, 25:256-263, August 1976.
29. Bellucci, S.; Tobelem, G.; Caen, J.P.: Inherited platelet disorders. Prog Haematol, 13:223-263, July 1983.
30. Grottum, K.A.; Hovig, T.; Holmsen, H. *et al*: Wiskott-Aldrich syndrome: qualitative platelet defects and short platelet survival. Br J Haematol, 17:373-388, October 1969.
31. Akkerman, J.W.N.; Van Brederode, W.; Gorter, G. *et al*: The Wiskott-Aldrich syndrome: studies on a possible defect in mitochondrial ATP resyntesis in platelets. Br J Haematol, 51:561-568, August 1982.
32. Mills, S.D. and Winkelmann, R.K.: Eczema, thrombocytopenic purpura and recurring infections. Archs Dermatol, 79:466-472, October 1959.
33. Blaese, R.M.; Strober, W.; Brown, R.S. *et al*: The Wiskott-Aldrich syndrome. A disorder with a possible defect in antigen processing or recognition. Lancet, i (18 May):1056-1061, 1968.
34. Parkman, R.; Remold-O'Donnell, E.; Kenney, D.M.; *et al*: Surface protein abnormalities in lymphocytes and platelets from patients with Wiskott-Aldrich syndrome. Lancet, ii (December 19-26): 1387-1389, 1981.
35. Mentzer, S.J.; Remold-O'Donnell, E.; Crimins, M.A.V. *et al*: Sialophorin, a surface sialoglycoprotein defective in the Wiskott-Aldrich syndrome, is involved in human T lymphocyte proliferation. J Exp Med, 165:1383-1392, May 1987.
36. Remold-O'Donnell, E.; Kenney, D.M.; Parkman, R. *et al*: Characterization of a human lymphocyte surface sialoglycoprotein that is defective in Wiskott-Aldrich syndrome. J Exp Med, 159:1705-1723, June 1984.
37. Parkman, R.; Rappeport, J.; Geha, R. *et al*: Complete correction of the Wiskott-Aldrich syndrome by allogeneic bone-marrow transplantation. N Engl J Med, 298: 921-927, April 1978.
38. Lum, L.G.; Tubergen, D.G.; Corash, L. *et al*: Splenectomy in the management of the thrombocytopenia of the Wiskott-Aldrich syndrome. N Engl J Med, 302:892-896, April 1980.
39. Pearson, H.A.; Schulman, N.R.; Oski, F.A. *et al*: Platelet survival in WiskottAldrich syndrome. J Pediatr, 68:754-760, May 1966.
40. Krivit, W.; Yunis, E.; White, J.G.: Platelet survival studies in Aldrich syndrome. Pediatrics, 37:339-341, February 1966.
41. Huntley, C.C. and Dees, S.C.: Eczema associated with thrombocytopenic purpura and purulent otits media. Pediatrics, 19:351-361, March 1957.
42. Faraci, R.P.; Hoffstrand, H.J.; Witebsky, F.G. *et al*: Malignant lymphoma of the jejunum in a patient with Wiskott-Aldrich syndrome. Surgical treatment. Arch Surg, 110: 218-220, February 1975.
43. Perino, K.E. and James, R.B.: Wiskott-Aldrich syndrome: review of literature and case report. J Oral Surg, 38:297-303, April 1980.
44. Boraz, R.A.: Dental considerations in the treatment of Wiskott-Aldrich syndrome: report of a case. J Dent Child, 56:225-227, May-June 1989.
45. Forsyth, K.; Matthews, S.C.; Seshadri, R. *et al*: Wiskott-Aldrich syndrome in identical twins: abnormality of CD4 and CD8 positive lymphocytes. Aust NZ J Med, 18: 73-76, February 1988.

ABSTRACTS

Vanderas, Apostole P.: Relationship between oral parafunctions and caraniomandibular dysfunction in children and adolescents: A review. J Dent Child, 61: 378-381, September-December 1994.

A review of the literature on the relationship between oral parafunctions and craniomandibular dysfunction is presented. The interpretation of the results was performed by applying the epidemiologic approach of "necessary and sufficient" which implies that there must be a one-to-one relationship between the factor and the disease. In all studies but one bruxism was constantly correlated with signs and/or symptoms of craniomandibular dysfunction indicating that a one-to-one relationship exists between this parafunction and the dysfunction of the masticatory system. Also, the reported correlations were highly significant. The strength and consistency of correlations support causality. The causal relationship between the rest of the oral parafunctions and craniomandibular dysfunction needs further investigation. On the basis of the evidence provided by the reviewed studies, bruxism cannot be considered as necessary, but a sufficient cause of craniomandibular dysfunction for a certain group of individuals.

Oral parafunctions; Craniomandibular dysfunction; Bruxism

Porter, S.R.; Sugerman, P.B.; Scully, C. et al: Orofacial manifestations in the Wiskott-Aldrich syndrome. J Dent Child, 61:404-407, September-December 1994.

The Wiskott-Aldrich (WAS) syndrome is a rare syndrome of thrombocytopenia, immunodeficiency, and eczema and hence is sometimes known as the TIE syndrome. Oral ulceration, petechiae and a bleeding tendency are the main orofacial manifestations but there are few detailed reports of this disorder in the dental literature. The present communication describes the clinical features of a child with mucocutaneous features of both Wiskott-Aldrich syndrome and chronic bullous disease of childhood.

Oral; Disease; Immunodeficiency; Wiskott-Aldrich syndrome

Järvinen, Seppo H.K.: Unerupted second primary molars: Report of two cases. J Dent Child, 61:397-400, September-December 1994.

Two cases with an unerupted primary left mandibular second molar are described. In one of them the succedaneous second premolar was congenitally missing, in the other the unerupted primary molar was positioned inferiorly and lingually to its permanent successor. Unerupted primary molars should be extracted, but the time of extraction should be defined carefully by taking into consideration the development of the succedaneous premolar and the space relations in the permanent dentition.

Unerupted primary molar; Extraction time

Wiedenfeld, Kenneth R.; Draughn, Robert A.; Welford, Joel B.: An esthetic technique for veneering anterior stainless steel crowns with composite resin. J Dent Child, 61: 321-326, September-December 1994.

The restoration of primary anterior teeth presents complicated esthetic and retention problems to the clinician. A technique is described for the chairside veneering of composite resin to stainless steel crowns, which results in well contoured restorations with superior durability and esthetics. A trimmed and fitted stainless steel crown can be veneered in three to five minutes. This provides the adaptability and gingival contour benefits of the stainless steel crown in conjunction with the cosmetics of the composite facings. The technique described produced composite veneers with a mean sheer bond strength of 3520 PSI (24.4 Mpa).

Composite resin veneer; Stainless steel crowns; Primary teeth

Faine, Mary P. and Oberg, Donna: Snacking and oral health habits of Washington state WIC children and their caregivers. J Dent Child, 61: 350-355, September-December 1994.

The purpose of this study was to assess the snacking and oral hygiene habits of Washington State WIC children and their caregivers. In a structured interview, 84 caregivers reported that their children, 12 to 48 months of age, were snacking two to three times daily. Nutritious foods—milk, fruit juice, crackers, bananas, apples and cheese—were the most frequendy reported snacks. However, 38 percent of children were given highly cariogenic raisins for snacks once or more times per week. Snack foods were purchased most often by caregivers because they were healhful or the child's favorite. Putting their child to bed with a bottle was a regular practice of one-fourth of the group. Caregivers stated that not brushing the teeth and sugar intake were the main causes of cavities. One-fourth of the children did not have their teeth cleaned by an adult and 17 percent did not appear to receive adequate fluoride. Tooth decay or bleeding gums when brushing were commonly reported dental problems of caregivers. One-half of WIC caregivers had not visited a dentist in the last year, whereas, 91 percent of WIC children had never had a dental examination.

Diet and dental caries; Dental nutrition; WIC program

Weinstein, Philip; Domoto, Peter; Koday, Mark; Leroux, Brian: Results of a promising open trial to prevent baby bottle tooth decay: A fluoride varnish study. J Dent Child, 61:338-341, September-December 1994.

One hundred thirty-three farm worker children with an average age of 17

months participated in a study of a fluoride varnish in WIC programs in the Yakima Valley of Central Washington. After parents were interviewed, the child was examined and a fluoride varnish applied to the maxillary incisors. At the six-month recall 62 children and their parents returned. Results indicated a significant decrease in decalcification from 35 percent to 21 percent and an increase in decay from 3 percent to 16 percent. The rate of decay was lower then the 30 percent found in this age-group in this population. Of 130 sound teeth at baseline, 13 percent were decayed or decalcified in six months; of 73 decalcified teeth at baseline, 51 percent were found to be sound in six months. Additional experimentation with fluoride varnish is warranted.

Fluoride varnish; Primary maxillary incisors

Lee, Chad; Rezaiamira, Nadia; Jeffcott, Ellen *et al*: Teaching parents at WIC clinics to examine their high caries-risk babies. J Dent Child, 61: 347-349, September-December 1994.

Seventy-seven parents/caretakers of children, averaging 24.4 months from four WIC clinics in King County, Washington, observed a five-minute videotape. These parents/caretakers were then asked to examine their own children, using a mirror and a brochure with illustrations of the progression of disease. Following completion of a questionnaire, a trained dental student or dentist examined the child and completed the same questionnaire. Over 80 percent of parents/caretakers responded the same as dentists. Dentists identified disease in 35.1 percent of the sample. Kappa = 59, Z = 4.4, and $p < .001$. Results support the training of nondental personnel to screen early childhood caries.

Dental examination; Parents; Dentists

Rizk, Salwa P.: Falling between the cracks: Oral health survey of school children ages five to thirteen having limited access to dental services. J Dent Child, 61:356-360, September-December 1994.

A survey of 367 Indianapolis school children, ages five to thirteen, revealed a group of children who have the least probability of receiving dental care, because of severe financial barriers in family income. These children belong to the lowest socioeconomic level of families whose income is not low enough to qualify them for Medicaid coverage. These children have higher rates of caries prevalence and gingivitis, and less filled teeth than children who belong to the same low socioeconomic group but whose families receive Medicaid or insurance coverage.

Children without Medicaid; Access to dental care

da Fonseca, Marcio A. and Mueller, William A.: Hallerman-Streiff syndrome: Case report and recommendations for dental care. J Dent Child, 61:334-337, September-December 1994.

Hallerman-Streiff syndrome is a rare congenital anomaly characterized by a peculiar bird facies, mandibular and maxillary hypoplasia, dyscephaly, cataracts, microphtalmia, hypotrichosis, skin atrophy, and short stature. Dental abnormalities are present in 80 percent of the cases and include malocclusion, crowding, severe caries, supernumerary and neonatal teeth, enamel hypoplasia, hypodontia, premature eruption of primary dentition, agenesis of permanent teeth, and anterior displacement or absence of condyles.

Very few cases have been described in the dental literature. The predisposition to severe caries, together with other problems, makes it imperative that young patients be started in a strong prevention program as early as possible. This is a case report of a five-year-eleven-month-old white male who presented for a dental examination at The Children's Hospital in Denver, CO. The findings and recommendations for treatment are discussed.

Hallerman-Streiff syndrome; Dental characteristics; Prevention program

Domoto, Peter; Weinstein, Philip; Leroux, Brian *et al*: White spots caries in Mexican-American toddlers and parental preference for various strategies. J Dent Child, 61:342-346, September-December 1994.

One hundred and thirty Mexican-American children ages nine to thirty-four months (Mean = 17.1 months), and their parents/caretakers were studied at a farmworkers clinic in rural Washington. Parents/caretakers participated in a bilingual interview; then each child received a dental examination, and a cotton swab was used to collect plaque for a caries activity test (Cariostat). Results showed that 7 percent had at least one maxillary incisor decayed and 30.4 percent had at least one incisor with a white spot lesion. The Cariostat was related to dental age, but not to disease. Forty percent of the parents/caretakers whose children had disease were aware of the problem. Immediate substitution of cup for bottle and the elimination of extra nighttime feedings were the least likely interventions endorsed by the subjects. Other interventions, including periodic visits for fluoride applications were much more likely.

Cariostat (caries activity test); Nighttime feedings; Fluoride application