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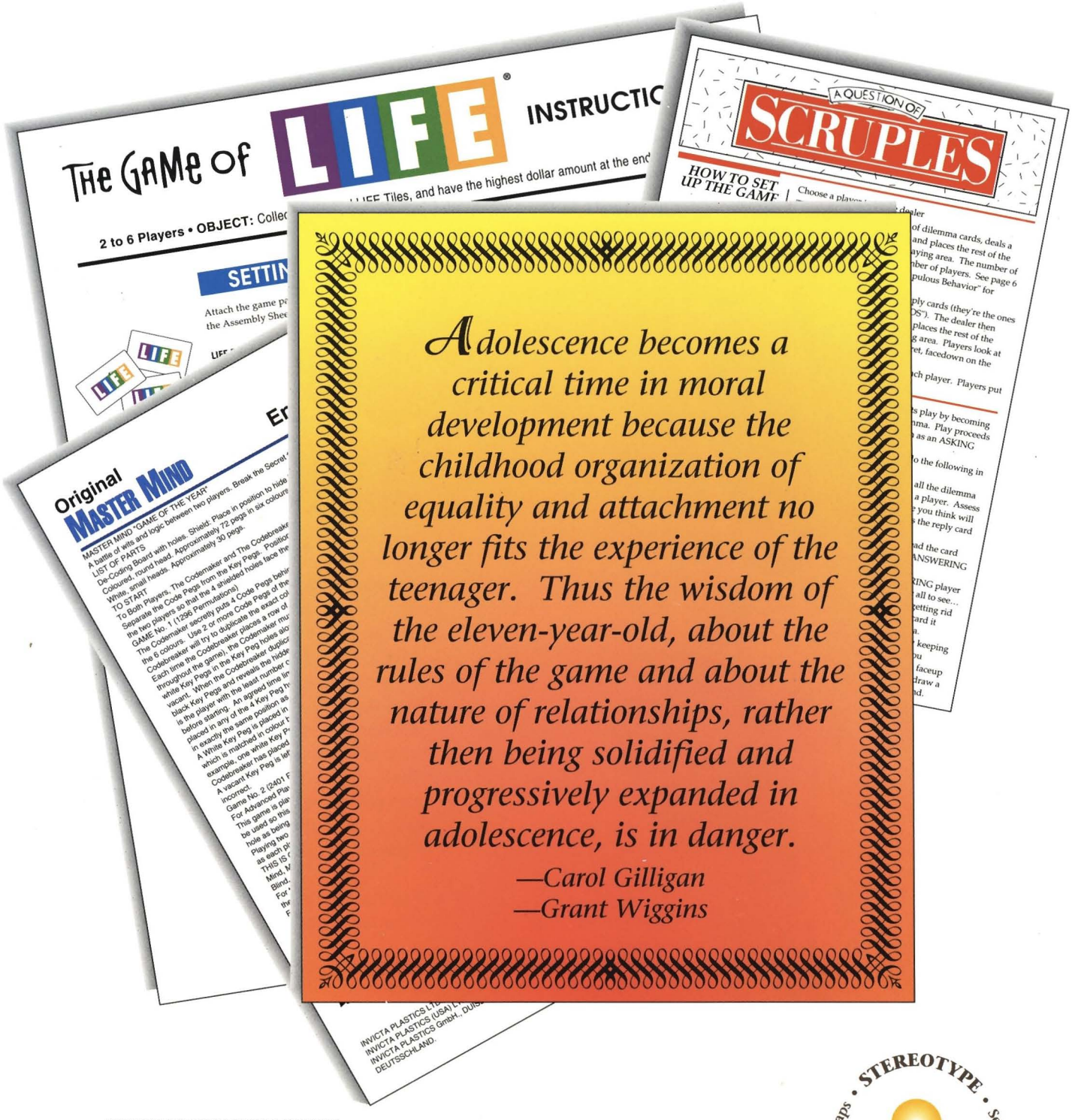
AMERICAN SOCIETY OF DENTISTRY FOR CHILDREN
JOURNAL OF DENTISTRY FOR CHILDREN
MARCH—APRIL 1995



VERACITY IS THE HEART OF MORALITY

—Thomas Henry Huxley

Handicaps • STEREOTYPE • Sexism
Children in Crisis

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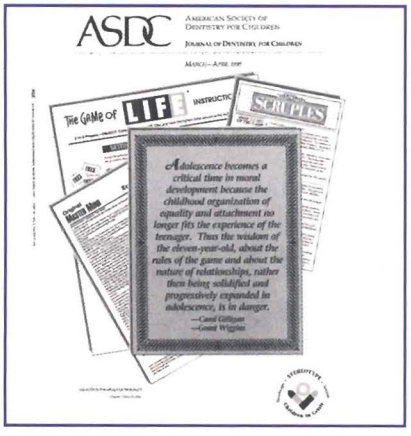

Little do we realize the influences that we have on children and their easy discernment of moral corruption among the adults in their environment.

Art and design by Sharlene Nowak-Stellmach

CLINIC

DEMOGRAPHICS

REPORTS

Calendar

1995

ASDC Annual Meeting, Palm Springs, CA, October 11-15
So. CA ASDC Unit, Red Lion Hotel, Culver City, May 23
Pennsylvania ASDC Unit, Westin William Penn, Pittsburgh, PA, June 9 & 10
Indiana ASDC Unit, French Lick, IN, June 10 & 11
International Assoc. Paediatric Dent, Góteborg, Sweden, June 8-11
Florida ASDC Unit, Gainesville, FL, September 8 & 9
American Conference of Dental Management of HIV Disease, ADA, Fairmont Hotel, Chicago, June 16-18

1996

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

1997

ASDC Annual Meeting, site to be determined, late October

For the busy reader

Neurological assessment of the child with head trauma—page 93

Approximately 25,000 children die in the United States each year from head injury. Because dental trauma is a subset of head trauma, the emergency appearance of a child in the dental office may present as a true medical emergency.

Requests for reprints should be directed to: Martin J. Davis, Columbia University, Dental & Oral Surgery, 630 W. 168th Street, New York, NY 10032.

Cariostatic and ultraconservative sealed restorations: Nine-year results among children and adults—page 97

Sealants have been available to the dental profession since 1965. Despite their documented success, they are still underutilized. The objections to their use were initially legitimate, but good research has proved the effectiveness of the currently recommended sealants. This study began nine years ago and covers a period of great activity in the use of these materials.

Requests for reprints to: Dr. Eva Mertz-Fairhurst, c/o Ms. Debbie Nunley, Dental Materials Group, Department of Oral Rehabilitation, MCG School of Dentistry, Augusta, GA 30912-1260.

A three-year follow-up of glass ionomer cement and resin fissure sealants— page 108

The authors compared the three-year clinical durability of the fissure-sealant glass ionomer cement with that of a chemically cured BIS-GMA resin. Retention rates for the resin were significantly better.

Requests for reprints to: Dr. G. Karlzén-Reuterving, FTV (Public Dental Health Service) Dental School Umeå, Umeå University, 901 87 Umeå, Sweden.

In vitro studies of the effect of a dental explorer on the formation of an artificial carious lesion—page 111

The early carious white-spot lesion can remineralize completely, provided that no mechanical damage has occurred. the author reports on the effect of using a dental explorer in dental examinations.

Requests for reprints to: Dr. Othman M. Yassin, P.O. Box 31 Al-Sareeh, 21156 Irbid, Jordan.

Validity of the mother's recall of her child's antibiotic use—page 118

The authors evaluated the validity of mother's recall of the child's use of antibiotics by comparing it with the child's health records, and also examined the potential determinants of the mother's recall of the child's use of antiobiotics.

Requests for reprints to: Dr. Ananda Dasanayake, Department of Oral Biology, School of Dentistry, University of Alabama at Birmingham, University Station, Birmingham, AL 35294.

Natal and neonatal teeth—page 123

Teeth present at birth are called *natal* teeth; those that erupt within thirty days after birth, are called neonatal teeth. A further classification refers to their degree of maturity: A mature natal or neonatal tooth is one that shows normal development; an immature one has immature development.

Requests for reprints to: Dr. Jian-Fu Zhu, The University of Texas, Health Science Center at San Antonio, 7703 Floyd Curl Drive, San Antonio, TX 78284-7888.

Glanzmann's thrombasthenia: The use of autologous fibrin glue in tooth extractions—page 129

The authors report on dental extractions in a thrombasthenic girl, without the use of platelet transfusion and with the use of autologous fibrin glue.

Requests for reprints to: Dr. Meir Rakocz, Division of Pediatric and Hospital Dentistry, The Chaim Sheba Medical Center, Tel Hashomer 52621, Israel.

Can your next pediatric patient hear you?—page 132

Few of us consider the consequences of hearing loss. But almost a million youths between the ages of three and seventeen years have hearing difficulties and of these, approximately 143,000 cannot hear and understand normal speech. It is important to remember that 4.1 million people (20.9 percent of all persons with hearing problems) report that the onset of their hearing problems occurred during childhood.

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

Asian American and Pacific Islander children: They will become an increasing reality in your practice—page 136

The 1990 U.S. Census indicates that as a result of a growing number of Asian and other immigrants, together with continuing increased numbers of births in different minority population groups, there are changes in the racial mixture of the nation that is more dramatic in the past decade than in any other time in the 20th century.

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

Radiographic considerations for supernumerary tooth extraction: Report of case—page 141

The authors discuss a rare case combining supernumerary teeth and dental fusion. The authors suggest an appropriate radiographic protocol for the extraction of supernumerary teeth.

Requests for reprints to: Dr. Curtis J. Creath, Department of Community and Public Health Dentistry, School of Dentistry, University of Alabama at Birmingham, 1919 Seventh Avenue, South, Birmingham, AL 35294.

Monostotic fibrous dysplasia in an eight-year-old male: Report of case—page 145

Reported is a condition that appears in the first decade of life. It has an obscure etiology, uncertain pathogenesis, and diverse histopathology.

Requests for reprints to: Dr. Noeen Arshad, 88 Greaton Road, West Roxbury, MA 02132.

Oral findings in Michelin Tire syndrome: Report of case—page 148

This a rare genetic disease whose primary clinical presentation includes symmetrical, ringed creases around all extremities and torso.

Requests for reprints to: Dr. Mark L. Helpin, Chairman, Department of Pediatric Dentistry, University of Pennsylvania, School of Dental Medicine, 4001 Spruce Street, Philadelphia, PA 19104-6003.

CLINIC

Neurological assessment of the child with head trauma

Martin J. Davis, BA, DDS
Lillian Vogel, BA, DMD

It is estimated that 30 percent of all children in the United States will, at some time, experience dental trauma.[1] Since dental trauma is a subset of head trauma, the emergency appearance of a child in the dental office may present as a true medical emergency.[2] Approximately 25,000 children die in the United States each year from head injury. In addition, it is a major cause of physical disabilities, seizure disorders, and developmental delay in children.[3]

A force that is strong enough to fracture, intrude, or avulse a tooth in a child is clearly strong enough to result in cervical spine or intracranial injury.[4] A critical need exists at the time of presentation in the dental office to triage the child who may have head and/or cervical injuries. The dental practitioner must be particularly alert to a number of difficult-to-diagnose problems associated with head trauma. Among these are acute subdural hematoma, skull fracture, facial bone fractures of the orbit, zygoma or midface, and subcondylar fracture. Internal bleeding or compromise of brain stem function may become obvious slowly over several hours or even days subsequent to the traumatic incident. This is true of the subdural hematoma, one of the most difficult to manage sequelae of head injury.[5] An acute subdural collection of blood is typically associated with laceration or contusion of the brain. Prospects for recovery are poor; the fatality rate approximates 90 percent.[6]

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EPIDEMIOLOGY

The incidence of dental trauma and head injury is escalating due to increased participation by both girls and boys in such sports as soccer, field hockey, lacrosse, volleyball, softball, football, basketball, skating, bicycling, and motorbiking.[7] Zeng recently reported that 60 percent of the visits at Seattle Children's Hospital from 1982-1991 were the result of trauma; further, the number of visits was 2.1 times greater in 1991 than in 1982.[8] Duus observed that 17 percent of children assessed in the hospital for so-called "minor" head injuries actually had intracranial complications.[9] Male children have about a 50 percent higher occurrence of head trauma than do female children, but the individual female sports participant has a generally higher incidence of injury. Collegiate female basketball players have an orofacial injury rate of 7.48 percent, for soccer 3.11 percent, for field hockey 2.48 percent, for lacrosse 2.27 percent, for volley ball 1.59 percent and for softball 1.55 percent. This compares with a rate of 0.5 percent for male football players.[10]

Dental practitioners must play an important role in prevention of head-trauma sequelae through encouraging patients, both female and male, to wear properly constructed and well-fitted mouthguards. Mouthguards not only offer protection from orofacial injury, including

fractures of teeth and jaws, but they also provide significant cushioning of blows to the chin, thereby obtunding potential cervical and intracranial injury.[11]

CLOSED HEAD-INJURIES

The most common type of head-injury in children and adolescents is the closed head-injury. In closed head-injury, serious damage may result from the brain oscillating within the skull, and in the process stretching or severing neural pathways within the brain or brain stem. Contusions and hematomas may form from the impact of the brain against the inner table of the skull. Brain lesions such as contusions, hematomas, and the mechanical shearing of neural pathways are considered to be primary injuries. Such crushing and shearing injuries may rapidly manifest signs or symptoms of closed head-injury. Alternatively, these injuries also create conditions that only appear as sequelae over time. This latter group of slowly evolving sequelae demands that particular attention be paid to the neurological status of a child presenting with dental injuries.[12] Critical to a positive prognosis for the patient with closed head-injury is the early effective triage and, when indicated, aggressive treatment in a special intensive care facility.

While primary injury is related to mechanical forces, secondary injury is related to physiologic disruption that can compromise otherwise viable cells. Only recently has the importance of aggressive, immediate treatment directed at interrupting and attenuating secondary types of subcellular, metabolic, and ultrastructural derangement been fully appreciated. These are the processes that render neurons and astrocytes of the brain extremely vulnerable to damage and destruction.[3] Common pathophysiologic factors in brain trauma include intracranial hypertension and cerebral ischemia, disturbances that are responsible in large part for secondary injury and cell death during the first few days following a head injury. Another pathophysiologic process responsible for tissue loss is secondary cerebral hypoperfusion due to posttraumatic cerebrovascular dysfunction.[3,12]

There is a reported clustering of dental trauma incidents at age one and again at age six.[13] This pattern reflects distinct physical and intellectual stages in a child's development. As an infant learns to walk and run, his or her balance is affected by having a greater body mass above the waist. The size of the infant's head is disproportionately large, as compared to body size, making the infant "top-heavy" and subject to falls. The propensity for a six-year-old to injure his head results in part from the child becoming more independent, expanding relationships and interests, and being exposed to more environmental hazards such as playground injuries.

OFFICE NEUROLOGICAL ASSESSMENT

In their earliest years, children may have difficulty communicating. Even at a later age, frightened and emotionally distressed children may report symptoms inadequately and may be unaware of the seriousness of their injuries.[2] Moreover, their emotional state may mirror that of the accompanying parent or guardian, a factor that can further complicate the diagnostic process. Unhurried and gentle handling of these children is critical in assuring cooperation and a favorable treatment outcome.[14]

If the child presents after head-injury to the dental office, rather than to a hospital, the child almost without exception will be conscious. For reference, a scale exists to describe comatose patients, the Glascow Coma Scale (GCS). It provides a practical means for monitoring changes in the level of consciousness based on eye opening and verbal and motor responses. The lowest score is 3; the highest is 15. Scores of 8 or lower are associated with permanent cognitive, emotional, and behavioral problems. It may be impossible to distinguish between mild and severe head-injury on initial examination. The patient's course after injury, as documented by sequential examinations, can truly reflect the original severity of injury. If in doubt about the significance of the injury, follow the safest course and refer the patient to the emergency department of a hospital, where the child can be evaluated and admitted for observation if necessary.

Following the two basic guidelines of all emergency management: "primum non nocere", first do no harm, and secondly, "never treat a stranger", a proper medical history must be obtained. Information should be secured about a loss of consciousness, neck or head pain, and numbness anywhere in the body. Assuming that the child is conscious, the patient should be asked to provide a history of the traumatic event. Amnesia of the episode may be indicative of a previous loss of consciousness. Additional signs meriting immediate concern include nausea, vomiting, drowsiness, or blurred vision. The child should be tested for "oriented x 3" and should respond appropriately when asked: "What is your name?", "What place is this?", "Is it morning, afternoon, or night time?" These queries should be made with sensitivity to the child's age, communication level and emotional overlay at the moment. The head should be carefully examined for signs of trauma such as scalp laceration, hematoma, and evidence of skull fracture. Blood

or clear fluid draining from the ear (otorrhea) or the nose (rhinorrhea) should alert the dentist to the possibility of a cerebrospinal fluid leak due to the presence of skull fracture. Temperature, pulse, respiratory rate and blood pressure should be recorded. A particularly slow pulse (bradycardia) in the presence of an elevated systolic blood pressure with widening between the levels of systolic and diastolic pressures is an indicator of rising intracranial pressure.

Certain of the Cranial Nerves (C.N.) should be assessed for normal function. Most important are C.N. III, IV, Vl, and VII. The oculomotor nerve, C.N. III, can be tested by observing whether "Pupils are Equal, Round, and Responsive to Light and Accommodate" (PERRLA). Failure of the pupils to constrict or the presence of eyelid ptosis suggest that damage has occurred to the third cranial nerve. "Extraocular Movements Intact", or EOMI, provides a rapid assessment of C.N. III, IV, and Vl. The dentist should check to see whether the child is able to track the examiner's finger as it moves laterally and vertically across the visual field. Failure of the child to track the dentist's finger may indicate that the midbrain or pons had been injured.[15] Abnormal ocular function or pupil alignment may signal impending cerebral herniation. Head trauma frequently involves C.N. VII, the facial nerve. Routine testing of motor function involves examination of the strength of facial muscles by asking the patient to close her eyes, smile and then frown. Facial muscle movements should be symmetrical. If a lesion is suspected, taste also may be impaired and should be assessed.

Damage to C.N. I, II, V, VIII, and IX through XII is uncommon. Testing the olfactory nerve, C.N. I, involves having the patient identify various scents while the eyes are closed. When testing children, familiar nonoffensive scents such as coffee or peppermint should be used. Testing the optic nerve, C.N. II, includes a light flashed in the eye. Loss of both direct and consensual pupillary constriction in an eye is diagnostic of damage, as is the absence of the blink reflex. Asking the patient to identify a letter or color is an additional test. Direct injury to the branches of the trigeminal nerve, C.N. V, causes a loss of touch or pain sensation and the experience of paresthesia. These are so troubling to the patient that evaluation is almost mandatory. A cotton swab or finger is brushed successively against each side of the forehead (ophthalmic branch), cheek (maxillary branch) and lower lip (mandibular branch). The patient is asked whether the sensation is of equal intensity bilaterally; if it is not, C.N. V probably has been harmed.[16] Injury to C.N. VIII, the acoustic nerve, will be evident through a loss of balance (vestibular nerve) or hearing (cochlear nerve). Spontaneous nystagmus is an objective sign of vertigo. The precise measurement of hearing is outside the scope of the usual dental office; gross hearing can be tested, nevertheless, by the dentist whispering into one of the patient's ears or rubbing the fingers while covering the patient's other ear. These simple tests will detect significant hearing loss.

Cranial Nerves IX through XII rarely sustain damage as a result of head trauma. Of possible clinical relevance to the dentist are C.N. IX and XII. Cranial nerve IX, the glossopharyngeal nerve, is tested by stimulating the soft palate to elicit the "gag reflex". Cranial nerve XII, the hypoglossal nerve, is responsible for motor functions of the tongue and is tested by asking the patient to extrude the tongue. Lateral deviation of the tongue suggests potential injury of the medulla.

These simple tests will facilitate the rapid evaluation of cranial nerve function in the dental office. The preceding neurologic assessment can be accomplished in a matter of minutes and will assist the practitioner in ruling out impending neurologic crisis subsequent to head trauma.

POSTTREATMENT PRECAUTIONS

If the history of the accident seems incompatible with severe brain-injury, the neurological examination is within normal limits, and necessary dental emergency care has been provided, the pediatric dental patient may be discharged to home provided that the patient is accompanied by a parent or reliable adult. The parent or guardian must understand the need for careful observation of the child at home and the following reasons why the patient should be brought to the hospital without delay: somnolence that cannot be easily interrupted, protracted vomiting, severe headache, and any other observation or symptomatic complaint thought to be abnormal by the adult. The patient should be scheduled to be seen by the dentist within a few days for a follow-up appointment.

LEGAL ASPECTS

The dental practitioner should be aware of the necessity for detailed chart entries regarding head trauma with dental involvement. The chart should contain the patient's medical and dental histories, a record of the incident, diagnoses and treatment rendered. The record may be a critical reference for the dentist asked to appear as an expert witness in future litigation or as the

Table ☐ Gross neurological examination summary.

- ☐ Medical history
- ☐ History of traumatic incident
  - ○ Loss of consciousness
  - ○ Amnesia of event
- ☐ Nausea, vomiting, drowsiness
- ☐ Blurred vision
- ☐ "Oriented × 3"
- ☐ Skull fracture; CSF leakage
- ☐ Lacerations; facial bone fracture
- ☐ Temperature, pulse, blood pressure, respiratory rate
- ☐ Cranial nerve examination
  - ○ EOMI
  - ○ PERRLA
  - ○ Sensory function
  - ○ Motor function
- ☐ Post-op instructions

subject of a professional liability claim. It is exceedingly important that the dental clinician keep accurate records of how the trauma occurred, as reported by the patient and parent, the nature and findings of the neurological examination, and how the patient was subsequently managed.

The use of a "trauma chart insert", placed in the patient's permanent record, that leads the practitioner through proper triage, an orderly neurological assessment, and dental treatment is strongly urged (see Table). Such forms are available generally and from the authors upon request.

SUMMARY

Dental trauma is a type of head trauma. It is essential that the dentist be able to assess the gross neurological status of the child presenting with head injury and to recognize acute and delayed signs of nerve injury. Time does not permit the dentist a leisurely review of necessary skills at the time of an emergency visit. He or she must be prepared in advance with a consistent and simple approach for the management of head trauma in children. Time of entry into care is critical to the prognosis.

REFERENCES

1. Andreasson, J.O. and Andreasson, F.M: *Textbook and color atlas of traumatic injuries to the teeth.* Third Edition, Copenhagen: Munksgaard, 1991, pp 19-45.
2. Hunter, J.G.: Pediatric maxillofacial trauma. Pediatr Emerg Med Pediatr Clin N.A., 39:1127-1143, October 1992.
3. Ghajar, J. and Hariri, R: Management of pediatric head injury. Ped Emerg Med Ped Clin N.A., 39:1093-1125, October 1992.
4. Kopel, H.M. and Johnson, R: Examination and neurologic assessment of children with oro-facial trauma. Endodo Dent Traumatol, 1:155-159, October 1985.
5. Phuenpathom, N.; Choomuant, M.; Ratanalert, S.: Outcome and outcome prediction in acute subdural hematoma. Surg Neurol, 40: 22-25, July 1993.
6. Baker, E.T. and Wepsic, J.G.: Neurosurgical Emergencies in Wilkins, Jr., E.W. *et al* (Eds.). *MGH Textbook of Emergency Medicine*, Baltimore: Williams & Wilkins, 1983, p. 602.
7. Pinkham, J. and Kohn, D.: Epidemiology and prediction of sports related traumatic injuries. Dent Clin N.A., 35:609-625, October 1991.
8. Zeng, Y.; Sheller, B.; Milgrom, P.: Epidemiology of dental emergency visits to an urban children's hospital. Pediatr Dent, 16:419-423, November-December 1994.
9. Duus, B.R.; Boesen, T.; Kruse, K.V. *et al*: Prognostic signs in the evaluation of patients with minor head injury. Br J Surg, 80:988-991, 1993.
10. Kumamoto, D.P.: Sports dentistry. Compend Contin Educ Dent., XIV:492-501, 1992.
11. Ranalli, D.N.: Sports dentistry. Dent Clin N.A., Philadelphia: W.B. Saunders Co., 35:609-858, 1991.
12. Johnston, M.V. and Gerring, J.P.: Head trauma and its sequelae. Pediatr Annals, 21:362-368, June 1992.
13. Henry, P.C.; Hauber, R.P.; Rice, M: Factors associated with closed head injury in a pediatric population. J Neuroscien, 24:311-316, December 1992.
14. Kaban, L.B.: Diagnosis and treatment of fractures of the facial bones in children 1943-1993. J Oral Maxillofac Surg, 51:722-729, July 1993.
15. DeJong, R.N.; Sahs, A.L.; Knight, K.V. *et al: Essentials of the neurological examination.* Philadelphia: Smith Kline and French, 1962.
16. Croll, T.P.; Brooks, E.B.; Schut, L. *et al*: Rapid neurologic assessment and initial management for the patient with traumatic dental injuries. JADA, 100:530-534, April 1980.

PEDIATRIC EQUESTRIAN INJURIES

During the two-year period of the study, 32 children were evaluated. Two children were injured when a horse stepped on them. Thirty children fell from or were thrown from a horse. Of these, 20 were wearing a helmet. Head injuries were more frequent in those patients not wearing helmets. The mean Modified Injury Severity Scale (MISS) score for riders without a helmet (12.9) was significantly higher (more severe) than that for helmeted riders (2.8). All three patients with a Glascow Coma Score <15 on arrival were not wearing a helmet at the time of injury. The frequency of hospitalization was significantly higher for those not wearing a helmet. Compared with other common mechanisms of childhood injury the mean Modified Injury Severity Scale score of injured riders was exceeded only by that of pedestrians struck by a car.

Bond, G.R. *et al*: Pediatric equestrian injuries: Assessing the impact of helmet use. Pediatrics, 95:487-489, April 1995.

Cariostatic and ultraconservative sealed restorations: Nine-year results among children and adults

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Pit and fissure sealants have been available to the dental profession since 1965.[1] Despite their documented success, they remain underutilized as a means of preventing Class I decay, the most common form of dental caries.[2,3] The initial reticence of dentists to use these products may have stemmed from several sources:

- ☐ A dentist's orientation toward restoration rather than prevention.
- ☐ Inadequate understanding of the meticulous technique required.
- ☐ Poor performance by some of the first-generation products.

- ☐ A concern for the possibility of sealing in clinically undetectable caries.

Training in the placement of sealants is now a part of every dental school curriculum. The technique and philosophy of sealant use are well understood, and the current generation of sealants have well-documented success rates. The concern for lesion progression beneath sealants has been addressed by numerous studies that have documented the arrest of caries beneath intact lesions.[4-20]

Despite this wealth of information, sealants are still underutilized.[3,21,22] In particular, therapeutic use of sealants has received only cursory exploration by general practitioners and pediatric dentists.

This paper reports the nine-year results of a study whose aim was:

- ☐ To evaluate the long-term success of arresting caries in pit and fissure lesions by placement of ultraconservative, sealed composite restorations.
- ☐ To compare the clinical performance of minimal (nonextended) sealed amalgam restorations versus amalgam restorations with the traditional Class I cavity outline form.

MATERIALS AND METHODS

The study design has been described previously.[14-18] A brief description follows: 123 patients were selected because they had one or more tooth pairs with frank Class I caries with radiographic extension into dentin. There were eighty females and forty-three males.

At baseline, there were five children, ages 8-10, twenty-eight children ages 11-15, and six children, ages 16-18. Thus, there were thirty-nine (32 percent) children and eighty-four (68 percent) adults ages 19-52. The ratio of children to adults (39 to 84) was approximately 1:2. The median age of all subjects was twenty-three years.

A tooth pair consisted of two permanent molars or two premolars. A total of 131 molar pairs and twenty-five premolar pairs was accepted. Seventeen patients had two pairs of study teeth, five had three pairs, one had four pairs, and one had five pairs; the remaining ninety-nine patients had only one pair of study teeth.

Each patient received a sealed composite restoration placed over active caries (CompS/C) that was paired either with a localized, nonextended sealed amalgam restoration (AGS) or with the traditional *extended for prevention* unsealed amalgam restoration (AGU). At baseline, 156 CompS/C restorations were paired with seventy-seven AGS and seventy-nine AGU restorations. Restorations were placed by nine operators, all dental school faculty, who had been instructed in the restorative techniques. Rubber dam was used for all but a few procedures in which it could not be adequately adapted.

The tooth preparation for the CompS/C restoration consisted only of a bevel in enamel around the entire periphery of the carious lesion. The bevel was at least 1 mm wide and placed in sound enamel. The demineralized, chalky enamel was removed until translucent, sound enamel was reached. No removal of caries below the bevel was attempted. Enamel undermined by the carious lesion was left intact. The bevel and the entire occlusal surface were etched for sixty seconds with a liquid etchant (37 percent phosphoric acid), rinsed, dried, and the enamel bonding agent was placed. A radiopaque self-cured composite material (Miradapt®—Johnson & Johnson Dental Products Company, East Windsor NJ) was placed with hand instruments. Additional contouring was carried out with a bur, if necessary. The sealant (Delton® Tinted [yellow] - Johnson & Johnson Dental Products Company, East Windsor, NJ) was then placed over the restoration and all major pits and fissures. The only retention form for this restoration was the etched bevel and the surrounding etched enamel. Some retention of the restoration could possibly be attributed to the sealant placed over it.[20]

The tooth preparation for the AGS restoration was localized to the carious area with no extension for prevention, since sealant was used for caries prevention in the adjacent pits and fissures. All caries was removed, and the pulpal floor was prepared in sound dentin. The localized preparation had a pear-shaped retention form and its cavosurface margins were 90°. After the amalgam restoration was placed, the entire occlusal surface including the amalgam restoration was etched for sixty seconds. The yellow sealant was placed over the localized amalgam restoration and all the remaining pits and fissures.

The traditional, unsealed AGU restorations were placed in preparations that removed not only the carious lesion but were also extended to include the remaining major pits and fissures.

The amalgam used in both the AGS and AGU groups was a high-copper product (Dispersalloy®—Johnson & Johnson Dental Products Company, East Windsor, NJ). After placement, the amalgam restorations were carved and burnished. No polishing procedures were performed.

Clinical evaluations of the three treatments were made by a team of fifteen calibrated dentists, using modified Ryge criteria (Table 1). Table 2 presents the criteria

Table 1 □ Modified Ryge criteria for sealed and unsealed Class 1 restorations.

Rating	Color match	Marginal discoloration	Marginal integrity	Anatomic form	Caries at margin
OSCAR	———	———	Restoration is fully sealed	———	———
OSCAR/ALFA	———	———	Restoration is partly sealed; no open margin*	———	———
ALFA	Good match with mirror and dental light	No marginal discoloration	No sealant; no open margin	Not undercontoured, not discontinuous	No caries at margin
OSCAR/BRAVO	———	———	Restoration is partly sealed; open margin present.** No dentin nor base is exposed	———	———
BRAVO	Mis-match, but within normal (tooth-like range)	Discoloration, but not penetrating toward pulp	No sealant; open margin present.** No dentin nor base is exposed	Undercontoured, discontinuous, but neither dentin nor base is exposed	———
CHARLIE	Mis-match outside normal (outside tooth-like range)	Discoloration penetrating toward pulp	Crevice** so deep that dentin or base is exposed	Sufficient material is lost (due to wear) so that dentin or base is exposed	Caries present at margin
DELTA	———	———	Dentin or base is exposed and restoration is mobile, fractured, or missing	Sufficient material is lost (due to previously observed wear) so that the restoration is mobile, fractured, or missing	Caries around the restoration is so extensive that the restoration is mobile, fractured, or missing
HOTEL	Restoration is metallic and color evaluations do not apply; or restoration is fully sealed with yellow sealant and color evaluations cannot be made		———	Restoration is fully sealed and discontinuities cannot be evaluated nor be expected "to be caused by wear"	———
NA	Not applicable:	ratings that involve sealant (such as OSCAR, OSCAR/ALFA, or OSCAR/BRAVO) but do not apply and cannot be used for unsealed restorations, such as the AGU group in this study			

*No open margin = no visible crevice; no explorer catch, or explorer catch one way only (ALFA Marginal Integrity)
**"Open margin" = "crevice:" these terms are identical and interchangeable; they denote a visible, V-shaped crevice at margin, with explorer catch both ways (BRAVO marginal integrity)
Note: OSCAR, ALFA, BRAVO and any combination thereof are considered as CLINICALLY ACCEPTABLE ratings. CHARLIE and DELTA are CLINICALLY UNACCEPTABLE ratings: clinical failures that need immediate attention

Table 2 □ Modified Ryge criteria for a sealant placed over a Class I restoration and all occlusal pits and fissures.

Rating	Marginal integrity of sealant	Clinical efficacy of sealant
ALFA	No bubble or void at the sealant margin; no sudden discontinuity or fracture of sealant at its margin	No caries at sealant margin nor in any previously sealed pits or fissures
BRAVO	A bubble or void present at the sealant margin; or a sudden discontinuity due to fracture of sealant at its margin	———
CHARLIE	———	Caries present at sealant margin or in any occlusal pit or fissure (but not at margin of a study restoration). This finding was considered the *Clinical failure of sealant* in caries prevention

used for clinical evaluations of the marginal integrity of sealant and for sealant effectiveness in caries prevention.

At baseline and each subsequent evaluation, two examiners evaluated each restoration independently. At the end of each patient's evaluation, the person recording the Ryge calls would look for any disagreements. Any disagreement was resolved by the two examiners by reaching a consensus. The evaluators were well calibrated. Usually the disagreements were due to an oversight by one of the examiners. Rarely a third evaluator would be asked to evaluate the tooth.

RESULTS

At year 9, fifty-seven patients (46 percent of the original sample) returned for examination: seventy-five CompS/C, forty AGS, and thirty-five AGU teeth were evaluated.

Among children who were eight to eighteen years old at baseline, twenty-three CompS/C, eleven AGS, and twelve AGU teeth were evaluated. Thus the ratio of paired restorations in children was twenty-three versus fifty-two pairs in adults, retaining a ratio of 1 :2 at year 9.

A clinical example of a CompS/C study tooth is shown in sequential intraoral photographs and radiographs in Figures 1-6. The patient was a fifteen-year old girl who received two separate CompS/C restorations in the central and distal areas of the permanent maxillary left first

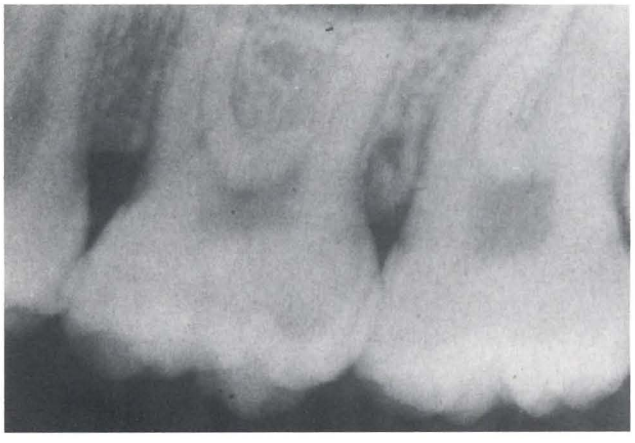


Figure 1. At baseline: A fifteen-year old female patient presented with clinically obvious occlusal caries in the central and distal pit areas of the permanent maxillary left first molar (tooth #14).
Figure 1a. Preoperative radiograph confirmed that this first molar had two separate carious lesions in the central and distal pit areas.

Figure 1b. The intraoral photograph shows that the tooth preparation consisted of placing only a bevel around the periphery of each carious lesion and that the caries below the bevel was not removed; also, enamel undermined by caries was likewise not removed. (Note that the intraoral photograph showing the mirror-image of tooth #14 was made with a dental mirror. The metal rim of the dental mirror has cut off most of the mesiofacial surfaces of this tooth, so that part of the large mesiolingual cusp as well as the entire mesiofacial cusp are not visible. Thus in this photograph, the tooth appears narrower on the buccal side than on the lingual side. There is a tendency at first glance to identify, therefore, the right side of the photograph as the lingual side of the tooth. Bearing this in mind, it is important to keep the orientation of the distolingual groove (DLG) as a reference area, indicating the lingual side of this tooth when viewing the subsequent photographs: the lingual side of this tooth is on the reader's left side when viewing these clinical photographs).

molar. The clinical performance of this tooth was satisfactory over the nine-year period: all the margins of the two localized restorations remained completely sealed, but there was a bare area in the middle of each of the restorations. With regard to the sealant ratings, there was a bare pit on the distolingual area of the occlusal surface. The color of the sealant remained yellow in some areas and became more tooth-colored in other areas. The sequential radiographs of this tooth do not show any obvious progress of the two study lesions. These findings are typical for the CompS/C group, i.e., the carious lesions appear dormant and do not progress.

Sealant retention over restorations and the incidence of open margins (Table 3)

The Marginal Integrity Ryge criterion in this study pertains not only to the status of the margins but also includes the degree of sealant retention over the margins of sealed study restorations. Table 3 demonstrates the proportion of restorations with closed and open margins. The sealed restorations are further subdivided into groups with complete or partial retention or no sealant.

Complete sealant retention was found in 28 percent of the CompS/C restorations and in 22.5 percent of the AGS restorations. Partial sealant retention with no open margin was observed in 41.3 percent of the CompS/C restorations and in 62.5 percent of the AGS restorations. Among the AGU restorations, 57.1 percent had no open margins.

Partial sealant retention with an open margin occurred in 6.7 percent of CompS/C restorations and in 7.5 percent of AGS restorations. Among the AGU restorations, 31.4 percent had open margins. As compared to the AGS group, the higher incidence of open margin in the AGU group was significant (Pearson Chi-Square, $P = 0.007$). The distribution of open margins is illustrated in the graph (Figure 7).

Table 4 shows sealant retention over margins of study restorations. The margins of 33.3 percent of the CompS/C and 22.5 percent of the AGS restorations remained completely sealed after nine years. Sealant retention

Figure 2. At 6 months:
Figure 2a. Radiographically, both lesions are definitely visible above the two separate radiopaque CompS/C restorations.

Figure 2b. Clinically, note the orange-yellow color of the sealant. Both restorations remained completely sealed. (Note that the large mesiolingual cusp and the mesiofacial cusp are fully visible here and at all subsequent visits).

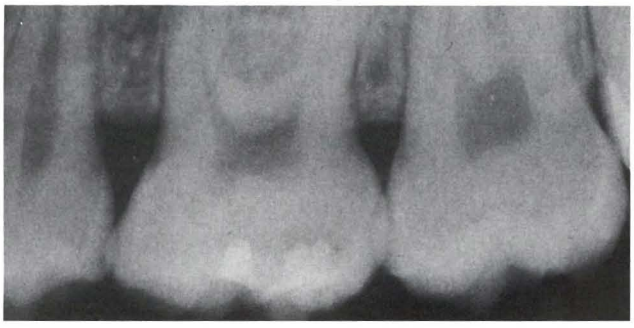


Figure 3. At year 2:
Figure 3a. Radiographically, the lesions do not appear to progress.

Figure 3b. Clinically, the color of sealant remained yellow. The two restorations remained completely sealed. (Note: The clinical photograph was taken on a diagonal and had to be cropped for proper orientation).

with >50 percent to 100 percent of the margins sealed was 64 percent among CompS/C and 82.5 percent among the AGS restorations.

Anatomic form of restorations (Wear)

At year 9, no clinically evident wear of composite or amalgam was found in the CompS/C and AGS groups. A single undercontoured AGU restoration was found. Wear of sealants, however, was observed.

Clinical failures of study restorations

Table 5 describes all clinical failures at year 9. Between years 6 and 9, there were no new failures in the AGS group. There were five (7 percent) new clinical failures of the CompS/C restorations. These included two unrelated failures that were due to proximal caries. Within the AGU group, there were two (5.7 percent) new failures. Table 6 summarizes the new, previous, and cumulative clinical failures for each group. The new and

Figure 4. At year 4:
Figure 4a. Radiographically, the lesions are not progressing.

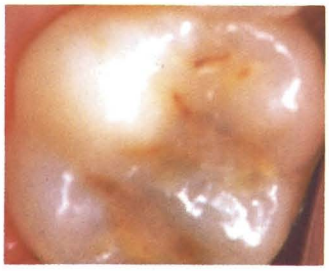


Figure 4b. Clinically, some areas of the sealant remained yellow but some areas have faded, and a comment was made that sealant seemed very thin in some areas. Both restorations remained completely sealed.

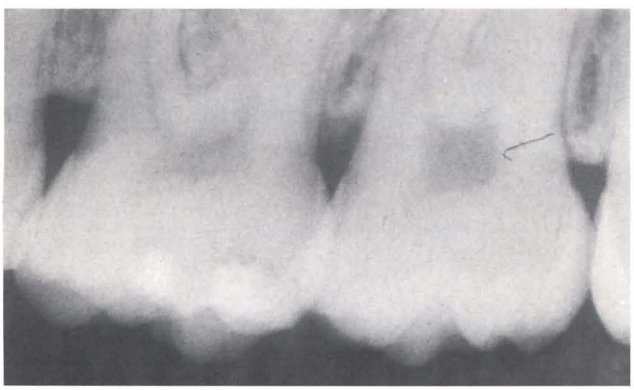


Figure 5. At year 6:
Figure 5a. Radiographically, the lesions appear to be no deeper than at 6 months (see Figure 2a)

Figure 5b. Clinically, the two restorations were rated the same as at year 4.

previous unrelated failures are also shown in Table 6. "Unrelated" failures denote failures that are not related to the study, such as removal of the study restoration to provide access to new proximal carious lesions, etc. Table 6 shows that there were 12 (16 percent) cumulative failures among the CompS/C, 1 (2.5 percent) among the AGS, and 6 (17.1 percent) among the AGU restorations.

Also, there were six CompS/C and two AGS restorations that could not be evaluated due to circumstances that could not be controlled during the unsupervised years 7 and 8 of the study. Four CompS/C restorations were replaced for unknown reasons (probably because they may have appeared radiographically unusual to dentists unfamiliar with the study). Two CompS/C study restorations were placed in third molars that were subsequently extracted, although neither patient knew why (had no pain or other symptoms). One patient was in military service and received numerous new restorations, as well as replacement of numerous existing restorations, including both of his CompS/C and AGS restorations; the latter were replaced with an MO and a DO Class II amalgam restoration, respectively, evidently due to proximal caries. One patient was in a car accident and had his AGS tooth avulsed.

Caries was the only mode of failure among the AGU restorations. Failures due to caries were as follows: 17 percent among AGU, 1 percent among CompS/C, and 2 percent among AGS restorations.

The survival of restorations was analyzed by means of the Wilcoxon's test for homogeneity of survival curves between materials.[24] The study design was a 4-celled rather than a 3-celled study, so that there were 77 AGS & CompS/C pairs and 79 AGU and CompS/C pairs of

Figure 6. At year 9:
Figure 6a. Radiographically, there was no change in lesion size as compared to the 6 months radiograph (Figure 2a).

Table 3 ☐ Sealant retention over study restorations and presence/absence of open margins.

	No open margins			Open margins present	
	Complete sealant retention	Partial sealant retention	No sealant	Partial sealant retention	No sealant
75 CompS/C	21 28.0%	31 41.3%	0	5 6.7%	0
40 AGS	9 22.5%	25 62.5%	0	3 7.5%	0
35 AGU	NA	NA	20 57.1%	NA	11 31.4%

Note: Total percentages across the table add up to less than 100%; the remaining percentages are shown in other tables.

study teeth.[16] Thus, CompS/C versus AGS and CompS/C versus AGU were compared directly using the same individuals; then the AGU group was compared versus the AGS group.

This statistical analysis for survival curves demonstrated:

- ☐ No difference between AGU versus CompS/C groups (p-value = 0.9073).
- ☐ A significant difference between AGS versus CompS/C (p-value = 0.0215).
- ☐ A significant difference between AGS versus AGU (p-value = 0.0441).

The AGS group was the most successful of the three groups with only one clinical failure.

Sealant retention over the remaining occlusal surfaces

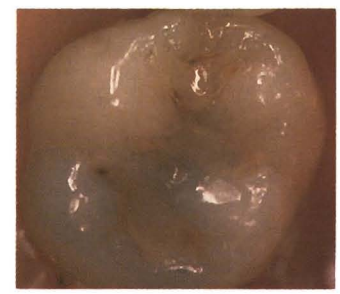
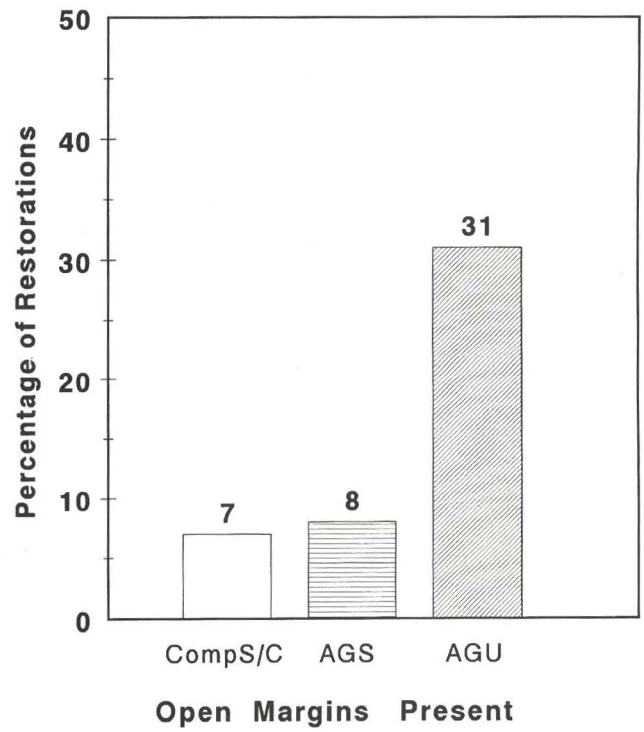


Figure 6b. Clinically, the sealant color remained yellow in some areas and faded in some areas. All margins of the restoration remained sealed, but some loss of sealant occurred in the middle of each restoration. A shallow bare pit without any softness was found in the occlusal portion of the lingual groove at the sealant margin, and the marginal integrity of the sealant changed, therefore, from ALFA to BRAVO (see Table 2). (Note: A blue mark on the distal marginal ridge was caused by the articulating paper at baseline. It was sealed over and could be seen in the subsequent photographs).



Fgure 7. Open margins observed for each restoration type. The difference in the incidence of open margins for AGS versus AGU was significant: Chi-square, P = 0.007.

Table 4 ☐ Sealant retention over margins of study restorations.

	Completely sealed restorations	Completely sealed margins with a bare area in the center of the restoration		Partially sealed margins		
	margins remaining 100% sealed		(Subtotal)	>50% sealed margins	(Subtotal)	<50% sealed margins
75 CompS/C	21	4	(25)	23	(48)	9
	28.0%	5.3%	(33.3%)	30.7%	(64.0%)	12.0%
40 AGS	9	0	(9)	24	(33)	4
	22.5%		(22.5%)	60.0%	(82.5%)	10.0%

Note: Total percentages across the table add up to less than 100%; the remaining percentages are shown in other tables

Table 5 ☐ Description of clinical failures of restorations at year 9.

Restoration type	Failure type	Tooth number	Description of failure and outcome
CompS/C	Charlie MI	3	Void at margin; resealed at Year 9
CompS/C	Charlie MI	31	Void at margin; resealed at Year 9
CompS/C	Charlie C/M	18	Caries at margin of study restoration at Year 9. Many broken restorative appointments
CompS/C	Unrelated proximal caries	5	Prior to year 9, study restoration had to be removed for access due to disto-gingival proximal caries
CompS/C	Unrelated proximal caries	15	Distal and mesial proximal caries; study restoration was replaced with an MOD amalgam
AGU	Delta C/M	13	Caries at margin so extensive that the study restoration was lost, leaving a large open lesion
AGU	Charlie OPF Caries	31	Pit and fissure caries, but NOT at margin of a study restoration

Ryge Criteria for failures given in Table 1
MI = Marginal Integrity
C/M = Caries at Margin
OPF = Caries in Occlusal Pit and Fissure

Table 6 ☐ Clinical failures of restorations.

	New failures at year 9	New unrelated failures	Previous failures	Previous unrelated failures	Cumulative failures minus unrelated failures
75 CompS/C	3	2	9	2	16−4=12
	4.0%	2.7%	12.0%	2.7%	16.0%
40 AGS	0	0	1	1	2−1=1
			2.5%	2.5%	2.5%
35 AGU	2	0	4	0	6−0=6
	5.7%		11.4%		17.1%

Table 7 ☐ Sealant retention over the remaining occlusal surface (occlusal areas not involved with the study restoration).

	Complete	Partial	No sealant	(Subtotal)	Clinical failures	Other*
75 CompS/C	50	7	0	(57)	12	6
	66.7%	9.3%		(76.0%)	16.0%	8.0%
40 AGS	27	10	0	(37)	1	2
	67.5%	25.0%		(92.5%)	2.5%	5.0%

*Other: "could not be evaluated" at year 9 (see text)

Table 8 ☐ Comparison of restoration performance in children (ages 8-18) and adults (ages 19 and older).

	No open margins			Open margins present		
	Complete sealant retention	Partial sealant retention	No sealant	(Subtotal: No open margins)	Partial sealant retention	No sealant
Children: 23 CompS/C	3	10	0	(13)	4	0
	13.0%	43.5%		(56.5%)	17.4%	
Adults: 52 CompS/C	18	21	0	(39)	1	0
	34.6%	40.4%		(75.0%)	1.9%	
Children: 11 AGS	2	7	0	(9)	1	0
	18.2%	63.6%		(81.8%)	9.1%	
Adults: 29 AGS	7	18	0	(25)	2	0
	24.1%	62.1%		(86.2%)	6.9%	
Children: 12 AGU	NA	NA	5	(5)	NA	4
			41.7%	(41.7%)		33.3%
Adults: 23 AGU	NA	NA	15	(15)	NA	7
			65.2%	(65.2%)		30.4%

Note: Total percentages across the table sum to less than 100% because clinical failures and "other" 8 restorations (see Table 7) are not included.

is shown in Table 7. Complete retention was identical for the CompS/C versus AGS groups. Added together, complete and partial retention were 76 percent and 92.5 percent, respectively.

Sealant retention over the remaining occlusal surface (Table 7)

At year 9, complete sealant retention over the remaining portions of the occlusal surfaces of the teeth in the CompS/C group was the same as that in the AGS group (66.7 percent and 67.5 percent, respectively). Partial sealant retention for the entire occlusal surfaces was 9.3 percent and 25 percent, respectively.

Marginal integrity of sealant

Sealant margins remained intact in 59 percent of CompS/C study teeth and in 87 percent of the AGS study teeth. A discontinuity or a void at the sealant mar-

gin was found in 17 percent CompS/C and in 5 percent AGS teeth.

Clinical failures of sealant

Sealant Failure is defined as *occlusal pit or fissure caries but not at margin of a study restoration.* Through year 9 there were four sealant failures in the CompS/C group and one sealant failure in the AGS group.

Comparison of children and adults

Table 8 shows the results among children aged eight to eighteen versus adults. There were forty-six restorations evaluated in children and 104 in adults.

The percentage of CompS/C restorations with no open margins was 56.5 percent in children and 75 percent in adults. The majority of AGS restorations survived with no open margins (81.8 percent in children and 86.2 percent in adults). A slightly lower percentage of AGU restorations in children (41.7 percent) maintained good marginal integrity as compared to adults (65.2 percent). Open margins were relatively evenly dispersed between children and adults for AGS restorations, but a higher percentage of *open margin* in CompS/C restorations was seen in children than in adults (17.4 percent versus 1.9 percent, respectively). The reason for this difference is unknown.

Chi-square analysis for complete versus partial sealant retention over the sealed restorations showed no significant differences between children and adults ($P = 0.143$ for CompS/C restorations and $P = 0.736$ for AGS restorations). The Fisher exact P values were 0.198 and 0.554, respectively.

Open margins versus no open margins in the AGU group showed no significant difference between children and adults (Chi-Square, $P = 0.801$).

DISCUSSION

Over the nine-year period, there were twelve (16 percent) clinical failures out of seventy-five CompS/C restorations and six (17.1 percent) out of thirty-five AGU. The Wilcoxon's test showed no difference in longevity of the experimental sealed composite restoration placed over caries (CompS/C) versus the traditional unsealed amalgam (AGU) restorations. Thus the traditional unsealed amalgam restoration has a clinical failure rate equivalent to the experimental CompS/C restoration. There was only one clinical failure in the localized sealed amalgam (AGS) group. Thus the longevity of AGS restorations was significantly superior to that of the CompS/C restorations. This difference may be attributed to the less complex clinical procedures involved with placement of the AGS restoration compared to the rigors required for successful bonding of the composite restoration. The latter involved a completely new concept of providing at least a 1 mm bevel in "sound-looking" enamel, regardless of whether it was undermined by caries, and then leaving soft caries below the bevel and under the sealed composite restorations.

An even greater significant difference in clinical longevity was found between AGS versus AGU restorations, favoring the AGS group. Thus the traditional AGU restoration had the worst record of survival as compared to the two types of sealed restorations. Of all the test groups, AGS demonstrated the greatest clinical longevity.

The AGU group was also the worst in the *Ryge Caries at Margin* criterion (Table 1). We recommend, therefore, that Class I amalgams be sealed, since the restoration margins in the AGS group were shown to be protected against recurrent caries, demonstrating a much lower incidence of open margins and caries at margin than the AGU group. It may also be of benefit to seal at least the occlusal surfaces of Class II restorations.

Among the CompS/C restorations, there were two cumulative failures due to caries at margin in children, and four such failures in adults. This finding was equivalent, therefore, for children and adults, since the ratio of children to adults was 1:2 in this study. Because only permanent teeth were used in this study, no marked deviations were expected and none was found. Even the incidence of recurrent caries was proportional for both age groups: two occurred in children and four in adults.

A previous SEM study has shown that sealant penetrates the interface gap between the cavity walls and freshly placed amalgam restorations.[20] This phenomenon may protect the amalgam margins against ditching, greatly reducing the potential for formation of new and recurrent caries at the margin. Bearing in mind the results of this long-term clinical study, there is no justification for placement of unsealed amalgams having the typical extension for prevention outline form in Class I restorations.

The basic cause of marginal deterioration in unsealed amalgams is poor tensile properties resulting from nonhomogeneous microstructure. During normal mastication, forces applied occlusally generate a high tensile component of stress as well as compressive and shear stresses. In unsupported amalgam margins with even

small interface gaps, sufficient deformation occurs to cause fracture from bending.

Sealant placed in the interface gap, which seems always to exist, acts as a cushion to support the amalgam, when masticatory force is applied. Thus the sealant minimizes the deformation of the amalgam to the extent that it remains intact at the margins.

Also it should be pointed out that none of the sealed cavosurface margins in the CompS/C group *caved in*. A clinician would be apprehensive of leaving caries for two reasons

- ☐ Continuing decay process.
- ☐ The possibility that remaining tooth structure might crumble under occlusal loading.

Neither of these events has been shown to happen and this is emphasized, to address the clinical concerns of practitioners.

The Anatomic Form criterion for sealant wear could not be assessed clinically by the Ryge criterion, since sealant cannot be undercontoured with respect to enamel or with respect to restorations; facets in sealant have not been observed. The tactile sense and the color match for the yellow sealant as compared to enamel were the main indicators for sealant wear, although it was also complicated by the actual fading of the orange-yellow sealant color.

The wear of sealant in various occlusal areas should not matter as long as the pits and fissures and the restoration margins remain sealed.

CONCLUSIONS

- ☐ Sealed composite restorations placed over caries arrested the progress of carious lesions over a period of nine years.
- ☐ The total of six clinical failures (17.1 percent) in the unsealed amalgam group (AGU) was due to caries; two occurred among the children and four in the adults. Thus the incidence of recurrent caries in the AGU group was equivalent for both of the age-groups. Caries at the margin occurred in only 1 percent of CompS/C and 2 percent of AGS restorations. We recommend, therefore, that Class I amalgam restorations in permanent teeth of children and in adults should be sealed at the time of placement.
- ☐ The incidence of open margins was the greatest (31 percent) in the AGU group, compared to 7 percent in the CompS/C and 8 percent in the AGS groups. This difference was statistically significant.
- ☐ The sealed restorations were superior to the unsealed restorations in conserving sound tooth structure, protecting margins, and preventing recurrent caries.

REFERENCES

1. Cueto, E.I.; Buonocore, M.G.: Adhesive sealing of pits and fissures for caries prevention. Abstract#400. J Dent Res, 44:137, July 1965.
2. Proceedings, National Institutes of Health Consensus Development Conference: Dental sealants in the prevention of tooth decay. J Dent Educ 48 (Supplement): 1-134, February 1984.
3. National Institutes of Health Consensus Development Conference Statement on dental sealants in prevention of tooth decay. J Am Dent Assoc, 108:233-236, February 1984.
4. Handelman, S.L.; Buonocore, M.G.; Hesek, D.J.: A preliminary report on the effect of a fissure sealant on bacteria in dental caries. J Prosthet Dent, 27:390-392, April 1972.
5. Handelman, S.L.; Buonocore, M.G.; Schoute, P.C.: Progress report on the effect of a fissure sealant on bacteria in dental caries. J Am Dent Assoc, 87:1189-1191, November 1973.
6. Handelman, S.L.; Washburn, F.; Wopperer, P.: Two-year report of sealant effect on bacteria in dental caries. J Am Dent Assoc, 93:967-970, November 1976.
7. Jeronimus, D.J.; Till, M.J.; Sveen, O.B.: Reduced viability of microorganisms under dental sealants. J Dent Child, 42:275-280, July-August 1975.
8. Going, R.E.; Loesche, W.J.; Grainger, D.A.*et al*: The viability of microorganisms in carious lesions five years after covering with a fissure sealant. J Am Dent Assoc, 97:455-462, September 1978.
9. Mertz-Fairhurst, E.J.; Schuster, G.S.; Williams, J.E. *et al*: Clinical progress of sealed and unsealed caries. Part I: Depth changes and bacterial counts. J Prosthet Dent, 42:521-526, November 1979.
10. Mertz-Fairhurst, E.J.; Schuster, G.S.; Williams, J.E. *et al*: Clinical progress of sealed and unsealed caries. Part II: Standardized radiographs and clinical observations. J Prosthet Dent, 42:633-637, December 1979.
11. Jensen, Ø.E. and Handelman, S.L.: Effect of an autopolymerizing sealant on viability of microflora in occlusal dental caries. Scand J Dent Res, 88: 382-388, October 1980.
12. Handelman, S.L.: Effect of sealant placement on occlusal caries progression. Clinic Prev Dent, 4:11-16, September-October 1982.
13. Mertz-Fairhurst, E.J.; Schuster, G.S.; Fairhurst, C.W.: Arresting caries by sealants: results of a clinical study. J Am Dent Assoc, 112:194-197, February 1986.
14. Mertz-Fairhurst, E.J.; Call-Smith, K.M.; Schuster, G.S. *et al*: Clinical performance of sealed composite restorations placed over caries compared with sealed and unsealed amalgam restorations. JADA, 115:689-694, November 1987.
15. Mertz-Fairhurst, E.J.; Williams, J.E.; Schuster, G.S. *et al*: Ultraconservative sealed restorations: Results through year three. J Public Health Dent, 51:239-250, Fall 1991.
16. Mertz-Fairhurst, E.J.; Williams, J.E.; Pierce, K.L. *et al*: Sealed restorations: 4-year results. Am J Dent, 4:43-49, February 1991.
17. Mertz-Fairhurst, E.J.; Richards, E.E.; Williams, J.E. *et al*: Sealed restorations: 5-year results. Am J Dent, 5:5-10, February 1992.
18. Mertz-Fairhurst, E.J.; Smith, C.D.; Williams, J.E. *et al*: Cariostatic and ultraconservative sealed restorations: six-year results. Quintessence Int, 23:827-838, December 1992.
19. Briley, J.B.; Dove, S.B.; Mertz-Fairhurst, E.J.: Radiographic analysis of previously sealed Class I restorations. IADR/AADR Abstract #2514, J Dent Res, 73:416, March 1994.
20. Mertz-Fairhurst, E.J. and Newcomer, A.P.: Interface gap at amalgam margins. J Dent Mat, 4:122-128, June 1988.

21. Brunelle J: Prevalence of dental sealants in U.S. school children. Abstract #12, J Dent Res, 68:183, March 1989.
22. Mertz-Fairhurst, E.J.: Guest Editorial: Pit and fissure sealants: a global lack of science transfer? J Dent Res, 71:1543-1544, August 1992.
23. Mertz-Fairhurst, E.J.: Reaction and discussion: retention and effectiveness of a single application of white sealant after 15 years. Proceedings, Symposium on Esthetic Restorative Materials, November 11-13, 1991. Sponsored by the American Dental Association, support provided by the National Institute of Dental Research. Published November 1993, pp 40-43.
24. Lee, E.T.: Statistical Methods for Survival Data Analysis. Belmont, CA: Lifetime Learning Publications, 1980, pp 76-88.

BICYCLE HELMETS

Bicycling has become extremely popular in the last 10 years. There were an estimated 100 million cyclists in the United States in 1993. In addition to providing efficient transportation, bicycling can be an enjoyable form of aerobic exercise. The sport is not without hazards, however. In 1991, bicycle-related injuries necessitated approximately 600,000 emergency-department visits and contributed to approximately 800 deaths. Many of those injured or killed were children and adolescents.

Head injury from cycling is the most common cause of death (70% to 80% of cases) and the leading cause of disability. One fourth of injured bicyclists treated in emergency departments have head injuries, as do half of those admitted to the hospital. Many children are injured falling from their bicycles, and collision with fixed objects or motor vehicles plays a significant role in making bicycle safety a major pediatric health issue.

According to the National Safety Council, 180,000 collisions between motor vehicles and bicycles occurred in 1991. Eight hundred of these resulted in fatalities. Forty-two percent of these deaths occurred in children younger than 15 years. With the proliferation of mountain bikes, these patterns of injury may change for the worse as more cyclists venture into potentially treacherous areas.

Included in the recommendations of the American Academy of Pediatrics are

- ☐ All cyclists should wear a properly fitted ANSI- or Snell-approved helmet specifically designed for cycling. Children riding as passengers must wear appropriate-size helmets in specially designed protective seats.
- ☐ Pediatricians must inform parents and patients of the importance of wearing bicycle helmets and the dangers of riding without a helmet.
- ☐ State or local municipal governments should be urged to enact legislation requiring helmet use by all bicyclists and mandating bicycle rental agencies to include helmets as part of the rental contract. The Academy has developed model state legislation entitled, "Child Bicycle Safety Act."
- ☐ Coalitions of physicians, parents, and community leaders need to be encouraged to develop and support community-based and school education programs to promote bicycle safety training and helmet usage.

Committee on Injury and Poison Prevention, Am Acad Pediatrics; Bicycle helmets. Pediatrics, 95:609, April 1995.

A three-year follow-up of glass ionomer cement and resin fissure sealants

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Jan W.V. van Dijken, DDS, PhD

Carious lesions in occlusal fissures develop most frequently during the first two years after eruption. Probably due to the complex anatomy of the fissures, oral hygiene practices or fluoride treatments are more effective in preventing caries on smooth surfaces than in fissures. Sealing the fissures with a low-viscous resin material used as a cariostatic agent was introduced in 1968 by Roydhouse.[1] A review of numerous clinical trials, where BIS-GMA resins were used, showed after one year a wide variation in retention rates (18 percent - 99 percent). This was explained mainly by differences in technical performance.[2] Caries may occur in fissures where the sealant is badly adapted, partially lost or totally lost. The effectiveness of the resin sealant is directly related to the resin impregnation of the enamel. Placement of the resin is very technique-sensitive and is influenced by several factors such as patient cooperation, operator variability and contamination of the operation field. In studies where the sealant is applied under optimal conditions a very low failure rate can be obtained.[3] Sealing is often performed, however, under more primitive conditions without an assistant, which partially explains the varying clinical results. The ability of a sealant to release fluoride as well as occlude pits and fissures would be a distinct advantage over the conventional resin-based sealants. In 1974 McLean and Wilson advocated the use of glass ionomer cement as pit and fissure sealant. They used ASPA II during two years, and showed a retention rate of 84 percent after one year and 78 percent after two years.[4] Type II glass ionomer cement was used in most previous trials. Initially, wide fissures were selected or the fissures were mechanically prepared in order to allow the viscous cement to penetrate the fissures. In these fissures a high success rate was obtained.[4,5] Variable retention rates were reported for fissures that had not been mechanically widened and which had been sealed with a type II glass ionomer. McKenna and Grundy showed 93 percent and 82.5 percent retention rates after six months and one year, respectively, with Ketac Fil.[6] Mills and Ball, using a glass ionomer cermet cement (Ketac Silver), showed 82 percent retention after two years.[7] Hickel and Voβ also used the glass cermet cement, but only in wide fissures, and showed 69.2 percent retention after eighteen months.[5] Williams and Winter used ASPA IV and reported retention rates of 47.1 percent and 35.4 percent after two and four years, respectively.[8] A specially formulated, glass ionomer cement has recently been developed as a sealant material. Investigations evaluating the retention of this sealant glass ionomer show varying retention rates of 75 percent at four months and 34 percent-39 percent at six months.[9,11,12] The aim of this study was to compare intraindividually the clinical durability of the fissure-sealant glass ionomer cement with a chemically cured BIS-GMA resin during a three-year period.

The study was supported by the Swedish Dental Society.

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MATERIALS AND METHODS

Patient group

The patients participating in the study included forty-seven children, (twenty-six girls and twenty-one boys), with an average age of seven years, one month (range: six years, seven months to seven years, ten months). They were chosen from the children attending a community dental clinic in Umeå during 1989. First permanent molars and their contralaterals were chosen for the study. For each patient, two or four fully erupted teeth, with no previous filling or clinical evidence of caries, were sealed. A total of 148 first molars were sealed, sixty-two in the maxilla and eighty-six in the mandible.

Experimental materials

The materials used were a chemically cured BIS-GMA resin sealant (Delton, Johnson and Johnson, East Windsor, NJ, USA) and a glass ionomer cement sealant (FUJI III, GC Dental Industrial Corp., Tokyo, Japan). The teeth were randomly assigned to treatment with either Delton or Fuji III. The contralateral tooth was always sealed with the other material. Before sealing, the fissures were cleaned with pumice and water-spray. When Delton was used, the fissure was etched with 37 percent phosphoric acid for twenty seconds, rinsed with water for twenty seconds and then thoroughly dried. The resin sealant was then applied according to the manufacturer's instructions. The teeth sealed with Fuji III were cleaned with 40 percent polyacrylic acid (Durelon liquid, ESPE, Seefeld, Germany) for twenty seconds, rinsed with water for ten seconds and then dried with an air syringe before sealing. The accessory applicator was used to apply the sealant. When the glass ionomer lost its gloss, Fuji varnish was applied. After setting, occlusion was carefully checked.

Evaluation

The sealants were observed at six months, and one, two, and three years after their placement. At each evaluation, presence of caries was noted and retention of the sealants was recorded as total retention, partial loss, or complete loss. All sealants were placed and controlled by one dentist working at the Public Dental Health Center at the Dental School, Umeå.

RESULTS

During the period of the study, forty-five patients were available for assessment at all appointed observation times. Due to moving, two patients with four sealed mandibular teeth could not be evaluated at all observation visits. The frequencies of completely retained and the cumulative frequencies of partially or totally lost sealings at each recall are shown in Figure. The number of teeth with retained glass ionomer cement sealant decreased markedly at each recall, from 79.7 percent at six months, 72.2 percent at one year, 43.1 percent at two years to 27.8 percent after three years. Retention rates for the resin sealant were significantly better with 98.7 percent at six months, 97.2 percent at one year, 90.3 percent at two years and 79.2 percent after three years. None of the resin sealants was totally lost. Caries was recorded in three (4.2 percent) of the resin sealed teeth (at six months, one year and three years) and in one (1.4 percent) of the glass-ionomer-cement-sealed teeth (at two years).

DISCUSSION

The use of a glass ionomer cement as fissure sealant has advantages, such as bonding to enamel and dentin, and release of fluoride. The insufficient mechanical and physical properties of the material, especially in loaded areas, resulted, however, in a high rate of retention failures; which is confirmed in other studies.[4-13] Compared with the earlier used type II glass ionomer cement, the type III cement specially made for fissure sealing showed higher loss rates.[9-13] Mejare and Mjör found that

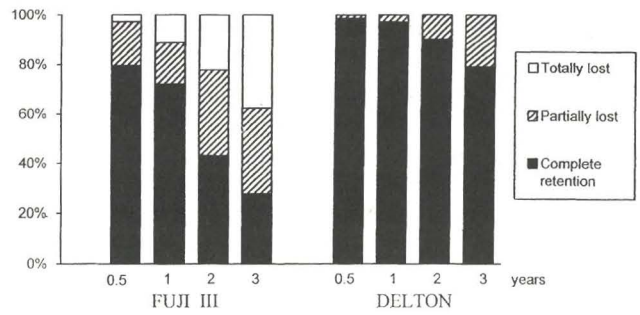

Figure. Retention rates and cumulative frequencies of partially lost and totally lost sealants (percent) during the three year period.

after thirty to thirty-six months, 84 percent of the sealants were totally lost.[9] Forss *et al* found that after two years, 48 percent were totally lost and 26 percent were partially lost; and Ishikawa *et al* found that after six months, 65.8 percent were totally lost.[10,11] Boksman discontinued his study after six months, because of a total loss of 94 percent.[13] He placed the sealants using rubber dam, which probably implies an operation field too dry for the hydrophilic glass ionomer cement. In our study the rates of totally and partially lost sealants were 11.1 percent and 16.7 percent, respectively, after one year; and 37.5 percent and 34.7 percent, respectively, after three years. These slightly better results with the type III cement can partly be explained by operator variability, as shown in other studies.[9,10]

Studies, where pressure was applied on the sealant during the application of the cement, showed better retention rates.[4-8,14] Mills and Ball showed only 6 percent of the sealants were lost after two years.[7] They etched the fissure with 37 percent phosphoric acid and then compressed the sealant into the fissure with a gloved finger or a plastic instrument. McKenna and Grundy, showed a total loss of 3.5 percent after one year; they had used a ball burnisher to insert the cement into the fissures.[6] The glass ionomer cement sealants in our study were applied according to the manufacturer's instructions, without pressure on the material.

The resin sealant showed significantly better retention rates in this study. None was totally lost during the experimental period, but 20.8 percent were partially lost. In most of these sealants, eleven of thirteen, only a minor portion was lost, while in the other two cases half of the sealant was lost. This is in contrast to the glass ionomer sealants, where a larger portion was generally lost.

Despite the high loss rate in the glass ionomer cement group, the caries incidence was very low for both groups, which is in accordance with other sealant studies.[3,7,9,10] The only child who developed caries in the glass ionomer cement group also developed caries in the contralateral tooth. The low caries incidence in the resin group can easily be explained by the high sealant retention. Caries can still develop, however, when the bond fails in only a small part of the sealant, especially in high-caries-risk patients. To explain the low caries incidence in the glass ionomer cement group is more difficult. One reason may be small amounts of glass ionomer cement left in the deepest parts of the fissures as was observed by scanning electron microscopy.[9,15] Another reason may be the better mineralization of the newly erupted tooth by the fluoride release of the glass ionomer cement. This continuous fluoride leakage from the sealant during long periods results in a more mature and acid-attack resistant tooth substance, even after a relatively early sealant loss.[16,17]

Recently light-cured, resin-reinforced glass ionomer cements were introduced; they have better mechanical and physical properties than the conventional ones. Rasmussen and Rasmussen evaluated two of these cements, marketed as base materials, as fissure sealants during one year.[18] They showed low retention rates, 15 percent after six months, for the nonetched fissures, while the acid etched ones showed 59 percent retention. The use of etching with a more aggressive acid than polyacrylic acid showed relatively good retention rates for a glass cermet cement in another study, when it was combined with pressure during insertion.[7]

REFERENCES

1. Roydhouse, R.H.: Prevention of occlusal fissure caries by use of a sealant: a pilot study. J Dent Child, 35:253-262, May-June 1968.
2. Ripa, L.W.: Occlusal sealants—rationale and review of clinical trials. Int Dent J, 30:127-139, June 1980.
3. Wendt, L.K. and Koch, G.: Fissure sealant in permanent first molars after 10 years. Swed Dent J, 12(5):181-185, 1988.
4. McLean, J.W. and Wilson, A.D.: Fissure sealing and filling with an adhesive glass-ionomer cement. Brit Dent J, 136:269-276, April 1974.
5. Hickel, R and Voβ, A.: Vergleichende untersuchung uber fissurenversiegelung: Komposit versus Cermet-zement. Dtsch Zahnärztl Z, 44: 472-474, June 1989.
6. McKenna, E.F. and Grundy, G.E.: Glass ionomer cement fissure sealants applied by operative dental auxiliaries—retention rate after one year. Austr Dent J, 32: 200-203, June 1987.
7. Mills, R.W. and Ball, I.A.: A clinical trial to evaluate the retention of a silver cermet-ionomer cement used as a fissure sealant. Oper Dent, 18:148-154, July-August 1993.
8. Williams, B. and Winter, G.B.: Fissure sealants. Brit Dent J, 150: 183-187, April 1981.
9. Mejàre, I. and Mjör, I.A.: Glass ionomer and resin-based fissure sealants: a clinical study. Scand J Dent Res, 98:345-350, August 1990.
10. Forss, H.; Saarni, U.M.; Seppä, L.: Comparison of glass ionomer and resin-based fissure sealants: a two year clinical trial. Community Dent Oral Epidemiol, 22:21-24, February 1994.
11. Ishikawa, M.; Dogon, I.L.; van Leeuwen, M.J.*et al*: A comparative study of two fluoride releasing sealant materials. J Dent Res, 68, 1014, Abstr No 1178, June 1989.
12. Torppa-Saarinen, E. and Seppä, L.: Short-term retention of glass ionomer fissure sealants. Proc Finn Dent Soc, 86:83-88, 1990.
13. Boksman, L.; Gratton, D.; McCuthrheon, E.*et al*: Clinical evaluations of a glass ionomer cement as a fissure sealant. Quint Int, 18:707-709, October 1987.
14. Smales, R.J. and Gerke, D.C.: The use of glass ionomer cements for restoring occlusal tooth surfaces. Austr Dent J, 35:181-182, April 1990.
15. Övrebö, R.C. and Raadal, M.: Microleakage in fissures sealed with resin or glass ionomer cement. Scan J Dent Res, 98:66-69, February 1990
16. Skvartveit, L.; Tveit, A.B.; Tötdal, B. *et al*: In vivo fluoride uptake in enamel and dentin from fluoride-containing materials. J Dent Children, 46:97-100, March-April 1990.
17. Komatsu, H.; Yoshimura, M.; Ohshima, K.*et al*: Retention of fluoride in enamel after removal of applied glass-ionomer cement. J Dent Res, 68:Abstr No 893, 978, June 1989.
18. Rasmusson, C.G. and Rasmusson, L.: Kliniska försök med ljushärdande glasjonomercement. Tandl tidn, 86:117-123, February 1994.

In vitro studies of the effect of a dental explorer on the formation of an artificial carious lesion

Othman Mahmoud Yassin, DDS, MSc

The anatomical structure of the tooth surface is an important factor in caries incidence. Occlusal caries is responsible for over half the total caries experience, because of the pits and fissures in occlusal surfaces. Specific factors such as plaque retention, type of plaque, and inaccessibility of fissures for traditional cleaning methods are probably important factors in fissure caries.

The fissures provide a niche for dental plaque that is difficult to remove. The caries process usually starts soon after tooth eruption, in pits and fissures: The first visible changes are soon followed by cavity formation, especially when oral hygiene and dietary habits are caries-promotive.

The early carious white-spot lesion can remineralize completely, provided that no mechanical damage has occurred.[1] This type of lesion cannot be detected radiographically, leaving the use of the explorer as the only diagnostic method.

The effect of dental probing on the early pit and fissure lesion should be investigated, because of the mechanical damage caused by probing.

This work was conducted at The London Hospital Medical College under the supervision of Dr. J.C. Elliott. I am very grateful to him for giving me his time and valuable advice during the course of the project.
I thank Miss Jocelyn Germain from the Department of Oral Pathology for providing all the necessary instructions, procedures for sectioning and embedding the teeth, and the staff of her department at The London Hospital Medical College.
I express my sincere gratitude to Dr. Mossa Marbut for assistance with statistical aspects of this work and his great help and encouragement in writing the thesis.
I also acknowledge the Jordan Armed Forces for granting me a training scholarship.

MATERIALS AND METHODS

Extracted human third molars of unknown history were collected. Caries-free teeth were selected only for macroscopic examination. The teeth were cleaned of soft tissues and calculus with a scalpel and washed thoroughly with tap water. Then the teeth were polished gently with flour of pumice in water, using a soft rubber cup in a slow-speed handpiece.

The sample size consisted of forty teeth, and all the teeth were stored in distilled water containing 1 percent by weight thymol as a disinfectant, to prevent bacterial contamination and growth.

The roots were removed from the crowns with a water-cooled high speed handpiece, using a fissure bur. Enamel from buccal surfaces of the forty teeth was obtained by cutting each crown in half mesiodistally, using a diamond disc on the low-speed handpiece under running tap water The buccal enamel surface was flattened by hand, using silicon carbide abrasive paper (Grit 360) to get approximately 25 mm^2 of flat enamel without removing an excessive amount.

Finally the teeth were painted with an acid resistant red color nail varnish, and allowed to dry fully overnight. Four U-shaped standardized grooves were made in each enamel slab, using a machine designed for this purpose (Figures 1, 2).

A preliminary study was done to observe the formation of the caries-like lesions. Five teeth were exposed to demineralizing solution (3 percent by weight of cellulose solution in 0.1 M lactic acid containing 1.08 mM KH_2PO_4 and 1.8 mM $CaCl_2$ buffer to pH 4.28), for each

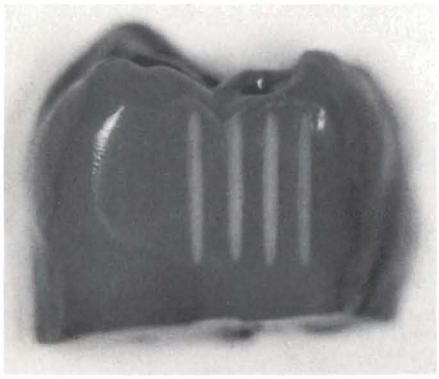

Figure 1. A photograph of the specimen with 4 grooves and coated with nail varnish before exposure to the demineralized solution.

time period of two, three, five, seven, and fourteen days. Teeth were removed from the demineralizing solution at the end of each time-period, washed in distilled water, and subjected to examination by microradiography.

The remaining thirty specimens were exposed to a fresh demineralized solution, the same system solution that was used previously for two weeks. The demineralized grooves of these specimens were probed with the same procedures used for the sound grooves.

The entire depth of each individual groove was filled completely with demineralizing solution, using a fine brush. The specimen teeth (30) were kept individually in a closed box with a dish of demineralizing solution to prevent dehydration at room temperature.

The teeth were kept for two weeks in demineralizing solution, then the specimens were removed, washed and cleaned thoroughly with distilled water to remove the cellulose solution from the grooves. The nail varnish also was removed with acetone. After the teeth had become dry, the white appearance of the demineralized areas could be seen easily along the whole depth and length of the grooves (Figure 2).

Three different weights were made from lead, 500 g, 300 g, 100 g, by casting in a suitable mold. Two holes were drilled in both sides of each weight, one for a screw fixed in the top to provide a hook, and the other in the bottom for the rod with the explorer.

Figure 3. A photograph of the explorer used in the study.

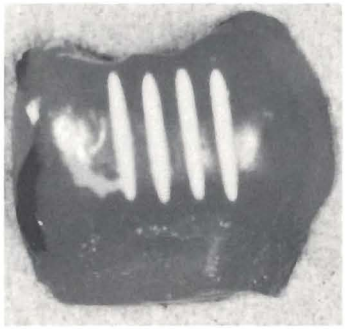

Figure 2. A photograph of the chalky (white) appearance of the grooves after two weeks of demineralization.

The tip of a sharp explorer (Probe 6, stainless steel, used in the Dental Institute of The London Hospital Medical College, Figure 3) was cut from its handle. The tip was then pushed into a prepared hole in a metal rod. This metal rod with the tip of the explorer was placed in the hole at the bottom of the weight (Figure 4).

To apply the explorer with a known force, a machine was designed with a pulley carrying the weight from the hook by a thread on one side and the thread connected to a screw on the other side. By rotating this screw, the weight could be lowered slowly until the explorer touched the bottom of the groove, so that the force of probing could be controlled. The specimen was mounted in an impression compound onto a removable bracket, which helped to fix it by a clamp on the base of the machine used for cutting the grooves. The enamel surface of the specimen was placed in a horizontal position. Then the explorer with the required weight was lowered slowly until the explorer reached the bottom of the groove. The sample was then moved horizontally by the screw, in

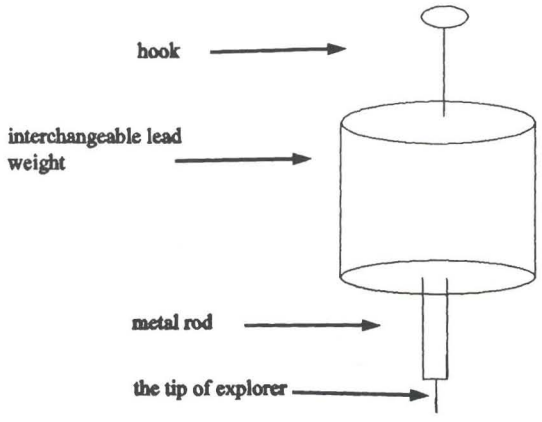

Figure 4. A diagram of the design of explorer and weight.

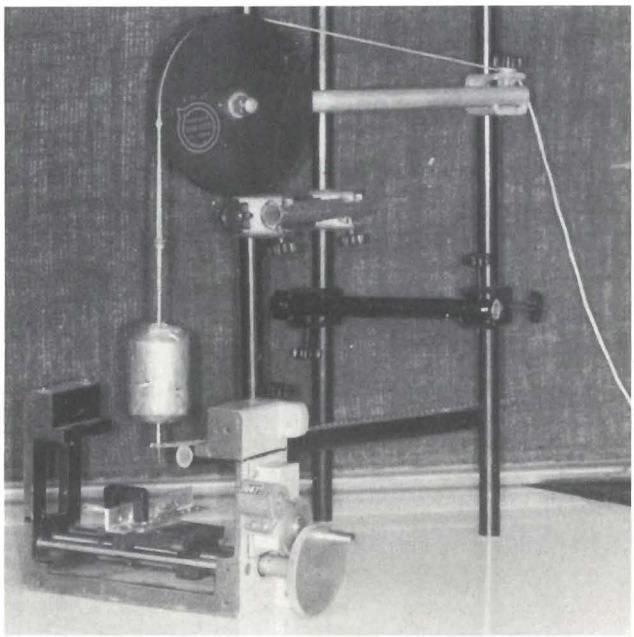

Figure 5. A photograph of the machine designed for applying controlled probing.

only one direction, under the effect of the explorer (the action was a scratching of the groove) (Figure 5).

Five specimens with sound enamel were chosen for probing of the grooves. The first groove of the specimen was used as a control without probing. The second groove was probed by a 100 g weight. This weight was replaced by a 300 g weight for probing the third groove, and a 500 g weight for probing the fourth groove. The same procedures of probing were followed in the demineralized grooves of thirty teeth. After each probing, the explorer was sharpened lightly with a medium sandpaper disc, and replaced by a new one after ten specimens had been probed. After finishing the probing, the specimens were prepared for embedding in hard resin, before sectioning and microradiography.

Before the specimens were subjected to microradiography, they were dehydrated in alcohol, embedded in resin, and sectioned in 200-micron thicknesses using the Leitz microtome (Model 1600, Ernst Leitz Wetzlar GmbH, Germany). Before sectioning, the specimen enamel surface was oriented with the length of the groove, perpendicular to the plane of the diamond saw in the microtome, and the saw also perpendicular to the enamel surface. Because of the difficulty of obtaining sound sections, only one section was obtained from each specimen. The sawed slices (twenty-three sections were obtained from the thirty specimens) were dried in air and stored in small boxes, to prevent the risk of deformation and to be ready for microradiography. During sectioning of the specimens, good quality sections failed to be obtained from seven teeth.

A Hilger & Watts (Y33 Microfocus X-ray generator, copper target, no filtration) X-ray machine was used. The distance between the X-ray source and the specimen was 30 cm. The X-ray set was operated at 40 kV high tension and 2 mA tube-current. The exposure time was twenty-five minutes. The microradiographs of the twenty-three sections were examined to detect the presence of the lesion and the effect of the explorer on the structure of the demineralized enamel grooves. The observable depth of radiolucency in the microradiographs was chosen as the lesion depth. A graticule (Graticules Ltd., Tonbridge, Kent, England, 200×0.01 μm=2 mm) was used to calibrate the ocular micrometer of the light microscope.

The lesion-depths in the bottom of the grooves were measured from the microradiographs, using the calibrated ocular micrometer of the light microscope at 30× magnification.

The depth values were measured from the surface layer at the deepest point at the bottom of the groove in a direction parallel to the center line of the groove. The penetration of the explorer into the lesion was indicated by the absence of the radiopaque layer (the surface layer). This penetration of the explorer was measured perpendicular from the point of the origin of the surface layer to the bottom of the cavity caused by probing. A hypothetical line was drawn to estimate the original position of the surface layer.

Statistical evaluation was performed with student's t-test (unpaired). All the data are expressed as the mean with standard deviation (SD). All the significant results reached a significant level of $P<0.05$, unless otherwise stated.[2]

RESULTS

There was no evidence of mechanical damage or any sign of explorer penetration, in the first experiment, in the sound enamel grooves, after probing with 100 g, 300 g, and 500 g of force (Figure 6).

In the second experiment with thirty teeth, the grooves were demineralized for two weeks. In the grooves which were probed with different weights, the photographs showed evidence that the surface layer (the white line) was broken down in the bottom of the groove (Figure 7). A cavity was formed in the subsurface zone, which

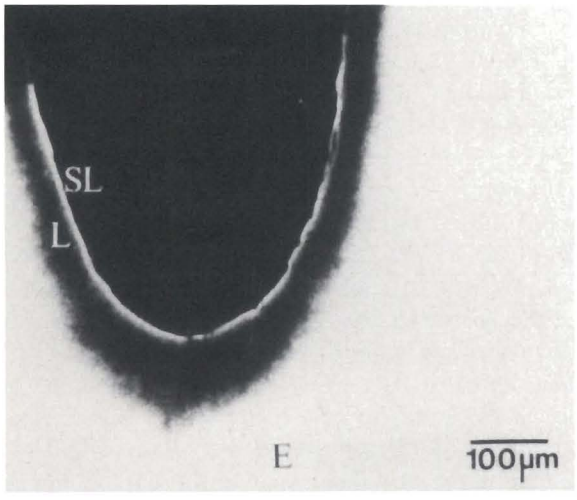

Figure 6. A microradiograph of demineralized enamel groove grown in 14 days in vitro. E = enamel, SL = surface layer, L + subsurface lesion. Note the width of the lesion in the bottom of the groove (130× magnification).

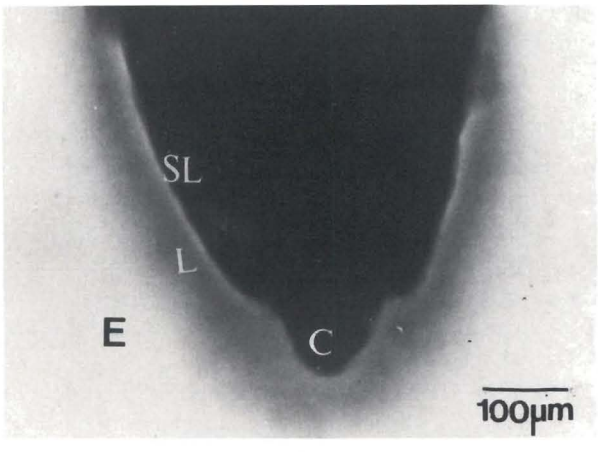

Figure 7. A microradiograph of demineralized enamel groove probed with 100 g force. E = enamel, SL = surface layer, L = subsurface lesion. C = cavity produced by the explorer (130× magnification).

represents the penetration of the explorer through the surface layer and the subsurface zones. The extent of explorer penetration was found to be different for the three probed grooves, with different weights (force) applied. For example, there was least penetration in the groove observed for probing with the lowest force (100 g) and greatest penetration in the groove that was probed with greatest force 500 g. Little difference could be seen between the 300 g and 500 g forces. After applying 500 g force, the explorer was seen to have penetrated nearly the whole depth of the lesion (Figure 7).

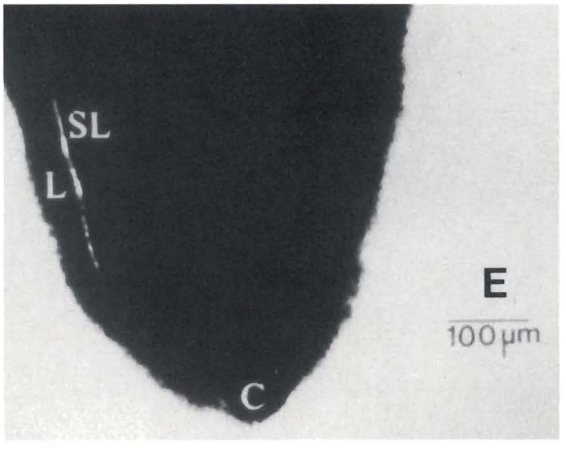

Figure 8. A microradiograph of demineralized enamel groove probed with 500 g force. E = enamel, SL = surface layer, C = cavity produced by the explorer (130× magnification).

Measurement of the lesion-depth in the nonprobed grooves was made at the deepest point in the bottoms of the grooves. The depth was measured from this point in a direction parallel to the center line of the groove (Figure 8). The mean lesion depth in the nonprobed grooves was found to be 101.9+12.9µm (+SD). The mean of the lesion depths in the probed grooves was found to be 99.8+12.9µm (+SD) for the group of 100 g, 102.8+13 8 µm (+SD) for the group of 300 g, and 102.8 + 11.2 µm (+SD) for the group of 500 g (Table 1).

Thus there were no significant differences in the lesion-depth measurements between the control grooves with intact surface layers, and measurements based on the hypothetical lines drawn in the bottoms of the grooves where there was an absence of surface-layer for the probed grooves (Figure 9). In other words, there was no significant error in estimating the original position of the surface layer.

The extent of explorer-penetration into demineralized enamel in the bottom of the groove was found to be correlated positively with the weight of force applied. In the first group, which was probed with the 100 g weight, the mean extent of penetration in twenty-three grooves was found to be 46.5 + 12.4 µm (+SD). The mean percentage of the extent of penetration in relation to the lesion-depth was found to be 46.7 percent.

In the second group, which was probed with 300 g, the mean extent of penetration into the twenty-three grooves was found to be 71.4 + 11.6 µm (+SD). The

Table 1 ☐ The mean lesion formed in four grooves after two weeks of demineralization (in microns). Control = unprobed grooves. Group 1 grooves probed with 100g force. Group 2 grooves probed with 300g force. Group 3 grooves probed with 500g force. (23 sections were used in each group).

Group	Mean	Std. Dev.	Std. Error	Coef. Var.
Control	102	13	3	13
1	100	13	3	13
2	103	14	3	14
3	103	11	2	11

Table 2 ☐ The mean depth of explorer-penetration into the lesion by different forces of probing (in microns). Group 1 = grooves probed with 100g force. Group 2 = grooves probed with 300g force. Group 3 = grooves probed with 500g force (23 sections were used in each group).

Group	Mean	Std. Dev.	Std. Error	Coef. Var.
1	46.5	12.4	2.6	26.6
2	71.4	11.6	2.4	16.3
3	101.0	11.3	2.4	11.2

Table 3 ☐ The explorer-penetration was compared by using unpaired t-test, the values for t and probability level for all groups are listed below. Group 1 = grooves probed with 100g force. Group 2 = grooves with 300g force. Group 3 = grooves probed with 500g force.

Group	t-value	Degree of freedom	P
1 versus 2	7.037	44	<0.001
1 versus 3	15.59	44	<0.001
2 versus 3	8.75	44	<0.001

Table 4 ☐ The percentage of mean explorer-penetration in relation to the lesion depth (23 sections were used in each group). Group 1 = grooves probed with 100g force. Group 2 = grooves probed with 300g force. Group 3 = grooves probed with 500g force.

Group	Mean	Std. Dev.	Std. Error	Coef. Var.
1	46.7	11.4	2.4	24.40
2	69.5	7.4	1.5	10.50
3	98.2	1.9	0.4	1.93

Table 5 ☐ The percentage of explorer-penetration in relation to the lesion depth was compared by using unpaired t-test, the values for t and probability level for all groups area listed below. Group 1 = grooves probed with 100g force. Group 2 = grooves with 300g force. Group 3 = grooves probed with 500g force.

Group	t-value	Degree of freedom	P
1 versus 2	8.67	44	<0.001
1 versus 3	21.35	44	<0.001
2 versus 3	18.14	44	<0.001

mean percentage of the extent of penetration in relation to the lesion-depth in the bottom of the groove was found to be 69.5 percent.

In the third group, which was probed with the 500 g weight, the mean extent of penetration into the twenty-three grooves was found to be 101.0+11.3 μm (+SD). The mean percentage of the extent of penetration in relation to the lesion depth in the bottom of the groove was found to be 98.2 percent (Table 2).

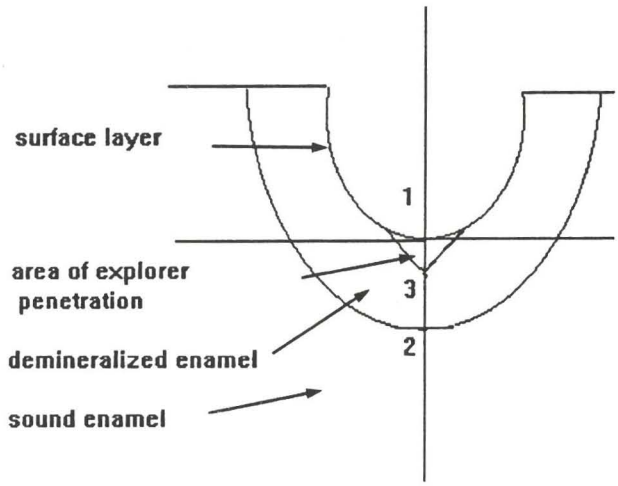

Figure 9. A schematic drawing of how the lesion depth and explorer penetration were measured. 1-2 = lesion depth, 1-3 = explorer penetration.

Comparing the effects of explorer-penetration between the different weight forces applied, there was a highly significant difference in penetration between the first (100 g) and second (300 g) groups (unpaired t-value = 7.037, n=23, $p<0.001$). Also there was a statistically significant difference in penetration between the first (100 g) and the third (500 g) groups (unpaired t-value = 15.59, n = 23, $p<0.001$). The same results were found between the second (300 g) and third (500 g) groups (unpaired t-value = 8.752, n = 23, $p<0.001$) (Table 3).

The percentages of explorer-penetration in relation to the lesion-depth were compared between the three grooves with the three different forces (100 g, 300 g, 500 g) (Table 4). A highly significant difference was observed between 100 g and 300 g (unpaired t-value = 8.674, n = 23, $p<0.001$). Also the same results were found between the 100 g and the 500 g (unpaired t-value = 21.35, n = 23, $p<0.001$) and 300 g and 500 g (unpaired t-value = 18.14, n = 23, $p<0.001$) (Table 5).

DISCUSSION

In the preliminary study, the lesions formed were observed in the first three teeth that were removed at intervals of two, three, and five days, although they were very small and difficult to see under the light microscope at 30× magnification. No X-ray dense surface layer was

visible in these specimens. The appearance was similar to that seen in acid-etching of the enamel. The fourth tooth was removed from the solution after seven days of demineralization. The lesion formed was larger than in the previous specimens, but no evidence of a surface layer could be seen. In the initial stages of lesion formation, it was found by Arends and Christoffersen that the lesion does not show the presence of a relatively sound surface layer; this appeared to develop during the later progress of lesions.[3]

This study used a method of creating artificial U-shaped grooves in the buccal surface of human teeth, as described by Smits and Arends.[4] They created two parallel artificial grooves in enamel of approximately 600 μm in depth and 200 μm in width.

In this study, four parallel artificial U-shaped grooves were made 500 μm in depth, 400 μm in width, and with 1 mm intervals. It has been found that the depths of the grooves were not the same along the entire length of the grooves; the greatest depth was in the middle of the grooves and the shallowest at the two ends. The reason for this was due to the circular shape of the disc. So during sectioning, the sections that were chosen for microradiography were those obtained from the middle areas of the specimens.

Artificial caries lesions may not be identical, however, with natural enamel lesions, but they are in general quite similar to natural lesions in some stages. Great caution must be taken in working with experimental models.

It was noticed that the subsurface lesion in the bottom of the groove was wider than the subsurface lesion in the lateral walls of the groove. This is similar to the findings in the study of Smits and Arends, who found that the progression of the lesion in the wall of a groove was about half as fast as the progression in the bottom portion.[4,5] They attributed the difference to the difference in direction of the prisms in the bottom of the grooves from the direction in the lateral walls.

The findings of the present experiments on the damaging effect of a sharp dental explorer on early enamel lesions are in agreement with the previous studies using a sharp explorer *in vitro* and with clinical studies.[6-8] In the study of Bergman and Linden, interproximal probing was used on extracted teeth with white spot lesions.[6] van Dorp *et al*, however, used probing on artificial grooves of bovine enamel in thin sections. Featherstone and Mellberg found that there is a different rate of lesion formation between bovine enamel and human enamel.[9] They found that lesion formation in bovine enamel was threefold faster compared with human enamel. Ekstrand *et al* found that the occurrence and size of the defects caused by the explorer were strongly related to the degree of tissue opacity, so that the extension of opacity apparently determines the severity of traumatic damage.[8]

Ten Cate and Exterkate compared the demineralization in bulk enamel and thin sections using quantitative microradiography.[10] They found that there was a difference in demineralization between single sections and bulk enamel. The depth of the lesions formed in single sections was found to be deeper than the ones formed in bulk enamel, and more mineral had been removed from the single sections. This discrepancy was explained by the authors to result from vibration experienced by the specimen during sectioning, which might cause an increase in the number of pores accessible to acids. Also cracks were observed in the sections, which might increase the acid susceptibility of the enamel. In contrast Strang *et al* concluded that cutting thick sections and hand-grinding the specimens does not affect them and no difference existed in the way sections and slabs behave, when subjected to demineralization *in vitro*. In this study enamel slabs were used, because it is probably more appropriate for the probing procedures.

The mean lesion-depth in the bottom of the grooves was found to be 101.9+2.7 μm (+SD) in this study. Smits and Arends measured the lesion-depth at the bottom of artificial grooves, using polarized light microscopy and microradiography. They found that there were no significant differences between these two methods for measuring lesion-depth. The mean lesion-depth in the bottom of the groove was 177.0+8.02 μm (+SD) after fourteen days of demineralization. No surface layer was formed in the lesions exposed to a 50 percent saturated HAP solution at a pH of 4.5 (1 ml liquid was added for each 1mm^2 enamel surface exposed). In the study by van Dorp *et al*, the lesion depth was found to be about 200 μm deep after five weeks of demineralization. These lesions were formed in bovine enamel by the single-sections technique exposed to 1 ml of 1 N lactate buffer at pH 4.5 containing 0.2 mM MHDP (methylenehydroxydiphosphonate). This is an inhibitor of crystal dissolution, and so helps the formation of a surface layer, and perhaps slows down the attack, but does not stop it.[12] These differences in the lesion-depth might be due to different groove depths and widths. The other possible reason may be due to the different demineralizing solutions that have been used. Another possibility is in the different techniques of measurements (they measured the lesion-depth from densitometric tracings). Furthermore, the composition of the enamel may be inherently variable, which might account for the difference in the lesion-depth.

The weight force of 500 g was used in the present study because this force had been used in the van Dorp *et al* study.[7] The mean depth of the cavity was found to be 46.5+12.4 μm (+SD) produced by 100 g force of probing, 71.4+11.6 μm (+SD) produced by 300 g force of probing, and 101+11.3 μm (+SD) produced 500 g force of probing. While in the study of Bergman and Linden, the depth of the cavity ranged from 0.1-2.0 mm in diameter produced from interproximal probing of extracted teeth with white-spot lesions.[6] The forces applied on probing ranged from 0.5-0.9 Kp. These results indicate that the traumatic cavity depth produced by probing was more than cavity depth produced in the present study. This was probably due to the force applied in the Bergman and Linden study, which appeared to be greater than the forces applied in the present study.

Different units and symbols of force have been used in the previous studies mentioned. In some instances the author's meaning is unclear. For example, in the Bergman and Linden study, the symbol kp was used to designate the unit of force. If this means *kilo poundals*, 0.9 kp would seem excessively large. If the unit intended is *Pascal (Pa)*, this is the *S.I.* unit for pressure with dimensions of $m^{1}kgs^{2}$. But the *S.I.* unit for force is the *Newton (N)* with dimensions of $mkgs^{2}$.

In the van Dorp *et al* study, they used a force of "0.5 N (0.5 kgf)". There appears to be an internal inconsistency, because the force exerted by 0.5 kg is 0.5 g N, where g is the acceleration of gravity, which is about $10ms^{2}$.

From the above, it is clear that great care is needed in specifying the force applied to an explorer. Clinically it is not possible to measure the force applied on the explorer during a routine diagnostic examination.

CONCLUSIONS AND PROPOSALS FOR FUTURE WORK

From the results of the present study the following conclusions could be drawn:

- ☐ The sharp dental explorer can convert a white-spot lesion with a relatively intact surface layer into a cavity.
- ☐ The extent of this cavity is positively related to the force applied in probing.
- ☐ The occurrence of mechanical damage is positively related to the presence of demineralization of the enamel.

More research is needed on the effect of forces of less than 100 g applied to early lesions, as when cleaning fissures with a dental explorer during a routine clinical examination. Further work is required to learn the influence of this procedure on caries progression and remineralization, and its effect on the ability of probed enamel lesions to resist further acid attack (demineralization).

REFERENCES

1. Von der Fehr, F.R.: Maturation and remineralization of enamel. Ad Fluor Res, 3: 83-95, April, May, June 1965.
2. Snedecor G.W. and Cochran, W.G.: *Statistical methods*. 6th edition. Iowa: Iowa State University Press, Ames, Iowa
3. Arends, J. and Christoffersen, J.: The nature of early caries lesions in enamel. J Dent Res, 65:2-11, January-February 1986.
4. Smits, N.T. and Arends, J.: In vitro demineralization of human enamel in artificial U-shaped grooves. Caries Res, 20:217-222, May-June 1986.
5. Silverstone, L.M.: Structure of carious enamel, including the early lesion. Oral Sci Rev, 3:100-160, 1973.
6. Bergman, G. and Linden, L.A.: The action of the explorer on incipient caries. Svensk Tandlakare Tidsskrift, 62:629-634, November-December 1969.
7. van Dorp, C.S.E.; Exterkate, R.A.M.; ten Cate, J.M.: The effect of dental probing on subsequent enamel demineralization. J Dent Child, 55:343-347, September-October 1988.
8. Ekstrand, K.R.; Qvist, V.; Thylstrup, A.: Light microscope study of the effect of probing in occlusal surfaces. Caries Res, 21:368-374, July-August 1987.
9. Featherstone, J.D.B. and Mellberg, J.R.: Relative rates of progress of artificial carious lesions in bovine and human enamel. Caries Res, 15:109-114, May-June 1981.
10. tenCate, J. M. and Exterkate, R.A.M.: Use of the single section technique in caries research. Caries Res, 20:525-528, November-December 1986.
11. Strang, R.; Damato, F.A.; Stephen, K.W.: Comparison of in vitro demineralization of enamel sections and slabs (short communication). Caries Res, 22:348-349, September-October 1988.
12. Featherstone, J.D.B.; Duncan, J.F.¤tress, T.W.: Surface layer phenomena in in-vitro early caries-like lesions of human tooth enamel. Archs Oral Biol, 23:397-404, August 1978.

Validity of the mother's recall of her child's antibiotic use

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In an epidemiologic investigation of the association between antibiotic use, oral colonization of mutans streptococci (a group of cariogenic bacteria), and tooth decay, we measured the early childhood use of antibiotics in a group of children.[1,2] Theoretically, in places where antibiotics cannot be purchased without a prescription, a valid measure of antibiotic use can be obtained by reviewing medical and dental records of the subjects. Under certain circumstances, however, investigators must rely upon the subjects' recall of their use of antibiotics. These circumstances include inadequate resources, confidentiality, unavailability of records, and the possibility of subject's noncompliance in purchasing or completing the course of antibiotics prescribed.[3-5] Self-reported data on most exposures are prone to error, however, and if the study subjects are children and the measurement of their antibiotic use is based upon parents' recall, these errors might be even greater.[6]

We evaluated the validity of mother's recall of the child's use of antibiotics by comparing it with the child's health records, and also examined the potential determinants of the mother's recall of the child's antibiotic use. To demonstrate the nature and the amount of information bias in the mothers' recall, we compared the association between the mothers' recall of their children's early use of antibiotics and the prevalence of mutans streptococci in children with the association between the prevalence of mutans streptococci and antibiotic use, obtained from health records.

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METHODS

The study subjects were African-American children in grades K-4 (5-12 years) in an inner-city elementary school in Birmingham, Alabama, and their mothers. The only selection criteria for the study were the availability of written parental consent and the assent of the child. These were obtained for each subject according to the guidelines of the Institutional Review Board for Human Use at the University of Alabama at Birmingham. Information related to the use of antibiotics by children since birth was obtained by sending a structured questionnaire home with children asking the mother of the child to complete it and return it to the child's teacher. In the absence of the mother, the father (<1 percent), siblings (<1 percent), aunts (2 percent), or grandparents (5 percent) filled out the questionnaire. A trained interviewer administered the questionnaire over the telephone to those who had telephone facilities and who did not return the completed questionnaire after two reminders. All mothers were asked for consent to review the medical, dental, and emergency records of the child. Consent was obtained from mothers of 152 subjects and the records were located for 126 subjects. Those records that were not located were either lost (N = 7) or sent for micro filming (N =12), or the health care providers declined consent (N = 7). Records were reviewed by one investigator (APD), and detailed antibiotic prescription information for each child was abstracted for each year of life. The investigator had no previous knowledge about the mother's recall of the child's antibiotic use, or

the mutans streptococci levels in the child at the time of the record review. Ninety-four percent of the records reviewed were from the county health department clinics and the Children's Hospital; only 6 percent were from private pediatricians and dentists.

The oral presence and levels of mutans streptococci were assessed in plaque and saliva of children.[7] The identification of mutans streptococci was based on colony morphology and biochemical characteristics.[8]

Mothers who had telephone interviews and those who gave incomplete responses to the questions regarding the child's use of antibiotics (N = 24) were excluded from the analysis. This report then is based on information regarding 102 subjects whose antibiotic usage was obtained from the mother, using a self-administered questionnaire, as well as from the health records.

Data analysis

Depending on the distribution of variables of interest, both parametric and nonparametric statistical methods were used in the analyses. Data on antibiotic use were dichotomized into *never* and *ever* use of antibiotics. The percentage of agreement and Cohen's kappa statistic were used to measure the agreement between mother's recall and health records.[9] McNemar's test with the Edward's continuity correction was used in evaluating the association between the antibiotic use of the child based on mothers' recall and the information obtained from health records.[10,11] Sensitivity, specificity, and positive and negative predictive values of mother's recall were calculated, using the information based on health records as the standard.

Multiple logistic regression analysis was used to evaluate potential determinants of the mother's ability to recall the child's antibiotic use. The concordance or discordance of the mother's response with the information obtained from health records was used as the dependent variable in the models. The determinants evaluated were

- ☐ Ages of the mother and child.
- ☐ Educational level of the mother.
- ☐ Gender of the child.
- ☐ Number of siblings.
- ☐ Age of the child at the first medical visit.
- ☐ A surrogate measure of the household income.

The number of antibiotic prescriptions and the number of emergency visits appearing in the health records of the child were also tested. Goodness-of-fit of the models was tested, using the methods described by Hosmer and Lemeshow.[12] For categorical variables, the regression coefficient obtained from these models estimates the logarithm of the ratio of the concordance odds in one subgroup to the concordance odds in another subgroup (e.g., the concordance log odds ratio for sons compared to daughters). For continuous variables such as age, the regression coefficient estimates the increase in concordance log odds per unit increase in the independent variable. The correlation between the number of antibiotic courses (episodes) reported for the child by the mother, and the actual number of prescriptions given, was assessed using Spearman's Rank Correlation Coefficient.[13] The difference between the median number of episodes obtained from each source was tested using the Median Test.[14] Fisher's Exact test and the Student's t-test were used where appropriate.

In our data, we observed a significant positive correlation between the number of antibiotic prescriptions given to the child during the second year of life and the presence of mutans streptococci in the child's mouth ($r = 0.18$, $p = 0.04$). In evaluating the effect of the source of antibiotic information on the association between mutans streptococci and the antibiotic use, we used, therefore, only those subjects who had complete data on the antibiotic use during the second year of life (N = 82). All statistical tests were two-tailed, and the type I error probability less than or equal to 0.05 was considered as the level of significance. Accordingly, the 95 percent confidence level was used to compute confidence intervals (CI).

RESULTS

Study children were between five and twelve years of age, and their mothers' ages ranged from eighteen to forty-one years. Half the study subjects had their first medical visit by 1.5 months of age. The number of emergency visits per child ranged from 0 to 10 (mean = 0.7, SD = 1.7). Table 1 compares the mother's response with the antibiotic prescription information obtained from health records. The agreement between the mother's recall and the health records was poor (concordance

Table 1 ☐ Comparison of mother's recall of child's antibiotic use with the information obtained from health records of the child.

Child's antibiotic use: Health records	Child's antibiotic use: Mothers' recall		
	Yes	No	Total
Yes	46	25	71
No	14	17	31
Total	60	42	102

Sensitivity = 65%; False negative proportion = 35%
Specificity = 55%; False positive proportion = 45%
Positive predictive value = 77%
Negative predictive value = 40%

Table 2 ☐ The association between child's antibiotic use obtained from health records and the oral presence of mutans streptococci.

Presence of mutans streptococci in child's mouth	Child's antibiotic use during second year: Health records		
	Yes	No	Total
Yes	16	31	47
No	5	30	35
Total	21	61	82

Odds Ratio = 3.1; 95% Confidence Interval = 1.03 – 9.3

Table 3 ☐ The association between child's antibiotic use obtained from the mother and the oral presence of mutans streptococci.

Presence of mutans streptococci in child's mouth	Child's antibiotic use during second year: Mothers' recall		
	Yes	No	Total
Yes	8	39	47
No	4	31	35
Total	12	70	82

Odds Ratio = 1.6; 95% Confidence Interval = 0.44 – 5.8

= 62 percent; kappa = 0.18, p = 0.06). The proportion of children exposed to antibiotics based on records was 70 percent and the proportion based on mother's recall was 59 percent.

None of the potential determinants of the mother's recall of child's antibiotic use evaluated in logistic regression models reached statistical significance. The only factors suggestive of being related to mother's recall, though not significant, were the gender of the child [OR (M/F) = 2.5, 95 percent CI = 0.91-6.9] and the number of emergency visits of the child (Unit OR = 0.66, 95 percent CI = 0.41-1.04), indicating that the mothers tend to recall better for sons than for daughters, and they recall better for children who had few emergency visits than for children who had many visits.

The mother's recall of the number of antibiotic episodes of the child since birth (mean = 1.3, SD = 1.8) was significantly lower (p = 0.02) than the number of episodes obtained from health records (mean = 2.3, SD = 3.2). There was a positive correlation, however, between the mother's recall of the number of antibiotic courses given to the child since birth and the number indicated in the health records (r = 0.17, p = 0.09).

Measurement of the use of antibiotics in children using the two sources of information yielded two different estimates for the association between mutans streptococci and antibiotic use. When the antibiotic information was based on health records, we observed a significant positive association (OR = 3.1, 95 percent CI = 1.03-9.3) between oral presence of mutans streptococci and the use of antibiotics by the child during the second year of life (Table 2). The strength of this association became weaker and nonsignificant (OR = 1.6, 95 percent CI = 0.44-5.8) when the antibiotic information was based on mother's recall (Table 3).

DISCUSSION

There are speculations that the global reduction of tooth decay may be due partly to the widespread use of antibiotics during early childhood.[1] In a study evaluating the association between antibiotic use and a group of cariogenic organisms (mutans streptococci), we addressed the methodological issues related to the measurement of antibiotic use among children. The validity of recall of antibiotic use that occurred in the distant past may often be questionable, especially when the recall is that of someone other than the study subject. We evaluated the validity of mother's recall as a surrogate for the child's use of antibiotics, since the mother was identified as the primary caretaker during the early childhood for all 102 children included in this study.

Our findings indicate that the agreement between the mother's recall and health records in identifying children ever exposed to antibiotics is poor. The mother's recall was more sensitive than specific. That is, the correct classification of the child's antibiotic use by the mother was greater when the child had actually been given antibiotic prescriptions than when the child had not been given antibiotic prescriptions. The sensitivity of mother's recall of the child's antibiotic use in our study (65 percent) was similar to the sensitivity of mother's recall of her own antibiotic use reported by Werler and coworkers.[15] They reported a sensitivity of 67 percent for the mothers of malformed children and 54 percent for mothers of nonmalformed children. In our study, the positive predictive value (i.e., the probability of finding a prescription in health records of the child when the mother indicated so) was greater than the probability of the mother being correct when she indicated that the child never received antibiotics (negative predictive value).

Mothers often recalled a fewer number of antibiotic courses than indicated in the records. This underestimation appeared to be consistent across different frequencies of antibiotic use.

We did not identify any significant predictors of the accuracy of the mother's recall of the child's antibiotic use, which is compatible with random misclassification of the child's antibiotic use by the mother. The mothers misclassified the amount of child's antibiotic use between twelve and twenty-four months of age to such an extent that the measure of association (odds ratio) between antibiotic use and mutans streptococci was biased

toward the null value of 1. This finding implies that the real association between antibiotic use and mutans streptococci should be greater than what is observed, when information on the child's antibiotic use is obtained from the mother.

There are threats to the validity of our results. Even though mothers whose children's records were not reviewed (N = 26) recalled a higher frequency of antibiotic use for their children compared with those whose records were reviewed (N = 126), there were no differences in mother's recall between the subjects whose records were reviewed but excluded from the analysis (N = 24), and those who were included (N = 102). Since none of the variables by which the two groups differ was a significant predictor of the accuracy of the mother's recall in our multiple logistic regression models, it is unlikely that nonparticipation led to any selection bias.

Because the original study was planned to evaluate the association of mutans streptococci and antibiotic use, the possibility of information bias has also to be considered. The differential misclassification of antibiotic data obtained from health records (i.e., abstraction of antibiotic data is uneven between those with and without mutans streptococci) is unlikely because, at the time of the record review, the investigator was blind as to the mother's recall as well as to the mutans streptococci and caries status of the child. Mother's misclassification of the child's antibiotic use may have also been random (i.e., maternal recall of antibiotic use is even for children with and without mutans streptococci) since she was blind as to the study hypotheses and to the mutans streptococci and caries status of her children.

The other possible pitfall of our study is related to the standard we chose to measure the validity of the mother's recall. There are some potential problems associated with the use of records as a standard. Noncompliance with prescribed medication, the possibility that children may have been given antibiotics prescribed to the others such as siblings, and incomplete medical records are among them. Noncompliance has been called primary when the prescription is not filled; and secondary when it is filled, but not taken as prescribed.[4,5] We cannot rule out the possibility that what we interpret as an underestimation by the mother reflects actual compliance. It seems unlikely, however, that noncompliance with prescriptions would be as high as what we observed (35 percent false negative). In a study examining primary noncompliance, Saunders reported that 21 percent of the antibiotic (or antifungal) prescriptions were not filled by patients discharged from the emergency department and 36 percent of these subjects identified insufficient funds as the reason for noncompliance.[3] Since more than 90 percent of our study subjects attended county health department clinics, where most were covered by Medicaid, we estimate the proportion of unfilled prescriptions among our study subjects to be lower than 21 percent. Antibiotics from other sources are less of a problem in the U.S. where antibiotics cannot be purchased without a prescription. Thus, the discrepancy we observed between mother's recall and health records most likely reflects underreporting by the mother.

The generalizability of these results to other dissimilar populations may be questionable, since all our study mothers were inner-city, low income, African-American mothers. We conclude that the mother's recall of the child's antibiotic use obtained by using a self-administered questionnaire is subjected to considerable underestimation at least among innercity, low income blacks. This mode of data collection in epidemiologic investigations should probably be limited, therefore, to circumstances where no other options are available and where there is sufficient power to detect an association, even in the presence of substantial misclassification of antibiotic use. Results of such studies should be interpreted with caution. Informativeness of future studies in this area could be enhanced by encompassing a wider range of the population, perhaps by utilizing interviewer administered questionnaires, and by comparing *mother's recall* to a better standard.

REFERENCES

1. Loesche, W.J.; Eklund, S.A.; Mehlisch, D.F. *et al*: Possible effect of medically administered antibiotics on the mutans streptococci: Implications for reduction in decay. Oral Microbiol Immunol, 4: 77-81, June 1989.
2. Staves, E. and Tinanoff, N.: Decline in salivary *S. mutans* levels in children who have received short-term antibiotic therapy. Pediatric Dent, 13:176-178, May-June 1991.
3. Saunders, C.E.: Patient compliance in filling prescriptions after discharge from the Emergency Department. Am J Emerg Med, 5: 283-286, July 1987.
4. Rashid, A.: Do patients cash prescriptions? Br Med J, 284:23-26, January 1982.
5. Hulka, B.S.; Cassel, J.C.; Kupper, L.L. *et al*: Communication, compliance, and concordance between physicians and patients with prescribed medications. Am J Public Health, 66:847-853, September 1976.
6. Strecher, V.J.; Becker, M.H.; Ckark, N.M. *et al*: Using patients' descriptions of alcohol consumption, diet, medication compliance, and cigarette smoking: the validity of self reports in research and practice. J Gen Intern Med, 4:160-166, March-April 1989.
7. Kohler, B. and Bratthall, D.: Practical method to facilitate estimation of *Streptococcus mutans* levels in saliva. J Clin Microbiol, 9:584-588, May 1979.
8. Shklair, I.L. and Keene, H.J.: A biochemical scheme for the separation of the five varieties of Streptococcus mutans. Arch Oral Biol, 19:1079-1081, November 1974.

9. Cohen, J.: A coefficient of agreement for nominal scales. Educ Psychol Meas, 20:37-46, August 1960.
10. McNemar, Q.: Note on the sampling error of the difference between correlated proportions or percentages. Psychometrika, 12: 153-157, September 1947.
11. Edwards, A.L.: Note on the "correction for continuity" in testing the significance of the difference between correlated proportions. Psychometrika, 13:185-187, August 1948.
12. Hosmer, D.W. and Lemeshow, S.: The importance of assessing the fit of logistic regression models: A case study. Am J Pub Health, 81:1630-1635, December 1991.
13. Spearman, C.: The proof and measurement of association between two things. Am J Psychology, 15:72-101, January 1904.
14. Mood, A.M.: *Introduction to the theory of statistics*. New York: McGraw-Hill, 1950, p 25.
15. Werler, M.M.; Pober, B.R.; Nelson, K. *et al.*: Reporting accuracy among mothers of malformed and nonmalformed infants. Am J Epidemiol, 129:415-421, February 1989.

SKATEBOARD INJURIES

Skateboarding has resurged and so have its associated hazards and injuries. There are an estimated 8 million skateboarders now in the United States. Pediatricians informed about skateboard activities in their areas can help prevent needless injuries to children and adolescents.

During the last skateboard injury epidemic, the annual incidence of injuries peaked at 150,000 in 1977 and subsequently decreased to 16,000 in 1983. It is likely that this decrease in injuries was primarily related to decreased skateboard activity and not to improved safety conditions. With increased popularity the number has risen, with an estimated 56,435 injuries being treated in emergency departments in 1992. In addition, an estimated 1900 hospitalizations occurred due to skateboard-related injuries during this period. The vast proportion of admissions were from head injuries.

The American Academy of Pediatrics makes the following recommendations:

- ☐ Children younger than 5 years of age should not use skateboards. Their center of gravity is higher, their neuromuscular system is not well developed, their judgment is poor, and they are not sufficiently able to protect themselves from injury. More developmentally appropriate activities need to be encouraged.
- ☐ Skateboards must never be ridden near traffic. Their use should be prohibited on streets and highways. Activities that bring skateboards and motor vehicles together ("catching a ride") are especially dangerous.
- ☐ Skateboarders need to be encouraged to wear helmets and protective padding for their elbows and knees to reduce or prevent injury. The experience of children wearing multi-purpose helmets needs evaluation.
- ☐ Communities should be encouraged to develop safe skateboarding areas away from pedestrian and motor vehicle traffic.

Committee on Injury and Poison Prevention, Am Acad Pediatrics: Skateboard injuries. Pediatrics, 95:611, April 1995.

Natal and neonatal teeth

Jianfu Zhu, DDS, MS, DSc
David King, DDS, PhD

Normal eruption of mandibular primary incisors starts at about six months of age. Periodically in the medical and dental literature, cases are reported of infants born with teeth already erupted. Various terms have been used to designate teeth that have erupted before their normal time, such as congenital teeth, fetal teeth, predeciduous teeth, and dentitia praecox. The classification advocated nearly forty-four years ago by Massler and Savara for teeth erupting prematurely is still the one most widely used at present; teeth present at birth are termed natal teeth, whereas those that erupt within thirty days after birth are called neonatal teeth.[1] It has been suggested by Spouge and Feasby, however, that such teeth also should be further classified according to their degree of maturity.[2] A mature natal or neonatal tooth is one that exhibits normal development, hence has a relatively good prognosis; while the term immature natal or neonatal tooth implies defective development and a poorer prognosis for retention.

HISTORICAL REFERENCES AND SOCIAL SIGNIFICANCE

Natal and neonatal teeth have been observed and recorded from very ancient sources, such as the cuneiform inscriptions found at Nineveh(59 BC).[3] Superstitions and folklore concerning these teeth have varied from claims that such children were exceptionally favored by fate to the belief that they were doomed. As examples of the former it is said that Louis XIV, Richard III, Napoleon, Mazarin, Mirabeau, Zoroaster, Hannibal, and Cardinal Richelieu were born with teeth.[4] In Poland, Africa, India, and China such children have been regarded as monsters and bearers of misfortune.[5,6]

PREVALENCE

The reported prevalence of natal and neonatal teeth has varied considerably from one in every 11.25 to 30,000 births (Table I), this wide range may result from inconsistencies in survey methodologies.[1,6-13] The highest frequencies are found in those studies that rely on prospective examination of patients. Ballantyne was probably the first to make an estimate of the prevalence. He reported three cases in 17,578 births (1 in 6,000) at the Paris Maternity Hospital.[8] In 1948, Massler and Savara reported the frequency of natal and neonatal teeth to be one in 2000 births at two hospitals in Chicago.[1] Allwright (1958) reported a prevalence of one per 3,400 births.[9] Recently, the results from two independent studies of natal and neonatal teeth in Hong Kong Chinese indicate that the prevalence to be 1:1,118.and 1:1,442.[12-13]

Natal teeth are encountered more often than neonatal teeth in an approximate ratio of three to one.[7,10] This is not surprising since mother and baby usually return home soon after the birth event and many neonatal teeth

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Table 1 ☐ Prevalence of natal and neonatal teeth.

Author	Total births	Number of infants with natal or neonatal teeth	Ratio
Puech, 1876	60,000	2	1:30,000
Ballantyne, 1897	17,578	3	1:6,000
Massler, 1948	6,000	4	1:1,500
Massler, 1948	5,400	3	1:1,800
Allwright, 1958	6,817	2	1:3,400
Mayhall, 1967	90	8	1:11.25
Kates, 1984	7,155	10	1:716
Leung, 1986	50,892	15	1:3,392
Bedi, 1990	28,850	20	1:1,442
To, 1991	53,678	48	1:1,118

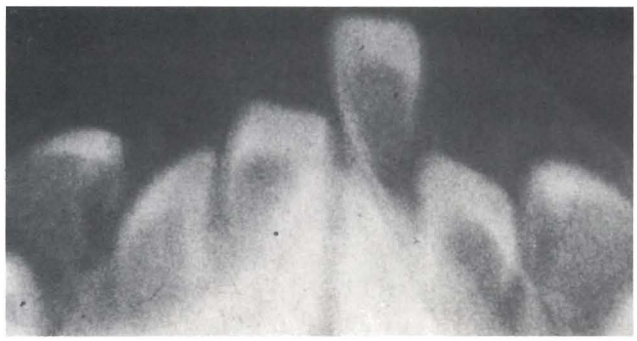

Figure 1. Occlusal radiograph of the mandibular anterior area shortly after birth. Note the rootless, shell like, superficially located tooth structures.

may not be documented. Rarely will a child exhibit both types of teeth.[10]

Some previous surveys suggest that more females are affected than males, but in two recent major studies no significant difference was shown.[1,2,6,12,13] Kates and his coworkers pointed out that more females were enrolled in the previous clinical studies, which might reflect a bias, such as greater parental or professional concern over affected females.[10]

TEETH AFFECTED

The teeth affected most often are the lower primary central incisors. Kates and coworkers in a three year study of 18,155 infants born at Boston Hospital reported sixty-one natal and neonatal teeth, all in the mandibular primary incisor position.[10] In another recent report, all of the natal and neonatal teeth found among 53,678 Hong Kong Chinese were also mandibular central incisors.[13] The strong predilection for the lower central incisors is not surprising in view of the fact that these teeth are normally the first to erupt into the oral cavity. According to Bodenhoff's study of natal and neonatal teeth, 85 percent are mandibular incisors, 11 percent are maxillary incisors, 3 percent are mandibular canines and molars, and only 1 percent are maxillary canines or molars.[14] The presence of natal and neonatal molars appears, therefore, to be rare. To date twenty cases of natal molars have been reported, but only six of them are mandibular molars.[6,8,15-21]

Most studies found between 38 to 76 percent of natal and neonatal teeth occurred in pairs.[1,6,10,12,22,23] Natal and neonatal teeth usually represent units of the normal primary complement. Several reports indicate only 1 to 10 percent are supernumerary.[1,6,7,10,22]

ETIOLOGY

Over the years there have been many suggestions regarding the cause of premature eruption, including hypovitaminosis, hormonal stimulation, trauma, febrile states, pyelitis, and syphilis, but a cause and effect relationship has not been established.

Currently, natal and neonatal teeth are attributed to a superficial position of the developing tooth germ, which predisposes the tooth to erupt early.[7,24] Boyd and Miles show this clearly in both their anatomical sections and radiographs of the fetal mandible. The erupted primary central incisors were located, not in an alveolus, but in a slight hollow on the surface of the alveolar bone, very much above the germ of the permanent successor.[25] We have observed similar eruption patterns in our patients (Figure 1). The explanation for this condition is uncertain, but it may be related to hereditary factors.

Many investigators have reported natal and neonatal teeth as a familial trait with a frequency ranging from 8 percent to 62 percent. This may reflect a hereditary factor in the occurrence of these teeth.[1,6,7,10,12,13,26] Holt and McIntosh reported a family in which natal teeth occurred in members of three successive generations.[27] Hyatt described a family in which five siblings were born with erupted teeth.[28] Herpin observed natal teeth in two children born of the same mother, but with different fathers.[29] On the other hand, Asana reported natal teeth in two children of the same father, but born of different mothers.[30] In a study by Kates and coworkers, of thirty-six affected individuals, seven had a positive family history of natal or neonatal teeth.[10] Recently Bedi and Yan, in their twenty cases, found only one infant with a positive family history.[12]

Table 2 ☐ Syndromes and developmental abnormalities associated with natal or neonatal teeth.

Adrenogenital syndrome (Leung, 1986)
Chondroectodermal dysplasia (Feingold *et al*, 1966)
Cutis gyratum and acanthosis nigricans (Beare *et al*, 1969)
Cyclopia (Boyd and Miles, 1951)
Oculomandibulodyscephaly (Russell and Bass, 1970)
Pachyonychia congenita (Murray, 1921; Anncroth *et al*, 1975)
Patent ductus arteriosus and intestinal pseudo-obstruction (Harris *et al*, 1976)
Pfeiffer syndrome type 3 (Alvarez, M.P., 1993)
Pierre Robin syndrome (Leung, 1986)
Rubinstein-Taybi syndrome. (Hannekam and Doorne, 1990)
Short rib-polydactyly syndrome. (Gorlin *et al*, 1976)
Steatocystoma multiplex (King and Lee, 1987)
Van der Woude syndrome (Hersh and Verdi, 1992)
Wiedemann-Rautenstrauch neonatal progeria (Devos *et al*, 1981)

Figure 2. Natal tooth partially covered with gingival tissue.

Evidence of genetic contribution is also seen in the association of natal or neonatal teeth with multisystem syndromes and developmental abnormalities.[11,25,31-46] A summary of these findings is presented in Table 2.

Whether or not natal or neonatal teeth occur in these syndromes with a greater frequency than found in the general population is not well established. Some of the syndromes or developmental abnormalities that include chondroectodermal dysplasia, oculomandibulodyscephaly, pachyonychia congenita, and cleft palate or cleft lip have been frequently found associated, however, with natal or neonatal teeth.[31-36]

In the spring of 1979, there was an epidemic of acne with other symptoms among boarders at a school for blind children in central Taiwan.[47] An epidemiological investigation together with analytical chemical studies indicated that the illness was a form of chloracne, due to the ingestion of rice-bran cooking oil that had been contaminated during manufacture with a mixture of polychlorinated biphenyls (PCBs). This was the second recorded epidemic of chloracne due to PCB contamination of cooking oil; the first had occurred in Japan in 1968 and involved over 1000 cases.[48] At birth, exposed children had increased rates of eyelid swelling and discharge, nail deformity, hyperpigmentation, acne, natal teeth, and swollen gums compared to controls. In addition they had more generalized itching, localized skin infections and hair loss.

In summary, most prematurely erupted teeth seem to occur in otherwise normal infants, with or without a family history of the condition. In some infants they may be a localized manifestation of an underlying syndrome or attributed to environmental factors, thus accentuating the importance of a thorough evaluation of infants with natal or neonatal teeth.

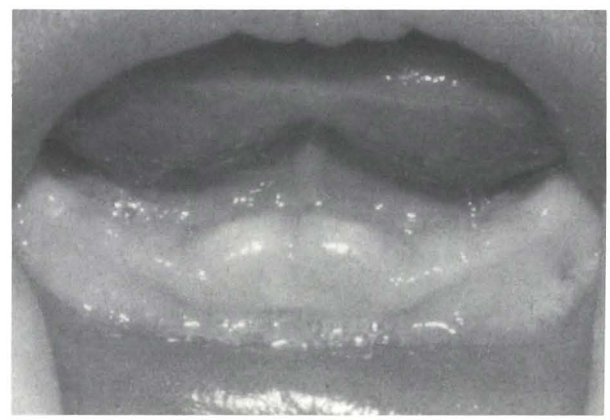

Figure 3. Clinical appearance of an elevated mucosal papilla within which the crown can be felt shortly after birth.

CLINICAL APPEARANCE

Natal and neonatal teeth may resemble normal primary teeth; but, in many instances, they are poorly developed, small, conical, yellowish, with hypoplastic enamel and dentin, and with poor or total failure of development of roots.[49] The appearance of each natal tooth can be classified in one of the following four categories, which are based on the observations of previous authors.[6,13]

- ☐ A shell-like crown structure loosely attached to the alveolus by a rim of oral mucosa; no root.
- ☐ A solid crown loosely attached to the alveolus by oral mucosa; little or no root.
- ☐ The incisal edge of the crown just erupted through the oral mucosa (Figure 2).
- ☐ A mucosal swelling with the tooth unerupted but palpable (Figure 3).

Figure 4. Intraoral photograph showing two mandibular natal teeth. The enamel is thin, rough, yellowish in color.

Figure 5. Clinical photograph showing the natal tooth and the large ulcerated area on the ventral surface of the tongue. Note sharp enamel spur on incisal which seems to be a consistent feature in Riga-Fede.

According to To, natal teeth of category 1, and those of category 2, if the degree of mobility is more than 2 mm, are candidates for extraction.[13]

HISTOPATHOLOGY

Histologically, natal or neonatal tooth-enamel may follow the normal pattern of mineralization of human primary enamel.[23] The majority of such teeth present abnormalities, however, in all tooth structures.[24,25] Enamel hypoplasia has been frequently observed (Figure 4). In the dentin, large interglobular spaces with abnormal cell inclusions have been found, as well as an irregular pattern of dentinal tubule orientation.24 Adjacent to and in part of the cervically situated dentin, changes in the dentin are more accentuated, and in some cases dentinal tubules are completely absent in smaller regions.[26] Both Hertwig's sheath and cementum may be absent.[18,26] Other findings include absence of Weil's basal layer and the cell-rich zone in the pulp, and an increase in the number of dilated blood vessels in the pulpal tissue.[7,24]

It has been noted that, after the hypoplastic enamel is exposed to the oral cavity, it will usually become a yellow brown and progressively deteriorate.[22] Other studies have confirmed this observation.[10,23] The severity of enamel deterioration increases with length of exposure.

CLINICAL SYMPTOMS AND COMPLICATIONS

The presence of a mobile, prematurely erupted tooth may elicit pain so that the baby refuses to nurse. Other symptoms such as infantile diarrhea, drooling, and malaise have, in some cases, been associated with the eruption of neonatal teeth, but only anecdotal evidence exists.[1,50] Some newly erupted neonatal or natal teeth are loose and movable in all directions. This is because the tooth is attached only at the cervix to the gingiva because root formation is incomplete and bony attachment by gomphosis is not possible. Hypermobility has caused concern over the years because of the likelihood of swallowing or aspirating the tooth.[1,6,13,51] Inhalation of one of these teeth has never been reported in the literature, however, and the danger is probably more imagined than real. Enamel spurs or sharp incisal edges may cause ulceration on the ventral surface of the tongue (Riga-Fede disease) (Figure 5), which can result in the infant's refusing to nurse. Additionally, the erupted tooth may cause laceration of the nursing mother's nipples, but this possibility is also probably more speculative than real.[24,27] Normally the infant's tongue overlies the lower incisors when nursing, and any trauma will be to the infant's tongue rather than mother's breast.

MANAGEMENT OF NATAL OR NEONATAL TEETH

If the tooth is not causing any difficulty to the infant or mother, it should be left alone. Neonatal teeth are usually less mobile, and natal teeth frequently become less mobile by one month of age.[1,52] Natal and neonatal teeth that survive beyond four months often have a good prognosis.[10] Generally the gingival tissues are normal; occasionally they are, however, edematous and hemorrhagic. King and Lee recommend that inflamed gingival tissue around teeth not scheduled for extraction be controlled by applying chlorhexidine gluconate gel three times a day.[52]

Ulceration of the tongue (Riga-Fede disease) is caused by the forward position of the tongue during sucking (thumb, breast or bottle) causing pressure of the tongue on the tooth. This condition may be relieved by grinding any sharp edges or projections on the tooth. In itself, Riga-Fede is not an absolute reason for extraction.

Extraction of a natal or neonatal tooth should not present significant difficulties; the underdeveloped cells of the dental papilla and those of the Hertwig root sheath are easily detached, however, from the calcified part of the tooth. These cells, which remain in the alveolus, may continue to develop into a toothlike structure.[53-55] This occurs in 9.1 per cent of the children and a few of them may result in an alveolar abscess.[52] Hence, extraction of natal and neonatal teeth should be followed by curettage of the socket to prevent continued development of the cells of the dental papilla.

Delaying surgical procedures on newborns until after the tenth postpartum day is no longer considered routinely necessary because of the prophylactic administration of vitamin K that is standard procedure in most hospitals.[56] If necessary, hemostasis may be enhanced by using topical hemostatic agents in combination with direct pressure. Where true space loss normally is not encountered after extraction of natal and neonatal primary incisor, extraction of a natal or neonatal molar is more likely to result in loss of arch space.[57] Each case must be evaluated independently and sound clinical judgment must be used to decide whether to retain or extract the tooth in question.

REFERENCES

1. Massler, M. and Savara, B.S.: Natal and neonatal teeth: a review of twenty-four cases reported in the literature. J Pediatr, 36:349-359, March 1950.
2. Spouge, J.D. and Feasby, W.H.: Erupted teeth in the newborn. Oral Surg, 22:198-208, August 1966.
3. Livius, Titus: Livy; with an English Translation by E.T. Sage and A.C. Schlesinger. Book XLI; Chap. 21, 1938, p 255.
4. Thoma, K.: *Oral pathology*, ed. 2, St. Louis: The C.V. Mosby Company, 1941, p 164.
5. Charon, L.F.: L'eruption precoce, description d'un cas. Arch Stomat (Liege), 10:272-275, 1955.
6. Allwright, W.C.: Natal and neonatal teeth: a study among Chinese in Hong Kong. Br Dent J, 105:163, September 1958.
7. Bodenhoff, J. and Gorlin, R.J.: Natal and neonatal teeth: folklore and fact. Pediatrics, 32:1087-1093, December 1963.
8. Ballantyne, J.W.: Note on three additional cases of congenital teeth. Tr Edin Obst Soc, 23:112, 1897.
9. Mayhall J.T.: Natal and neonatal teeth among the Tlinget Indians. J Dent Res, 46:748-749, February 1967.
10. Kates G.A.; Needleman H.L.; Holmes L.B.: Natal and neonatal teeth: a clinical study. J Am Dent Assoc, 109:441-443, September 1984.
11. Leung, A.K.C.: Natal teeth. Am J Dis Child, 140:249-251, March 1986.
12. Bedi R. and Yan S.W.: The prevalence and clinical management of natal teeth-a study in Hong Kong. J Paediatr Dent, 6:85-90, October 1990.
13. To, E.W.H.: A study of natal teeth in Hong Kong Chinese. Int J Paediatr Dent, 2:73-76, August 1991.
14. Bodenhoff, J.: Dentitio connatalis et neonatalis. Odont Tidskr., 67: 645-695, 1959.
15. Friend, G.W.; Mincer, H.H.; Curruth, K.R.: Natal primary molar: case report. Pediatr Dent, 13:173-175, May-June 1991.
16. Kakarantza-Angelopoulou, E.; Papagiannoulis, L; Nikas, G.: Multiple immature natal teeth in a newborn. J Clin Pediatr Dent, 17: 157-162, Spring 1993.
17. Alvarez, M.P.; Crespi, P.V.; Shanske, A.L.: Natal molars in Pfeiffer syndrome type 3: a case report. J Clin Pediatr Dent, 18:21-24, Fall 1993.
18. Oriola J.P.: The presence of teeth at birth. Dent Cosmos, 49: 1104,1907.
19. Wong H.B.: Natal and neonatal teeth in Singapore. J Singapore Paediatr Soc, 4:74-82, 1962.
20. Soni N.N.; Silberkweit, M.; Brown, C.H.: Polarized light and microradiographic study of natal teeth. J Dent Child, 34:433-438, November-December 1967.
21. Sigal, M.J.; Mock, D.; Weinberg, S.: Bilateral mandibular hamartomas and familiar natal teeth. Oral Surg, 65:731-735, June 1988.
22. Bjuggren, G.: Premature eruption in the primary dentition-a clinical and radiological study. Swed Dent J, 66:343-355, July 1973.
23. Berman, D.S. and Silverstone, L.M.: Natal and neonatal teeth: a clinical and histological study. Br Dent J, 139:361-364, November 1975.
24. Hals, E.:Natal and neonatal teeth: histologic investigations in two brothers. Oral Surg, 10:509-521, May 1957.
25. Boyd J.D. and Miles A.E.W.: Erupted teeth in a cyclops foetus. Br Dent J, 91:173-181, October 1951.
26. Anneroth, G.; Isacsson G.; Lindwell, A.*et al*: Clinical, histological, and microradiographic study of natal, neonatal, and preerupted teeth. Scand J Dent Res, 86:58-66,January 1978.
27. Holt, L.E. and McIntosh, R.: *Holt's diseases of infancy and childhood.* 2nd ed. New York: D. Appleton-Century-Crofts Company, Inc., l940, p 41.
28. Hyatt, H.W.: Natal teeth: its occurrence in five siblings. Clin Pediatr, 4:46-48, January 1965.
29. Herpin, A.: Cases of the presences of the teeth at birth. Dent Cosmos, 54:121, 1912.
30. Asana, D.J.: Infant born with two lower central incisor teeth. Indian M Gaz, 56:16, 1921.
31. Feingold, M.; Jankoski, J.; Johnson *et al*: Ellis-van Creveld syndrome. Clin Pediatr (Phila) ,5:431-436, July 1966.
32. Gorlin, R.J.; Pindborg, J.J.; Cohen, M.M.: *Syndromes of the head and neck.* New York: McGraw-Hill, p 82.
33. Russell, M.A.J. and Bass J.W.: Hallermann-Streiff syndrome: clinical and prognostic considerations. Am J Dis Child, 120:462-465, November 1970.

34. Murray, F.A.: Congenital anomalies of nails associated with erupted teeth at birth. Br J Dermatol, 33:409-411, December 1921.
35. Anncroth, G.; Isacsson, G.; Lagerholm, B. *et al*: Pachyonychia congenita. Acta Derm Venereal, 55:387-394, 1975.
36. Herpin, A.: Les dents a la naissance, abstracted. Dent Cosmos, 54: 121, 1912.
37. McDonald, R.M. and Reed, W.B.: Natal teeth and steatocystoma multiplex complicated by hidradenitis suppurativa. Arch Dermatol, 112:1132-1134, January 1976.
38. King, N.M. and Lee, A.M.P.: Natal teeth and steatocystoma multiplex: a newly recognized syndrome. J Craniofac Genet Dev Biol, 7:311-317, August 1987.
39. Demarquay J.N.: Quelques considerations sur le bec-de-lievre. Gaz Med Paris 13:52-53, 1845.
40. Hersh, J.H. and Verdi, G. D.: Natal teeth in Monozygotic twins with Van der Woude syndrome. Cleft Palate-Craniofacial J, 29: 279-281, May 1992.
41. Bocian, M. and Walker, A.P.: Lip pits and deletion lq32-41. Am J Hum Genet, 26:432-433, February 1987.
42. Murray, J.C.; Nishimura D.Y.; Buetow, K.H. *et al*: Linkage of an autosomal dominant clefting syndrome (Van der Woude) to loci on chromosome lq. Am J Hum Genet, 46:486-491, March 1990.
43. Devos, E.A.; Leroy, J.G.; Frijns, J-P.*et al*: The Wiedemann-Rautenstrauch or neonatal progeroid syndrome: report of a patient with consanguineous parents. Eur J Pediatr, 136:245-248, July 1981.
44. Hannekam, R.C.M. and Doorne, J.M.W.: Oral aspects of Rubinstein-Taybi syndrome. Am J Med Genet, Suppl, 6:42-47, 1990.
45. Beare, J.M.; Dodge, J.A.; Nevin, N.C.: Cutis gyratum, acanthosis nigricans and other congenital anomalies: a new syndrome. Br J Derm, 81:241-247, April 1969.
46. Harris, D.J.; Ashcraft, K.W.; Beatty, E.C. *et al*: Natal teeth, patent ductus arteriosus and intestinal pseudo-obstruction: a lethal syndrome in the newborn. Clin Genet, 9:479-482, May 1976.
47. Gladen, B.C.; Taylor, J.S.; Wu, Y-C. *et al*: Dermatological findings in children exposed transplacentally to heat-degraded polychlorinated biphenyls in Taiwan. Br J Dermatol, 122:799-808, June 1990.
48. Kuratsune, M.; Yoshimura, T.; Matsuzaka, J. *et al*: Epidemiologic study of Yusho, a poisoning caused by ingestion of rice oil contaminated with a commercial brand of polychlorinated biphenyls. Environ Health Perspect, 1:119-128, 1972.
49. Wei, S.H. (ed.). *Pediatric dental care*. Am Acad Pedodont Monograph, ed 1. New York: Medcom, Inc., 1978, p 9.
50. King, D.L.: Teething revisited. Pediatr Dent, 16:179-181, May/ June 1994.
51. Chow, M.H.: Natal and neonatal teeth. J Am Dent Assoc, 100: 215-216, February 1980.
52. King, N.M. and Lee, A.M.P.: Prematurely erupted teeth in newborn infants. J Pediatr, 114:807-809, May 1989.
53. Ryba, G.E. and Kramer, I.R.H.: Continued growth of human dentine papillae following removal of the crowns of partially formed deciduous teeth. Oral Surg, 15:867-875, July 1962.
54. Southam, J.C.: Retained dentine papillae in the newborn. Br Dent J, 125:534-538, December 1968.
55. Ooshima, T.; Mihara, J.; Saito, T. *et al*: Eruption of tooth-like structure following the exfoliation of natal tooth: Report of case. J Dent Child, 53:275-278, July-August 1986.
56. Holder, T.M. and Ashcraft, K.W.: Pediatric Surgery. Philadelphia: W.B. Saunders Co. 1980, p 67.
57. Gardiner, J.H.: Erupted teeth in the newborn. Proc R Soc Med, 54:14-16, June 1961.

SUBSTITUTION OF FLUORIDE RINSES

Our aim was to study the dental health development after substitution of fluoride rinses and supervised toothbrushings by unsupervised use of fluoride toothpaste.

In conclusion our results suggest that discontinuation of supervised mouthrinses and toothbrushings at schools decrease the number of caries-free individuals and increase the number of carious teeth needing restorative treatment. From a clinical perspective the present results further suggest that during eruption and maturation of permanent teeth, fluoride mouthrinses and supervised toothbrushings should be continued at schools or replaced by an equivalent.

Karjalainen, S. *et al*: Caries development after substitution of supervised fluoride rinses and toothbrushings by unsupervised use of fluoride paste. Community Dent Oral Epidemiol, 22:421-424, December 1994.

Glanzmann's thrombasthenia: The use of autologous fibrin glue in tooth extractions

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Gavriel Lavie, DMD
Uri Martinowitz, MD

Glanzmann's thrombasthenia is an inherited autosomal recessive bleeding disorder, due to functional platelet abnormality.[1] The biochemical lesion is quantitative and/or qualitative abnormality of the platelet fibrinogen receptor glycoprotein IIb/IIIa.[2-4] Since normal platelet aggregation requires fibrinogen binding to the platelet membrane, thrombasthenia platelets are unable to form aggregates.[5] This rare disorder appears more often among certain ethnic groups; in Israel they are found only among Jews of Iraqi origin and Arabs.[6] The clinical symptoms are characterized by varying degrees of mucosal and cutaneous bleeding, epistaxis, gingival bleeding petechiae, purpura, menorrhagia, gastrointestinal bleeding and hematuria.[5,7,8] Serious bleeding may follow surgery in patients not prepared with platelet transfusion. Many of the patients are becoming refractory to platelet transfusions, due to the development of alloantibodies against platelets.

It is recommended, therefore, to leave platelet transfusion only to life-threatening, bleeding episodes and to use HLA-matched platelets in such cases. This report describes dental extractions in a thrombasthenic girl, without the use of platelet transfusion and with the use of autologous fibrin glue.

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The rationale behind the use of fibrin glue is to induce clot formation at the site of the surgical wound (Figure 1). The fibrin glue mimics the final phase of blood clotting, thus bypasses the missing or defected links in the coagulation cascade. Thrombin causes fibrinogen to coagulate; factor XIII, as an adjuvant, provides for the cross-linkage and stabilization of the fibrin clot; aprotinin. an antiprotease, prevents early disruption of the clot during the fibrinolysis process.

CASE REPORT

A fifteen-year-old Iraqi origin girl, suffering from Glanzmann's thrombasthenia, was found to have numerous carious lesions and four teeth 15, 26, 36, 46 were indicated for extraction (Figure 2). For the last four months she was treated by contraceptives due to severe menorrhagia. Because of a history of severe bleeding complication after dental treatment that required platelet transfusion, a decision was made to start first with the dental operative treatments and only later to approach the surgical procedures.

Five sessions of operative dental treatments were conducted with the use of local regional or intraligamentary anesthetic injections and prophylactic swish and swallow of 1 gr of tranexamic acid in a solution, one hour before treatment. In the next four days, swish and swallow routine was followed twice a day of the same dosage of 500 mg (an ampule). No postoperative bleeding occurred at

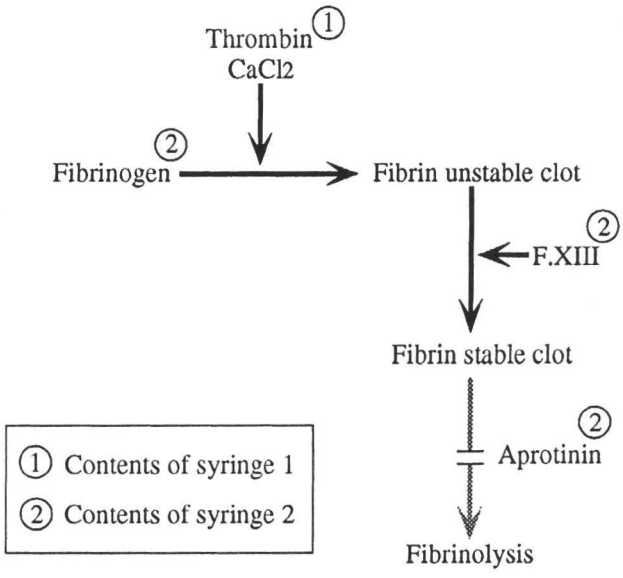

Figure 1. Mechanism of action of fibrin glue.

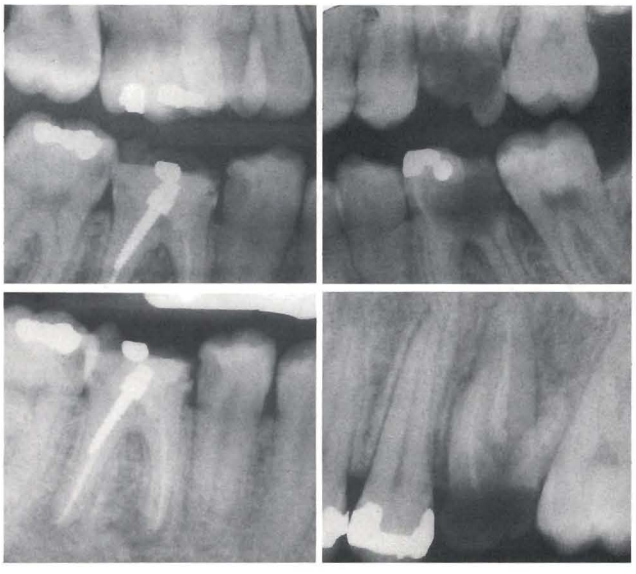

Figure 2. Radiographs of teeth indicated for restorative dental care and extractions (#15, #26, #36, #46).

the sites of the injection, matrix bands, or rubberdam clamp placement. As the dental operative phase was completed, the patient was prepared for the surgical phase. The patient donated 300cc of blood and autologous cryoprecipitate was prepared in the blood bank. In the three extractions, the fibrin glue was prepared from the autologous cryoprecipitate (Figure 3). The cryoprecipitate was mixed with a protease inhibitor-aprotinin (Trasilol®, Bayer, Germany) 20,000 KIU/ml in a ratio of 3:1, respectively, and transferred to one syringe, while thrombin (Thrombinar®, Armour Pharmaceuticals, USA) dissolved in 40 millimoles of calcium chloride at a concentration of 500 units/ml was transferred to the second syringe. The teeth were anesthetized intraligamentarily. The extractions were performed as atraumatically as possible. Following the extraction, the socket was filled with a thrombin-soaked gauze for two to three minutes, and thereafter the socket was filled with fibrin glue by the double syringe technique and gelfoam was placed in the socket (Figure 3). The soft tissue margins were sutured with a nonresorbable suture and a thin film of fibrin glue was placed over the sutured margins. Antifibrinolytic swish and swallow rinse of 1 gr tranexamic acid was performed by the patient one hour presurgery, followed by 500 mg q.i.d. for seven days.

Postoperative oozing started twelve hours postextraction of tooth #15. The bleeding was stopped by firm pressure of a petroleum-gel soaked gauze. The gauze is covered with a thin layer of vaseline in order to prevent

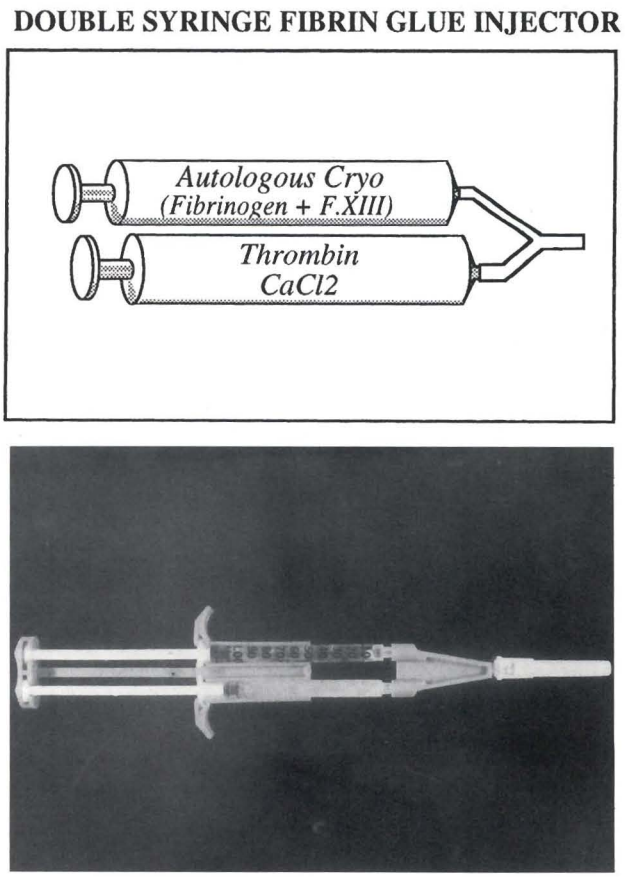

Figure 3. Double syringe fibrin glue injector.

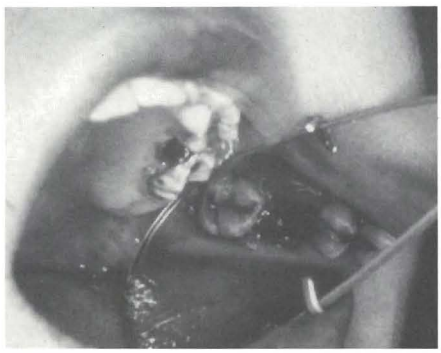

Figure 4. Extraction site of tooth #26, eight days postsurgery.

dislodging of the clot that could be incorporated into the dry gauze fibers.

The sutures were removed in all three sessions, eight to ten days postextraction (Figure 4).

Tooth #46 was not extracted, due to patient's disappearance from our clinic. Recently it was found that the family had moved to the northern part of the country. Attempts will be made to complete the treatment.

DISCUSSION

There are two major strategies to encounter hemorrhagic crisis in patients with Glanzmann's thrombasthenia undergoing dental extraction.

- ☐ Systemic transfusion of platelet concentrate.[9]
- ☐ Local hemostatic measures at the site of extraction:
- ☐ Microfibrillar bovine collagen with an antifibrinolytic inhibitor.[10]
- ☐ Oxidized cellulose with pressure inserted by soft-acrylic splints.[11]

Due to the increased risk of developing antiplatelet alloantibodies after platelet transfusion, it is recommended to use platelet infusions only in major or life-threatening bleedings.

It is our strategy to control the hemostasis at the site of the surgery, therefore, by local measures. The use of fibrin glue derived from the patient's own blood eliminates the risk of blood born viral infections. The use of fibrin glue in patients on anticoagulant therapy, in conjunction with antifibrinolytic rinses, and synthetic collagen or Gelfoam, was found to be successful.[12] Perkin *et al* in 1979 demonstrated a remarkable series of thirty-six extractions in two patients where he controlled the local hemostasis by the use of microfibrillar bovine collagen combined with antifibrinolytic systemic therapy (EACA) without any bleeding complication.[9] Our experience with soft acrylic splints postextraction as demonstrated by Jasmin *et al* in patients who suffer from severe hemophilia A was not successful and we were reluctant to use it in this case.[11] This technique is dependant upon the cooperation of the patient that should not move the splint and by that may dislodge the blood clot. Secondly we are interested in the local contact of the antifibrinolytic swish and swallow rinse of tranexamic acid and the splint may prevent that contact.[13] Resorbable sutures are not recommended in our hemostasis protocol, because they are easy to rupture, bulkier in contact with moving soft tissues, such as tongue and lips, cause, therefore, the unneeded attention of the patient, especially if he is a child. Nonresorbable sutures are placed and removed within seven to ten days.

CONCLUSION

The combination of fibrin glue, antifibrinolytic swish and swallow rinses of tranexamic acid were found to be a safe way to prevent bleeding at the extraction site, in a patient with Glanzmann's thrombasthenia.

REFERENCES

1. Glanzmann, E.: Hereditare hamorrhagische thrombasthenie. Ein beitrag zur plättchen. J Kinderkr, 88:1, 1918.
2. Nurden, A.T.: Glycoprotein defects responsible for abnormal platelet function in inherited platelet disorders in platelet membrane glycoproteins. New York: Plenum, 1985, p 357.
3. Phillips, D.R. and Agin, P.P.: Platelet membrane defects in Glanzmann's Thrombasthenia. J Clin Invest, 60:535-545, September 1977.
4. Weiss, H.J.; Turitto, V.T.; Baumgartner, H.R.: Platelet adhesion and thrombus formation on subendothelium in platelets in glycoproteins IIB-IIIa, Ib, and storage granules. Blood 67:322-330, February 1986.
5. Bennett, J.S. and Sandford, J.S.: Congenital qualitative platelet disorders in *Hematology*. New York: McGraw-Hill, 1990, p 1409.
6. Seligsohn, U. and Rososhansky, S.: A Glanzmann's thrombasthenia cluster among Iraqi Jews in Israel. Thromb Haemost, 52:230-231, December 1989.
7. Reichert, N.; Seligsohn, U.; Ramot, B.: Clinical and genetic aspects of Glanzmann's thrombasthenia in Israel. Thromb Diath Haemorrh, 34:806-820, December 15, 1975.
8. Bennett, J.S.: *Hereditary disorders of platelet function in hematology - basic principles and practice.* New York: Churchill Livingstone, 1991, p 1918.
9. Sugar, A.W.: The management of dental extractions in cases of thrombasthenia complicated by the development of isoantibodies to donor platelets. Oral Surg Oral Med Oral Path, 48:116-119, August 1979.
10. Perkin, R.F.; White, G.C.; Webster, W.P.: Glanzmann's thrombasthenia. Report of two surgical cases using a new microfibrillar collagen preparation and EACA for hemostasis. Oral Surg Oral Med Oral Path, 47:36-39, January 1979.
11. Jasmin, J.R.; Dupont, D; Velin, P.: Multiple dental extractions in a child with Glanzmann's thrombasthenia: Report of case. J Dent Child, 54:208-210, May-June 1987.
12. Martinowitz, U.; Mazar, A.; Taicher, S. *et al*: Dental extraction for patients on oral anticoagulant therapy. Oral Surg Oral Med Oral Path, 70:274-277, September 1990.
13. Sindet-Pedersen, S.; Ramstrom, G.; Bernvil, S. *et al*: Hemostatic effect of tranexamic acid mouthwash in anticoagulant-treated patients undergoing oral surgery. N Engl J Med, 320:840-843, March 30, 1989.

DEMOGRAPHICS

Can your next pediatric patient hear you?

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

Picture the following scene: You stopped your car at a traffic light. A car driven by a teenager pulls alongside of yours. The windows of both cars are closed. You become aware of a deep bass rhythmic throbbing sound emanating from one of the two cars. No, nothing is mechanically wrong with either vehicle. You're listening to the "music" from the next car. In disbelief, you wonder about the decibel level inside the next car and the impact on the hearing of the teenage driver.

Most of the time, few of us consider the consequences of hearing loss. It's just one of the infirmities of old age—is it not? Yes, a large proportion (43.0 percent) of the 20.3 million individuals with hearing trouble are sixty-five years of age or over. The proportion of persons with normal hearing in the older age population is only 9.7 percent. But almost one million youths (968,000) between three and seventeen years of age have hearing difficulties and of these, about 143,000 cannot hear and understand normal speech. In all, a total of 4.8 million people (three years and older) cannot hear and understand normal speech.[1]

From the perspective of practitioners providing services to youngsters, it is important to remember that 4.1 million individuals (20.9 percent of all persons with hearing trouble) report that the onset of hearing difficulties occurred during childhood (Figure 1).[1] (One wonders about the consequences to the hearing of the teenager (and his/her generation) who is waiting at the red light in the next car with the "rhythmic throbbing sound.") Hearing difficulties are one of the most prevalent of chronic conditions and when they affect children there can be major developmental and educational consequences. (Note: the data in this presentation refer only to the civilian noninstitutionalized population of the country. In addition, except where specified, the data do not include persons who report chronic tinnitus [a noise in the ears, as ringing, buzzing, roaring, clicking, etc.] but who do not report other hearing trouble.)

Communication with pediatric dental patients at times can be difficult under the best of circumstances. You would need to consider the added difficulties when hearing impairment complicates the relationship between a young patient, yourself and your staff. The following presentation will provide some dimensions to the extent of this problem in the younger general population cohorts in our communities—and probably in your practice.

SOURCE OF DATA

The information for this presentation was drawn from the extensive series of reports from the National Health Interview Survey (NHIS).[1-3] The NHIS is a continuing nationwide household interview survey. Each week a probability sample of the civilian noninstitutionalized population of the country is interviewed by personnel of the U.S. Bureau of the Census. The interview sample for 1990-91 was composed of 92,237 households con-

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taining 239,663 persons. The total noninterview rate was 4.4 percent.[1]

HEARING TROUBLE IN THE GENERAL POPULATION

The consequences of some chronic illnesses tend to be "fashionable"—not so the effects of others. Consider how much time you (or a member of your family) spent in selecting those "just right" designer eye glass frames which add a sense of professionalism, fashion or maybe just some sort of statement. No doubt more time was spent on frame selection than the actual physical examination by your physician or optometrist.

By contrast, hearing aids (if they are even used) often are hidden in eye glass frames, in the external auditory canal or just about any place that will ensure the "all important secret" of diminished hearing.

Between 1971 and 1991, there was an increase from 13.2 million to 20.3 million persons, three years and over, who reported hearing trouble. The number of individuals with hearing trouble per one thousand population increased from 69 to 86 persons (a 25 percent increase in the reported prevalence). When the data are age adjusted (hearing trouble is associated highly with an increase in age and there has been an aging of the population during the period) there was still a 14 percent increase in the prevalence of hearing trouble.[1] The most frequently reported cause of hearing trouble (by respondents) was "getting older" (28 percent), followed by "...noise from machinery, aircraft, power tools, loud music, appliances, Walkman personal stereos, hair dryers, etc." (23 percent).[1]

Table 1 ☐ Number of children (less than 18 years of age) per 1,000 with hearing impairment: 1982-1992.[2]

Year	Number
1982	19.7
1983	18.8
1984	24.0
1985	19.2
1986	20.1
1987	16.0
1988	17.0
1989	15.6
1990	21.0*
1991	16.1
1992	15.0

Note: 1. The NHIS survey did not report annually hearing impairment as a separate category prior to 1982.
2. These data include persons with tinnitus. In 1992, there were 76,000 individuals with tinnitus.

* Additional questions were used in the 1990 survey. The use of extra questions tends to increase the report of the particular condition.

- ☐ Although the female population is larger and older than the male population, there are far more males with hearing trouble (12 million) than females (8.3 million).
- ☐ White persons are more likely to have hearing trouble than Afro-Americans.
- ☐ Hispanics are less likely to have hearing trouble than non-Hispanics.
- ☐ Persons with hearing problems are proportionately overrepresented in families with an annual income of less than $10,000 and underrepresented in families with incomes of $50,000 and over.
- ☐ Individuals not in the labor force, in service and blue collar occupations and those with under twelve years of education, have higher rates of hearing trouble than their respective counterparts.[1]

CHILDREN WITH HEARING PROBLEMS

Since the early 1980s, there has been a general decreasing trend in the reported prevalence of children with hearing impairments, from approximately 19-20 per

Table 2 ☐ Number and percent of children 3-17 years with all levels of hearing trouble by various demographic characteristics: 1990-1991.[1]

	Percent distribution of children		Number of children with hearing problems
	All children	Hearing problems	(in thousands)
Race			
White	80.5%	86.2%	834
Black	15.6	10.5	102
Other	3.9	3.3	32
Total	100.0%	100.0%	968
Ethnicity			
Hispanic*	13.6	15.1	145
NonHispanic	86.4	85.0	816
Total	100.0%	100.0%	968
Economics			
<$10,000	11.7	16.3	142
$10,000-$24,999	26.9	31.6	276
$25,000-$49,999	37.9	33.2	290
$50,000+	23.6	18.9	165
Total	100.0%	100.0%	968
Regions			
Northeast	18.4	16.1	156
Midwest	24.4	28.0	271
South	34.9	22.7	321
West	22.3	22.7	220
Total	100.0%	100.0%	968
Residence			
Metropolitan			
Central City	29.4	28.2	273
Non Central City	48.0	46.5	450
Nonmetropolitan	22.6	25.3	245
Total	100.0%	100.0%	968

* May be of any race

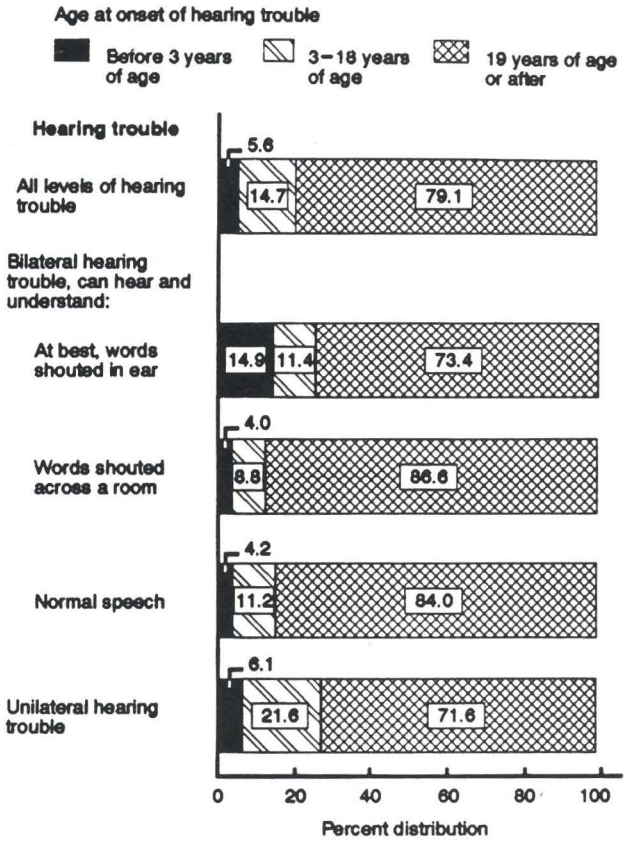


Figure. Average annual percent distribution of persons 3 years of age and over by age at onset of hearing trouble by type of hearing trouble: 1990-1991.[1]

thousand children to approximately 15-16 per thousand children (Table 1).

- ☐ White children are more likely to have hearing trouble than Afro-American children.
- ☐ Contrary to the findings for adults, Hispanic children are more likely to have hearing trouble than non-Hispanic children.
- ☐ Children with hearing problems are proportionately overrepresented in families with annual incomes below $25,000 and underrepresented in families with incomes of $50,000 and over.
- ☐ Children with hearing problems are proportionately underrepresented in the population that resides in metropolitan areas, in the Northeast and especially in the Southern region and overrepresented in the population that resides in the nonmetropolitan and in Midwestern region of the nation (Table 2).
- ☐ Twenty-seven percent of children (5-17 years of age) with hearing troubles (265,000) have some limitations in activities (including 21 percent with school-related limitations), compared to 6.2 percent of children with no hearing trouble.[1]
- ☐ 148,000 children and young adults (14 percent of persons with hearing trouble) use hearing aids.
- ☐ Approximately 22,000° children and young adults (2 percent of persons with hearing trouble) use TDD/TTY systems (a typewriter-like device for the deaf that communicates over telephone lines using text).[4,5]
- ☐ Among the estimated 127,000 youths who do not hear and understand normal speech, 48,000 were reported to be attending special schools or classes. This does not include the approximately 10,000 students between five and seventeen years of age in resident schools for the deaf.[1]

FROM THE PERSPECTIVE OF THE PEDIATRIC DENTIST

Practitioners increasingly are providing care to diverse populations of youngsters. In an effort to help practitioners and their staffs recognize the demographic dimensions and the particular characteristics of the needs of youngsters with special problems, an extended series of presentations in the *Journal of Dentistry for Children* has been offered, including reviews on the developmentally disabled, fetal alcohol syndrome, emotional problems, AIDS, changed family structure, child neglect and abuse, drug addiction and chronic illnesses.[6-12] The almost one million youngsters with hearing problems who will frequent the practices of pediatric and general dentists also will need the additional attention that is necessary for special patients.

While many children with hearing trouble have only slight hearing loss, you and your staff will need to work closely with the parents and guardians in an effort to establish systems for communication as you develop the necessary confidence and working relationship with your young patient. For example, the implementation of OSHA regulations with requirements for masks and any number of other barriers has eliminated the potential system of lip reading that could be used by individuals with diminished hearing. But no one ever said that caring for children would be easy!

As to the teenager at the traffic light, I have found that when the light turns green, if you hesitate for a moment or two, the teenager in the next car with the "deep bass rhythmic throbbing sound" will zoom off into

the distance—permitting you to listen to your Mozart or Vivaldi CD.

REFERENCES

1. U.S. Department of Health and Human Services, Ries, P.W.: Prevalence and characteristics of persons with hearing trouble: United States, 1990-91. Vital and Health Statistics, Series 10, No. 188. DHHS Pub. No. (PHS) 94-1516. Hyattsville, MD: National Center for Health Statistics, 1994.
2. U.S. Department of Health and Human Services. Current Estimates from the National Health Interview Survey: United States, 1982 through 1992. Vital and Health Statistics, Series 10, No. 150; 154; 156; 160; 164; 166; 173; 176; 181; 184; 189. DHHS Pub. No. (PHS) 85-1578; 86-1582; 86-1584; 86-1588; 87-1592; 88-1594; 89-1501; 90-1504; 92-1509; 93-1512; 94-1517. Hyattsville, MD: National Center for Health Statistics, 1985; 1986; 1986; 1986; 1987; 1988; 1989; 1990; 1991; 1992; 1994.
3. U.S. Department of Health and Human Services, Ries, P.W.: Hearing ability of persons by sociodemographic and health characteristics: United States. Vital and Health Statistics, Series 10, No. 140. DHHS Pub. No. (PHS) 82-1568. Hyattsville, MD: National Center for Health Statistics, 1982.
4. U.S. Department of Health and Human Services, LaPlante, M.P., Hendershot, G.E., Moss, A.J.: Assistive technology devices and home accessibility features: prevalence, payment, need, and trends. Advance Data, No. 217. Hyattsville, MD: National Center for Health Statistics, September 16, 1992.
5. U.S. Department of Commerce. Statistical Abstract of the United States: 1993. Washington, DC, Government Printing Office, 1993.
6. Waldman, H.B.: Special pediatric population groups and their use of dental services. J Dent Child, 56:211-215, May-June 1989.
7. Waldman, H.B.: Fetal alcohol syndrome and the realities of our time. J Dent Child, 56:435-437, November-December 1989.
8. Waldman, H.B.: Almost eleven million special children. J Dent Child, 58:237-240, May-June 1991.
9. Waldman, H.B. and Waldman, M.E.: Increasing numbers of pediatric AIDS patients. J Dent Child, 58:400-404, September-October 1991.
10. Waldman, H.B.: Problems your pediatric patients may face. J Dent Child, 60:38-42, January-February 1993.
11. Waldman, H.B.: Is your next pediatric patient an addict? J Dent Child, 60:136-139, March-April 1993.
12. Waldman, H.B.: Almost twenty million chronically ill children. J Dent Child, 61:129-133, March-April 1994.

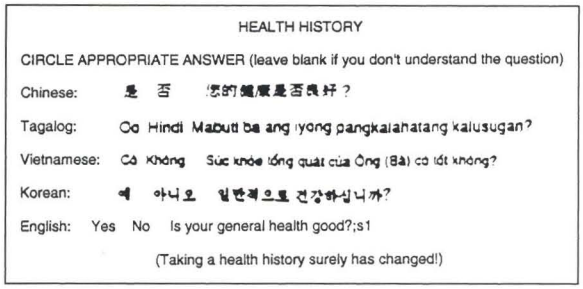
CONSIDERATIONS FOR DETECTING HEARING LOSS IN INFANTS

A successful infant hearing program must detect hearing loss that will interfere with normal development of speech and oral language. Because normal hearing is critical for speech and oral language development as early as the first 6 months of life (Kuhl, Williams, Lacerda, Stephens, and Lindbloom, 1992), it is desirable to identify infants with hearing loss before 3 months of age.

Facilities or agencies that implement infant hearing programs must develop protocols to achieve identification of all infants with hearing loss. To gain access to most infants, the Joint Committee on Infant Hearing recommends the option of evaluating infants before discharge from the newborn nursery. For infants discharged early or delivered at an alternative birthing site, it is desirable to have their hearing assessed before 3 months of age.

Concern for hearing should not stop at birth. Some children may develop delayed-onset hearing loss.

Joint committee on infant hearing, AAP: 1994 position statement.
Pediatrics, 95:152-156, January 1995.



Asian American and Pacific Islander children: They will become an increasing reality in your practice

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

When you were in high school or college, you probably studied French, Spanish, German, Russian or a variety of other European languages. Chinese, Tagalog (the chief native language of the Philippine Islands), Vietnamese, Korean and any number of other Asian languages either were not one of the school offerings, or you saw limited utility of spending time learning such an "alien" language (unless you planned a vacation in some far off "exotic" land). The reality is that those far off "exotic" lands, or at least their populations have come to the United States in increasing numbers.

"Unusually large waves of immigrants from Southeastern Asia and South Asia in the 1980s transformed the Asian community in the United States, propelling an explosion of diversity among Asian Americans, the nation's fastest growing minority."[2] The 1990 U.S. Census indicates that as a result of a growing number of Asian and other immigrants, together with continuing increased numbers of births in different minority population groups, there are changes in the racial mixture of the nation that is more dramatic in the past decade than in any other time in the 20th century. In 1990, "...nearly one in every four Americans (were of) African, Asian, Hispanic or American Indian ancestry."[3]

The predominant racial group in this country still traces its origin to Europe and will continue to be in the majority for some time into the future. Nevertheless, in some states (e.g. California and New Mexico) the percent of the population that is white and not of Hispanic origin descent has decreased so dramatically, that the once dominant group could become a minority by the year 2000.[3]

As a result of the relatively small number of individuals, who in the past were classified as being in "other groups" (i.e. not "white" or "black") limited emphasis was placed in the dental literature on the developing diversity and numeric significance of the Asian American and Pacific Islander populations in our communities.

The following presentation will review results from the 1990 Census of the Population (detailed sections of which have become available in the mid 1990s) in an effort to encourage a greater awareness of the increasing realities of Asian American and Pacific Islander children in dental practices.

Note: the classification of race and ethnicity is increasingly difficult. The number of interracial unions increase from 0.4 percent of all marriages in 1960 to 2.2 percent in 1992.[4] As a result, the federal government is rethinking its current system of separating the various population groups. Consider the following example. "Teja Arboleda's maternal grandparents are European. His father's mother is African-American. His father's father is the product of a marriage between a Filipino and a Chinese.

Although listed as white on his birth certificate, Mr. Arboleda refused to state his ethnic or racial identity on the 1990 census form. And so an employee of the Census Bureau noted his name, olive-toned skin and dark hair, and marked him down as Hispanic."[4]

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Table 1 ☐ US population by race: 1990 Census of the Population.[5]

	Number
	(in millions)
White	199.7
Black	29.9
American Indian, Eskimo or Aleut	1.9
Asian American	6.9
Chinese	1.6
Filipino	1.4
Japanese	.8
Asian Indian	.8
Korean	.8
Vietnamese	.6
Cambodian	.1
Loatian	.1
Hmong	<.1
Thai	<.1
Other Asian	.3
Bangladeshe, Burmese, Indonesian, Malayan, Okinawan, Pakistani, Sri Lankan, Others	
Pacific Islander	.4
Hawaiian	.2
Samoan	<.1
Guamanian	<.1
Other Pacific Islanders	<.1
Tongan, Tahitian, North Mariana Islander, Palauan, Fijan, Others	
Other races	9.8
Total	248.7

Table 2 ☐ States with the highest Asian or Pacific Islander concentration: 1990.[3]

State	1990 population	Change from 1980	Percent of state population
	(000s)		
Hawaii	685.2	17.5%	61.8%
California	2,845.7	127.0	9.6
Washington	210.9	105.7	4.3
New York	693.7	123.4	3.9
Alaska	19.7	144.9	3.6

Table 3 ☐ States with the largest increase in Asian American and Pacific Islander population: 1980–1990.[3]

State	1990 population	Change from 1980	Percent of state population
	(000s)		
Rhode Island	18.3	245.6%	1.8%
New Hampshire	9.3	219.0	0.8
Georgia	75.8	208.6	1.2
Wisconsin	53.6	195.0	1.1
Minnesota	77.9	103.5	1.8

OVERALL NUMBERS

Between 1980 and 1990, the number of Asian Americans and Pacific Islanders in increased by 95 percent (from 3.7 million to almost 7.3 million).[2] Half of the 400,000 Pacific Islanders were Hawaiians† (Table 1). Political upheavals and economic hardships in the various Asian nations have brought more than two million immigrants during the 1980s. As a result, the fastest growing Asian subgroups under the Asian American rubric are Vietnamese (135 percent growth in the 1980s), Asian Indians (126 percent increase), and Koreans (125 percent increase).

While the Chinese remain the dominant Asian American group in this country, there have been significant changes in the last decades.

- ☐ In the past twenty-five years the number of Chinese immigrants has equalled or surpassed the number that had come in the first eighty years of immigration.
- ☐ Japanese, who thirty years ago represented 52 percent of the Asian American population, now represent 11.7 percent.
- ☐ Filipinos are no longer one of the fastest growing Asian minorities.[2]
- ☐ The Hmong (agricultural people from the mountains of Laos whom the Central Intelligence Agency recruited to help fight the North Vietnamese) have moved to Eau Claire, Wisconsin and Fresno, California.
- ☐ There are more Asian Americans in the State of New York than in the State of Hawaii (694,000 vs. 685,000) (Table 2).
- ☐ During the past decade, the States of Rhode Island and New Hampshire had the largest percent increase in Asian Americans and Pacific Islanders (Table 3).

NUMBER OF CHILDREN

There are almost two million Asian American children (30.7 percent of whom were foreign born) and 127,000 Pacific Islander children (5 percent of whom were foreign born) recorded by the 1990 Census (Table 4).

- ☐ The number of Asian American children ranged from 387,000 Filipino and 385,000 Chinese to less than 6,000 Indonesians (Table 5).
- ☐ The number of Pacific Islander children ranged from 72,000 Hawaiians to almost 4,000 Polynesians (Table 6).

Table 4 ☐ Number of Asian American and Pacific Islander children by age: 1990 Census of Population.[6]

	Asian American	Pacific Islander
	Number (000s)	
< 5 yrs	537.8	36.6
5-9 yrs	550.7	37.0
10-14 yrs	516.8	34.7
15-17 yrs	319.9	18.8
Total	1,925.2	127.2
Percent foreign born	30.7%	5.0%

Table 5 ☐ Asian American children less than 18 years of age: 1990 Census of Population.[6]

	Number (000s)		
Filipino	387.1	Cambodian	69.5
Chinese	384.8	Laotian	66.3
Korean	245.5	Hmong	57.3
Asian Indian	232.9	Pakistani	25.6
Vietnamese	199.3	Thai	22.7
Japanese	158.1	Indonesian	5.8

Table 6 ☐ Pacific Islander children less than 18 years of age: 1990 Census of Population.[6]

	Number (000s)		
Hawaiian	71.8	Guamanian	15.9
Samoan	24.7	Tongan	7.6
Micronesian	18.4	Polynesian	3.8

Table 7 ☐ Children 5-17 years of age enrolled in school but who do not speak English "very well": 1990 Census of Population.[6]

	Percent
Total population except Asian Americans and Pacific Islanders	4.9%
Asian American	28.7
Hmong	78.8
Cambodian	68.2
Loatian	59.1
Vietnamese	48.5
Chinese	33.9
Indonesian	33.3
Korean	23.9
Pakistani	20.0
Japanese	19.2
Thai	18.7
Filipino	11.7
Asian Indian	10.0
Pacific Islander	7.2
Melanesian	33.1
Tongan	25.0
Samoan	13.3
Guamanian	10.0
Micronesian	9.1
Polynesian	5.8
Hawaiian	2.1

Table 8 ☐ Children less than 18 years of age living with two parents: 1990 Census of Population: 1990.[6]

	Percent
Total population except Asian Americans and Pacific Islanders	72.7%
Asian American	84.6
Pakistani	91.9
Asian Indian	91.8
Korean	89.0
Chinese	87.6
Japanese	86.1
Hmong	86.2
Thai	84.6
Indonesian	84.6
Loatian	82.6
Filipino	80.9
Vietnamese	76.6
Cambodian	71.0
Pacific Islander	68.0
Melanesian	83.2
Tongan	79.8
Micronesian	73.7
Guamanian	72.1
Samoan	69.9
Polynesian	66.6
Hawaiian	63.9

NOT A HOMOGENEOUS POPULATION

It is all too common for some to assume that "all Asian Americans do well in school," "that they are willing to work long hours" and any number of other stereotypes.[7] The reality is that the Asian American population is as diverse as any of the other groups in our nation.

- ☐ The Hmong are in many cases illiterate in their own language and "...are ill equipped to find a place in the American economy."[8]
- ☐ Almost 29 percent of Asian American children enrolled in school do not speak English "very well" (ranging from 79 percent of Hmong children to 10 percent of Asian Indian children).
- ☐ Seven percent of Pacific Island children enrolled in school do not speak English "very well" (ranging from 33 percent of Melanesian children to two percent of Hawaiian children) (Table 7).
- ☐ Filipino and Asian Indians tend to be much more in the mainstream economy than other immigrants.
- ☐ Asian Indians and Koreans have a large percentage of people who are reasonably well educated; have friends, connections and relatives and have a perception of the opportunities available in this country.

† In an attempt to eliminate repetition, except for the term "Asian American," "American" is not repeated for all subpopulations.

- ☐ The most widely spoken languages of the Asian American immigrant population are Chinese, Filipino, Vietnamese, Japanese, Korean, Khmer (Cambodian) and Laotian.

There are, however, eighty-eight dialects in China, eighty-seven dialects in the Philipines, and three main Vietnam.[8]‡

- ☐ Approximately 73 percent of the total population of children less than eighteen years of age (not including Asian Americans and Pacific Islanders) live with two parents. Almost 85 percent of Asian American children live with two parents (ranging from 92 percent of Pakistani and Asian Indian children to 71 percent of Cambodian children).
- ☐ By contrast, 68 percent of Pacific Islander children live with two parents (ranging from 83 percent of Melanesian children to 64 percent of Hawaiian children) (Table 8).
- ☐ In 1989, 13 percent of the total population (not including Asian Americans and Pacific Islanders) lived below the poverty level. Similarly 14 percent of Asian Americans lived below the poverty level (but ranging from 63 percent of the Hmong to 6 percent of Filipinos).
- ☐ A somewhat higher percent of Pacific Islanders lived in poverty (17 percent) (ranging from 40 percent of Guamanians to 9.5 percent of Melanesians) (Table 9).
- ☐ In one study, South East Asian refugee high school youths (all of whom had been in the United States for five years or less) maintained strong ties to their native foods and traditional meal patterns. Fruits, meats and soft drinks remain highly preferred items. While milk is well-liked, cheese remains a strongly disliked food item. Fruits and vegetables are consumed quite often. American foods, such as candy bars, cake and potato chips are not consumed frequently.[10]
- ☐ Social science research testing and measurement protocols that were developed using Judaeo-Christian value systems may require reassessment when applied to Asian American and Pacific Islander families.

Table 9 ☐ Persons below the poverty level: 1989 Census of Population.[6]

	Percent
Total population except Asian Americans and Pacific Islanders	13.1%
Asian American	14.0
Hmong	63.6
Cambodian	42.6
Loatian	34.7
Vietnamese	25.7
Indonesian	25.2
Pakistani	15.1
Chinese	14.0
Korean	13.7
Thai	12.5
Asian Indian	9.7
Japanese	7.0
Filipino	6.4
Pacific Islander	17.1
Guamanian	33.9
Samoan	25.8
Tongan	23.1
Micronesian	17.6
Polynesian	17.2
Hawaiian	14.3
Melanesian	9.5

GENERAL HEALTH CONDITIONS

"Because of their small number until the last two decades, many health care workers have had little exposure to this minority, (Asians) their culture, and health problems."[12]

Infectious diseases that are uncommon in the general U.S. population may be quite common in the Asian American population; including a chronic carrier state for hepatitis B and (particularly in refugees and recent immigrants) parasitism and tuberculosis.

- ☐ Folk remedies used by both Chinese and South East Asian refugees may have toxic levels of lead and arsenic.
- ☐ Misdiagnosis of child abuse may occur, resulting from lack of awareness of the use of some folk remedies used by some Asian Americans, (particularly those from South East Asia) including dermabrasive procedures and moxibustion—a form of treatment in which cones of sunflower pith or down from the leaves of the plant "Artesisia Box" are stuck in the skin and ignited. The heat produced by the smoldering cones act as a counterirritant and is reputed to cure a variety of disorders.[12]
- ☐ There are few nationally published data on the oral health status of the general population of Asian Americans and Pacific Islanders and even fewer data on the status of the various subgroups. Some local studies of children of recent refugees and immigrants who are enrolled in Head Start programs do provide indications of greater unmet restorative treatment needs. But because of economic and social reasons, new nonrefugee immigrants tend to

‡For an extended review of Asian American and Pacific Islander cultural differences, see a previous publication.[9]

seek less treatment than the general population. On the other hand, "refugee status" confers welfare benefits (such as Medicaid for the first eighteen months, as well as English as a second language programs and job training for parents). As a result, refugee children have a higher utilization of dental services.[13]

AN EVOLVING PROFESSION TO SERVE A CHANGING POPULATION

In 1978, Asian Americans represented 4.2 percent of the entering classes in dental schools in the United States. By 1993, one out of every five (20.8 percent) first-year dental students was an Asian American; 67.2 percent were reported as white, 6.1 percent as African-American, 5.5 percent as Hispanic, and 0.4 percent as American Indian.[14,15] Increasingly the profession will reflect the diversity in our general population—but that will take time. For the present, we will need to increase our awareness of the bewildering diverse population of youngsters (and their parents) who will come to dental practices with a seemingly infinite variety of perceptions and experiences that are far different from those of current practitioners. Many of these new children will be Asian Americans and Pacific Islanders. Are you and your staff prepared for these new children in our communities?

REFERENCES

1. Jacobsen, P.L. and Fredekind, R.: A multiple language health history for dental practice. Cal Dent Assoc J, 21:25-36, May 1993.
2. Barringer, F.: Immigration brings new diversity to Asian population in the U.S. New York Times, June 12, 1991, p A1, D25.
3. Barringer, F.: Census shows profound change in racial makeup of the nation. New York Times, March 11, 1991, p A1, B8.
4. Holmes, S.A.: Federal government is rethinking its system of racial classification. New York Times, July 8, 1994, p A18.
5. U.S. Department of Commerce, Bureau of the Census. 1990 Census of Population: General Population Characteristics, United States. Pub. No. 1990 CP-1-1. Washington, DC: Government Printing Office, 1992.
6. U.S. Department of Commerce, Bureau of the Census. 1990 Census of Population: Asian and Pacific Islanders in the United States. Pub. No. 1990 CP-3-5. Washington, DC: Government Printing Office, 1993.
7. Slaughter-Defoe, D.T. and Nakagawa, K.: Toward cultural/ecological perspectives on schooling and achievement in African and Asian-American children. Child Development, 61:363-383, April 1990.
8. Cheng, L.L.: Cross-cultural and linguistic considerations in working with Asian populations. American Speech-Language Health Association, 29:33-37, June 1987.
9. Waldman, H.B.: Asian Americans: an increasing reality in the population and the dental profession. Ohio Dent J, 66:38-43, Spring/Summer 1992. (Note: some of the differences between the population groups reported in the current report were presented in this earlier publication.)
10. Story, M. and Marris, L.J.: Food preferences, beliefs and practices of Southeast Asian refugee adolescents. J School Health, 58:273-276, September 1988.
11. Morris, T.M.: Culturally sensitive family assessment: an evaluation of the family assessment device used with Hawaiian-American and Japanese-American families. Family Process, 29:105-116, March 1990.
12. Lin-Fun, J.S.: Population characteristics and health care needs of Asian Pacific Americans. Public Health Reports, 103:18-27, January-February 1988.
13. Pollick, H.G.; Rice, A.J.; Echenberg, D.: Dental health of recent immigrant children in the Newcomer School, San Francisco. Amer J Public Health, 77:731-733, June 1987.
14. Minority Report: supplement #3. 1978/79 Annual Report on Dental Education. Chicago: American Dental Association, 1979.
15. Applicant Analysis: 1993 entering class. Washington, D.C.: American Association of Dental Schools, 1994.

CASE REPORTS

Radiographic considerations for supernumerary tooth extraction: Report of case

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Supernumerary teeth and dental fusion are rare anomalies of the maxillofacial complex. Although their recognition is usually straightforward for dentists, their treatment is sometimes not as clear. This paper discusses a rare combination of these two dental anomalies and suggests considerations for an appropriate radiographic protocol for supernumerary tooth extraction.

Anomalies of the permanent and primary dentitions can range from changes in tooth morphology to variations in tooth number. Supernumerary teeth represent a dental anomaly that consists of one or more extra teeth. There are two morphological types of supernumerary teeth: supplemental and rudimentary. Supplemental are supernumerary teeth of normal shape and size. These usually resemble the teeth of the group they are associated with (e.g., incisors, molars). Rudimentary are often smaller and tuberculate in shape.[1]

The most common supernumerary teeth are mesiodens (maxillary midline area), representing 90-98 percent of all supernumerary teeth.[1-4] The next most common sites for supernumerary teeth are distal to the maxillary third molar (the "fourth molar"), the mandibular premolars, and the maxillary lateral incisors.[2] There are several reports of supernumerary teeth in the mandibular anterior region, usually in the area of the canines and incisors.[5] Krayer was the first to report the presence of a supernumerary in the mandibular lateral incisor region.[6]

Supernumerary teeth occur the majority of the time in the permanent dentition and are usually found as a single tooth (76-78 percent of the cases).[1] Pairs (12-23 percent) and groups of three or more (1 percent) have also been described.[7-9] Multiple supernumerary teeth are characteristic of abnormalities such as cleidocranial dysplasia, Gardner's syndrome, and cleft lip and palate.[2] There is a two to one predilection of males over females for supernumerary teeth.[9-11]

The literature describes different etiologies for supernumerary teeth. The dichotomy theory associates these teeth with an extra tooth bud. A more widely supported theory describes hyperactivity of the dental lamina resulting in its splitting.[1,2,12,13]

There are a variety of complications due to unerupted supernumerary teeth, including:

- ☐ Enlarged follicular sacs.
- ☐ Cystic formations.
- ☐ Eruption of the teeth into the nasal cavity.
- ☐ Interference with the eruption of adjacent teeth.
- ☐ Displacement of teeth.
- ☐ Formation of diastema.
- ☐ Root resorption.
- ☐ Malformation.
- ☐ Loss of vitality of the teeth adjacent to the supernumerary.[1,14]

When any of these complications are anticipated, im-

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mediate removal of the supernumerary is indicated.

There are different opinions related to the optimal time for surgical intervention of supernumerary teeth. Some authors recommend immediate removal, when any of these complications are diagnosed.[1,8,10] They suggest that the removal of these teeth will induce spontaneous eruption of the permanent tooth and prevent anterior space loss and midline shift; thereby avoiding extensive orthodontic and/or surgical treatment. Other authors favor postponement of surgical removal until root development of the neighboring teeth is complete. This delay would help prevent loss of vitality or malformation of the adjacent teeth.[1]

A study by Hogstrom and Anderson demonstrated the complications related to surgical removal of supernumeraries.[15] They concluded that the prognosis of the adjacent teeth was not affected by whether the supernumerary teeth were surgically removed upon diagnosis or surgery was postponed until root development of adjacent incisors was completed. The median time between removal of a supernumerary tooth and the clinical appearance of the affected unerupted tooth is thirty months, when not surgically exposed, and sixteen months with exposure.[16]

Fusion is a growth and developmental abnormality. Fused teeth often have the clinical appearance of a bifid crown and arise from the union of two separate tooth germs.[17] Sometimes fusion occurs between two normal teeth; other times it involves a supernumerary and a normal tooth.

The incidence of fused teeth is 0.5 to 1.0 percent in the primary dentition and 0.1 percent in the permanent, with an equal distribution in females and in males.[13] The most common site for this anomalous condition is the incisor and canine region, and it can occur unilaterally or bilaterally with a predilection for the mandible.[17,18]

Fused teeth are usually asymptomatic; they can cause clinical problems, however, such as:

- ☐ Crowding and impaction of a tooth when a supernumerary is fused with a normal permanent tooth.
- ☐ Diastemas when the fusion includes two normal anterior teeth.
- ☐ Various other esthetic problems.
- ☐ Periodontal problems if labial and lingual grooves continue into the root surface.[17]

The treatment of choice for asymptomatic fusion in the primary dentition is observation until exfoliation. In the permanent dentition, the treatment and retention of the fused tooth is indicated, when a good crown-root ratio exists for the retained portion of the tooth. Root canal therapy can be successfully completed, if necessary, and reshaping will provide satisfactory esthetics and function.[19]

For both supernumerary and fused teeth the radiographic evaluation is important to assess the location and nature of these anomalies. When located in the anterior region a periapical or occlusal radiograph is preferred to a panoramic film. For supernumerary teeth, taking two films of the same area at different angles assists in locating the tooth position. The Clark's and parallax techniques have been described for this procedure.[20,21]

Postoperative radiographs are often not obtained following surgical procedures. Concerns for total patient radiation and actual clinical benefits have limited such use. The relative risk of untoward results from certain procedures, however, may suggest specific exceptions.

CASE REPORT

A seven-year-old African-American female presented for routine dental care at The Children's Hospital of Alabama Dental Clinic. The mother's chief complaint at the time of the examination was the noneruption of a mandibular anterior permanent tooth and a retained primary tooth. The medical history was noncontributory to dental treatment. The oral examination revealed that teeth #25 and #26 were clinically absent and tooth Q was retained. Radiographs were recommended to evaluate the position of #25 and #26. An occlusal radiograph revealed they were fused and impacted, due to presence of a rudimentary supernumerary tooth located incisally. Because the patient was seven years old, the decision was made to observe the fused teeth in anticipation of delayed eruption. After a year of observation with occlusal radiographs taken every four months, no improvement of the condition was seen (Figure 1). The extraction of tooth Q and the supernumerary was recommended because of continued root formation of fused #25/#26.

Using a local anesthetic, tooth Q was extracted without complication. A mucoperiosteal flap was incised to improve access to the supernumerary. Approximately 2mm of alveolar bone were removed and the supernumerary tooth was located buccal to the extraction site and removed without complication (Figure 2). Silk sutures were placed. The patient tolerated the procedure without difficulty. Postoperative instructions for care were given to mother as well as a follow-up appointment for two weeks.

At the two-week appointment, the patient presented with no complaint and the tissue was healing in satisfactory manner. An occlusal radiograph was taken, and it revealed the presence of a supernumerary tooth at the

Figure 1. Preoperative radiograph revealing fusion of teeth #25 and #26. A supernumerary tooth is also seen. (Outlines of two supernumerary teeth can be observed)

Figure 3. Radiograph taken two weeks after the extraction of supernumerary tooth, revealing a second supernumerary tooth.

Figure 2. Clinical view of supernumerary extraction.

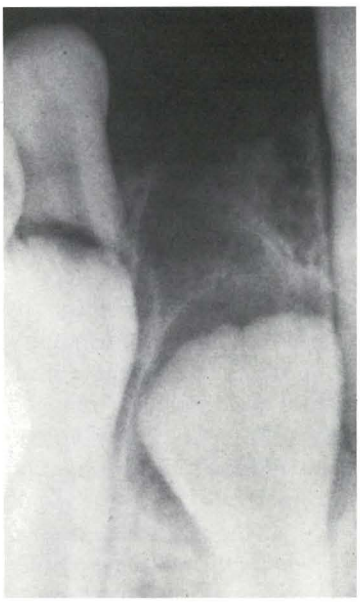

Figure 4. Postoperative radiograph taken after extraction of second supernumerary tooth.

same position of the previous one (Figure 3). The decision to remove the second supernumerary was made. The tissue was re-opened using a local anesthetic.

After removal of approximately 1mm of bone, the second supernumerary was located lingual to the healing extraction site and removed without complication. An occlusal radiograph was negative for the presence of any additional supernumerary teeth (Figure 4). Silk sutures were placed. The patient returned for follow-up two weeks postoperatively, and the bone was healing satisfactorily.

We explained to the mother that there was still the possibility that the fused tooth could remain impacted and orthodontic/surgical treatment might be required. The patient will be maintained on routine dental care at our clinic, with periodic radiographs to evaluate the position of the fused tooth. If after a year no progress is observed, the patient will be referred to the Orthodontic Clinic for evaluation and treatment.

DISCUSSION

This case is an unusual one in several respects. First, supernumeraries are more common in males than females.[9-11] In addition supernumeraries are rare in the mandibular, lateral incisor region. In fact this is only the second report of a supernumerary tooth in this area.[6] Paired supernumeraries are also more unusual (12-23 percent of all cases).[7-9] The buccal and lingual positions of the supernumeraries are also highly unusual. Fusion in the permanent dentition has an incidence of 0.1 percent and 0.5 percent to 1.0 percent in the primary dentition.[13]

The determination that the union of teeth #25 and #26 was fusion and not gemination was based on the *two-tooth rule* explained by Mader.[17] He stated that if a fused tooth is counted as two and there is a normal number of teeth for this region, the case probably represents an example of fusion. If there is an extra tooth present in the region, then the case may represent an example

of gemination or a case of fusion between a normal and a supernumerary tooth.

A case such as this one reinforces the importance of a thorough radiographic evaluation for all patients for whom extraction of supernumerary teeth is planned. In the clearer perspective usually available in hindsight, two radiographs taken from different angulations would probably have revealed the second supernumerary tooth in this case. It is possible that the small size of one of the supernumeraries, however, could have still been sufficiently overlapped to prevent detection. The unusual buccal and lingual positions may not have necessarily been evident either. Upon careful reevaluation of the initial radiograph (Figure 1), two definite outlines can be observed. If two supernumerary teeth had been suspected, a postoperative radiograph after extraction of the first one would have been indicated.

The literature does not discuss the importance of postoperative radiographs in cases of supernumerary tooth extraction. *Stafne 's Oral Radiographic Diagnosis* does express the necessity for postoperative radiographic examination to observe the progress of repair and healing following surgical removal of cysts and tumors.[22] Postoperative radiographs have become a more common practice following the extraction of teeth. By this method the dentist will have on record that no tooth fragments or foreign bodies were left, that there was no alveolar fracture or injury to the adjacent teeth; and that no other conditions were present that might prevent or hamper healing. Surgical procedures for dental anomalies such as supernumerary teeth may also need special consideration regarding postoperative radiographs.

CONCLUSIONS

This paper reports an unusual case in number and location of supernumerary teeth and fused permanent incisors. More important in this case, however, is the implied radiographic protocol for supernumerary teeth.

Before extracting a supernumerary tooth, two preoperative radiographs should be taken at different angulations, in order to reveal the number and location of such anomaly. The findings in this case report also suggest, however, the need for a routine postoperative radiograph immediately following extraction of a supernumerary tooth. Although rare, the presence of more than one supernumerary tooth (or tooth fragment or other odontogenic anomaly) may not always be clearly evident on the preoperative radiograph. An immediate postoperative radiograph would reveal any incomplete extraction and eliminate the need for a patient to return for an additional surgical procedure, as unfortunately occurred in this case.

REFERENCES

1. Primosch, R.E.: Anterior supernumerary teeth-assessment and surgical intervention in children. Pediatr Dent, 3:204-215, June 1981.
2. Shaefer, W.G.; Hine, M.K.; Levy, B.M.: *A Textbook of Oral Pathology*. Fourth Edition. Philadelphia: W.B. Saunders Co., 1983, pp 47-50.
3. Luten, J.R.: The prevalence of supernumerary teeth in primary and mixed dentition. J Dent Child, 34:346-353, September 1967.
4. Tay, F.; Pang, A.; Yuen, S.: Unerupted maxillary anterior supernumerary teeth: report of 204 cases. J Dent Child, 51:289-294, July-August 1984.
5. Stafne, E.C.: Supernumerary teeth. Dent Cosmos, 74:653-659, 1932.
6. Krayer, J. W.: A supernumerary tooth located at the facial of a mandibular lateral incisor-a case report. J Periodontol, 60:410-412, July 1989.
7. Rosenzweig, K.A. and Garbarski, O.: Numerical aberrations in the permanent teeth of grade school children in Jerusalem. Am J Phys Anthropol, 23:277-284, September 1965.
8. Grahnen, H. and Lindahl, B.: Supernumerary teeth in the permanent dentition: A frequency study. Odont Revy, 12:290-294, 1961.
9. Parry, R.R. and Iyer, V.S.: supernumerary teeth amongst orthodontic patients in India. Brit Dent J, 11:257-258, October 1961.
10. Bergstrom, K.: An orthopantomographic study of hypodontia, supernumeraries and other anomalies in school children between the ages of 8-9 years: An epidemiologic study. Swed Dent J, 1:145-157, January-February 1977.
11. Buenviaje, T.M. and Rapp, R.: Dental anomalies in children: A clinical and radiographic survey. J Dent Child, 2:569-574, January-February 1983.
12. Sedano,H.O. and Gorlin, R.J.: Familial occurrence of mesiodens. Opt Acta, 27:360-362, March 1969.
13. Knychalska-Karwan, Z.; Pawlicki, R.; Jakob-Dolezal, K.: The mesiodens teeth under an electron scanning microscope and X-ray microanalyzer. J Int Assoc Dent Child, 15:7-13, June 1984.
14. Bodin, I.; Tulin, P.; Thomsson, M.: Hyperdontia llI. supernumerary anterior teeth. Dentomaxillofac Radiol, 10:35-42, February 1981.
15. Hogstrom, A. and Andersson, L.: Complications related to surgical removal of anterior supernumerary teeth in children. J Dent Child, 54:341-343, September-October 1987.
16. Mitchell, L. and Bennett, T.G.: Supernumerary teeth causing delayed eruption - a retrospective study. Brit J Orthodont, 19:41-46, February 1992.
17. Mader, C.L.: Fusion of teeth. J Am Dent Assoc, 98:62-64, January 1979.
18. Ravn, J.J.: Aplasia, supernumerary teeth and fused teeth in the primary dentition. An epidemiologic study. Scand J Dent Res, 79: 1-6, 1971.
19. Clem, W.H. and Natkin, E.: Treatment of fused tooth: Report of a case. Oral Surg, 21:365-370, March 1966.
20. von Arx, T.: Anterior maxillary supernumerary teeth: A clinical and radiographic study. Austr Dent J, 37:189-195, June 1992.
21. Goaz, P.W. and White, S.C.: *Oral Radiology, Principles and Interpretation*. Second Edition. St. Louis: The C.V. Mosby Co., 1987, pp 118-120.
22. Gibilisco, J.A.: *Stafne's Oral Radiographic Diagnosis*. Fifth Edition. Philadelphia: W.B. Saunders Co., 1985, pp 366-370.

Monostotic fibrous dysplasia in an eight year old male: Report of case

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Fibrous dysplasia is one of the most perplexing diseases of osseous tissue. It is a lesion of unknown etiology, uncertain pathogenesis and diverse histopathology. The condition is divided into polyostotic and monostotic forms, depending on the number of bones involved.[1]

In 1937, Albright and associates described a syndrome characterized by osteitis fibrosa disseminata, areas of skin pigmentation and, in females, endocrine dysfunction and precocious puberty. In the polyostotic form of fibrous dysplasia multiple bones exhibit evidence of disorder, in comparison to the monostotic condition involving only one bone.[2]

The condition appears in the first decade of life. Etiology of the condition is obscure. Various theories have been proposed. There is clinical evidence that indicates that local infection or trauma may result in the disease. Some investigators insist that the monostotic form is a peculiar reparative reaction on the part of bone to any one of a variety of injuries.[1] Alternatively, focal bone expression of a complicated endocrine disturbance has been suggested as a possible etiology.[2]

The primary differential diagnoses for fibrous dysplasia of the jaw are, ossifying fibroma, Paget's disease of bone and chronic osteomyelitis which may mimic the radiographic appearance of fibrous dysplasia.[2] Fibrous dysplasia frequently stabilizes or slows considerably after the onset of puberty. The incidence of malignant transformation is very rare (less than 1 percent).

Fibrous dysplasia should be considered in the differential diagnosis of all bony lesions in the head and neck region. Since the condition is frequently seen in the first decade of life, it is possible that a pediatric dentist is presented with or consulted to evaluate the condition. It is important, therefore, to have a basic understanding of the condition.

LITERATURE REVIEW

The syndrome described by Albright and associates was subsequently accepted as an entity by Lichtenstein and Jaffe who coined the term *Fibrous Dysplasia of bone*. In addition they recognized that the condition may be monostotic.[3]

Monostotic fibrous dysplasia generally exhibits an equal gender distribution, although, according to some authors, it is more common in females, and usually has its onset during the first decade of life.[2]

Clinically craniofacial monostotic fibrous dysplasia presents with a slowly enlarging, painless, nonmobile, nontender, nonfluctuant and hard swelling. The lesion may involve any facial bone but is more commonly seen in the maxilla.[4] Pain and pathological fractures are rarely a complication in craniofacial fibrous dysplasia.[6] Symptoms can include nasal obstruction, proptosis or anosmia, depending on the site of bony involvement.[5] Neurological symptoms resulting from the involvement of foramina

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Figure 1. The arrow points to a firm, diffuse swelling.

Figure 2. The teeth on the affected side were buried in a bony mass, only occlusal surfaces showed.

Figure 3. A panograph was taken to determine the extent of the lesion.

have been reported but are not common. Dental findings include displacement of teeth, interference with normal eruption and/or missing teeth at the site of the lesion. Mobility of erupted teeth is not a feature of fibrous dysplasia.[2]

Radiographically monostotic fibrous dysplasia appears like a diffuse mass with ill-defined margins and a thin egg shell cortex. Radiographic appearance varies greatly from irregular unilocular or multilocular radiolucencies to mottled radiolucent, radiopaque or ground glass appearance of the involved bone.[1,2]

Histologically the lesion is essentially fibrous, made up of proliferating fibroblasts in a compact stroma of interlacing collagen fibers. Irregular trabeculae of bone are scattered throughout the lesion with no definite pattern of arrangement. Characteristically, some of these trabeculae are C-shaped and resemble Chinese characters. The trabeculae are usually coarse woven bone but may be lamellar, although not as well organized as normal lamellar bone. Bone formation by stellate osteoblasts can be observed. Osteoclastic activity may be seen where the calcification of osteoid extends to the surface of the trabeculae.[4] The margins of this metaplastic bone blends imperceptively with the surrounding stroma, giving the appearance of irregular contours, often described as feathering or streaming into the stroma.[6] Malignant transformation is rare (less than 1 percent) and is more common in polyostotic than in monostotic forms.

Treatment of craniofacial monostotic fibrous dysplasia is usually limited to surgical recontouring of bone. Occasionally, in the maxilla, the lesion may show rapid aggressive growth with resultant grotesque facial deformity. In these instances partial resection of the maxilla may be necessary to preserve orbital contents and the globe. Treatment with radiotherapy is contraindicated as malignant transformation is a possibility. The frequency of postradiation sarcoma is high after radiotherapy.[8]

CASE REPORT

An eight-year-old male of Hispanic origin was referred to the Department of Pediatric Dentistry at Boston University by his family dentist for the evaluation of a right-sided, mid-face swelling.

Patient's medical history was noncontributory. Extraoral examination revealed a mid-face swelling involving the right maxillary region below the infraorbital margin. Intraoral examination identified a firm, diffuse swelling involving the upper right quadrant from the canine region to the retromolar area (Figure 1). Swelling was nontender, nonfluctuant, nonmobile and involved both the labial and palatal alveolar surfaces. Teeth on the affected side

were buried in the bony mass, showing only their occlusal surfaces (Figure 2).

A panograph (Figure 3) was taken to determine the extent of the lesion. Radiographic analysis showed a typical ground glass appearance of the bone in the area of the lesion, with missing first and second premolars. The first permanent molar on the affected side was embedded in the lesion. The upper right first and second primary molars were grossly carious. The patient was referred to the Department of Oral and Maxillofacial Surgery for further evaluation and treatment.

The patient was diagnosed as having monostotic fibrous dysplasia, after clinical, radiographic and histologic examinations. Treatment was provided using general anesthesia and included osseous recontouring of the bony lesion and surgical exposure of the upper right first molar to aid its eruption. Grossly carious upper right primary molars were also extracted.

REFERENCES

1. Shafer, W.G.; Hine, M.K.; Levy, B.M.: *Text Book of Oral Pathology.* Philadelphia: W.B. Saunders Company, pp 694-699.
2. Regezi, J.A. and Seiubba, J.: *Oral Pathology.* Philadelphia: W.B. Saunders Company, pp 372-374.
3. Harris, W.H. and Dudely, H.R.: The natural history of fibrous dysplasia. J Bone Joint Surg, American Vol. 44-A.:207-233, March 1962.
4. Eversole, L.R. and Sabes, W.R.: Fibrous dysplasia: A nosologic problem in the diagnosis of fibro-osseous lesions of the jaws. J Oral Pathol, 1:189-220, October 1972.
5. Eich, G.F.; Babyn, P.; Armstrong, D. *et al*: An unusual case of craniofacial fibrous dysplasia presenting in early infancy. Pediatric Radiol, 20:495-498, February 1990.
6. Arendt, D.M.; Whitt, J.C.; Hon, C.B.: Facial asymmetry. JADA,120: 668-690, June 1990.
7. Mizuno, A.; Kuroyanagi-Nakajima, M.; Akiyama, Y.: Facial fibrous dysplasia: Report of a case. Oral Surg Oral Med Oral Pathol, 72: 284-288, September 1991.
8. Tanaka, Y.; Tajima, S.; Maejima, S.: Craniofacial fibrous dysplasia showing marked involution postoperatively. Annals Plastic Surg, 30: 71-76, January 1993.
9. Dachille, H.D. and Goldberg, J.S.: Fibrous dysplasia-induced hypocalcemia/rickets. J Oral Maxillofacial Surg, 48:1319-1322, December 1990.

ORTHODONTIC TREATMENT NEEDS OF MALE ARMY RECRUITS

The final major finding from this study is the markedly higher preference for orthodontic care by recruits with more than 12 yr of education (58.2%) versus those with 12 yr or less (13.9%). Previous studies have shown that demand for orthodontic care is highly correlated with family income and socioeconomic status.

The potential future impact of education level on demand for orthodontic care by US Army recruits could be substantial. According to 1992 data from the Defense Data Manpower Center, only 7.7% of US Army enlisted personnel have more than 12 years of education. However, current demographic trends show the Army is recruiting better-educated, enlisted personnel. One-third of the recruits in this sample had more than a high school education. If the Army is able to retain these better-educated soldiers, this recruiting trend coupled with the drawdown of military personnel could result in a considerably more highly educated active duty enlisted force than present. Data from this study suggest that such a force will have a higher desire for orthodontic care.

Future demand for orthodontic care by US military recruits may be further stimulated by the emerging acceptance of adult orthodontic care by the general public. A poll of practicing orthodontists conducted by the American Association of Orthodontists in 1970 found that 90% of orthodontists were treating patients between 21 and 30 yr of age and 48% were treating patients between 30 and 40 yr of age.

When viewing the demand for orthodontic services by US Army recuits, it is important to remember that other factors may dampen effective demand for orthodontic care in the military setting. The major cap on demand for orthodontic care for active duty soldiers appears to be restricted access. Restricted access to orthodontic care by active duty US soldiers is driven by combat readiness. Too, assignment stability is important for allowing an orthodontic case to be taken from inception to completion.

Searcy, V.L. and Chisick, M.C.: Perceived, desired, and normatively determined orthodontic treatment needs in male US army recruits. Community Dent Oral Epidemiol, 22:437-440, December 1994.

Oral findings in Michelin Tire syndrome: Report of case

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Michelin Tire syndrome is a genetic disease whose primary clinical presentation consists of symmetrical, ringed creases around all extremities and torso.[1-7] This distinctive picture is caused by underlying hamartoma formation of smooth muscle tissues.[3-5] It is generally thought to be an autosomal dominant disease, which may present as an isolated finding or in association with other abnormalities. It is a rare disease with only fourteen cases reported in the literature.[1,2] Interestingly there are no functional problems related to the skin folds; further, the depth and number of creases appear to decrease with time.[1,3,4] The name *Michelin Tire syndrome* was first used by Ross in 1969, because the physical findings resembled the corporate symbol of the Michelin Tire Company (Figure 1).[1,5,6] Of all previous reports in the literature, only Schnur *et al* and Kunze mention abnormal oral findings.[1,7] Other reports describe skin anomalies, but make no mention of the mouth or describe any remarkable oral findings.[2-6] Schnur reported clefting of the corners of the mouth, a submucous cleft, high-arched palate, midline notching of the upper lip, multiple oral frenula, missing (primary) lateral incisors, pegged mandibular incisors, and extensive dental decay.[1] Kunze reported a median cleft in each of his two patients, with one of these children also demonstrating micrognathia.[7] The following is a report based on detailed oral examination of the child whom Schnur *et al* had described. There have been no previous reports in the dental literature on patients with *Michelin Tire syndrome.*

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CASE REPORT

The patient, a three-year-old male with diagnosed *Michelin Tire syndrome*, was referred to the Dental Division for oral evaluation. The child was the product of a term gestation. The mother was thirty years old and the father thirty-eight years old at the time of conception. There was no history of parental consanguinity. At birth, the child was noted to have an increased number of deep skin creases, excessive hair, and a dark complexion. Clefting of the lateral commissures of the mouth, midline notching of the upper lip, a submucous cleft, and other orthopedic anomalies were also present. Further, the child had long eyelashes, thick eyebrows, hypertelorism, bilateral epicanthal folds, and an antimongoloid slant to the eyes. Developmental delay was also found, as were generalized and focal seizures.[1]

The child was extremely apprehensive in the out-patient setting and thorough examination was stressful and difficult for him. In light of the fact that he was already scheduled for a general anesthetic procedure by the Otorhinolaryngology Division, we determined it would be in the patient's best interest to defer further evalu-

Figure 1. Symmetrical, ringed creases of extremities are seen in Michelin Tire syndrome.

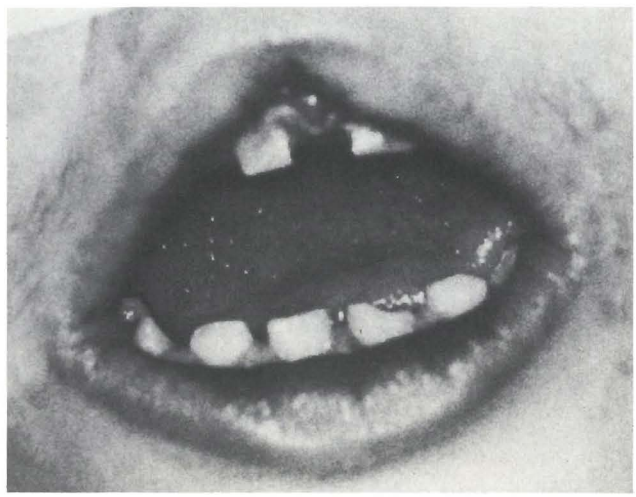

Figure 2. Note the midline notching of the upper lip. Surgery had been performed to repair bilateral clefted commissures.

Figure 3. A heavy maxillary frenum and shallow buccal vestibule were noted in this patient. The severe caries was associated with nocturnal bottle feeding.

ation and treatment until that time. Three weeks after our initial consultation the child received bilateral myringotomy tubes, using a general anesthestic. At that time we performed a detailed clinical examination, a radiographic survey, prophylaxis and topical fluoride application, restorations, sealants, and extractions, where such treatment was indicated. Our examination revealed a slightly tight oral opening, a sequela to the previously performed surgery to close the wide, clefted commissures. Notching at the midline of the upper lip was present. Intraoral soft tissues were remarkable for maxillary lateral and central frenula which were short and heavy. The maxillary buccal vestibule was noted to be shallow. In the mandible we noted similar findings of multiple short, heavy frenula and a shallow buccal vestibule. No lingual frenum was noted; the family later reported to us, however, that they had forgotten to mention that a lingual frenectomy procedure had previously been performed. The dental arches were wide, U-shaped, and symmetrical. There was a tendency toward a crossbite of the maxillary and mandibular right canines. The palate was wide but normal in form; it was not high arched. All primary teeth were present, though they had not fully emerged from the gingiva. Color, size, form, and texture were within normal limits, except for the presence of severe caries in the maxillary anterior teeth, a situation not unexpected in light of this child's nighttime bottle feeding history. No hypoplasia was found. Large interdental spacing was present (Figures 2, 3, 4, and 5). Mild to moderate plaque and calculus accumulations were noted. Radiographic examination revealed a normal developing dentition.

Microscopic examination of one of the extracted teeth revealed that dentin and cementum were within normal limits. Because of the extensive caries, no report was made regarding the enamel.

At the postoperative clinic visit, we discussed in detail with the family nursing caries, oral hygiene techniques, and fluoride supplementation. Soft tissues were healing well and all restorations were intact. At this time we also

Figure 4. A shallow buccal vestibule was also noted in the mandible.

Figure 5. Lateral frenula were found on the right and left sides of the maxilla and the mandible as shown in this example. A frenectomy had been performed on the tongue.

examined the mother's oral cavity. No unusual soft tissue or dental conditions were found and the alveolar arches were normal in size and configuration. There were no heavy frenum attachments and the depths of the maxillary and mandibular buccal vestibules were within normal limits. The father was not present at this time.

DISCUSSION

This paper reports the oral findings of a patient with *Michelin Tire syndrome*, a rare genetic disorder characterized by diffuse smooth muscle hamartomas that produce skin creases or folds on the extremities and torso. Oral examination revealed some notable findings:

- ☐ Wide alveolar arches.
- ☐ Large interdental spacing.
- ☐ Clefting of mouth commissures.
- ☐ Midline notching of the upper lip.
- ☐ Multiple maxillary and mandibular central and lateral frena.
- ☐ Shallow buccal vestibules (though not so shallow as seen in chondroectodermal dysplasia).
- ☐ Ankyloglossia.
- ☐ Primary teeth that are normal in number, size, shape, and texture.
- ☐ Delayed full-eruption of primary teeth into the mouth.

Since there are other conditions that present with skin creases, this oral picture may assist in the diagnosis of *Michelin Tire syndrome*. The authors urge others who examine *Michelin Tire syndrome* patients to report their findings, in order to verify or modify those described here.

REFERENCES

1. Schnur, R.E.; Herzberg, A.J.; Spinner, N. *et al*: Variability in the Michelin Tire syndrome. J Amer Acad Derm, 28:364-370, February 1993.
2. Bass, H.N.; Caldwell, S.; Brooks, B.S.: Michelin tire baby syndrome: familial constriction bands during infancy and early childhood in four generations. Amer J Med Genet, 45:370-372, February 1993.
3. Glover, M.T.; Malone, M.; Atherton, D.J.: Michelin-tire baby syndrome resulting from diffuse smooth muscle hamartoma. Pediatr Derm, 6:329-331, December 1989.
4. Niikawa, N.; Ishikiriyama, S.; Shikimani, T.: Michelin tire syndrome—an autosomal dominant trait. Amer J Med Genet, 22:637-638, November 1985.
5. Burgdorf, W.H.C.; Doran, C.K.; Worret, W-I.: Folded skin with scarring: Michelin tire baby syndrome? J Amer Acad Derm, 7:90-93, July 1982.
6. Ross, C.M.: Generalized folded skin with an underlying lipomatous nevus, the Michelin tire baby. Arch Derm, 100:320-323, September 1969.
7. Kunze, J. and Riehm, H.: A new genetic disorder: autosomal-dominant multiple benign ring-shaped skin creases. Eur J Pediatr, 138: 301-303, July 1982.

ABSTRACTS

Mertz-Fairhurst, Eva J.; Adair, Steven M.; Sams, Deirdre R. *et al*: Cariostatis and ultraconservative sealed restorations: Nine-year results among children and adults. J Dent Child, 62:97-107, March-April 1995.

The objective of this clinical study was to evaluate the long-term efficacy of placing sealed posterior composite restorations for arresting active caries. The tooth preparation for this bonded and sealed restoration was limited to placing a bevel in the enamel only, without the removal of the carious lesion. The radiographic and clinical performance of these ultraconservative sealed composite restorations placed over caries (CompS/C) was compared over a period of nine years with: 1) ultraconservative, localized sealed amalgam (AGS) restorations with no extension for prevention, and 2) traditional, unsealed amalgam restorations (AGU) with the usual extension for prevention outline form.

Sealant retention with >50 percent to 100 percent of the margins occurred in 64 percent of CompS/C and 82.5 percent of AGS restorations. After nine years the cumulative failure rates were 16 percent for CompS/C, 2.5 percent for AGS, and 17.1 percent for AGU restorations. Thus, the clinical performance of CompS/C restorations was slightly superior to that of the traditional AGU restorations. The AGS restorations were definitely superior to the traditional AGU restorations and to the CompS/C restorations in both children and adults alike.

Complete sealant retention over CompS/C and AGS restorations was equivalent between children and adults (P=0.14 and 0.74, respectively). A higher percentage of open margins in CompS/C restorations was seen, however, in children (17.4 percent) than adults (1.94 percent).

This study has shown that Class I caries can be arrested by the CompS/C restoration.

Arresting active caries; Sealed posterior composite restorations; Localized sealed amalgam restorations; Unsealed amalgam restorations

Rakocz, Meir; Lavie, Gavriel; Martinowitz, Uri: Glanzmann's thrombasthenia: The use of autologous fibrin glue in tooth extractions. J Dent Child, 62:129-131, March-April 1995.

Three teeth were extracted in three sessions in a 15-year-old girl with Glanzmann's thrombasthenia who suffered in the past severe bleeding and required platelet transfusions. The extraction sites were treated locally with autologous fibrin glue in adjunct of tranexamic acid swish and swallow rinses. No systemic platelet infusion was employed as a preventive measure. No postoperative bleeding occurred.

Glanzmann's thrombasthenia; Tooth extraction; Autologous fibrin glue

Yassin, Othman Mahmoud: In vitro studies of the effect of a dental explorer on the formation of a carious lesion. J Dent Child, 62:111-117, March-April 1995.

This project deals with *in vitro* mechanical damage of early carious lesion (enamel lesion) in artificial U-shaped grooves caused by a sharp dental explorer. The lesions were formed in enamel grooves in 3 percent by weight cellulose solution in 0.1 M lactic acid containing 1.08 M KH_2PO_4 and 1.8 mM $CaCl_2$ buffer to pH 4.28 after two weeks of demineralization. A relatively intact surface layer was observed in all the grooves.

Three demineralized grooves were probed with different forces 100 g, 300 g and 500 g. The traumatic damage was assessed by using microradiography. There was no damaging effect in the sound enamel grooves probed with a sharp explorer up to a force of 500 g. Breakdown of the surface layer was assessed in all probed demineralized enamel grooves, converting the white spot lesion with apparently a sound surface layer into a cavity. Explorer penetration into the lesion was found to be strongly related to the force applied. The use of a sharp dental explorer should be reconsidered in examination and diagnosis of early carious lesions in pits and fissures and an alternative technique should be sought.

Probe forces; Enamel resistance

Waldman, H. Barry: Asian American and Pacific Islander children: They will become an increasing reality in your practice. J Dent Child, 62:136-140, March-April 1995.

Asian Americans and Pacific Islanders are some of the fastest growing minorities in the United States. But most dentists have limited knowledge about and experience with children in these populations. A review is provided of Census data and cultural differences in an effort to bring increasing recognition of this population of children.

Asian Americans; Pacific Islanders; Children of minorities; Cultural differences

Waldman, H. Barry: Can your next pediatric patient hear you? J Dent Child, 62:132-135, March-April 1995.

Communication with pediatric patients can be complicated when the child has deminished hearing capacity. The prevalence of this problem in various population groups is provided in an effort to assist practitioners as they increase care to diverse population groups.

Hearing loss; Communication

Dasanayake, Ananda P.; Maclauso, Maurizio; Roseman, Jeffrey *et al*: Validity of the mothers' recall of her child's antibiotic use. J Dent Child, 62:118-122, March-April 1995.

We compared the mother's recall of her child's antibiotic use with the informa-

tion obtained from the child's health records, while studying the association between antibiotic use and prevalence of mutans streptococci among 102 (5-12 years) inner-city black children. Poor recall of the mother (concordance ratio = 62 percent; kappa = 0.18, p = 0.06) and the lower frequency reported by her (p = 0.02) biased the positive association between child's antibiotic use during the second year of life and oral presence of mutans streptococci toward the null value (Odds Ratio [OR] based on health records = 3.1, 95 percent Confidence Interval [CI] = 1.03-9.3; OR based on mother's recall = 1.6, 95 percent CI = 0.44-5.8). Multiple logistic regression analysis failed to identify any significant determinant of the accuracy of the mother's recall. Since mothers considerably underestimate child's use of antibiotics, at least among inner-city low income blacks, this method of data collection should be limited to circumstances where no other options are available, and such results should be interpreted with caution.

Antibiotic use; Streptococcus mutans; Recall

Davis, Martin J. and Vogel, Lillian: Neurological assessment of the child with head trauma. J Dent Child, 62:93-96, March-April 1995.

Dental trauma is a type of head trauma and may have serious life compromising sequelae. These sequelae may be immediate or may appear only over an extended period of time. It is essential that oral health care providers recognize neuropathology resultant from head trauma. The article provides a clear and appropriate neurological assessment of the child with dental and, therefore, head trauma and gives recommendations for the management of the child who may show significant sequelae to the event.

Dental trauma; Head trauma; Neuropathological sequelae

Helpin, Mark L. and Rosenberg, Howard M.: Oral findings in Michelin Tire syndrome: Report of case. J Dent Child, 62:148-150, March-April 1995.

Michelin Tire syndrome is a genetic disease caused by smooth muscle hematoma formation, a condition which causes symmetrical, ringed creases around all extremities and the torso. Oral findings in a child with this syndrome were unusual and are presented in this case report.

Michelin Tire syndrome

Karlezén-Reuterving, Gunlög and van Dijken, Jan W.V.: A three-year follow-up of glass ionomer cement and resin fissure sealants. J Dent Child, 62:108-110, March-April 1995.

The aim of the present study was to compare intraindividually a type III fissure-sealant, glass ionomer cement with a resin-based sealant. One hundred and forty-eight first permanent molars were sealed in forty-seven children. After three years 20.8 percent of the resin and 34.7 percent of the glass ionomer cement sealants were partially lost, and 0 percent and 37.5 percent, respectively, were totally lost. One tooth (1.4 percent) in the glass ionomer cement group and three teeth (4.2 percent) in the resin group developed caries.

Glass ionomer cement; Resin-based sealant

The Academy of Dentistry for the Handicapped Changes Its Name

The Academy of Dentistry for the Handicapped, originally founded in 1952, recently announced a name change to *Academy of Dentistry for Persons with Disabilities* (ADPD). The name change acknowledges the preferred "people first" language and maintains the progressive image earned by the organization since its inception.

The mission of the Academy of Dentistry for Persons with Disabilities is to improve the oral health of persons with disabilities by involving dental professionals and other health care providers in the planning and delivery of quality, compassionate and comprehensive care. Included are networking activities for professionals regarding the special oral health needs of persons with disabilities; educational opportunities for professionals on the topic of special needs dentistry; and involvement in national and local projects which heighten awareness among professionals and the general public about the need for specialized dentistry.

A good example of a local activity which became a national program is the Special Athletes, Special Smiles program spearheaded by Dr. Steve Perlman of the ADPD. This program provides dental screenings for athletes who participate in Special Olympics. To date, over 5,000 athletes have been screened in 15 major cities across the United States and Canada. This program was piloted at Boston University in 1992 and spread throughout North America with the help of many dedicated individuals and corporate sponsors. It has been successful in allowing dentists, dental hygienists and dental students to experience first-hand the issues and rewards of treating persons with disabilities.

If you require information about joining ADPD, call us at 312-440-2660.