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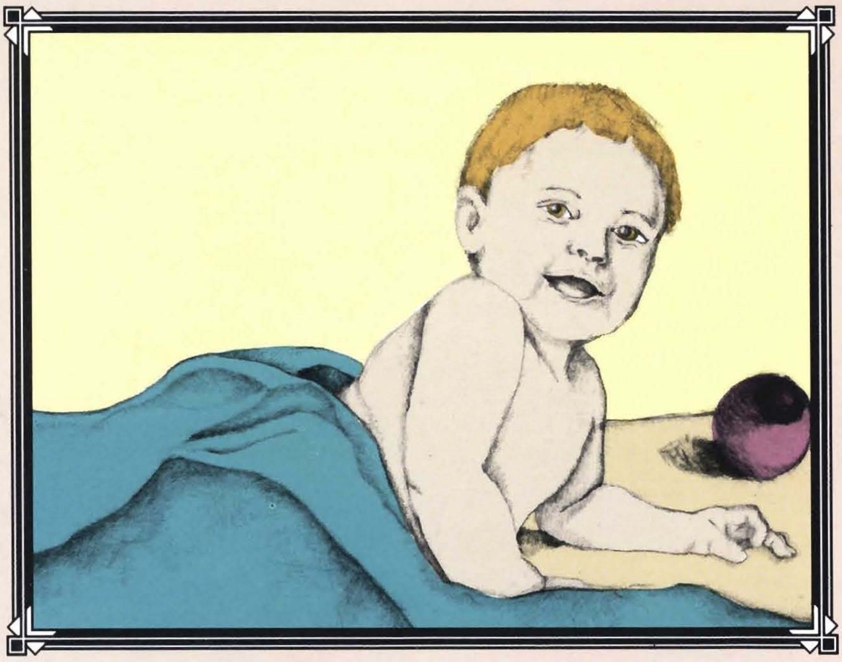
SEPTEMBER-OCTOBER 1988

JOURNAL OF DENTISTRY FOR CHILDREN

Phase IV (5.5 months to 8 months) is portrayed as the final prelocomotion period of life. The baby has much greater command of his body than he had at birth; and his eyes and ears function on a par with those of a young adult. Although he has gained appreciably in controlling his body, he still cannot move about.

Phase IV, like Phase III, is a period of chronically good humor. Enjoy it while you can. After eight months and locomotion is acquired, there will be no more dull moments.

-Jean Piaget, 1963
-Burton L. White, 1975



**I SEE THY BEAUTY GRADUALLY UNFOLD,
DAILY AND HOURLY, MORE AND MORE.**
—Alfred, Lord Tennyson

DIFFERENTIATION MULTIPLICATION MOVEMENT

ASDC

AMERICAN SOCIETY OF DENTISTRY FOR CHILDREN

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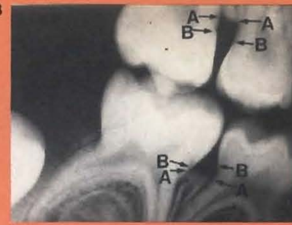

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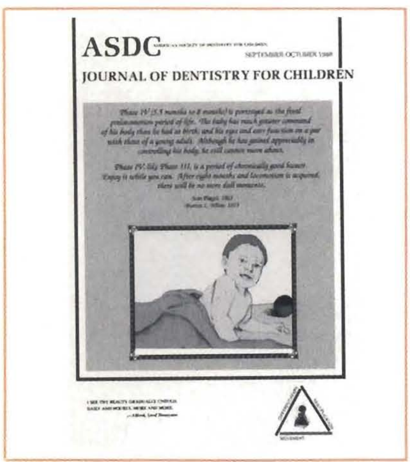

As if emerging from a cocoon, the Phase IV baby is escaping from the protection of virtual immobility. New interests, new things, and new strength propel him rapidly from place to place. Design and art by Sharlene Nowak-Stellmach.

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CASE REPORTS

For the busy reader

Monitoring of sedated pediatric dental patients—page 329

Changes in vital signs are most often secondary to respiratory depression. Pulse oximetry allows for earlier detection of respiratory changes than do other methods of monitoring. As some patients are unable to maintain an adequate airway, leading to hypoxemic episodes, careful monitoring can prevent their slipping into unconsciouness.

Requests for reprints should be directed to Dr. David P. Durr, Department of Pediatric Dentistry, Eastman Dental Center, 625 Elmwood Avenue, Rochester, NY 14620.

Dentofacial trauma in children—page 334

Most of the literature on dental injury pertains to the classification, incidence, prevalence, and treatment of fractured teeth. A major finding here is that the five leading causes of dentofacial injuries, regardless of severity, were falls, being struck by an object, bicycle accidents, assaults, and motor vehicle accidents.

Requests for reprints should be directed to Dr. Marilyn S. Harrington, School of Dentistry, University of Missouri-Kansas City, 650 E. 25th Street, Kansas City, MO 64108.

Dental emergencies presenting to a children's hospital—page 339

Most of the 222 dental patients with emergency situations, observed during the period described, had injuries involving the eight incisor teeth (86.1 percent). Patients or their parents facing these situations are often referred to the hospital emergency room, especially those that provide dental services.

Requests for reprints should be directed to Dr. Robert F. Majewski, Department of Pediatric Dentistry, Tufts University, School of Dental Medicine, One Kneeland Street, Boston, MA 02111.

The effect of dental probing on subsequent enamel demineralization—page 343

By using an explorer on demineralized fissures, an entrance may be created through which cariogenic microorganisms and their metabolic products can directly penetrate into the softened subsurface. The effect of probing on the rate of formation and growth of lesions was quantified *in vitro*.

Requests for reprints should be directed to Dr. C.S.E. van Dorp, Department of Cariology and Endodontology, Academic Centre for Dentistry, Amsterdam (ACTA), Louwesweg 1, 1066 EA Amsterdam, THE NETHERLANDS.

A radiographic study of interproximal alveolar bone crest between the primary molars in children—page 348

This study establishes baseline data regarding the normal relationship between the cementoenamel junction and the alveolar crest in the primary dentition, using bitewing radiographs. Measurements greater than those presented in this study may indicate an early stage of periodontal disease in the primary dentition, a tendency of the patient to develop the disease, or a combination of these.

Requests for reprints should be directed to Dr. Enrique Bimstein, Department of Pediatric Dentistry, Hadassah Faculty of Dental Medicine, Hebrew University of Jerusalem, P.O. Box 1172 Jerusalem, ISRAEL.

The attached gingiva in children: diagnostic, developmental and orthodontic considerations in its treatment—page 351

In the clinical context, the width of the attached gingiva is established by substracting the sulcus depth from the width of the keratinized gingiva. Summarizing all factors, it becomes clear that control of marginal inflammation appears to be the most important key to limiting—and possibly reversing—an otherwise progressive recession. A nonsurgical approach should be the clinician's first choice.

Requests for reprints should be directed to Dr. Enrique Bimstein, Department of Pediatric Dentistry, Hadassah Faculty of Dental Medicine, Hebrew University of Jerusalem, P.O. Box 1172 Jerusalem, ISRAEL.

The statistical unit for analysis of developmental changes in the attached gingiva in children—page 357

The issue of an adequate statistical unit in periodontal studies—sites or mean per patient—has been a subject of discussion for reasearchers and clinicians. The significant differences in values between these two groups, and some types of teeth, indicate that it would be inappropriate to obtain a mean per patient.

Requests for reprints should be directed to Dr. Enrique Bimstein, Department of Pediatric Dentistry, Hadassah Faculty of Dental Medicine, Hebrew University of Jerusalem, P.O. Box 1172 Jerusalem, ISRAEL.

Anomalies of form and number, fused primary teeth, a correlation of the dentitions—page 359

Patients with fused primary lateral incisors and canines have about a 75 percent chance of lacking the succedaneous lateral incisor. Patients with fused incisors have less than a 20 percent chance of a missing permanent tooth.

Requests for reprints should be directed to Dr. F. Thomas Hagman, Department of Pediatric Dentistry, College of Dentistry, Ohio State University, 305 West 12th Avenue, Columbus, OH 43210.

A complete fusion in the primary human dentition: a histological approach—page 362

A trace of cementum totally surrounded by dentine could not be found here; this histological evaluation gave no further information about the origin of this double tooth. The fact that the dental pulp was complex could not be observed radiographically.

Requests for reprints should be directed to Dr. Luc C. Martens, Universitair Ziekenhuis Gent, Kindertandheelkunde, De Pintelaan 185, B-9000 Gent BELGIUM.

Delayed eruption of a maxillary central incisor associated with an odontome: report of case—page 368

Odontomes—tumor-like lesions found in the jaws—are composed of enamel, dentine, cementum and pulp tissue. They are classified as either compound or complex. Findings in this case permitted the histopathological diagnosis of a complex odontome.

Requests for reprints should be directed to Dr. Richard G. Oliver, Department of Child Dental Health, Dental School, University of Wales College of Medicine, Health Park, Cardiff CF4 4XY, WALES.

Ameloblastic fibrodentinoma: report of case—page 372

The classification, progression, and treatment of the mixed odontogenic tumors remain controversial. In the case reported here, examination of a patient who presented with delayed eruption of a primary central incisor led to the discovery of an impacted incisor and an associated isolated radiolucency.

Requests for reprints should be directed to Dr. Stephen I. Brandt, Division of Pediatric Dentistry, Scott & White Clinic, 2401 South 31st Street, Temple, TX 76508.

Long-term interdisciplinary management of multiple mesiodens and delayed eruption: report of case—page 376

This report adds to existing knowledge on the treatment of mesiodens, emphasizing the need to individualize the treatment plan, and demonstrates the importance of interdisciplinary cooperation in total patient care.

Requests for reprints should be directed to Dr. Dennis N. Ranalli, University of Pittsburgh, School of Dental Medicine, Department of Pediatric Dentistry, 333 Salk Hall, Pittsburgh, PA 15261.

Monitoring of sedated pediatric dental patients

Behavior

B. Gene Whitehead, DMD
David P. Durr, DMD, MS
Steven M. Adair, DDS, MS
Howard M. Proskin, PhD

Goodson and Moore report that continuous monitoring of respiratory function and early recognition of respiratory difficulties are essential for the successful management of sedated pediatric dental patients.

Traditional methods of monitoring patients, including measurement of pulse, blood pressure, and respiratory rate, and visual observation of the patient for cyanosis, are useful, but may not reflect hypoxemia until severely lowered levels of blood oxygen are reached. The oxygen saturation of the hemoglobin (SaO_2), as noninvasively measured by a pulse oximeter, is reportedly an earlier sign of respiratory distress than are other factors.[2]

In room air, the normal SaO_2 of a healthy child is 97 percent to 100 percent. Adequate oxygenation of the tissues occurs above 95 percent. Levels below 95 percent are, therefore, considered hypoxemic, and those below 75 percent severely hypoxemic.[3] Detectable changes in other vital signs may not occur until the SaO_2 reaches the severely hypoxemic value. When using only the traditional methods of monitoring, the patient's respiratory status may, therefore, become severely compromised before any problem is recognized.[2]

Dr. Whitehead was a postdoctoral student, Department of Pediatric Dentistry, Eastman Dental Center; he is now in private practice, Clearwater, FL. Dr. Durr is Assistant Chairman, Department of Pediatric Dentistry, Eastman Dental Center. Dr. Adair is Chairman, Department of Pediatric Dentistry, Eastman Dental Center. Dr. Proskin is Coordinator of Biostatistics, Eastman Dental Center.

Chloral hydrate is a commonly used sedative agent in pediatric dentistry and can be administered alone or in combination with an antihistamine sedative and/or nitrous oxide and oxygen. It has been generally accepted that, in therapeutic doses, chloral hydrate has little direct effect on patients' physiologic functions. Studies by Houpt *et al* have shown that heart rate, blood pressure, and respiratory rate remain essentially unchanged throughout dental treatment in child patients sedated with various combinations of chloral hydrate, promethazine, and nitrous oxide and oxygen.[4,5]

To date, only one study has evaluated SaO_2 in pediatric dental patients sedated with chloral hydrate. In that experiment, Mueller *et al* sedated twenty-four-month to seventy-two-month-old patients with 100 mg/kg body weight chloral hydrate in conjunction with 50 percent nitrous oxide and 50 percent oxygen.[2] Seven of the twenty subjects had an SaO_2 decrease below normal physiologic levels with no detectable changes in heart rate, blood pressure or respiratory rate. These data suggest that significant periods of oxygen desaturation may occur in sedated pediatric dental patients without observable changes in the traditional vital signs.

The purpose of this study was to evaluate the SaO_2 and traditional vital signs in patients receiving chloral hydrate and hydroxyzine, along with nitrous oxide and oxygen, in doses more conservative than those used by Mueller *et al.*[2] These factors were also evaluated in patients sedated with only nitrous oxide and oxygen. As a secondary purpose, the effectiveness of these sedative regimens in managing patient behavior was analyzed.

MATERIALS AND METHODS

Twenty-four children, aged two to five years inclusive, were selected for this study. Each had minimum treatment needs of at least two Class I restorations and no previous dental experiences other than an initial examination and cleaning appointment. Behavior at the initial examination was rated, using the scale developed by Frankl, Shiere, and Fogels (Table 1).[6] Twelve children exhibiting *definitely negative* behavior at the initial examination appointment were assigned to a chloral hydrate sedation group. Another twelve children who exhibited *negative* behavior were assigned to a nitrous oxide sedation group. All subjects accepted into this study presented with unremarkable medical histories and had received medical clearance for sedation from their pediatricians.

Children assigned to the chloral hydrate sedation group were given 50 mg/kg body weight chloral hydrate* syrup and 25 mg hydroxyzine pamoate** oral suspension. Forty-five minutes after administration, the patients were brought to the dental operatory, placed in a supine position in the dental chair, and supported in a specially modified Papoose Board†.[7] The chloral hydrate-hydroxyzine sedation was supplemented with nitrous oxide administered via a nasal mask‡ during the dental treatment. All subjects were given 100 percent oxygen for the first three minutes, then 40 percent nitrous oxide and 60 percent oxygen during the course of dental treatment, and 100 percent oxygen for five minutes at the end of the procedure.

Children selected for the nitrous oxide sedation group were brought to the dental operatory and placed in a supine position in the dental chair. Nitrous oxide was administered according to the protocol described for the chloral hydrate sedation group.

Vital signs monitored in this study included oxygen saturation of hemoglobin (SaO_2), heart rate, blood pressure, and respiratory rate. SaO_2 and heart rate were monitored by a pulse oximeter★ with the sensor★★ placed on the patient's right great toe. Blood pressure was recorded by an automatic monitor♣ with the cuff placed on the patient's left arm. A pretracheal stethoscope was used to monitor respiratory rate. Patients were also observed for cyanosis.

The patient's behavior during treatment was determined as either interfering (I) or not interfering (NI) with completion of the dental procedure. Interfering behavior was defined as disrupting treatment while noninterfering behavior did not impede treatment progress. When more than 90 percent of the behavior ratings for a particular patient were coded NI, the sedation was considered effective; between 80 percent and 90 percent, it was considered moderately effective; and less than 80 percent, ineffective.

Baseline vital signs were taken prior to the administration of any medication. Vital signs and behavior ratings were recorded at three-minute intervals during the dental procedure, beginning when the child was

* - Noctec Syrup, 500 mg/5ml, E. R. Squibb and Sons, Inc., Princeton, NJ.

** - Vistaril, 25 mg/5ml, Pfizer Laboratories, Div., Pfizer Inc., New York, NY.

† - Olympic Papoose Board, Olympic Medical Corp., Seattle, WA.

‡ - Brown N_2O Scavenging Mask, N 100, McKesson, Moncks Corner, SC.

★ - Nellcor Pulse Oximeter, N 100, Nellcor Corp., Hayward, CA.

★★ - Nellcor Digit Oxisensor Transducer, D-25, Nellcor Corp., Hayward, CA.

♣ - Dinemap Adult/Pediatric Vital Signs Monitor, 845 XT, Critikon, Inc., Tampa, FL.

Table 1 □ categories of behavior defined by Frankl, Shiere, and Fogels.[6]

Rating 1:	Definitely negative Refusal of treatment; forceful crying; fearful; any other overt evidence of extreme negativism.
Rating 2:	Negative Reluctant to accept treatment; some evidence of negative attitude but not pronounced, i.e. sullen, withdrawn.
Rating 3:	Positive Acceptance of treatment; at times cautious; willingness to comply with the dentist, at times with reservation but patient follows the dentist's directions cooperatively.
Rating 4:	Definitely positive Good rapport with the dentist; interested in the dental procedures; laughing and enjoying the situation.

Table 2 □ Vital sign ranges for children two to five Years Old.[8]

Systolic blood pressure	78-112
Diastolic blood pressure	48-79
Heart rate	105± 35
Respiration rate	20-30

Table 3 □ Distribution of age, hypoxemic episodes, and sedation effectiveness for patients in the chloral hydrate group.

	Age				
	2	3	4	5	Total
Age distribution	5	6	1	0	12
Patients exhibiting hypoxemia	1[a]	1	0	0	2
Effective sedation	4	4	0	0	8
Moderately effective sedation	1	0	0	0	1
Ineffective sedation	0	2	1	0	3

[a] Patient exhibiting 2 episodes of hypoxemia.

Table 4 □ Distribution of age, hypoxemic episodes, and sedation effectiveness for patients in the nitrous oxide group.

	Age				
	2	3	4	5	Total
Age distribution	0	2	3	7	12
Patients exhibiting hypoxemia	0	0	0	0	0
Effective sedation	0	1	2	7	10
Moderately effective sedation	0	0	1	0	1
Ineffective sedation	0	1	0	0	1

placed in the dental chair and ending when an alert, responsive, and fully recovered child left the operatory. Vital signs were also recorded anytime SaO_2 fell below 95 percent, indicating mild hypoxemia, for more than three seconds or when any vital sign dropped below the normal age-related range as listed in Table 2, or if the patient became cyanotic.[8]

All dental treatment in this study was provided by the same pediatric dentistry postdoctoral student (BGW), with the assistance of the same dental auxiliary. All appointments were performed in the same operatory at the same time of day. A third party (DPD) recorded all vital signs and behavior ratings.

The study design was reviewed and approved by the Institutional Review Board of the Eastman Dental Center.

RESULTS

Table 3 reports the distribution of age, hypoxemic episodes, and sedation effectiveness for patients in the chloral hydrate group. The mean age of these twelve patients was two years, eight months (2.66 years) with a range from two years, one month (2.08 years) to four years, one month (4.08 years). Two patients in the chloral hydrate group exhibited episodes of hypoxemia, i.e. SaO_2 less than 95 percent for three or more seconds. One of these patients, aged two years, one month (2.08 years) exhibited two periods about thirty minutes apart, during which SaO_2 dropped to 92 percent. The other patient, aged three years, one month (3.08 years), exhibited one episode during which SaO_2 dropped to 90 percent. In all three instances, SaO_2 gradually rose when the head was repositioned. In no patient did there appear to be a relationship between SaO_2 and other vital signs. Blood pressure, heart rates, and respiratory rates remained within the normal age-related range (Table 2) for all patients; no patients exhibited cyanosis. Statistical analysis, correlating SaO_2 with other vital signs for each patient, showed no significance at the $p<0.05$ level. Behavior management in the chloral hydrate group was classified as effective for eight patients, moderately effective for one patient, and ineffective for three patients. All of the effectively sedated patients slept during the procedure, but were easily aroused at the end of the appointment. Both patients exhibiting hypoxemia were in the effective category. Ineffective behavior was primarily related to head movement. One patient in the chloral hydrate group exhibited mild nausea and vomiting. There were no other adverse reactions.

Table 4 reports the distribution of age, hypoxemic episodes, and sedation effectiveness for patients in the nitrous oxide group. The mean age of these twelve patients was four years, five months (4.42 years) with a range from three years, four months (3.33 years) to five years, ten months (5.83 years). There were no episodes of hypoxemia. All patients maintained an SaO_2 of 98 percent or higher. Blood pressures, heart rates, and respiratory rates remained within normal age-related ranges (Table 2) for all patients. Individual statistical analysis for each patient again showed on significant correlations between fluctuations in SaO_2 and other vital signs at the $p<0.05$ level. No patients exhibited cyanosis. Behavior management was classified as effective for ten patients, moderately effective for one patient, and ineffective for one patient. There were no adverse reactions to the nitrous oxide regimen.

DISCUSSION

Of all the physiologic functions which can be monitored, it appears that those related to respiratory function are most important.[1] Changes in vital signs are most often secondary to respiratory depression. A drop in heart rate or blood pressure, a change in skin color, or even some changes in respiratory rate occur only after a significant hypoxemia, i.e. a significant drop in SaO_2. Pulse oximetry allows for earlier detection of respiratory changes than do other methods of monitoring.[2]

Yelderman and New found the pulse oximeter to be precise and accurate with a highly positive correlation ($r = 0.98$) between values obtained via pulse oximetry versus those obtained from arterial blood gases.[9] According to Deckardt and Stewart, changes in blood pressure or hematocrit did not affect the accuracy of the pulse oximeter.[10] In the present study, it was noted that resistive movements by uncooperative patients could displace the toe sensor of the pulse oximeter resulting in abnormal readings. The cuff and hoses of the automatic blood pressure monitor were also affected by such movements, and aberrant readings were noted. Such problems only occurred with unsuccessfully managed patients who were conscious, overtly responsive, and obviously in no physiologic distress. Adequately sedated patients with minimal body movement showed no such abnormal readings. Artifacts were not included in evaluation of patient data.

The significant finding of this study was that children sedated even with conservative doses of chloral hydrate and hydroxyzine in conjunction with nitrous oxide and oxygen can have respiratory difficulties that are not detected by traditional methods of monitoring. SaO_2 levels fell to as low as 90 percent to 92 percent without changes in blood pressure, heart rate, respiratory rate or skin color in two of twelve patients. The use of the pulse oximeter allowed early detection at a time when correction was simply a matter of readjusting head position. Had detection been delayed, more extensive resuscitation measures may have been needed.

The hypoxemic episodes corrected by repositioning the head indicate that some patients were unable to maintain their own airway. Airway maintenance is an essential element in the definition of consciousness. Although the pediatric dentist's intent with this chloral hydrate regimen is conscious sedation, it is obvious that some patients are more deeply sedated, i.e. their physiologic status does not fulfill all criteria for consciousness. Slipping to an unconscious state may go undetected without careful monitoring.

There were no signs of respiratory difficulties among patients managed with only nitrous oxide and oxygen. None of these children exhibited hypoxemic episodes, and the lowest SaO_2 recorded during treatment was 98 percent. These results certainly do not preclude the development of respiratory difficulties in patients managed only with nitrous oxide and oxygen. Although a weak anesthetic agent, nitrous oxide can produce unconsciousness which, like that produced by the chloral hydrate regimen, may go undetected without careful monitoring. For the purposes of this study, the concentration of nitrous oxide was standardized at 40 percent. In actual practice the concentration of the agent is titrated until the desired level of patient sedation is reached. Titration with appropriate monitoring may help the dentist avoid placing the patient in an unconscious plane, especially when nitrous oxide is used as an adjunct to other pharmacologic agents.

Study group selection was based on the patients' behavioral ratings at the initial examination visits according to the categories defined by Frankl, Shiere, and Fogels.[6] Management of the *definitely negative* or *negative* patients was considered successful only if less than 10 percent of behavior ratings were scored as interfering. Based on these arbitrarily conservative criteria, eight of twelve patients in the chloral hydrate group and ten of twelve in the nitrous oxide group were managed effectively, and the sedation techniques, therefore, appear adequate for the patients selected. Nevertheless, a further effort to refine selection criteria, especially for patients potentially to receive chloral hydrate, was retrospectively attempted. Identification of behavioral patterns associated with ineffective management, however, was unsuccessful.

It should be noted that the two study groups were not homogeneous, and any comparison of results must be made cautiously. Patients selected for the nitrous oxide group had a mean age one year, nine months (1.75 years) greater than those of the chloral hydrate group. This was not a surprising observation, since one would expect older children to be less apprehensive about dental treatment and to require a milder means of pharmacologic management. Nevertheless, a larger population from which to select appropriate patients, if available, may have yielded more homogeneous groups.

SUMMARY

The results of this study showed that children sedated with 50 mg/kg chloral hydrate and 25 mg hydroxyzine in conjunction with 40 percent nitrous oxide and 60 percent oxygen may have respiratory difficulties which are not detected by the traditional monitoring of blood pressure, pulse, respiratory rate and skin color. The use of a pulse oximeter that noninvasively measures oxygen saturation of hemoglobin (SaO_2) allowed earlier detection of respiratory distress, when correction was a relatively simple matter of readjusting head position. This combination of sedative agents was effective in managing two-thirds of the patients originally classified in a behavioral category of *definitely negative*.[6] No children sedated with only 40 percent nitrous oxide and 60 percent oxygen exhibited respiratory problems, and over 80 percent of the patients classified in behavioral category of *negative* were managed effectively with this technique.[6]

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PRINCIPLES OF RADIOGRAPHIC EVALUATION OF MAXILLOFACIAL INJURY: INDIRECT SIGNS

Indirect signs of injury evident on radiographic studies should alert the examiner to the possibility of skeletal injury. Because most of the indirect signs of injury may also be found in association with sinus inflammatory disease, these findings only suggest the presence of possible injury.

If blood fills a maxillary sinus after injury, the sinus will become opaque on radiographic examination. Incomplete filling of the sinus results in an air-fluid level between the blood accumulation and residual air. If blood is trapped under a sinus mucosal surface it may produce a dome-shaped density similar to that produced by intrasinus cysts or polyps. Soft-tissue swelling produced by edema or hematoma may produce an area of increased density on plain films. Computed tomography is especially helpful in locating soft-tissue changes, especially in the periorbital and retrobulbar area.

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Philadelphia: Lea & Febiger, 1988, p 46.

Trauma

Dentofacial trauma in children

Marilyn S. Harrington, RDH, PhD
Adele B. Eberhart, RDH, MS
Jane F. Knapp, MD

BACKGROUND

Injury is the leading cause of death in the United States for people one to forty-four years of age.[1] Injury is also a leading cause of physician contacts, emergency room visits, disability and disfigurement, and years of productive life lost.[1] Given the magnitude of this public health problem, little is known about most injuries, especially nonfatal injuries. Trauma to the face, teeth, and oral soft tissues can result in physical and emotional complications. Like most other injuries, little is known about trauma to the face and the hard and soft tissues of the oral cavity. Most of the literature on dental injury pertains to the classification, incidence, prevalence, and treatment of fractured teeth.

Available information concerning the different types of dental injuries seems to vary with the site in which the study was completed.[2-3] The more severe dental injuries in children, such as luxations and bone fractures, are seen in the hospital setting, while fractured teeth are seen more frequently in health clinics, school programs and dental offices.[2-9]

The frequency of a particular type of injury can also vary with the age of the child. Displacement of teeth is typical in the primary dentition, while crown fractures

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are more common in the permanent dentition. Authors attribute this difference to the softness and resiliency of bone at an early age and the ability of bone and periodontal ligament to absorb more energy.[2,3,5,6,8]

An increased frequency of dental injuries in children has been reported in three different age-groups: one to three years old; seven to ten years old; and sixteen to eighteen years of age.[3,4,6,7,12,13] Among school-aged children, the eight-to- ten-year-old group has been reported to be at greatest risk for tooth fractures.[7-12]

Among school-aged children with coronal fractures, males accounted for more of the fractures than females.[2,6,10,14] Injuries to the primary dentition, however, did not vary according to sex.[2,3,8]

In addition to tooth fractures, facial, oral, and other dental injuries of interest to the dental professional have been reported. Such injuries included luxations, subluxations, avulsions, intrusions, soft tissue lesions, and fractures of alveolar and facial bones.[3,8,9,16,17] Rowe in a review of 1500 facial fractures found 4.8 percent were in children under eleven years and only 1 percent were in children under the age of five.[18] Among 1,420 patients who had facial fractures, Van Hoof *et al*, found 8 percent had occurred in the zero-to-ten-year-old age-group and 30 percent in the eleven-to-twenty-year-old group. Other than nasal bones, the most common facial bone fracture in children is the mandibular fracture.[22,23]

Falls have been reported as the leading cause of dentooral injuries during childhood.[3,4,9,14,15] Falls during early childhood, among the one-to-two-year-old children, can be accounted for by the instability and experimentation of the young toddler.[4] Aggressive and risk-taking behaviors have been cited as contributing factors in some injuries.[3] Other primary causes of orofacial injuries included automobile crashes, falls from or collisions on bicycles, sports, electrical burns, animal bites, and child abuse.[9,14-16,18,21,22] It has been reported that trauma to the head and associated areas occurs in 50 percent of child abuse cases with soft tissue injuries, such as bruises, being the single most common injury in child abuse.[18]

PURPOSE

The causes and circumstances surrounding dentofacial injuries in children need further understanding; epidemiological data, for example, can assist the dental profession in identifying appropriate preventive measures. The purposes of this study, therefore, were to:

- ☐ Describe and compare children presenting with dentofacial injuries to children presenting for treatment of all types of injuries.
- ☐ Describe the circumstances surrounding the leading causes of dentofacial injuries.
- ☐ Identify the use of pediatric dentists and oral surgeons in the acute treatment of dentofacial injuries by hospital emergency department personnel.

METHODS

This study was conducted at Children's Mercy Hospital, a privately owned, not-for-profit, tertiary-care hospital located in Kansas City, Missouri. The hospital is solely devoted to children and serves seventy counties in Missouri and Kansas and is the pediatric teaching hospital for the School of Medicine at the University of Missouri-Kansas City. The Advanced Pediatric Dentistry Program offered by the UMKC School of Dentistry is physically housed in and cooperatively financed by the hospital. In 1985, Children's Mercy Hospital developed and implemented the CHARTS: Injury Surveillance System. This system was designed to monitor the preevent, event, and postevent circumstances surrounding an injury event, Information stored in the computerized, on-line system was obtained directly from the patients' medical records. To standardize the data abstracted from the medical records, the International Classification of Diseases, 9th Revision (ICD-9-CM) and the Application of International Classification of Diseases to Dentistry and Stomatology, (ICD-DA) were used.[25,26] A specially designed form (Figure) was used by emergency department staff to elicit specific environmental circumstances surrounding the incident and such data were entered into the system. Data from CHARTS are reported in this study.

In 1986, 7,283 children presented to the emergency department as a result of an injury. Of that total population, 501 involved dentofacial related injuries. The dentofacial injuries included in this study are identified below along with the ICD/ICD-DA code numbers:

Figure. International classification of diseases (ICD) and its application to dentistry and stomatology (ICD-DA).

Injury	ICD/ICD-DA codes
Extrusion/Intrusion (includes Luxation)	525.1;525.8; 525.88; 873.66; 873.67
Avulsion	873.68
Open wound to lip or jaw	873.43; 873.44; 873.53; 873.54
Internal wound to the mouth	873.60; 873.61; 873.62; 873.64; 873.65; 873.70; 873.71 873.72; 873.74; 873.75; 935.0
Fractured teeth	873.63; 873.73
Burns to the mouth	941.03; 941.13; 941.23; 941.33; 941.43; 941.53; 947.0
Fractured maxilla/mandible	802.0; 802.2; 802.20; 802.25; 802.30; 802.4; 802.40 25,26

Table 1 □ Comparison of all injuries vs. dental injuries by sex.

	Male		Female		Total	
	N	(%)	N	(%)	N	(%)
All injuries	4270	(59)	3013	(41)	7283	100
Dental injuries	304	(61)	197	(39)	501	100

RESULTS AND DISCUSSION

The sex and racial characteristics were similar for both the dentofacial injury population and total injury population. Males comprised approximately 60 percent of both injury groups (Table 1). Seventy-five percent of the children with dental injuries who required admission to the hospital, however, were males, which indicates that males sustained more serious injuries than females. Over half of the children were black, 40 percent white, 4 percent hispanic, and less than 1 percent Asian and Indian children (Table 2). The fact that most injured children reporting for treatment at Children's Mercy Hospital were black is consistent with the urban physical location of the hospital and the total population served by the hospital. Many of the families immediately surrounding the hospital use it as their primary source for all pediatric medical and dental care. When looking at the more seriously injured child who requires admission, the racial distribution changes with an equal distribution between white and black children. Also, over two-thirds of the presenting handicapped children were white.

The age composition of the children with dentofacial injuries differed slightly from the total injured population. As shown in Table 3, dentofacial injuries were seen more frequently in younger age-groups. Seventy percent of the children with dental injuries were six years of age and under, compared to 54 percent of all injuries occurring in that age-group. In this study, as well as in previous studies, the peak incidence of presenting injuries was in the one-to-two-year-old age-group.[5,6,12,14] It is not surprising that very young children experience dentofacial injuries due to the active but unstable nature of toddlers.[6,9]

In this emergency department population, the most frequent injury was open wound to the mouth, accounting for over 60 percent of the reported injuries. Consistent with previous literature, displacement of teeth occurred frequently in younger age-groups (Table 4). Some authors have indicated that the supporting structures (alveolar bone and periodontal ligament) in the primary dentition are resilient, thereby favoring dislocations rather than fractures.[2,6,8,12] In this study, however, 42 percent of the fractures occurred in the primary dentition. Mandibular or maxillary fractures occurred primarily in children thirteen years and over (48 percent), but there were occurrences in six-and-under age-groups (22.5 percent), much higher than previously reported.[4] The maxillary and mandibular bone fractures were caused by, in descending order, fights, assaults, falls, motor vehicle accidents, pedestrian and bicycle incidents. The increased incidence of tooth fractures in very young children may be due to the fact that this study was conducted in a hospital setting, where injuries tend to be more serious.[2]

The seven types of dentofacial injuries were collapsed into the three categories of hard tissue injury, soft tissue injury, and maxillary and mandibular fractures and then ten leading causes of those injuries were placed in rank order (Table 5). Severity of injury was not taken into consideration. Falls continued to be the leading cause of dental injury. Of interest is the fact that intentional injury (fights, assaults) was one of the five leading causes of dental injuries in this pediatric population.

As is well known, most injuries occur in the home. The same was true in this study, since 62 percent of them occurred in the home. Street and highway (10 percent) along with public buildings (9 percent) accounted for another 19 percent of the primary locations where dental injuries occurred. Schools and day care centers accounted for 92 percent of the public buildings where injuries occurred.

Forty (8 percent) of the children with dental-related injuries required admission to the hospital. Likewise, 8 percent of the children with any type of injury required admission. The leading cause of injury for both groups was falls. The surface of impact in which falls occurred was a hard surface such as concrete, asphalt, handle bars of bicycles, tile, and wooden gymnasium floors. As indicated in Table 6, the three leading causes of injury which resulted in admission for the dental-injury group were falls, MVAs, and pedestrian incidents with the leading diagnoses being maxillary and mandibular fractures, open wounds to the mouth, and burns of the mouth. The number of admissions for black and white children was the same. Of the 40 children admitted to the hospital with dental-related injuries, eighteen (45 percent) were black and eighteen were white.

Of the 7,283 children presenting with some form of injury, 308 (4 percent) had a mental and/or physical handicap or both. Of the 501 children with dental injuries, eighteen or (3.5 percent) had a mental or physical handicap. It is estimated that 1 percent of the children in the United States have handicaps; it appears, therefore, that children with handicaps utilize emergency department services more and/or are at somewhat of a higher risk for injury than children without handicaps. The most common handicaps in this population were seizure disorders, cerebral palsy, mental retardation, hemophilia, diabetes, behavioral problems, and sickle cell anemia. Sixty-seven percent of the children with hand-

Table 2 □ Comparison of all injuries vs. dental injuries by race.

	Black	White	Hispanic	Asian	Indian	Unknown	Total
All injuries	3927(54)*	2943(40)	282(4)	21(<1)	4(<1)	106(1.5)	7283 (100)
Dental injuries	273(55)	201(40)	20(<1)	2(<1)	—	5(<1)	501 (100)

*Number in parenthesis is row percent.

Table 3 □ Distribution of all injuries vs. dental injuries by age.

Age	All injuries		Dental injuries	
	N	%	N	%
1	510	(7)	26	(5)
1-2	1543	(21)	151	(30)
3-4	1061	(15)	101	(20)
5-6	811	(11)	72	(14)
7-8	793	(11)	49	(10)
9-10	701	(10)	28	(6)
11-12	720	(10)	29	(16)
13-14	710	(10)	30	(6)
15-over	434	(5)	15	(3)
Total	7283	(100)	501	(100)

Table 4 □ Frequency of type of injury by age category.

Type of injury	Age									
	1	1-2	3-4	5-6	7-8	9-10	11-12	13-14	15+	Total
Avulsion	2	4	1	0	1	0	0	0	1	9
Burns	1	10	1	1	2	1	1	0	0	17
Max/Mand. Fracture	0	0	6	1	3	2	4	10	5	31
Tooth fracture	0	10	2	2	8	6	3	1	1	33
Ex/Intrusion	2	6	8	10	0	2	2	4	1	35
Internal wound	9	20	12	12	7	1	4	0	0	65
Open wound	12	101	71	46	28	16	15	15	7	311
501										

icaps were white and 22 percent were black, which is a reverse distribution from the total injury and dentofacial injury population.

Pediatric dentists, who are on call twenty-four hours a day at Children's Mercy Hospital, were asked to consult on a case by emergency department personnel for 50 (10 percent) of the children with dentofacial injuries and oral surgeons were consulted on ten cases. The oral surgeons were consulted only when there was a maxillary or mandibular fracture and pediatric dentists were primarily consulted on injuries that resulted in fractured, avulsed, or loosened teeth and not soft tissue injury.

Finally, other circumstances surrounding the injury incidents were identified. Almost half (47 percent) of the children were unsupervised during the incident, and for children between the ages of four and nine, 66 percent of the incidents occurred with no supervision present. Of the children with dentally related injuries, 196 (39 percent) were reported to be involved in risk taking or aggressive behavior and 85 percent of those behaviors were exhibited when there was no supervision. Of the nineteen children receiving an injury as a result of a motor vehicle crash, only one was restrained in a safety belt. None of the forty-one children involved in bicycle accidents was wearing a helmet at the time of the injury.

Table 5 □ Rank order of dental-injury incidents by dental-injury categories.

Incident	Hard tissue	Soft tissue	Max/Mand Fracture	Total	
	N	N	N	N	%
Falls	41	229	7	277	(55%)
Struck	7	47	2	56	(11%)
Bicycle	14	25	2	41	(8%)
Assult	4	20	11	35	(7%)
MVA	6	8	5	19	(4%)
Nature	—	15	—	15	(3%)
Non-fire burns	—	11	—	11	(2%)
Sports	2	6	—	8	(2%)
Pedestrian	1	3	3	7	(1%)
Child abuse	1	6	—	7	(1%)
Other	2	22	1	25	(5%)
	78	392	31	501	99%

Table 6 □ Children admitted with dental facial injuries by cause and diagnosis.

Incident	N	%	Diagnosis	N	%
Falls	11	28	Max & mand fracture	15	38
MVA	8	20	Open wound	9	23
Pedestrian	4	10	Burns of mouth	8	20
Burns	3	7	Internal structures	6	15
Fight	3	7	Other	2	4
Other	11	28		—	—
	40	100		40	100

CONCLUSIONS

In summary, the major findings include:

- □ The demographic characteristics of children experiencing dentofacial injuries were similar to children experiencing all types of injury.
- □ Children six years of age and under were at high risk for dentofacial injury, with the one-to-two-year-old being at the highest risk.
- □ Pediatric dentists and oral surgeons were consulted on only 12 percent of the dentofacial injuries and only for hard tissue trauma.
- □ Children with physical or mental handicaps appeared to utilize emergency department services more, and/or are at greater risk for injury than children without handicaps.
- □ Most of the injuries occurred at home and of public places, schools and day care centers provided a more common environment for injury to occur.
- □ Almost half of the dental injuries occurred when children were unsupervised and between the ages of four and nine; 62 percent of the children were unsupervised when their injuries occurred.
- □ Risk-taking and aggressive behavior were associated with more than a third of the dentofacial injuries and most of those behaviors were exhibited when there was no supervision.
- □ The five leading causes of dentofacial injuries, regardless of severity, were falls, being struck by an object, bicycle incidents, assault, and motor vehicle accidents.

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Dental emergencies presenting to a children's hospital

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Children's Hospital of Buffalo is a private hospital affiliated with the State University of New York at Buffalo Medical and Dental Schools. The hospital provides maternal, neonatal, pediatric, and adolescent care to patients from a wide geographic area around the eastern Great Lakes. The hospital has an accredited pediatric dentistry program, where four residents are in training under the supervision of full-time and part-time faculty from the State University of New York at Buffalo School of Dentistry. The Dental Department of Children's Hospital provides complete dental services to children and young adults. Emergency services are provided to patients presenting to the hospital on a twenty-four-hour-a-day basis, every day of the year.

This paper presents data on the number and type of dental emergencies presenting to the hospital during the evenings, weekends and holidays.

METHODS

Data were collected from all patients requiring after-hours dental emergency services at Children's Hospital of Buffalo. These patients were evaluated and treated by four pediatric dental residents during the period from July 1, 1983, to June 30, 1984. After-hours emergency patients were those patients presenting for dental care outside of the normal clinic hours of Monday through Friday, 8:30 AM to 4:30 PM.

Diagnosis and treatment of the patients were based on Emergency Hospital Dental Service guidelines set forth by the American Society of Oral Surgeons and the American Dental Association.[1]

RESULTS

During the period described, a total of 222 patients presented for after-hours dental emergency service. Information on sex and age distributions of the patients is presented in Table 1 and Figure 1.

The age of the patients ranged from one hour to thirty-one years. The distribution for both males and females was concentrated in the range one to eleven years of age, with a decrease of frequency between four to seven years in females and five to eight years in males.

Further examination of the data revealed seasonal variations in the number of patients presenting for emergency dental care. There was a decrease from October through February in all types of emergencies, with the largest decrease evident in trauma-related emergencies. Figure 2 presents a monthly distribution of the number of patients categorized according to etiological

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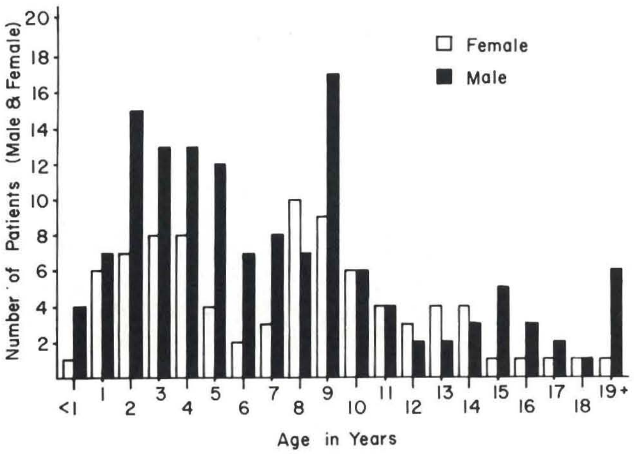

Figure 1. Number of patients versus age and sex.

Table 1 □ Number of patients presenting for emergency dental services, divided by sex and race.

Male/white	105	Female/white	51
Male/black	25	Female/black	28
Male/other	7	Female/other	6
Total male	137	Total female	85
Total number of patients = 222			

Table 2 □ Other etiological factors, listed in order of frequency.

9	failure of existing restoration
8	breakdown of orthodontic appliance
5	normal exfoliation
4	sequelae of untreated trauma
3	no definitive diagnosis
2	neonatal tooth
1	primary herpetic gingivostomatitis
1	mumps

factors, i.e. trauma, caries and other. Table 2 lists reasons for presenting for emergency care other than trauma and caries.

A distribution of the teeth involved in the emergency situations and the primary etiological factors related to each type of tooth is depicted in Tables 3A and 3B. Analysis of the tables reveals that of the permanent teeth involved, maxillary central incisors comprised 58.3 percent (91 of 156) and maxillary central and lateral incisors comprised 75.6 percent (118 of 156). Also, of the primary teeth involved, maxillary central incisors comprised 55.6 percent (85 of 153) and maxillary central and lateral incisors comprised 73.8 percent (113 of 153).

Combining the figures on permanent and primary teeth yielded a total of 309 teeth involved in emergency situations. Of this total, maxillary central incisors comprised 57.0 percent (176 of 309), with maxillary incisors overall yielding 75.0 percent (231 of 309).

The overwhelming majority of the dental emergency situations observed involved the eight incisor teeth (86.1 percent). Further emphasis will be placed on the condition of these teeth.

Table 4 shows the distribution of the various condi-

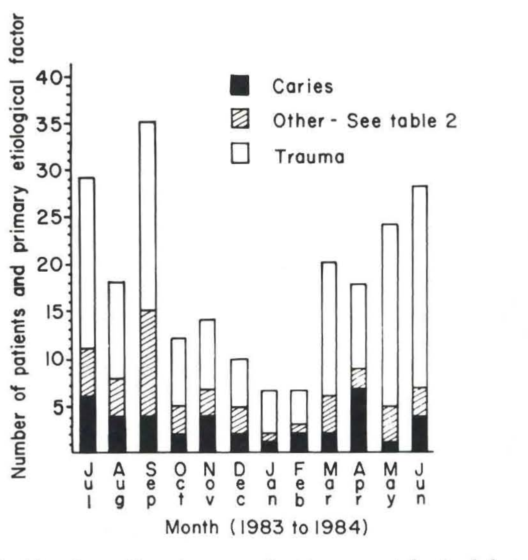

Figure 2. Number of patients and primary etiological factor versus month.

The authors would like to thank Dr. Kenneth R. Banas; Chi F. Chan; and Michael K. Conley for their assistance in collecting data.

Table 3 A □ Distribution of permanent teeth by primary etiological factor.

Other								1	1			1				
Caries			2											2		
Trauma						1	15	47	42	12						
	1	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**	**11**	**12**	**13**	**14**	**15**	**16**
	32	**31**	**30**	**29**	**28**	**27**	**26**	**25**	**24**	**23**	**22**	**21**	**20**	**19**	**18**	**17**
Trauma						1	4	10	9	4						
Caries														3		
Other														1		

Table 3 B □ Distribution of primary teeth by primary etiological factor.

other	2	3			3	1	1			1
caries	1		1	4	5	7	4		3	2
trauma		1	3	7	34	35	12	1		
	A	**B**	**C**	**D**	**E**	**F**	**G**	**H**	**I**	**J**
	T	**S**	**R**	**Q**	**P**	**O**	**N**	**M**	**L**	**K**
trauma				1	3	3	1			
caries	4	7							2	5
other	2	1								3

Table 4 □ Maxillary and mandibular distributed by presenting condition.

Primary	fracture (enamel)	Fracture (enamel, dentin)	Fracture enamel, dentin, pulp)	Fracture (root)	Mobile, but not displaced	Displaced	Partial avulsion	Complete avulsion	Intruded	Caries related
Maxillary										
D						1		4		4
E	1	1	4	3	4	8	2	7	4	9
F		6	3	3	7	7	3	2	6	11
G				1	5	1	1	1	3	5
Mandibular										
N						1				
O					1	1		1		
P					1	1				1
Q						1				
Total Primary	1	7	7	7	18	21	6	15	13	30
Permanent										
Maxillary										
7	1	4	1		6	1	3	2		
8	4	24	4	5	7	5	7	5		1
9	3	20	10	3	7	5	6	2	1	2
10	3	4		1	4	1				
Mandibular										
23		1			2	1				
24	1	2	1		3	1		1		
25	3	1	1		4			1		
26	1	1			2					
Total Permanent	16	57	17	9	35	14	16	11	1	3
Total	17	64	24	16	53	35	22	26	14	33

tions of the primary and permanent, maxillary and mandibular incisors treated during the study. If one tooth presented with more than one of the conditions listed, all such conditions were listed separately; for example, a subluxated maxillary incisor with a fracture involving enamel and dentin would be listed under both conditions.

Table 4 shows that the most commonly observed conditions of primary incisors were subluxation and displacement. The most commonly observed conditions of permanent incisors were coronal fractures, subluxation and displacement.

DISCUSSION

Dental emergencies can pose a difficult treatment situation for patient and dentist. This may be most obvious with a child who has not received previous dental care. Although most private-practice dentists have provisions for treating emergencies during office hours, many do not provide for after-hours emergency treatment. Patients, or their parents, facing such emergency situations must often seek treatment outside the private sector. Often these patients are referred to a hospital emergency room, especially to those hospitals providing dental services.

The provision of emergency dental care is a requirement of all hospital dental departments, according to the definition of a hospital dental service clarified by the Council on Hospital and Institutional Dental Services.[2]

A definition of emergency dental care has been proposed by the Society of Oral Surgeons:

"Emergency dental care is the management or treatment of hemorrhage, upper airway impairment, trauma, infection, or acute inflammation involving the oral and maxillofacial structures (including teeth and dentoalveolar processes) which threatens the person's life or substantially impairs the functioning of such structures."[1]

The American Society of Oral Surgeons further proposed a number of treatment areas involving emergency dental care. These areas are similar to those in the American Dental Association definition of Emergency Dental Care Services.[1]

Health care providers involved in the provision of emergency dental treatment, however, are likely to realize that not all dental patients presenting for emergency care can be categorized in accordance with the above definitions. DeLuke emphasized this issue by stating that "...the overriding factor is not whether the problem is determined to be a true emergency or not...but rather what the patient himself or his family believes is an emergency and what the community expects and anticipates that the hospitals located within it will provide."[1]

As a case in point, few dentists would feel that an uncomplicated normally exfoliating primary central incisor presents a dental emergency; the parent who insists, however, that the exfoliating tooth is a permanent incisor views the situation differently.

It appears that the patient or, as in the present study, the parent, usually determines when to seek emergency dental care. This may result in a possibility of abuse, or at least misuse, of the availability of emergency dental services by those, who for any number of reasons, do not receive regular dental care.

This was not the case, however, during the period of this study. For the majority of patients treated, the primary etiological factor was trauma. All such cases were considered to be true emergencies.

Of the remaining patients seen, most presented with acute pain or swelling secondary to the primary factor, such as caries, resulting in the emergency situation. Although most of these situations would likely have been prevented with routine dental care, the presenting condition of the patient would usually be described as one in need of 'emergency dental care' by all involved, i.e., patient, parent and dentist.

Thus, although the potential for misuse or abuse does exist, our data seem to concur with previous reports of emergency dental services, which indicate that non-emergency utilization of dental emergency facilities does occur, but not to a major extent.[3-5] Berger further comments that it would be difficult to eliminate such abuses without affecting the overall service.[3]

In other areas, interpretation of the data obtained concurs with a previous report on traumatic injuries to children's teeth.[6] Similarities were noted in the distributions of patients by sex, and in distributions of teeth injured, and of presenting conditions of the teeth. Although age distributions were similar, current results indicate a greater proportion of patients under the age of five years.

Another slight difference between past results and the present study, is the previously noted decrease in number of patients presenting between the ages of five to seven years in females and six to eight years in males.[6] In a more recent study, Galea describes similar distribution of traumatic dental injuries with peaks between one to three years and seven to ten years of age.[7] Berkowitz describes another peak at sixteen to eighteen years of age, which was not observed in this study.[8] The present data indicate a decrease in the numbers of patients older than eleven years. This decrease may be related to the availability of emergency dental care at three nearby hospital dental services provided, in part, by oral surgery and general practice residents.

It appears that the decrease in number of patients noted between five to eight years of age might be explained by the normal exfoliation of primary anterior teeth in similar age ranges.[9] Since the majority of patients requiring treatment presented with trauma to anterior teeth, the absence of such teeth in the child population in general would decrease the number of teeth likely to be injured.

Other similarities were also noted between the present study and a recent report on traumatic dental injuries.[7] The most common traumatic injuries to both the primary and the permanent dentitions were parallel in both reports. In the previous study, subluxation followed by displacement were the most common injuries to the primary dentition; the present study found displacement followed by subluxation (mobile, but not displaced) as the predominant injuries. Both studies report that in the permanent dentition, uncomplicated crown fracture followed by subluxation were the most common injuries.

Comparison of present data to reports on emergency dental care for the general population yields one major difference.[3-5] Whereas the majority of children's dental emergencies observed were related to trauma to anterior teeth, the most common presenting complaints in the general (adult) population were related to posterior teeth, with dental disease being the primary etiological factor.

CONCLUSION

In summary, present findings indicate that provision of emergency dental care for children is a unique and vital part of hospital dental care and of dentistry in general. The majority of the treated cases involved traumatic injuries to anterior teeth. It was also our finding that although abuses of the services provided were minimal, unfortunately, it is difficult, if not impossible to prevent.

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The effect of dental probing on subsequent enamel demineralization

Clinic

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For centuries, caries diagnosis has been performed by mirror and explorer. In spite of this long tradition, standardization of this diagnostic procedure seems virtually absent. The many ways in which caries becomes manifest, the quality and use of the diagnostic instruments, and the interpretation by the dentist are highly complex variables that jeopardize attempts to rationalize caries diagnosis. Caries of the fissures develops shortly after tooth eruption, when the enamel of the fissures as well as of the cervical region is still relatively weak and immature.[1] Especially when oral hygiene and dietary habits are caries-promotive, demineralized enamel can be observed as a white belt in the cervical region.[2] These white spots, however, can remineralize completely, provided that no mechanical damage has occurred.[3] In principle, the same applies to fissures, although the possibility of attaining proper plaque control in this area is small and depends on the particular type of fissure.[4] The type of fissure and its degree of mineralization, however, cannot be determined radiographically, leaving the use of the explorer the most prominent diagnostic method of fissure-caries detection.[5-7]

Recently, a number of studies have been published that show the deleterious effects of the use of the explorer.[8-12] The explorer has been shown not only to act as

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a vehicle of transfer of cariogenic microorganisms to not yet infected fissures, but also to cause mechanical damage to enamel already softened as a result of a carious attack. The degree of damage to the enamel structure depends on factors such as the quality of the tool (i.e. the shape of the tip of the explorer), the way (force and thoroughness) by which it is manipulated, and by the degree of mineralization of the enamel.[13] By using an explorer on demineralized fissures, an entrance may be created through which cariogenic microorganisms and their metabolic products can directly penetrate into the softened subsurface. The aim of this study was to quantify *in vitro* the effect of probing on the subsequent rate of formation and growth of lesions.

MATERIALS AND METHODS

The experiment was completed on single sections of bovine enamel.[14,15] These sections were provided with grooves and exposed to a demineralizing solution in order to create fissure- caries-like lesions. Half of the grooves on each section were probed after five weeks demineralization. To obtain an answer to our starting hypothesis, whether or not probing contributes to the rate and extension of demineralization in presoftened enamel, demineralization was continued for two more weeks. The lesion depths of the probed and nonprobed fissures were registered by microradiography to quantify a possible difference. Labial enamel slabs measuring 1 cm^2, were cut from bovine incisors. The slabs were embedded in cold-curing PMMA resin (Vertex (R)). The enamel was flattened and polished with silicon carbide abrasive paper (Gritt 600), removing up to 300 μm from the surface. Next the slabs were placed with the polished enamel perpendicular toward the diamond wheel of a sectioning machine, and sections were cut measuring 400 μm in thickness. The sections were placed under the stereomicroscope and sealed off individually using cellulose varnish. After the varnish had been fully dried, the sections were regrouped and clasped with the remaining end blocks in their original order with the (cellulose covered) enamel side facing the diamond wheel perpendicularly. Four standardized grooves were then cut in the block, i.e. in each section, measuring the width of the diamond wheel: 250 μm, at $\pm$ 450 μm depth, and at 1500 μm intervals. Ten sections were prepared in this way and placed in separate aliquots of 1 ml of 0.1 N lactate buffer at pH 4.5, containing 0.2 mM methylenehydroxydiphosphonate (MHDP) and stored at 37°C. The progression of demineralization was monitored using the technique of contact microradiography after one, three, five, six and seven weeks of demineralization, respectively; the experimental conditions used to make the microradiograms were modified because of the thickness of the specimens.[16-20] Regular X-ray film (Kodak Ultra-speed safety 2) was used and the X-ray source was operated at 20 KV and 20 mA for forty seconds. After five weeks of demineralization, two grooves, randomly chosen, per section were probed at a force of 0.5N (0.5 kgf)(Figure 1). For this purpose, a dental explorer was trimmed down to make the tip measure no more than 220 μm in diameter over a length of $\pm$ 600 μm. An additional set of microradiograms was taken before as well as immediately after probing to detect any direct damage caused by the explorer. Lesion depths were measured by projecting each of the microradiograms at 50x magnification, and the depths values were measured from the bottom of the groove perpendicular towards the dentinoenamel junction (Figure 2).

RESULTS

The microradiograms show a sharp demarcation line for each lesion, allowing accurate measurements. Regarding the progress of demineralization we found a correlation between demineralization time and progression of the enamel lesion, in both horizontal and vertical directions. The surface enamel was sealed off from the environment, and no demineralization originating from the surface could be observed. Before probing, the demineralized grooves showed a higher degree of radiopacity at the surface than at the subsurface. The mechanical damage brought about by the explorer could be observed on the microradiograms as an area of tip-shaped destruction. The measured lesion depth, plotted as a function of the time of demineralization are presented in Figure 3. The shaded bars represent the demineralization of the grooves, which were probed after five weeks of demineralization. These results show that a week after probing, a significant difference between the lesions in the probed and nonprobed fissures was measurable. In some of the probed fissures, the area of destruction had even reached the dentinoenamel junction, an observation that was never made in the nonprobed fissures. Two weeks after probing, the difference was even more pronounced: in almost all the probed fissures, demineralization had penetrated into dentine, whereas demineralization in the nonprobed fissures had not even reached the dentinoenamel junction. An example of this finding is shown in Figure 4, where the observation is exaggerated, due to the accidental difference in enamel thickness between probed and unprobed grooves. The difference between the lesion depths was statistically significant as shown by a

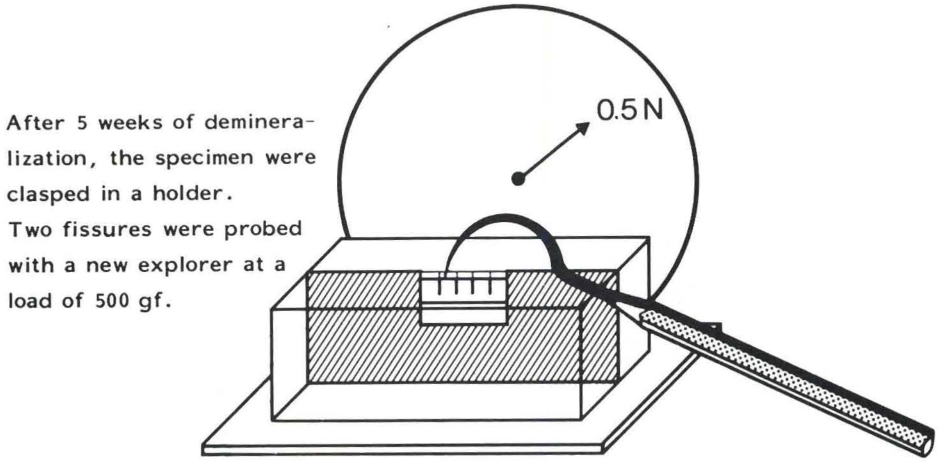

Figure 1. Experimental set-up showing the enamel sections with standardized grooves being probed at constant force on a weighing balance.

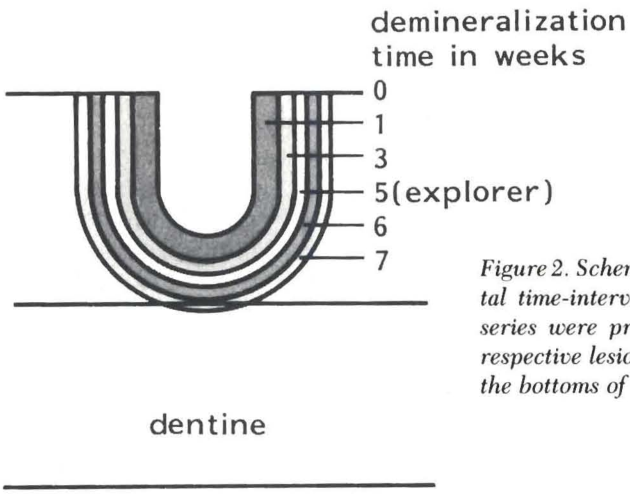

Figure 2. Schematized appearance of sections after experimental time-intervals. The respective microradiograms of each series were projected at fifty times magnification, and the respective lesion depths were determined by measuring from the bottoms of the artificial fissures.

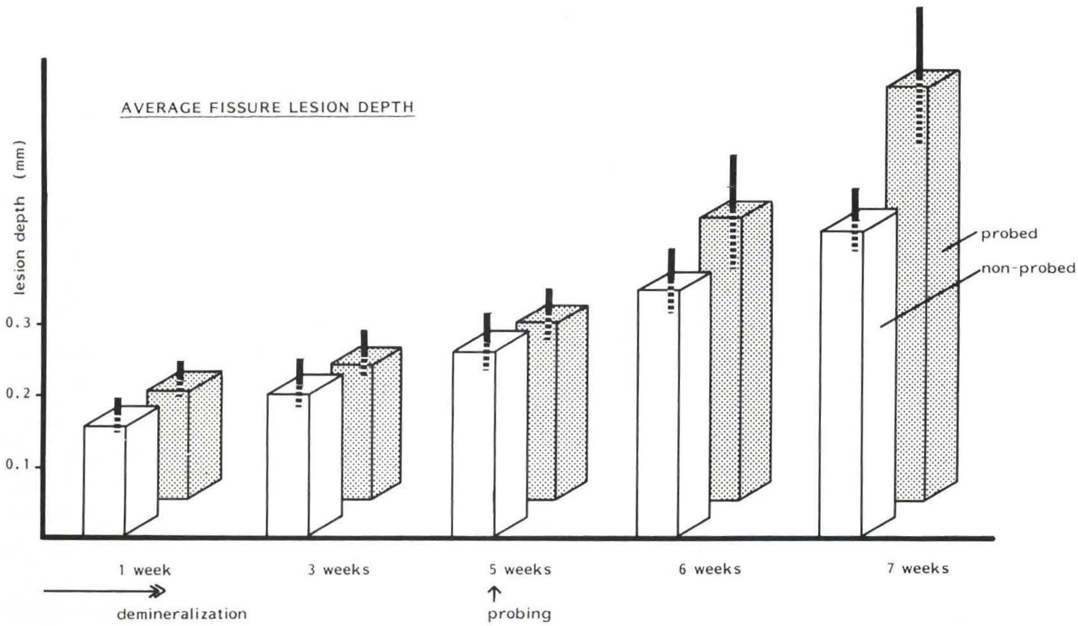

Figure 3. Lesion depth with time of experimentation for the probed and non-probed grooves. Standard deviations are given in the bars.

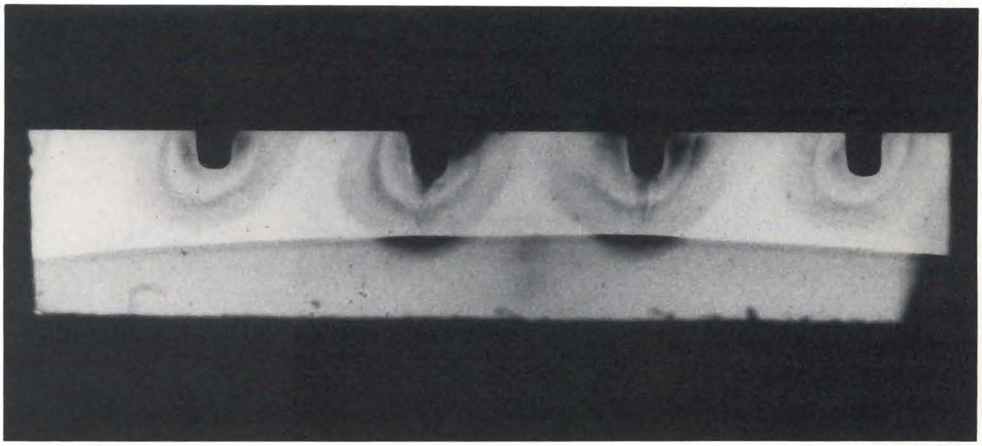

Figure 4. Microradiograph of section after 7 weeks of demineralization. For this particular section the two central grooves were probed after 5 weeks.

paired T-test comparison for the twenty probed and twenty nonprobed fissures (Table).

DISCUSSION

The present study clearly demonstrates that probing of artificial fissures with white spot lesions enhances the rate of subsequent tissue breakdown. And it might be presumed, that the use of an explorer for detection of fissure-caries during biannual check-ups contributes to the rate of decay, especially in the fissure area. The study was performed on bovine specimens. Demineralization and remineralization processes proceed more rapidly in bovine enamel as compared to human enamel, because enamel prisms, and more importantly, the interprismatic and intercrystalline spaces, are larger. Bovine enamel has the considerable advantage that larger specimens can be made, which implies that a number of fissures can be created in a similar tissue environment. The single section technique was chosen, as it is the most direct method to monitor changes occurring in one specimen.[14] As a modification to this method, we used thicker specimens, firstly because the demineralization tends to proceed faster and the surface behaves anomalously, when thin sections are used.[21] And secondly, because of the necessity to use sections with a width larger than the tip of the explorer. In artificial fissures, the prism orientation is different from natural enamel fissures, resulting in a rather homogeneous demineralization in all directions. In natural fissures there is a preferential demineralization of the fissure wall. This is a result of clogging of the fissure base with densely packed material, and the inaccessibility of this region to fermentation products from the plaque, rather than to intrinsic

Table □ Results of paired t-test on lesion depth of probed and non-probed grooves.

demineralization (weeks)	t value	p
1	0.1	
3	-0.6	
5	-1.1	
probing		
+1	3.2	<0.005
+2	7.5	$\ll 0.005$

enamel properties. With respect to the present study, this does not seem to impose a problem. During probing the exerted force can always be thought to be composed of a vector, parallel and at right angle to the enamel prisms. This applies, irrespective of the region of the tooth being probed, to either the fissure or smooth surface. Thus, the way in which the enamel prisms may suffer mechanical damage from probing of a fissure does not seem to depend on the type of fissure nor does it essentially differ from the type of damage on smooth surfaces.

A linear relationship between lesion depth and demineralization time in the bottom as well as in the walls of the grooves was previously demonstrated by Smits and Arends (1986), who used a comparable set-up to study fissure caries.[22] In our study, the lesions were about 200 μm deep after five weeks of demineralization; but probably the actual lesion front was deeper, due to the limited sensitivity of the photographic material.[23]

Particularly with respect to fissure caries, a critical reassessment of its diagnosis and therapy seems desirable. If plaque is removed, the naked eye is well able to detect lesions greater than 0.1 mm in diameter, an area which is comparable to the tip-size of a new explorer. Before caries is diagnosed, removal of plaque is advisable as a standard procedure. As enamel of newly erupted teeth is vulnerable to demineralization in its immature zones, such as fissure walls and cervical regions, mechanical loads easily cause damage. The use of an explorer in such zones should be discussed. The explorer often is used for plaque removal, but this might readily cause a transfer of plaque from infected enamel to uninfected enamel. Considering that probing, in various ways, may contribute to caries and caries progression, the question needs to be asked whether there is an acceptable alternative in caries diagnosis of the first region. Summarizing some available methods which may serve as alternatives to probing are:

- ☐ Visual inspection after plaque prophylaxis.
- ☐ Visual inspection with light sources that facilitate caries detection.
- ☐ The use of color solution for detecting caries.
- ☐ The use of fissure sealant in areas at risk. In cases of doubt, a mark on the patient's record will serve as a reminder for the next biannual check-up; and if initial caries was present, progression would make it more distinguishable then. In cases of doubt, however, a fissure sealant seems to be a safe therapy for preventing a progression of caries.[24]

CONCLUSIONS

The use of an explorer for probing demineralized enamel leads to mechanical damage of the tissue structures, and an increased rate of subsequent demineralization.

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A radiographic study of interproximal alveolar bone crest between the primary molars in children

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Periodontal disease in children is usually limited to marginal gingivitis, while more advanced forms of the disease are characteristic of the adult population.[1-3] Evidence of destructive periodontal disease in the primary dentition has been found, however, in 25.2 percent of a five-to-fourteen year-old child-population, and in 94 percent of extracted stained molars.[4-5] Evidence of periodontitis in the primary dentition may also be found related to systemic diseases, due to local trauma caused by habits or inadequate restorations; and in cases with juvenile periodontitis and/or prepubertal periodontitis.[2,6-9]

Several authors have described the usefulness of bitewing radiographs in the early diagnosis of incipient bone loss in the permanent dentition of teenagers.[10-13] On the other hand, information on the normal relationship between dental landmarks and the alveolar bone in the primary dentition is lacking. The purpose of the present study was to establish baseline data regarding the normal relationship between the cementoenamel junction and the alveolar crest in the primary dentition, using bitewing radiographs.

MATERIALS AND METHODS

The dental records of seventy-five children from the Jerusalem area were used in this study. From these, radiographs were selected based on the following criteria:

- ☐ Minimal evidence of distortion.
- ☐ Minimal overlapping between the proximal surfaces of the teeth.
- ☐ Clear image of the cementoenamel junction.
- ☐ No evidence of periodontal pathosis as determined by the presence of an intact lamina dura at the interdental crest.

After selection, one or both radiographs of the pair of bitewing radiographs from sixty-three children remained for measurement. There were twenty-eight boys and thirty-five girls whose ages ranged from three to eleven years (Table 1).

The radiographs were mounted in 35 mm slide frames and projected onto the screen of a Singer Clearmate Caramate II SP slide viewer (enlargement X7.2). On the projected image, measurements of the distance from the cementoenamel junction to the interproximal alveolar bone crest, in millimeters, were made at the interproximal site between the two primary molars, using dividers and a ruler. An attempt was made to measure the distance from the highest point on the occlusal surface to the cementoenamel junction; it was not possible,

Table 1 ☐ Distribution of children by age.

Age of child	3	4	5	6	7	8	9	10	11	Total
Number of children	11	8	6	8	2	7	11	8	2	63

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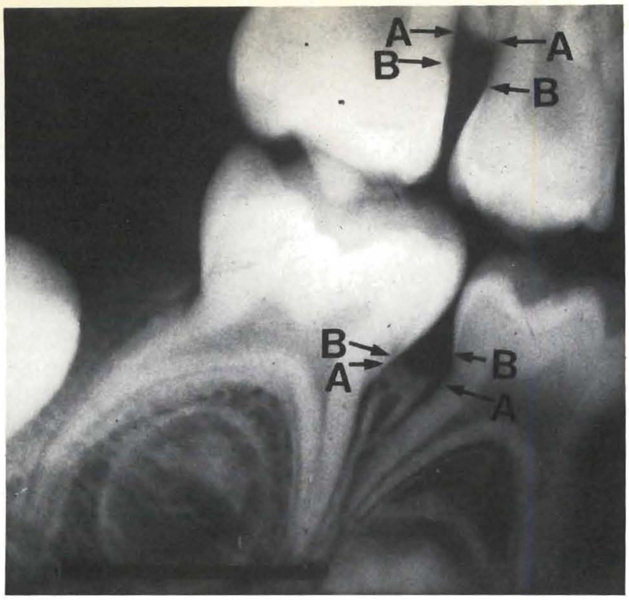

Figure 1. Bitewing radiograph of a five-year-old child in which the reference points utilized for the measurements are indicated. A = alveolar bone, B = cementoenamel junction.

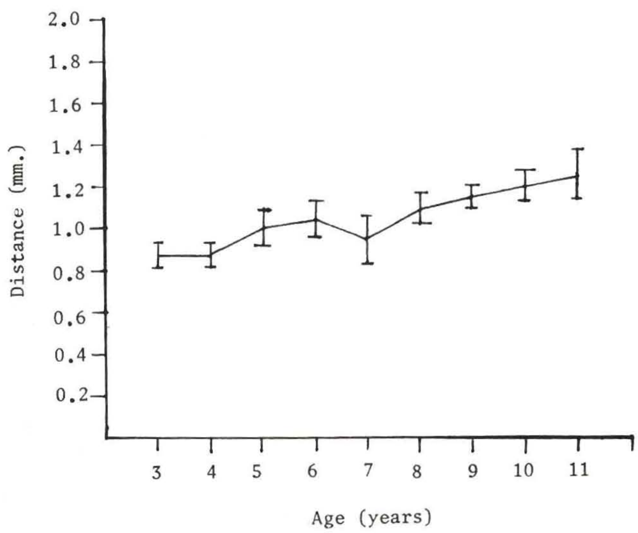

Figure 2. Graph showing the relationship between the age of the child and the distance from the cementoenamel junction to the interproximal alveolar bone, (r = 0.25, p<0.02).

however, to define clearly the former point. No further attempt was made, therefore, to use this measurement. The distance between the interproximal alveolar crest and the cementoenamel junction was measured on a total of 350 sites: 172 at the distal of the first primary molar and 178 at the mesial of the second primary molar (Table 2; Figure 1). All measurements were made by a single examiner.

The Student's T test was used to compare measurements between groups. The correlation between the patient's age and the distance from the cementoenamel junction to the alveolar crest was examined, using linear regression analysis from a standard computer software package. A significance level of 95 percent was chosen.

RESULTS

No significant difference was found between the mean ages of the boys (6.3 ± 2.4 years) and the girls (6.3 ± 2.6 years). One hundred and ninety-nine measurements from the cementoenamel junction to the alveolar crest were made in girls and 151 in boys; the mean distance for all the measurements from the cementoenamel junction to the alveolar crest was 1.01 ± 0.49 mm. The mean distance for the girls (1.04 ± 0.5 mm) was greater than for the boys (0.97 ± 0.5 mm); this difference, however, was not significant ($p<0.05$). The greatest distances between the cementoenamel junction and the alveolar crest were found at the distal surfaces of the mandibular first molars, with the mesial surfaces of the mandibular second molars showing the shortest measurements. No significant differences were found between left and right sides at the same sites (Table 2). The distance from the cementoenamel junction to the alveolar crest was greater at the distal surfaces of the first molars than at the mesial surfaces of the second molars; this difference being significant ($p<0.001$) for the mandibular teeth (Table 2).

A positive significant correlation ($r=0.25$, $P<0.02$) was found between age and the distance from the cementoenamel junction to the interproximal alveolar crest at all surfaces examined (Figure 2). When the measurements were separated into groups according to surface and jaw (i.e., four groups), there was a significant correlation between the age and the distance from the cementoenamel junction to the alveolar crest at three of the surfaces (Table 3).

DISCUSSION

In previous studies, both standardized and unstandardized radiographs have been used to examine the al-

Table 2 ☐ Distance, in millimeters, between the alveolar crest and the cementoenamel junction at the interproximal site between primary molars.

First molars	N*	Mean	S.D.	t value
Lower left	46	1.20	0.52	0.52**
Lower right	44	1.15	0.33	
Upper left	41	1.01	0.00	0.27**
Upper right	41	0.97	0.63	
Second molars				
Lower left	48	0.88	0.32	0.07**
Lower right	46	0.87	0.38	
Upper right	41	1.08	0.50	0.90**
Upper right	43	0.98	0.51	

* Number of measurements.
**Statistically not significant difference (P<0.05).

Table 3 ☐ Correlation between the ages of the children and the distances from the cementoenamel junctions of the primary molars to the alveolar crests at the four surfaces studied.

Surface	R*	P**
Mesial of the lower second molar	0.12	N.S.
Distal of the lower first molar	0.28	<0.01
Mesial of the upper second molar	0.40	<0.001
Distal of the upper first molar	0.26	<0.02

* R = correlation coefficient
**P = level of provability

veolar bone height.[10-15] Although it would be preferable to use standardized techniques, so that inter and intra-examiner errors could be minimized, this situation does not exist in clinical practice. In the present study, an attempt was made to measure the crown's cervicoocclusal dimension; as in the Selikowitz *et al* study, however, in which the use of unstandardized radiographs is discussed, the occlusal surface was distorted in a large number of radiographs and this measurement could not be made with any degree of accuracy.[12]

In the age-group examined in the present study, the normal distance between the cementoenamel junction and the interproximal alveolar crest in the primary molar area is 1.0 ± 0.5 mm, whereas Stoner indicated that the normal distance from the cementoenamel junction to the alveolar crest in the permanent dentition ranges between 1 to 2 mm in 91.2 percent of the surfaces.[14] This distance coincides fairly closely with the 2 mm distance regarded as the normal biological width of the attachment of the gingival fibers to the root surface.[16]

It was stated that the alveolar crest in the primary dentition parallels the cementoenamel junction of adjacent teeth.[17] The present findings indicate, however, that the distance between the cementoenamel junction and the alveolar crest, at the distal surface of the first primary molar may be slightly, but significantly, greater than at the mesial of the second primary molar. The lack of parallelism, therefore, does not necessarily indicate a bone defect. It was also noted that the shape of the alveolar crest was convex in some cases, not a straight line.

It was reported that in teenagers, the distance between the cementoenamel junction and the alveolar crest does not appear to be related to the time since eruption.[14] While in the present study, a correlation between the age of the patient and the distance from the crest of the alveolar bone to the cementoenamel junction was found, this correlation may be related to continuous eruption of the teeth, due to attrition of the primary tooth. In previous studies we showed that the junctional epithelium migrates apically onto the primary tooth root surface, and that this migration is not related to the shedding process.[18] The migration could be related to gingival inflammation, although we have not been able to confirm this histologically, and may also be related to continuous eruption.[19,20]

This study provides useful baseline data on alveolar bone height and shape for use in the clinical assessment of the periodontal condition of patients and for further studies on periodontal disease in children. Greater measurements than those presented in this study may indicate an early stage of periodontal disease in the primary dentition and/or a tendency of the patient to develop periodontal disease. These patients should be identified as risk populations, and receive special attention with the intent to cure present incipient disease and, most importantly, establish proper measures to prevent the chronic and continuous progress that is characteristic of periodontal disease.

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The attached gingiva in children: diagnostic, developmental and orthodontic considerations for its treatment

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Flap surgery or free gingival grafting has been recommended as an acceptable procedure for the treatment of localized gingival recession in children.[1,2] While in some cases mucogingival surgery is undoubtedly indicated, caution is recommended in labelling what appears to be abnormal in children. The clinician is advised that the diagnosis of an "adequate" band of attached gingiva is not dependent solely on its width, especially during the transition from the primary to the permanent dentition, and that improvement of the gingival architecture as the result of tooth alignment is also feasible.

The purpose of this report is to review relevant information, in order to help the clinician assess the adequacy of the width of the attached gingiva in children and improve his ability to choose when a conservative approach is more appropriate than a surgical approach in cases where gingival recession has been diagnosed.

DIAGNOSIS

Clinical considerations

Gingival recession on a particular tooth may be determined when comparison is made with the average height of the gingiva on the adjacent teeth.[3] Since inflammation and/or hyperplasia of the papillae of adjacent teeth, however, may give a false impression of recession or inadequate attachment, more accurate criteria are necessary. Using the cementoenamel junction of the affected tooth as a line of reference allows for differentiation between "true recession" (Figures 1,2), in which exposure of the root surface is included, and "pseudorecession", where it is not (Figures 3,4).[4]

Lang and Löe in 1972 studied the "adequacy" of the attached gingiva and concluded that a 2 mm band of keratinized gingiva, which corresponds to 1 mm of attached gingiva, is necessary to maintain gingival health.[5]

Figure 1. Clinical picture of gingival recession at the mandibular left permanent incisor. Note that the recession extends to beyond the cementoenamel junction.

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Further studies have demonstrated, however, that gingival health may be achieved in areas with less than 1 mm in width of attached gingiva.[6,7] Furthermore, developmental changes involved in the transition from the primary to the permanent dentition include stages in which the attached gingiva is normally less than 1 mm in width.[8-11]

The fact that the width of the attached gingiva correlates with the number of recession areas has led to the supposition that a narrow zone of attached gingiva is an etiological factor. It may indicate equally well, however, that a narrow band of attached gingiva is the consequence of the recession.[12]

Developmental considerations

The width of the attached gingiva is not an independent entity, but a component of a gingival unit, in which the depth of sulcus, the width of the keratinized gingiva, and the width of attached gingiva correlate closely; ie, in the clinical context, the width of the attached gingiva is established by subtracting the sulcus depth from the width of the keratinized gingiva.[13-15] These three components of the gingival unit may be subjected to morphological changes during oral development, and the clinician must be aware of these changes in order to be able to diagnose normal or pathological situations, especially in cases where an incisor has a gingival margin attachment apical to that of the adjacent teeth, but above the cementoenamel junction.

In general, it has been established that the width of the attached gingiva is greater in adults than in children in the primary dentition stage.[13,16] This change is not the result of a continuous cumulative process; but, as has been demonstrated by longitudinal and cross-sectional studies, a decrease in the width of the band of attached gingiva that takes place during the transition from the primary to the permanent dentition.[10,11]

In the newly erupted permanent tooth, an attached gingiva per se is nonexistent, since the depth of the gingival sulcus exceeds the width of the keratinized gingiva.[8,11] Furthermore, for several years after eruption, the attached gingiva is normally less than 1 mm in width. Tenenbaum and Tenenbaum, in a cross-sectional study, have shown that it may take more than eight years after eruption, until the attached gingiva reaches its adult width.[11] In a longitudinal study comparing the morphology of the gingival unit of the primary dentition of seven-to-nine year-old children to that of the permanent dentition, five years later, we indicated that for most of the teeth, the attached gingiva in the permanent

Figure 2. Clinical picture of gingival recession at the mandibular right central incisor, which extends beyond the cementoenamel junction. Note the topography of the recession facilitates plaque accumulation and makes its removal difficult.

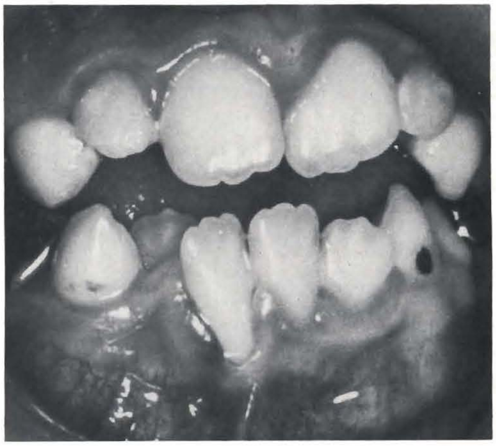

Figure 3. Clinical picture f a seven-year-old child, with severe anterior crowding. Gingival pseudorecession above the cementoenamel junction is evident at the buccally erupted mandibular right permanent incisor.

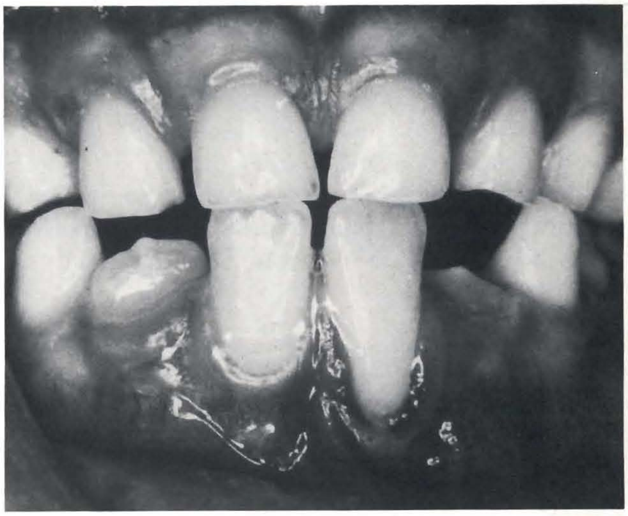

Figure 4. Clinical picture of gingival pseudorecession at a buccally erupted mandibular left permanent central incisor in a seven-year-old child with anterior mandibular crowding.

Figure 5. Lateral view of the case presented in Figure 3. Note the shallow vestibulum; and high mandibular frenum attachment, which deflects the marginal gingiva; and the lingual inclination of the mandibular right central incisor.

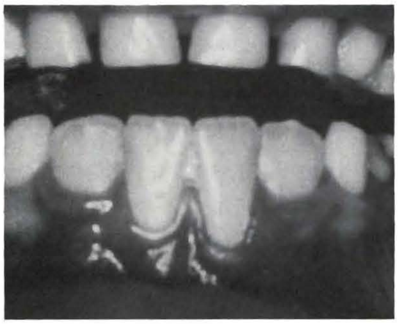

Figure 6. A six month follow-up picture of the same case presented in Figure 4, showing the results of meticulous oral hygiene, intercanine growth and spontaneous tooth alignment. The gingival recession has diminished significantly.

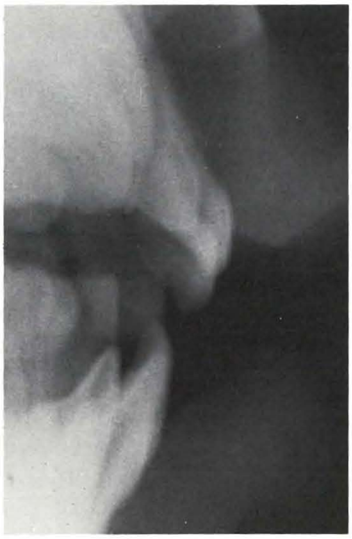

Figure 7. Lateral extraoral radiograph in which the buccolingual position of the permanent mandibular incisors is evident. In this case, the inclination of the most prominent tooth, which exhibits gingival recession, indicates that its apex is located away from the buccal plate of bone.

teeth was still narrower than in their primary predecessors.[17]

The differences in width of the attached gingivae in the primary and the early permanent dentitions and the gradual increase in the width of the attached gingiva that takes place in the permanent dentition after eruption, have been found to be the result of changes in sulcus probing depth, although there appears to be no similar trend in the width of the keratinized gingiva.[8,10,11,17] Based on the finding that the mucogingival junction remains stationary throughout life, these results indicated that, in the early permanent dentition, the occlusal movement of the newly erupted tooth is not accompanied by a parallel occlusal movement of the marginal gingiva.[18] In addition, in cases with a high frenum attachment (Figure 5), one must consider that frenum position changes with age.[19,20]

GINGIVAL ARCHITECTURE AND CONTROL OF INFLAMMATION

While much attention has been given to the decrease in the width of attached gingiva, which may result in areas of localized gingival recession, little has been said of its effect on the gingival architecture. Localized gingival recession seems to alter the normal topography of the free gingival margin, thus creating an environment different from the normal.

Recently, Smukler and Machtei studied the effectiveness of plaque control in areas of localized gingival recession and found that plaque control was less effective compared to control nonrecessive sites.[21,22] In addition, the Gingival Index and Bleeding Index were found to be significantly higher in the localized recession sites (Figure 2).

The pathogenesis of gingival recession is not yet completely clear; the inflammatory mechanism proposed by Baker and Seymor would indicate, however, that a self-perpetuating circle takes place: gingival recession facilitates plaque accumulation, which causes inflammation, which leads to further recession and so on.[21-23]

In light of this recent information, monitoring of marginal gingival inflammation should become a major factor in the treatment of localized gingival recession. Furthermore, in cases where a surgical approach is indicated, its aim should not be solely to increase the band of attached gingiva, but also to alter the gingival architecture via root coverage and coronally positioned flap.[24,25] Such an approach will not only serve as a corrective measure, but it will also have a beneficial effect on oral hygiene performance, which in turn will serve as a preventive measure for further loss of attachment.

IMPROVEMENT OF GINGIVAL CONTOUR DUE TO TOOTH ALIGNMENT

The path of eruption and the labiolingual position of teeth influence the thickness of the periodontium and, specifically, the width of the attached gingiva (Figures 3-5).[1] If a tooth erupts in labioversion and its attached gingiva has minimal width, there will be no increase in the width of this tissue. Furthermore, orthodontic alignment of the tooth will not enhance its attached gingiva.[1] Nevertheless, several studies have made more optimistic claims by demonstrating that, in some cases, an increase in width of the attached gingiva may take place as the result of tooth alignment, either with or without orthodontic treatment.[20,26-28] The explanation for improvement of the gingival contour implies movement of the tooth away from the thin attachment apparatus and a creeping reattachment.[20] In fact, the improved contour may be the result of regrowth of the gingival soft tissue on the affected tooth and/or apical migration of the gingival margin of the adjacent tooth (Figures 4,6).[4,28]

The treatment plan for gingival recession must take into consideration the buccolingual position of the root. The use of lateral extraoral radiographs (cephalometrics or tangential view) may be helpful in diagnosing tooth inclination, position of apices, and the amount of labial alveolar bone (Figure 7). Tooth alignment and a consequent improvement of the gingival contour may be achieved, provided the root apices are located away from the labial plate of bone (Figure 7), due to intercanine growth, stripping or extraction of the primary canines and/or orthodontic treatment.[20]

Intercanine growth

Changes in incisor position, related to the growth pattern of the jaws, may influence the improvement potential of gingival recession in children, due to an increase of the intercanine width. This occurs mainly during the emergence of permanent mandibular lateral incisors and presumably under the influence of the activity of the tongue and lips (Figures 4,6,7).[27]

Extraction or stripping of primary canines

In cases with anterior mandibular crowding and rotation of the mandibular permanent incisors, extraction of the primary canines may provide room for the spontaneous alignment of the incisors.[20] Paradoxically, improvements in arch alignment and gingival contour have been found to be greater in untreated subjects than in those receiving serial extractions. Although extractions of the primary canines provide space for spontaneous alignment, they may limit intercanine growth.[27] When extraction of the canines is contraindicated, partial relief of the incisor crowding may be achieved by mesial stripping of the primary canines.

Orthodontic treatment

When extraction is not essential and yet the potential for spontaneous tooth alignment becomes exhausted (due to age of the developing dentition vis-a-vis intercanine growth), orthodontic treatment may be indicated for the treatment of anterior mandibular crowding. Repositioning of a crowded tooth with gingival recession (in the presence of good plaque control) improves the bony architecture at the immediate site by moving the root deeper into the alveolus, with an accompanying improvement in form of the alveolar bone. Such changes may either render surgical repair unnecessary or provide an environment that will improve the prognosis of a gingival graft performed subsequent to the tooth movement. Periodontal surgery aimed at covering the root, before orthodontic treatment, is likely to fail, due to the prominence of the root immediately beneath the periosteum and the relative absence of labial alveolar bone.[20] Occasionally, one may see either well-aligned or rotated mandibular incisors, which, on closer examination, reveal that their roots are labially displaced, leaving a thin covering of labial alveolar bone (Figure 5). Orthodontic intervention is then advised to move the root lingually.[20] It must be remembered, however, that this type of orthodontic maneuver is difficult to achieve. Good control is rarely possible unless orthodontic attachments are placed on all teeth; this generally dictates waiting for the full permanent dentition, which may defeat the purpose, when the problem is diagnosed at age seven or eight years. It follows that treatment at this early age means accepting reduced mechanical efficiency. Furthermore, any routine orthodontic problems that may later occur with the eruption of all the permanent teeth will have to be addressed in a second stage of treatment, at age twelve or later.

Orthodontic alignment may contribute to improving the gingival contour.[20,26] On the other hand, when orthodontic treatment involves excessive forces (that do not permit repair and remodeling of alveolar bone), or movement of "wide" teeth into areas of "narrower" teeth or with gingival attachment deficiency, it may result in loss of attachment, localized recession, and crestal bone loss.[20,28] When considering the intimate relationship between position and width of attached gingiva during orthodontic treatment, the deleterious effect of the treatment on the gingival tissues may be none to minimal, limited to hyperplastic gingivitis that is transient, and without permanent damage.[29,30]

Occasionally, the appearance of recession during orthodontic treatment may be attributed to the presence of plaque. In persons who form supragingival calculus rapidly and retain debris, despite good routine oral hygiene, surgical intervention before orthodontic movement may be preferred.[20]

PATIENT COMPLIANCE

Several factors have been described as predisposing or precipitating of gingival recession: inadequate attached gingiva, plaque, marginal inflammation, crowding, tooth rotations, ectopic eruption, abnormal frenum attachment, shallow depth of the vestibulum which impedes toothbrushing (Figure 5), iatrogenic (such as orthodontic treatment) and unnecessarily aggressive toothbrushing.[15,20] There is general agreement in the literature that, of all these, the most important etiologic factor in gingival recession is plaque-induced inflammation and that labial positioning merely accelerates the process. It is also believed that improvement of the gingival contour is obtained when inflammation is controlled and that this is enhanced by good alignment of the teeth.[27] It has been stated further that, when marginal inflammation is controlled, frenal involvement declines.[27] Summarizing all these factors, it becomes clear that control of marginal inflammation appears to be the most important key to limiting and, possibly, reversing an otherwise progressive recession.

CONCLUSIONS

The clinician's first choice for the correction of gingival recession of the mandibular permanent incisors in children should be a nonsurgical approach, a decision based on the fact that in children there is a potential for correction of the condition when:

- ☐ Oral hygiene eliminates gingival inflammation.
- ☐ An increase in width of the attached gingiva takes place as teeth erupt.
- ☐ Tooth alignment may take place as the result of intercanine growth, mesial stripping, extraction of the primary canines and/or orthodontic treatment.

It should be noted that the position of the frenal attachment changes with age as a normal factor of growth.

A surgical approach should be considered when:

- ☐ Healing of gingival inflammation cannot be achieved by oral hygiene methods.
- ☐ The recession is beyond the cementoenamel junction.
- ☐ The gingival architecture cannot be maintained.
- ☐ Improvement of the gingival contour is not obtained by nonsurgical means.
- ☐ The oral development precludes the probability of an increase in width of the attached gingiva or if spontaneous tooth alignment is minimal or none.

The surgical approach may prove more successful, if preceded by orthodontic treatment. When orthodontic treatment, however, might increase the accumulation of plaque (thus inflammation), or its feasibility or effectiveness are low, surgery should be performed first.

In any case, plaque control is the most important factor in the successful treatment of gingival recession, regardless of method, conservative, orthodontic and/or surgical.

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GINGIVAL HEALTH OF INDO-CHINESE CHILD REFUGEES

Epidemiological studies have confirmed the prevalence and severity of periodontal disease in the Indo-Chinese population when in their country of orgin. Reports of refugees in transit camps and at the time of resettlement have shown that calculus and gingival bleeding are prevalent for all age groups, with significant pocket depth and loss of attachment in adults. Scheutz, Heidmann, and Poulsen reported loss of attachment in children aged between ten and fourteen.

Di Angeles and Rojas concluded that adolescent Indo-Chinese refugees were prone to severe gingivitis and incipient destructive periodontal disease when access to dental care was available after resettlement in other countries. A study of the gingival health of 230 Indo-Chinese children aged 5-12 years who had had dental care after resettlement in Australia demonstrated this trend in a younger age group. Calculus, and in particular subgingival calculus, characterized these children for all ages studied. Fifty-seven per cent of the children had subgingival calculus, with 28 per cent of the children each having more than five sites recorded. Furthermore, there were some children who displayed loss of attachment in association with gingival crevices deeper than 3.5 mm on permanent teeth at the time of examination.

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The statistical unit for analysis of developmental changes in the attached gingiva in children

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The issue of an adequate statistical unit in periodontal studies, sites or mean per patient, has been the subject of discussion on several occasions. Haffajee *et al*, in a study on the comparison of statistical methods of analysis of data from clinical periodontal trials, concluded that examination of the results on a mean per patient basis, may be sensitive to small changes in a large number of sites, but insensitive to major changes in a smaller number of sites, which may be of significance to the research worker or the clinician.[1] This approach was criticized by Dr. Fidler in a letter to the editor of the Journal of Clinical Periodontology (February 1984), in which he recommended to consider the individual as a statistically independent unit, and the observations made on each individual as related. Dr. Haffajee *et al* responded to the letter of Dr. Fidler (in the same journal issue), defending their technique, mainly by emphasizing that, in the case of periodontal disease, sites in the same patient may differ dramatically, and that treatment is often done on diseased sites, and not on "patients".

A sequel of this argument was an article by Dr. Blomqvist, in which he stated that in trials where experimental units of different levels are employed, the highest value limit should be used, since the use of a lower level unit would underestimate the standard error and level of significance.[2] The purpose of the present study was to identify an adequate statistical unit for the analysis of developmental changes in the width of attached gingiva in the mixed dentition of children.

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POPULATION AND METHODS

The five-year longitudinal changes in the width of the attached gingiva of fifty-four children, previously reported, were further analyzed, using a repeated measures-analysis-of-variance that allows one to ascertain the magnitude of the contribution of each source.[3-5] The Student's T test was used to analyze the significance of the difference between pairs of estimated mean values. The children had mixed dentitions and two groups of teeth were considered:

Group A — A primary tooth present at baseline and its permanent successor at the second examination.
Group B — The permanent tooth present at both examinations.

RESULTS

The findings of the repeated measures analysis of variance and the comparison between estimated means indicated that:

- The estimated mean per patient for group A (-0.45 ± 0.52 mm) was significantly different ($p<0.001$) to the one for group B (0.96 ± 0.52 mm).

- □ The estimated mean for the group of all maxillary gingival sites showed significant differences between patients ($p<0.015$).
- □ No significant differences ($p<0.05$) were found between the estimated means for type of tooth (central or lateral incisor, canine etc.) within the maxillary teeth in groups A or B and the mandibular teeth in group A. Among the mandibular teeth in group B a significant difference ($p<0.001$) was found between the central incisor and first molar.
- □ In group A, no statistically significant difference was found within the upper teeth and their lower antagonists ($p<0.05$); whereas in group B, significant differences were found between the incisors ($p<0.001$).

DISCUSSION

The significant differences in values between the two groups and some types of teeth, indicate that it would be inappropriate to obtain a mean per patient. These differences, for example, include the decrease in width of the attached gingiva due to the transition from the primary to the permanent dentition (group A), and the increase in width that takes place after eruption of the permanent teeth (group B); the value of one change will obscure the value of the other. On the other hand, since the changes in width of the attached gingiva for group A or group B were mostly similar, and all gingival areas within a group followed the same trend, a mean per group may be utilized. It appears that the key word in choosing the adequate statistical unit in clinical periodontal trials is *dependence*, in the case in which changes at all gingival sites are dependent and/or a common trend of change is investigated, a patient mean can be utilized. On the other hand, in cases where, due to developmental changes or differences in the examined factors within the subjects (primary or permanent teeth, inflamed or healthy areas), the expected change follows different trends at different sites, the use of a patient mean would be inadequate. In the particular case of the attached gingiva width, this measurement is subject to developmental changes throughout life.[3,4,6-8] Research on this subject should be controlled for the patient's age or developmental stage, in order to minimize the effect of developmental differences between subjects, in the results.

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Anomalies of form and number, fused primary teeth, a correlation of the dentitions

Anomalies

F. Thomas Hagman, DDS

With the exception of fusion, anomalies of form and number occur less frequently in the primary dentition than the permanent dentition.[1-5] The frequency of these anomalies is summarized in Table 1.

Many early reports of double formations did not differentiate between fused and geminated teeth. "Fusion may be defined as a union between the dentin and/or enamel of two or more separate developing teeth. Gemination is an attempt by a tooth bud to divide."[4] Fusion may be clinically differentiated from gemination by simply accounting for the number of teeth. Where fusion is suspected there should be fewer teeth than would be normal for a given dental age; the double formation, bifid crown, is counted as one unit. Fusion with a supernumerary tooth would not answer to the above criteria. This, however, would be a rare occurrence, since hyperdontia has a predilection for the maxilla, and fusion a preference for the primary mandibular incisors.[2,6] There is one reported case of fusion with a supernumerary tooth in this literature review and two reports of combined fusion and gemination.[5-7]

Heredity and sex seem to be a factor with numerical variations related to fusion. Moody and Montgomery demonstrated familial and sex disposition in three pedigrees involving lower primary incisors.[8] The fusion occurred in and was conferred on three succeeding generations by female members of the family.

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Table 1 ☐ Anomalies of form and number in children.

Author	Children N	Missing N / %	Supernumerary N / %	Fused N / %	Geminated N / %
2	1,173	(5) 0.4	(3) 0.3	(5) 0.43	(1) 0.09
3	2,209	(2) 0.09	(5) 0.23	(3) 0.14	
5[a]	CR			(2)	
6	4,564	(25) 0.54	(26) 0.56	(39) 0.85	
7	2,539	(25) 0.54	(26) 0.56	(39) 0.85	
8	CR			(22)	(6) 0.24
9	CR			(11)	
10[b]	285			(7) 2.46	
10[c]	616				(1) 0.16
11	CR			(1)	
12	CR			(2)	
13	1,000	(7) 0.7	(2) 0.2	(4) 0.4	
14[d]	3,557	(43) 1.2			

Author, the number is indexed to the references
CR, Case Reports
a, One case "germinated composite odontoma" i.e. incisor fused with a supernumerary
b, Primary dentition intact, biologically related group
c, eighteen or more primary teeth, biologically related group
d, Seventeen (17) "double formations" .47 percent

Table 2 ☐ Correlation of fused lower primary anterior teeth to aplasia of the succedaneous teeth.

Source of data	Sex M	Sex F	Fusion lateral Canine	Right or left R	Right or left L	Fusion of incisors	Aplasia lateral incisor	Aplasia canine	Percent
Bruszt*	1	10	13	10	3		9	2	85
Grahnen	1	2	3				3		100
Grahnen			1			4			0
Hagman	1	1	2	1	1		1		50
Levitas		2	2		2		2		100
Niswander			34			11	26		58
Ravn			8				8		100
Ravn						17	3		18
Toth		4	4	4			2		50

*Two of eleven patients had bilateral fusion, one patient with bilateral fusion of primary lateral incisors and canines had aplasia of both lower succedaneous lateral incisors, the other had aplasia of both lower succedaneous canines.

Grahnen and Granth reported three of eight siblings, twin sisters (monozygotic) and one brother, with fusion involving lower primary canines.[2] These three cases also had aplasia of the succedaneous lateral incisor. Bruszt reported eleven cases of fusion involving lower primary canines and lateral incisors, ten in girls and one in boys.[9] Of the ten cases evaluated, nine had aplasia of the succedaneous lateral incisor. Fusion occurred bilaterally in two of the eleven cases, making a total of thirteen incidences of fusion and eleven incidences of aplasia (ten female, one male) of the succedaneous lateral incisor. Niswander and Sujaku reported a high incidence of fusion among Japanese children in the primary dentition (2.5-3.0 percent).[10] About a half of these children were offspring of biologically related parents. Dependorf as cited by Grahnen and Granth reported bilateral fusion of mandibular incisors in two sisters.[2] Levitas reported two sisters with fusion of the primary lateral and canine with aplasia of the succedaneous lateral incisor.[11] Hagman reported a brother and sister with fusion of the primary canine and lateral; the brother had aplasia of the succedaneous lateral incisor.[12] Toth and Cesmi reported four cases of fusion between primary lateral and canine with aplasia of the succedaneous lateral incisor 50 percent of the time; all cases involved females.[7] Ravn reported eight cases of fusion between primary lateral incisors and canines, with aplasia of the succedaneous lateral incisor in every instance (Table 2).

DISCUSSION

There appears to be a correlation between the two dentitions. When fusion occurs in the primary canine-lateral position, there is at least a 50 percent chance that the succedaneous lateral incisor will be missing (Range, 50 percent to 100 percent; average, 78.2 percent). Fusion occurs most frequently in the mandibular anterior area with the canine and lateral incisor predominantly involved. In the reports that differentiated aplasia related to primary lateral-canine fusion and primary incisor fusion, succedaneous incisor aplasia occurred from zero percent to 18 percent, when the incisors were fused. Niswander's data showed 76 percent aplasia of succedaneous laterals, when the location of the aplasia was related to fusion of the primary lateral incisors and canines. Fusion occurs more frequently in females. Aplasia of the permanent lateral incisor occurred two out of three times among the males. Genetics appears to be a factor, when increased incidence is found.

It would be of great benefit for future reports or studies to be more specific as to location, teeth involved, sex, developmental disturbances, familial relation, and definition of terms.

SUMMARY AND CONCLUSIONS

Fusion is rare in the primary dentition (range .14 percent to 3 percent). Patients with fused primary lateral incisors and canines have about a 75 percent chance of lacking the succedaneous lateral incisor. Patients with fused incisors have less than a 20 percent chance of having a missing permanent tooth.

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PERSUASION BASED ON LEARNING PRINCIPLES

Employing programs soundly based on learning principles, effective persuasion has been achieved during the 1980s in the area of smoking. The persuasive messages evaluated have been soundly based on three learning principles:

☐ The level of fear employed must be relatively low, so that inappropriate responses (such as denial) are not required to deal with the fear. This has been achieved by concentration on immediate physiological effects of smoking such as blood pressure and lung capacity.

☐ This concentration on the short-term effects also allows for a more immediate result from the cessation of smoking, i.e., the results of increased lung capacity rather than the very long-term reinforcer of possible relief from earlier death. It is a well-established principle of learning that a reinforcer of shorter delay is more effective than one of long delay, and even with long delays of reinforcement less time for "associative interference" enhances learning.

☐ The principle of behavior shaping is employed as specific responses are offered rather than naive advice to simply not smoke. Typically this has involved teaching specific skills to resist the social pressures to smoke. These principles have been employed in numerous studies reporting positive results.

Job, R.F. Soames: Effective and ineffective use of fear in health promotion campaigns. Am J Pub Health, 78: 163-167, February, 1988.

A complete fusion in the primary human dentition: a histological approach

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Luc C. Martens, DDS, PhD
Luc G. De Craene, DDS

In October 1985, a four-and-a-half-year-old boy attended our clinic. He complained about acute pain and a swelling in the maxillary midline area. Examination of the oral cavity revealed a nonfluctuant swelling, located apically to a severely decayed "double tooth" (Figure 1). No pulp exposure had occurred. His oral hygiene was fairly good. A periapical radiograph showed a very large pulp system and an apical radiolucency around a large apex (Figure 2). A chronic periapical inflammation, which had become acute, was diagnosed.

It was decided to prescribe an analgesic and antibiotics for several days. During the following visit, the tooth was extracted. Consequently, we possessed by accident a "double tooth". According to the definition presented by Kelly, this case was considered to be a "complete fusion" of the primary maxillary right central and lateral incisor teeth.

Because the tooth was almost complete and intact after the extraction, it was decided to examine the double tooth histologically, to obtain more information on the pulp system and the etiology of double formations. In the meantime, the opportunity for endodontics could be evaluated.

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REVIEW OF THE LITERATURE

Fusion and gemination are terms used to describe the joint or double formation of teeth. Many authors have tried to explain the origin of both numerical variations.[1-5] In general, two approaches are possible (Figure 3).

Hitchin and Morris defined "fusion" as the result of the embryological persistence of the lamina interdentalis between two buds, while Kelly defined the same phenomenon as the incomplete attempt of two tooth buds to fuse into one.[6,7]

Kelly defined "gemination" as the incomplete attempt of one tooth bud to divide into two, whereas Hitchin and Morris described the phenomenon as the development of a supernumerary tooth from the remnants of the lamina interdentalis, after it is broken near a neighboring tooth germ.

Kelly also stated that by means of radiography, the difference between fusion and gemination can be determined. Fusion is usually characterized by two separated root canals, whereas gemination usually displays one large root canal. It has been hypothesized, however, that, if the two tooth buds make contact before calcification, one large root canal can be seen in a fusion. In that case, the fusion is called a "complete fusion".[8]

According to many epidemiological studies, the prevalence of double formations is 0.5 to 1.0 percent in the

Figure 1. Clinical view of the double tooth.

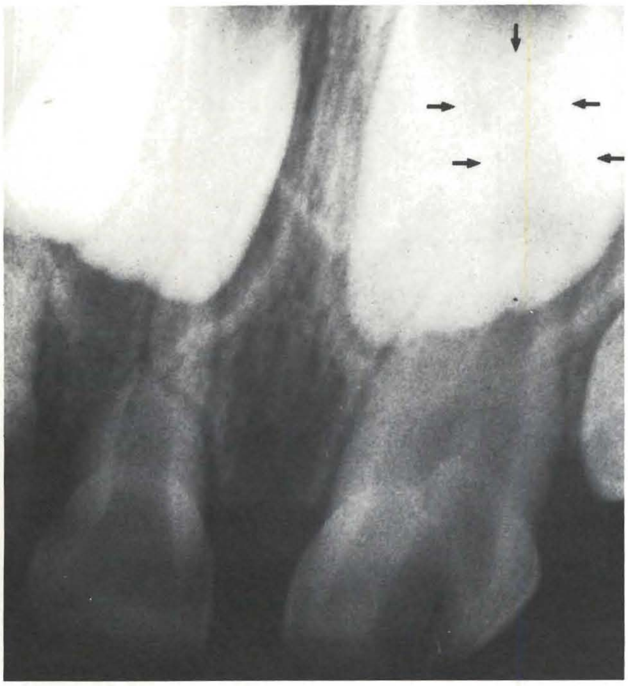

Figure 2. Periapical radiography of the maxillary midline area. The black arrows indicate the apical radiolucency.

primary dentition and 0.1 percent in the permanent dentition, with an equal distribution in females and in males.[9-11,14,15] Fusion is most often seen in the mandibular primary incisors, while gemination usually occurs in the maxillary primary incisors and canines.[13]

In addition to retarded root resorption of the fused teeth and consequently retarded eruption of the permanent successor(s), several authors report aplasia or malformation of the successor(s), following a fusion in the primary dentition.[4,9-11,13,14] Anomalies of the permanent teeth are most often found following a fusion, and appear to prevail in the mandible over the maxilla.[13]

MATERIAL AND METHODS

Immediately after the extraction, a radiograph was taken and the tooth was stored in 10 percent formalin.

Figure 3. Schematical representation of the two different approaches to fusion and gemination according to Kelly (1978) and Hitchin-Morris (1966).

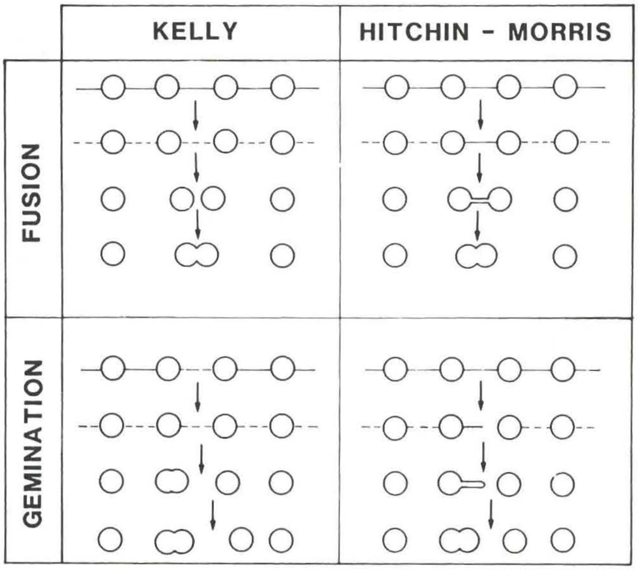

Figure 4a. Buccal view of the extracted double tooth.

Figure 4b. Lingual view of the extracted double tooth.

The size of the crown and the longitudinal groove buccally and palatally were the only clinical visible signs of this double formation. The severely carious lesion was situated palatally, while the buccal enamel remained almost intact. The size and flatness of the root and the discrete depression in the buccal and palatal plane indicated that this root originated from a double tooth (Figures 4a,b).

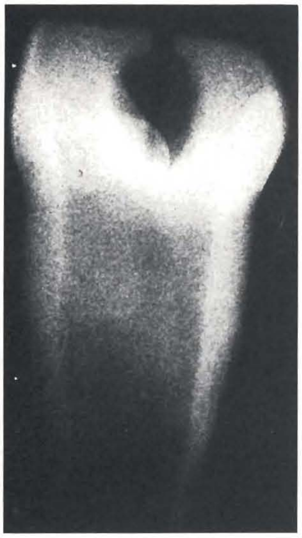

Figure 5. Radiography of the extracted double tooth.

Figure 6a. Overall view of a representative histological section.

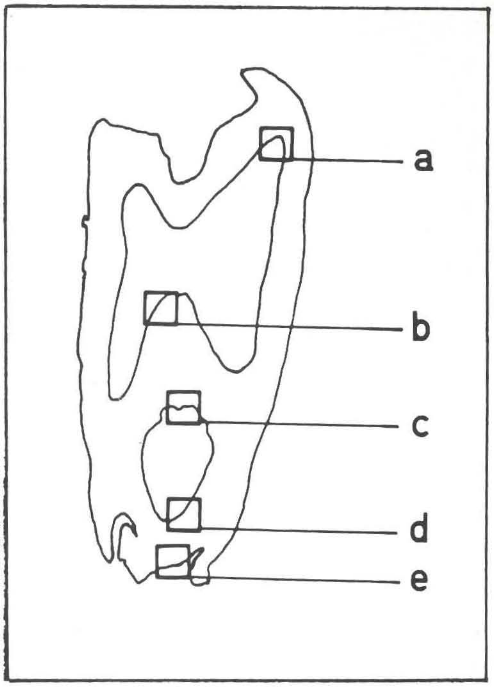

Figure 6b. Schematical representation of the same section.

Decalcification took place in 25 percent formic acid for a period of thirty days. The tooth was embedded in paraplast and cut in the mesiodistal plane, starting buccally, with a Reichert-Jung microtome. The sections were cut at approximately 6 μ and stained with Mayer Haematoxyline Eosin.

RESULTS

Instead of a simple, large pulp system, as determined from the radiograph (Figure 5), all histological sections displayed a H-shaped dental pulp (Figure 6). This confirmed that both the dentin and the pulp systems were united. Additionally, there were indications of a complex internal structure.

After a brief examination of all subsequent sections, a definite concavity in the middle of the root, on the palatal surface, was found. In some sections, this concavity appeared as an empty circular area, bordered apically by a discrete zone of cementum and coronally by fibrous tissue (Figures 7c,d).

The carious lesion was separated from the pulp by only a very irregular, small amount of tertiary dentine. The bacterial invasion, in some areas, had already reached the dental pulp. In the pulp we found a large number of inflammatory cells. The latter were also present in the most apical regions of the pulp system, which was in close relationship with the periapical region by an open apex (Figure 7e).

Surrounding the pulp, lined by quite regularly arranged odontoblasts, an extended, well-defined zone of tertiary dentine could be seen. The primary and secondary dentine were characterized by evenly distributed dentinal tubules, whereas the dentinal tubules of the tertiary dentine were clustered in groups of ten to fifteen. Those groups appeared in a totally disorganized

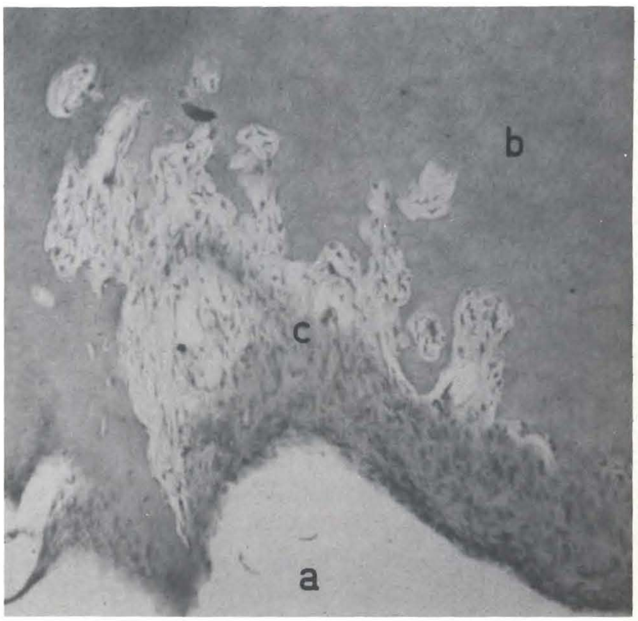

Figure 7c. Detail of zone "c" defined in Figure 6b, showing the upper border of the concavity (a) consisting of dentin (b) lined by fibrous tissue (c).

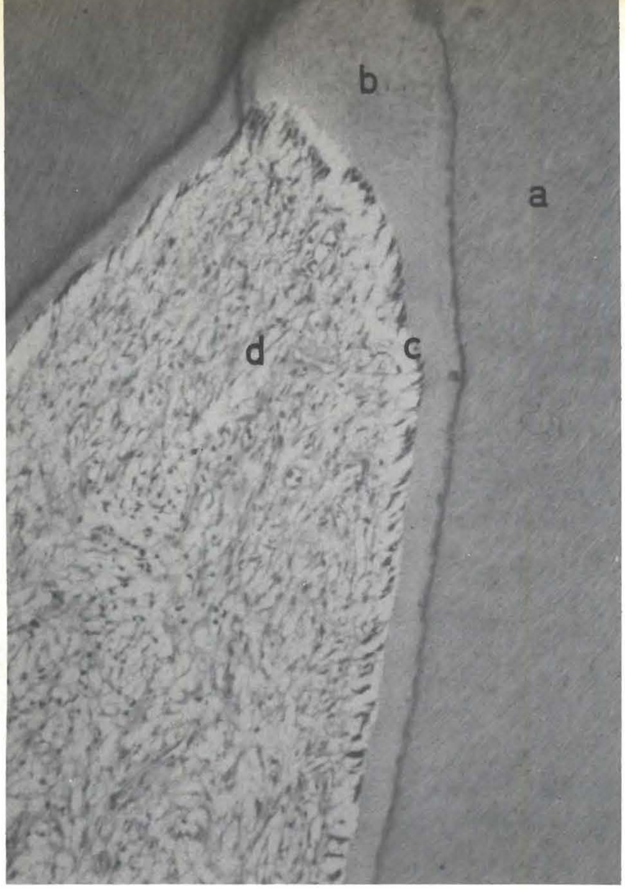

Figure 7a. Detail of zone "a" defined in Figure 6b, showing primary dentin (a), secondary dentine (b), odontoblasts (c) and a part of the coronal pulp (d).

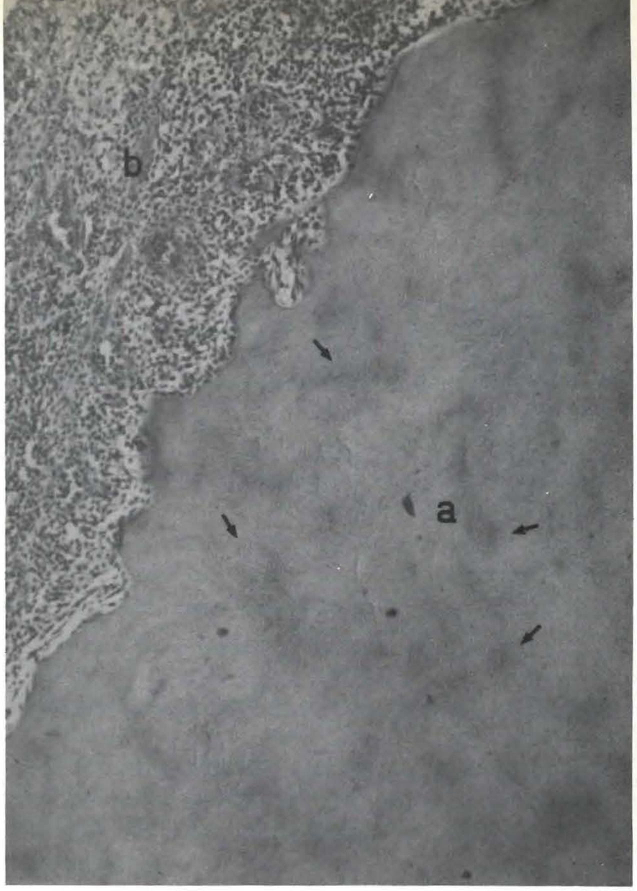

Figure 7b. Detail of zone "b" defined in Figure 6b, showing tertiary dentine (a) with groups of tubuli (see arrows) and a part of the pulp system (b) with inflammatory cells.

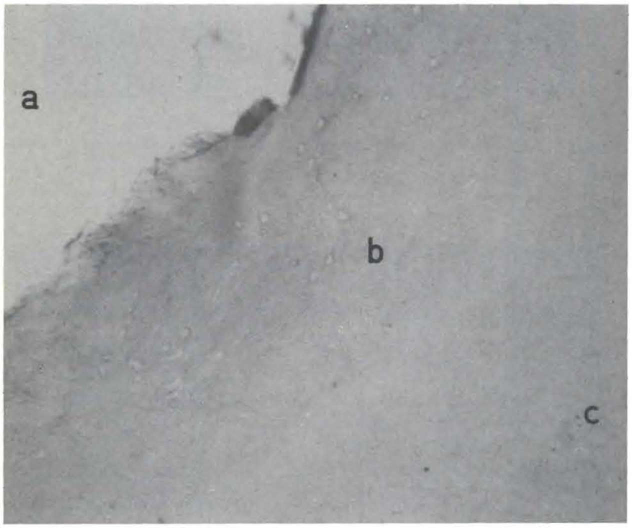

Figure 7d. Detail of zone "d" defined in Figure 6b, showing the lower border of the concavity (a) consisting of a discrete zone of cementum (b) lining the dentine (c).

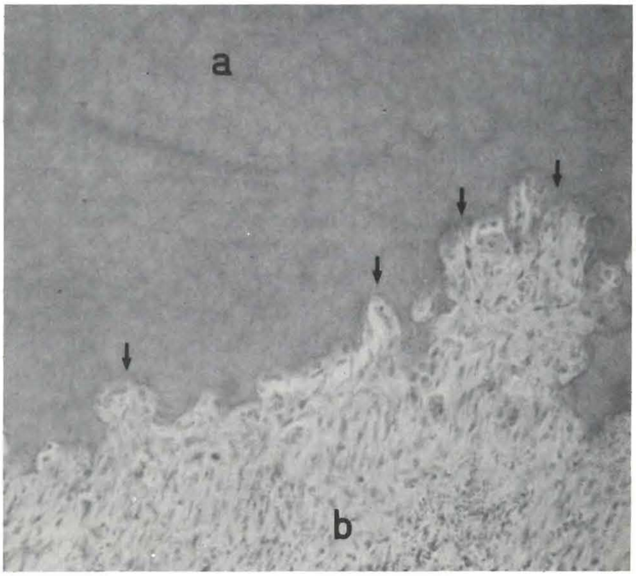

Figure 7e. Detail of zone "e" defined in Figure 6b, showing a part of the broad apex with dentine (a), part of the pulp system (b) and odontoblastic activity (see arrows).

pattern in the matrix (Figure 7b). The tubules from secondary and tertiary dentine made an angle (Figure 7a). A trace of cementum totally surrounded by dentine, however, could not be found. Around the root, a uniform layer of root cementum, sometimes disrupted by little groups of odontoclasts, was observed.

DISCUSSION

The definitions presented by Kelly are clinically acceptable. If the double formation is considered as one tooth, fusion results in a smaller number of teeth, while gemination results in an equal number of teeth. In the latter anomaly, more tooth substance on a single element can be seen, athough many authors claim that counting the normal teeth present in the mouth gives no assurance as to whether a case can be determined as a gemination or as a fusion.[9,10,16]

Figure 8a illustrates that either a gemination or a combination of a fusion with a supernumerary bud can result in the same clinical appearance. According to Kelly (1978) this clinical situation could only be considered as gemination. Figure 8b illustrates that fusion as a combination of agenesis and gemination can result in the same clinical appearance. When only the number of teeth present in the mouth is taken into account, however, only fusion can be concluded. These findings could not confirm or weaken either definition. The fact that a remnant of cementum totally surrounded by dentine was not observed, can lead to the conclusion that either the two originating buds were never separated, or the two originating buds were already joined in a very early stage, before mineralization or cementum formation began.[6-8]

The histological evaluation did confirm several radiographical and clinical findings. In this respect, the unity of the two pulp systems was found, but the root anatomy in the mesiodistal plane could not be evaluated adequately on radiographs. Only after superpositioning of the subsequent sections, was the large palatal concavity found. This concavity was also observed in the radicular pulp. The broad apex was seen radiographically as well as histologically.

Taking the findings into account, it can be concluded that a radiograph can be of great value, but sometimes is of little value in understanding the three dimensional anatomy of an abnormal dental pulp.[4,12] Lack of such knowledge may result in endodontic failures.[16-19] It can be presumed that such a complex internal structure is more a rule than an exception in fusions or geminations with joined pulp systems, even in the permanent dentition.

The bacterial infiltration seemed histologically to be more profound than expected from the radiograph. The caries attack stimulated tertiary dentine formation. This regeneration, however, was not enough to compensate for the mineral breakdown from the carious lesion. The physiological resorption process had already started through the activity of the odontoclasts.

The clinical systems, acute pain and swelling; the apical radiolucency; the wide-open apex; the proximity of the bud(s) of the successor(s); and finally the age of the patient, indicated extraction of this anomalous tooth was justified. The loss of primary incisors at this age, moreover, is very unlikely to cause an orthodontic problem. In the permanent dentition, however, a strong effort should be made to retain double tooth formations.[20-27]

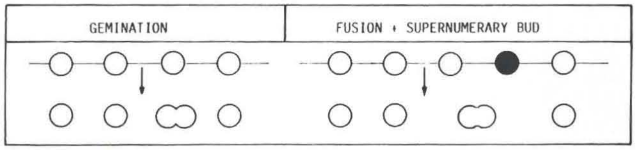

Figure 8a. Schematical representation of two different origins that result in the same clinical appearance of gemination.

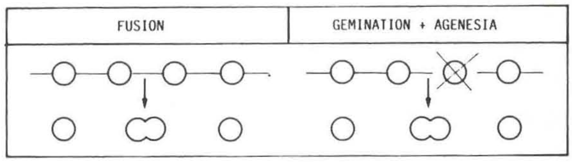

Figure 8b. Schematical representation of two different origins that result in the same clinical appearance of fusion.

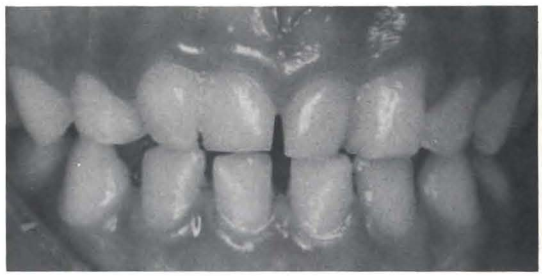

Figure 9a. Clinical view of two double formations before fissure sealing.

Figure 9b. Clinical view of two double formations after fissure sealing.

Rather than extraction, a preventive approach to primary double teeth is preferable.[19] Sealing of the fissure between the two components and fluoridation of the smooth surfaces is advised (Figures 9a,b).

CONCLUSIONS

Since a trace of cementum totally surrounded by dentine could not be found, this histological evaluation gave no further information about the origin of this double tooth. Although the radiograph did give some information, the fact that the dental pulp was complex could not be observed radiographically. For many reasons, endodontic therapy would have been inopportune in this case. Deep carious destruction in a primary double formation with obvious clinical symptoms is an indication for extraction. If one has to deal with a double formation in the primary dentition, a preventive approach to the phenomenon by sealing the fissure and by fluoridating the smooth surfaces, can be recommended.

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CELLS ARE THE PATIENT'S PROPERTY

The California Court of Appeal, in a precedent-setting decision, has ruled that researchers must get permission from patients before using tissues and body fluids obtained in the delivery of health care. The court also indicated that if research reveals that a patient's tissues may yield products of commercial value, the donor has a right to some compensation unless he specifically relinquishes any financial interest.

Crawford, M.: Court rules cells are the patient's property.
Science, 241: 653,654, 5 August, 1988.

Case reports

Delayed eruption of a maxillary central incisor associated with an odontome: report of case

Richard G. Oliver, MScD, FDS
Christopher G.L. Hodges, BDS, MScD

Odontomes are tumor-like lesions found in the jaws, and are composed of enamel, dentine, cementum and pulp tissue. They may be classified as either compound (in which histologically the material resembles a tooth or teeth) or complex (in which histologically the material is an unorganized collection of dental tissue). Opinions differ as to the relative frequency of either type, although the majority view suggests a higher incidence of the compound type.[1,2] Odontomes are more frequently found in the maxilla with an equal sex distribution. The etiology is obscure with local factors such as trauma or infection, and systemic factors such as genetic suggested. Despite their obvious odontogenic origin, they do not seem to be associated with either supernumerary teeth, or with tooth aplasia.[1,3] The most common sequel to the development of an odontome is the impaction of the adjacent tooth, with displacement or malformation of the adjacent tooth a rarer phenomenon.[1] Morning found that malformation of the root of adjacent teeth was more common than crown malformation.[4] This may be the result of inadequate space for continued normal root formation due to the impaction. Morning also found that less than half of the impacted teeth erupted spontaneously following surgical removal of the odontome.[4]

CASE REPORT

A male caucasian (SM) was referred to the Orthodontic Department at age seven years, nine months by his

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Figure 1. Radiograph at age seven years and nine months, showing unerupted maxillary left central incisor. No evidence of odontome.

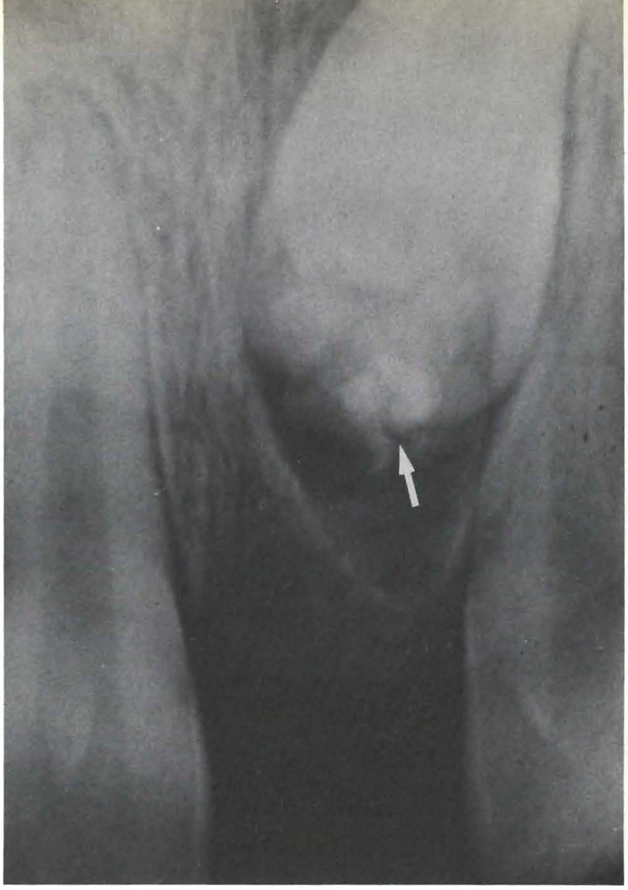

Figure 2. Radiograph before surgery, showing unerupted maxillary left central incisor and associated odontome.

general dental practitioner because of failure of eruption of his maxillary left central incisor. The antimere had erupted approximately fourteen to eighteen months previously and the maxillary left lateral incisor was now erupting in a mesial position. There was no history of trauma to this area of his mouth. An intraoral radiograph taken at this time (Figure 1) showed the maxillary left central incisor to be surrounded by bone, with little evidence of a crown follicle. There was no evidence of a developing odontome.

Twenty-five months later, a radiograph taken before surgical intervention showed a well-developed calcified structure, which was provisionally diagnosed as an odontome (Figure 2).

At operation (carried out under a general anaesthetic), the odontome was found to be about the size of a garden pea (Figure 3) and lying palatal to the crown of the unerupted central incisor. The incisor was not markedly displaced, but was high in the buccal sulcus, and consequently at the same time as the odontome was removed, gold chain was directly bonded to the labial surface of the unerupted tooth using the method described by Oliver and Hardy (Figure 4).[5]

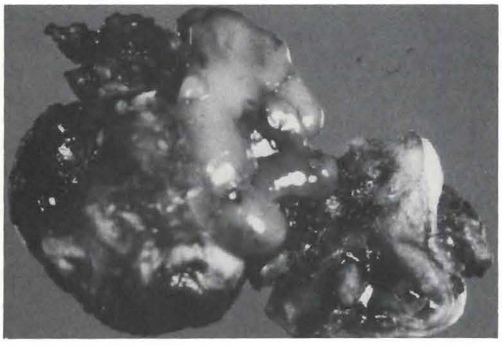

Figure 3. Odontome, following surgical removal. It was approximately 8 mm in diameter.

Figure 4. Gold chain bonded to the labial surface of the crown of the unerupted incisor. The flap was replaced over the crown of the tooth.

At the time the sutures were removed, traction was commenced from an edgewise appliance, which had been placed before surgery. Traction was continued for twelve months, when the central incisor erupted; a further twelve months of orthodontic treatment resulted in a Class I occlusion (Figure 5). Usually, no retention is required for cases in which a tooth has extruded; the newly erupted tooth, however, showed a tendency to move labially and consequently it was moved back into line with a Hawley bow and a multistranded wire was bonded palatally to both central incisors and the left lateral incisor for retention. The clinical crown length of the left central incisor is greater than its antimere; it is anticipated, however, that equal gingival heights will be achieved spontaneously over the next few years. The gingival margin of the left central incisor comprises keratinized mucosa, and the bony height is satisfactory radiographically (Figure 6). Mamelons are present on the newly erupted incisor and they contrast with the fractured incisal edge of the right central incisor. The normal process of attrition should reduce the mamelons with time. Oral hygiene was consistently poor throughout treatment and there was evidence of decalcification around the lateral incisors at the time the orthodontic appliance was removed. Treatment time would undoubtedly have been reduced, if the patient had managed to keep the appliance intact.

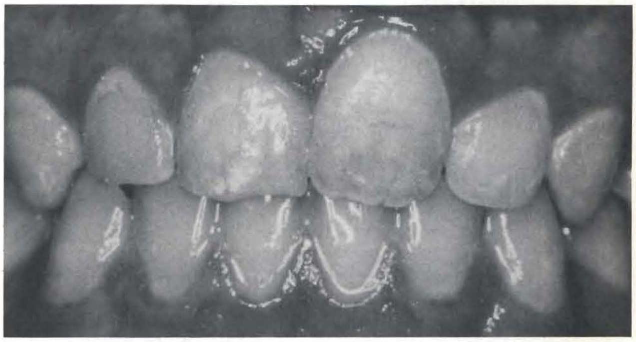

Figure 5. Dental appearance, fourteen months after removal of the fixed appliance. Note uneven gingival contour on the central incisors and poor oral hygiene.

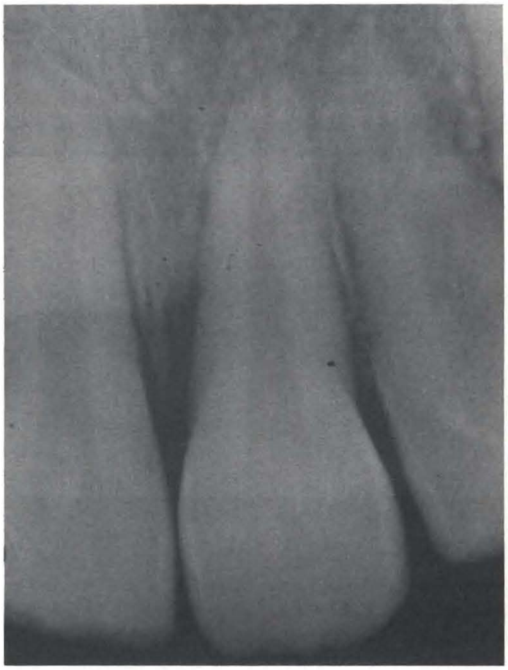

Figure 6. Radiograph of incisor, fourteen months after removal of the fixed appliance.

Histologic findings

Histopathological examination of a decalcified section of the excised lesion revealed elements of dentine, cementum, pulpal tissue, and immature enamel matrix, arranged haphazardly. Within the soft tissues, there was a fairly marked odontogenic epithelial component. These findings permitted the histopathological diagnosis of a complex odontome.

DISCUSSION

Shulman and Corio state that complex odontomes exhibit a predilection for the posterior mandible in females, whereas compound odontomes are found in the anterior maxilla with no sex bias.[6] They go on to report a case of delayed eruption of the maxillary left central incisor in a thirteen-year-old male. Although a small calcified structure was visible on a radiograph, it was not seen at the subsequent exposure and bonding of an edgewise bracket to the crown of the unerupted tooth. At the end of orthodontic treatment, a follow-up radiograph revealed the continued presence of the radiopaque body. This was subsequently removed and histologically shown to be a complex odontome.

At initial examination, the cause of the delayed eruption of the left central incisor for SM was unknown. It is likely, however, that the primordium of the odontome was present and was interfering with the normal process of eruption. Current thought on the mechanism of eruption suggests that the follicle plays a central role in the eruption process by providing a source of preosteoclastic monocytes in the coronal area of the follicle.[7] It may be that the presence of the primordium of the odontome inhibited the normal sequence of cytological events in the follicle of the unerupted incisor.

SUMMARY

A case is presented in which a maxillary left central incisor was delayed in its eruption. At initial examination, there was no known etiological factor; two years later, however, an intraoral radiograph revealed a substantial odontome. Treatment consisted of surgical removal of the odontome and application of orthodontic traction to the unerupted tooth. Histological examination confirmed a diagnosis of complex odontome.

The authors would like to thank Dr. Jeremy Bagg for his help with the histopathology report.

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LAW WEAKENS TENURE, UNIVERSITY AUTONOMY

Britain's academic community is nervously awaiting the implementation of a new law that will make sweeping changes in the organization and funding of British universities. Finally approved by Parliament last week after a protracted and intense political debate, it will, among other measures, abolish security of tenure both for all new university appointments and for those promoted to higher academic posts.

According to the Conservative government of Prime Minister Margaret Thatcher, the measures in the bill, which cover all levels of education from primary schools through to higher education, are intended to produce the most wide-ranging structural changes in Britain's education system for the past 40 years.

The most significant aspect of the new law from the universities' point of view is that, as from last November, nobody appointed to a university post either from outside or promoted from within his or her own department, will be guaranteed security of tenure. This would make it possible for universities to fire those whose academic performance becomes unacceptable.

The universities fought this measure. Although they did not get it deleted, they did at least persuade Education Secretary Kenneth Baker to temper the abolition of tenure by including a clause explicitly protecting academic freedom—something the government had originally been firmly resisting because it considered it unneccessary.

The new clause protecting academic freedom is the result of an amendment passed by the House of Lords, which the government decided not to challenge. It states that all academic staff "will have freedom within the law to question and test received wisdom, and to put forward new ideas and controversial or unpopular opinions, without placing themselves in jeopardy of losing their jobs or the privileges they may have at their institutions."

The government also backed away from its original proposal that universities should have the right to replace a highly paid research worker with one prepared to do the same work for a lower salary. Universities had warned that such a clause would threaten the ability of universities to attract and keep world-class research workers, and the government subsequently accepted new wording ensuring that academics whose research performance is judged satisfactory can be laid off only if their job disappears.

Dickson, D.; Law weakens tenure, university autonomy.
Science, 241: 652, 653, 5 August, 1988.

Ameloblastic fibrodentinoma: report of case

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Michael H. Mason, DDS
Randall Barkley, DDS

The ameloblastic fibrodentinoma is a mixed odontogenic tumor that is closely associated with the ameloblastic fibroma, the ameloblastic fibroodontoma, and the complex odontoma. The purpose of this report is to present a case of ameloblastic fibrodentinoma, briefly review the mixed odontogenic tumors and discuss the controversy surrounding the classification and progression of mixed odontogenic lesions.

AMELOBLASTIC FIBROMA

The ameloblastic fibroma is a relatively uncommon neoplasm composed of both epithelial and mesenchymal elements, but without evidence of calcified tissue.[1-3] The average age of onset for this lesion is 14.6 years; 40 percent of involvement is in children less than ten years of age. Males are more commonly affected than females. The posterior mandible is the most frequent location, and the tumor is often associated with an unerupted tooth, mimicking the ameloblastoma.[4] Only twelve cases of ameloblastic fibroma of the maxilla have been reported and only three of these were found in the anterior region.[5] Slow, asymptomatic growth is the usual finding, with enlargements of the jaw and migration of teeth common.[2] Radiographically, the lesion presents as a unilocular or multilocular radiolucency that may or may not include cortical plate expansion. In histologic section, epithelial cells are present in scattered islands and resemble primitive odontogenic epithelium, while the mesenchymal component appears as a primitive connective tissue with large cells similar to those of dental pulp.

AMELOBLASTIC FIBROODONTOMA

The ameloblastic fibroodontoma was initially grouped with the ameloblastic odontoma, which is an aggressive tumor with neoplastic elements of the ameloblastoma and tendencies toward local invasion of bone and recurrence after treatment. Hooker, in 1967, reviewed twenty-six cases diagnosed as ameloblastic odontoma and separated a slowly growing, noninvasive, and histologically distinct entity he subsequently classified as the fibroodontoma.[6]

Since that time many cases of ameloblastic fibroodontoma have been reported.[7-13] The tumor occurs with equal frequency in the maxilla and mandible, usually in the posterior region. Almost all cases are found in the first two decades of life, pain is normally absent, and no apparent sex predilection is noted. Radiographic presentation is characterized by a well-defined unilocular or multilocular lesion with multiple opacities. This

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slowly growing, expansile central neoplasm is frequently associated with an unerupted tooth, and can cause displacement of adjacent teeth. Histologically, primitive odontogenic epithelium and connective tissue similar to the ameloblastic fibroma are present, along with evidence of enamel and dentin calcification, resembling the complex odontoma, but usually in a more undifferentiated and less organized state.[4]

COMPLEX ODONTOMA

The complex odontoma is a well-differentiated, easily recognizable lesion composed of functional ameloblasts and odontoblasts, along with enamel, dentin, pulp, and cementum.[4,14,15] These structures may lie in a normal relationship to one another and the calcified masses may resemble teeth, or they may appear randomly organized and bear little resemblance to normal dental units. Mean age of occurrence is 14.8 years with a slight preference for males. The location of the compound (toothlike) odontoma is most frequently the anterior maxilla while the composite (nontoothlike) most often occurs in the maxillary or mandibular posterior regions. The lesions are usually asymptomatic and are detected upon routine radiographic examination as calcified masses, but occasionally they will cause the impaction of normal teeth or enlarge and present as a swelling. They do not invade adjacent structures.

AMELOBLASTIC FIBRODENTINOMA

Cases of mixed odontogenic tumors have been reported that consist of undifferentiated odontogenic epithelium in strands and cords similar to that found in both the fibroma and fibroodontoma, and with evidence of the dentin-like calcification found in the fibroodontoma, but without any sign of enamel.[16-20] Pindborg and Clausen describe this tumor as an immature dentinoma: Gorlin *et al* labeled it as a fibroameloblastic type of dentinoma; and Van Wyk and Van der Vyer called it an ameloblastic fibroma with dentinoid formation.[16,18,20] More recently the term ameloblastic fibrodentinoma has been used.[4,17]

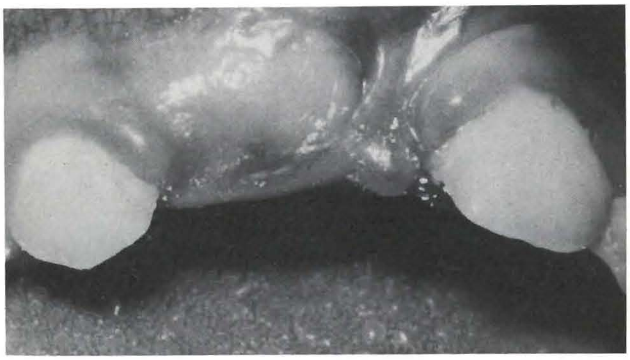

Figure 1. Appearance of maxillary incisor region. Note missing right primary central incisor and slight alveolar enlargement.

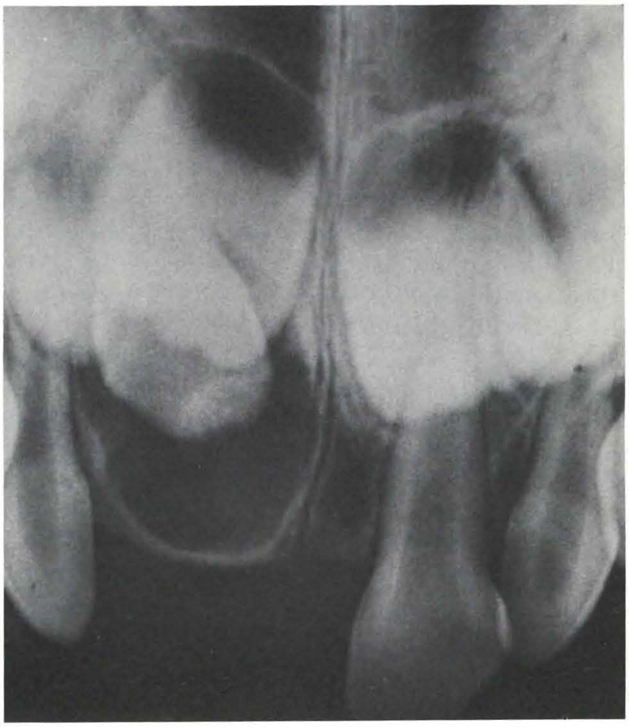

Figure 2. Periapical radiograph demonstrating an unerupted right primary central incisor and associated radiolucency.

CASE REPORT

A two-year-old Caucasian female presented for evaluation with a chief complaint of an unerupted primary central incisor. Clinical examination revealed a completely erupted primary dentition with the exception of the maxillary right central incisor. A slight enlargement of the labial alveolar cortical plate was noted in the edentulous area (Figure 1). The child's health history was negative and the remainder of the clinical examination was within normal limits.

Periapical radiography revealed an unerupted primary central incisor surrounded by a well-circumscribed radiolucency (Figure 2). A provisional diagnosis of dentigerous cyst was rendered and a decision made to perform an excisional biopsy under general anesthesia. The primary incisor and associated cyst (Figure 3) were removed without incident and healing was uneventful.

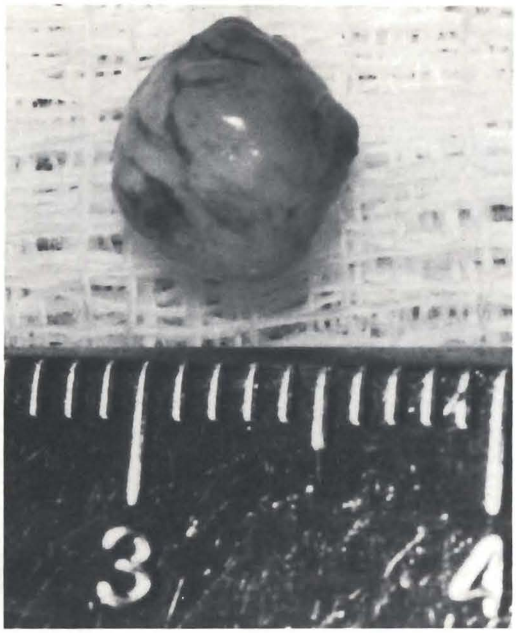

Figure 3. Well-encapsulated lesion removed from radiolucent area.

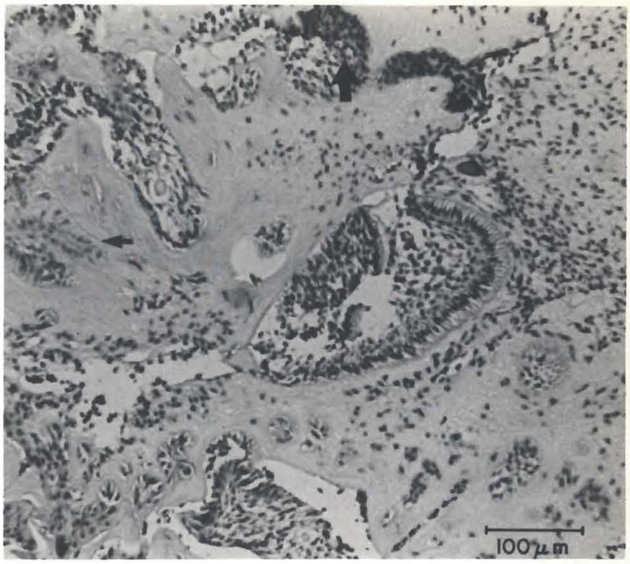

Figure 4. Cells resembling ameloblasts with polarized, palisaded nuclei and centrally located stellate reticulum are intimately associated with eosinophilic materials with features of dentinoid. A few irregular tubular structures (large arrow) are noted as well as cells with features of odontoblasts (small arrow). (Original magnification 40 X)

Histopathological examination of the cystic lesion revealed a cellular myxomatous stroma containing strands and cords of odontogenic epithelium resembling enamel organ components. Evidence of inductive influence was present and in some areas cells with features of odontoblasts were noted in association with dentinoid and showing focal evidence of irregular calcification (Figure 4). No features of ameloblastoma were found, and the final diagnosis was ameloblastic fibrodentinoma.

At the six-month follow-up evaluation, the surgical site was well healed and bone-fill evident (Figures 5,6). Recurrence of the lesion was not observed nor is it anticipated.

DISCUSSION

Most authors agree that the treatment of choice for isolated lesions of the jaws is simple enucleation and removal of involved tooth structures.[4,9,21] Shafer and Bhaskar, however, suggested a somewhat more aggressive approach for the ameloblastic fibroma because of the tendency for recurrence and possible malignant transformation (to ameloblastic fibrosarcoma).[4,22] A conservative approach is recommended for the ameloblastic fibroodontoma and the ameloblastic fibrodentinoma since the recurrence rate in the only study with long-term follow-up was 1 in 29.[23] Treatment of the odontoma is simple surgical removal and there is no expectancy of recurrence.[4]

It is appealing to classify all four discussed lesions as different stages of the same entity. In normal odontogenesis, epithelium is necessary for the differentiation of odontoblasts, and dentin deposition is necessary for and followed by enamel formation. Some authors feel, therefore, that the ameloblastic fibroma will eventually produce dentin-like material, becoming the ameloblastic fibrodentinoma.[2,4] Enamel formation would then signal the fibroodontoma stage, and the replacement of soft tissue by highly differentiated calcified tissue represents the final state, or odontoma.

Eversole, to the contrary, suggests that progression of the immature fibroma into the odontoma does not occur, and each stage is an end in itself characterized by aborted odontogenesis.[19] More recently, however, Eversole has accepted the theory that the fibrodentinoma represents a fibroma that has differentiated to the extent that dentin is elaborated.[24] Slootweg concluded that while the ameloblastic fibroma represents a separate entity that does not develop further, the ameloblastic fibroodontoma is in fact an immature odontoma.[7] Trodahl points out that odontomas must go through a developmental stage that is histologically identical to the ameloblastic fibroma.[25] Van Wyk and Van der Vyver suggest that the epithelial/connective tissue interface on their "ameloblastic fibroma with dentinoid formation" contained some features of the developing tooth and did not rule out its eventual differentiation into an odontoma.[18]

Gardner suggests that the nomenclature of mixed odontogenic tumors should be standardized and simplified[26] Terms such as ameloblastic fibrodentinoma and immature dentinoma should be changed to ameloblastic fibroma with dentin, and ameloblastic odontoma should be reclassified as odontoameloblastoma.

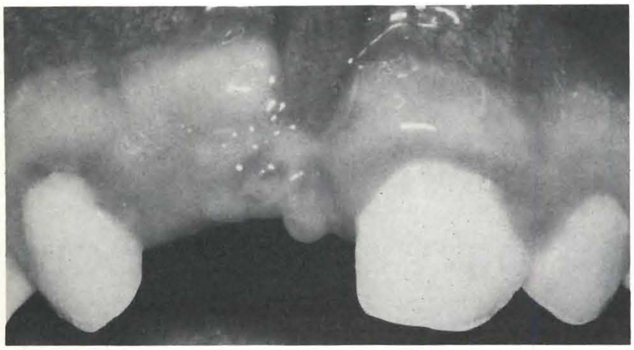

Figure 5. Appearance of surgical site at six-month recall visit.

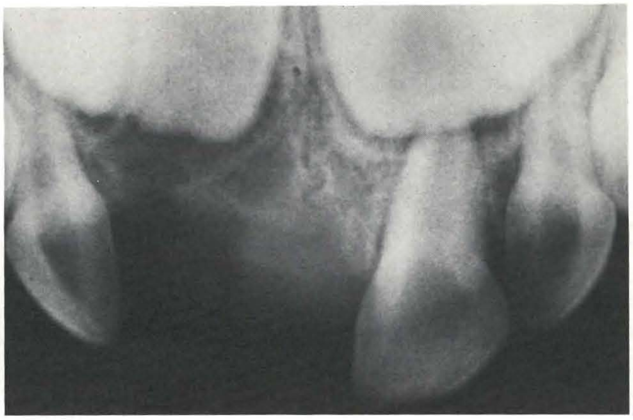

Figure 6. Periapical radiograph of surgical site at six-month recall visit. Bone fill can be observed.

CONCLUSION

The classification, progression, and treatment of the mixed odontogenic tumors remain controversial. In the case reported here, examination of a patient who presented with delayed eruption of a primary central incisor led to the discovery of an impacted incisor and an associated isolated radiolucency. By histological examination, it was determined that the lesion was an ameloblastic fibrodentinoma, which is an ameloblastic fibroma with dentinoid formation. This case is presented because of the unusual location of the lesion and its unique histologic appearance.

Delayed eruption of primary teeth should be investigated with the same thoroughness as retarded eruption of the permanent dentition. If, upon radiographic examination, a radiolucency associated with an unerupted tooth is discovered, prudence dictates immediate intervention. Biopsy is a necessity since the only reliable means of establishing a definitive diagnosis is by careful coordination of histologic and clinical findings. Because the most common lesions will not recur, a conservative approach is indicated, and should include enucleation of the lesion and extraction of the associated primary tooth. Close follow-up is recommended with all mixed odontogenic tumors, considering the paucity of adequate long-term studies and the aggressiveness of some closely related entities, including the ameloblastic fibrosarcoma, ameloblastoma, and ameloblastic odontoma.

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Long-term interdisciplinary management of multiple mesiodens and delayed eruption: report of case

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Hyperdontia, variation in the number of teeth in excess of that found in the normal dentition, occurs during the initiation phase of tooth development and may manifest itself clinically as either supernumerary or supplemental teeth. Supernumerary teeth are malformed morphologically, while supplemental teeth resemble more closely the adjacent unaffected teeth. The presence of hyperdontia has been reported variously to occur in 0.45 percent to 2.0 percent of the general population, but with much greater frequency in individuals with cleidocranial dysostosis and cleft lip and palate.[1-7]

The most common site for hyperdontia is in the anterior region in the midline of the maxillary arch. Supernumerary teeth in this location are termed mesiodens and have been reported to represent 80 percent of all impacted supernumerary teeth.[8] Mesiodens may be: single or multiple; unilateral or bilateral; conical, tuberculate, or normal in size and shape; erupted or impacted; inverted with the crown directed toward the nasal cavity or positioned with the crown directed toward the dental arch; and can occur simultaneously with the congenital absence of other teeth.[9]

The presence of mesiodens often has deleterious effects upon the dentition and dental arch.[10] Complications to the developing occlusion may result in impaction, delayed eruption, or ectopic eruption of the permanent incisors, or could contribute to the development of a maxillary midline diastema.[11] Left untreated, mesiodens can contribute to bone destruction, root resorption, pulpal pathosis, or the development of cysts.[12,13]

Numerous case reports have been presented which describe the clinical management of supernumerary teeth.[14-16] Few investigative studies that quantify data to facilitate appropriate treatment planning, however, have been reported.[8,17] Primosch, in 1981, presented a thorough review and proposed guidelines for a three-step approach to surgical management of anterior supernumerary teeth in children.[17] This author recommended:

- ☐ Immediate surgical removal.
- ☐ Surgical exposure of the unerupted incisor crown after six months.
- ☐ Orthodontic traction of the tooth in the absence of spontaneous eruption.[17]

In 1983, Nazif, Ruffalo, and Zullo reviewed fifty cases of impacted supernumerary teeth.[8] These authors rec-

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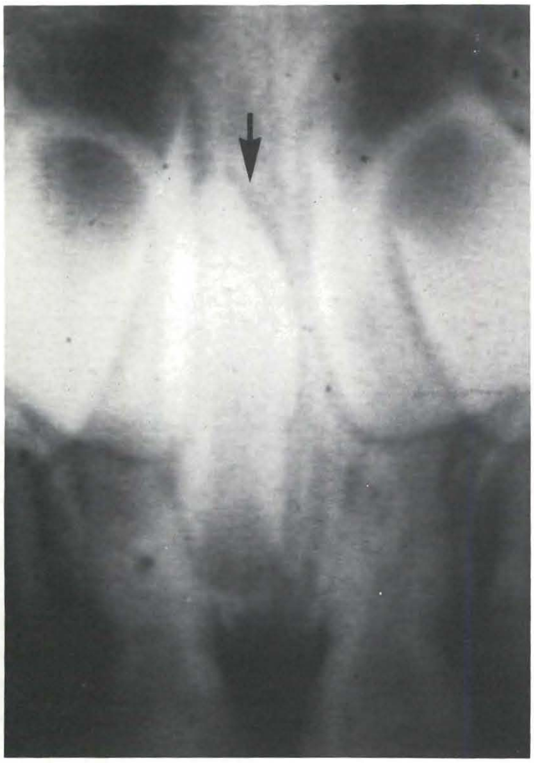

Figure 1. Periapical radiograph, at age 4.1 years, demonstrating the presence of a single impacted inverted mesiodens.

ommended early surgical removal of the supernumerary tooth:

- ☐ If the eruption of the permanent tooth had been impeded.
- ☐ If the supernumerary tooth appeared inverted or rudimentary.
- ☐ Or if the supernumerary tooth was associated with pathosis. Further, they suggested that complications associated with early removal were either infrequent or minor.[8]

The purpose of this paper is to present a report of a patient with multiple mesiodens and delayed eruption over a ten-year period. Further, it emphasizes the need for patient individuality and interdisciplinary cooperation in long-term treatment planning.

CASE REPORT

The patient was first seen for an emergency visit following trauma to the primary maxillary anterior teeth as the result of a fall sustained earlier that day. The patient was a well-nourished, well-developed, 4.1 year old white male in no apparent distress. The medical history was noncontributory except for periodic episodes of pneumonia.

Clinical examination revealed a complete primary

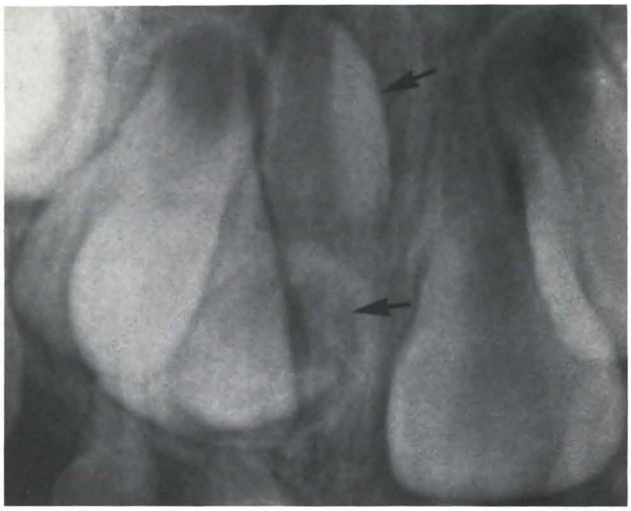

Figure 2. Maxillary anterior occlusal radiograph, at age 6.1 years, demonstrating the presence of the second mesiodens.

dentition with no caries or previous restorations. A Class I primary occlusion with anterior spacing was noted. The traumatized teeth showed no crown fractures, no mobility, no discoloration, and the surrounding soft tissues were not damaged. Radiographic examination revealed no root fractures or displacement. A single, inverted, impacted mesiodens, however, was discovered (Figure 1). It was decided that the mesiodens would be observed for a time, relative to the resorption of the roots of the primary central incisors and the eruption of the permanent central incisors.

The patient was seen over the next two years for routine preventive services and for follow-up of the mesiodens. At the age 6.1 years, a second impacted mesiodens was discovered (Figure 2). Additionally, during this same period, the medical condition of the patient became more complicated. Due to repeated episodes of pneumonia, the patient was referred by his pediatrician to the allergist. Allergy testing was negative and a subsequent diagnosis of childhood asthma was made. Asthmatic attacks continued and the patient was placed on 200 mg doses of theophylline every twelve hours and albuterol metered dose inhaler, two inhalations repeated every four to six hours as needed. At age seven years, the patient experienced a severe asthmatic attack which required paramedic transport to the hospital emergency room. On the advice of the allergist, surgical intervention to extract the mesiodens was delayed.

By age 7.5 years, the maxillary left primary central incisor had exfoliated and the maxillary left permanent central incisor had erupted, while the maxillary right primary central incisor remained present clinically and

Figure 3. Maxillary anterior occlusal radiograph, at age 7.5 years, demonstrating delayed eruption of the maxillary right permanent central incisor.

Figure 4. Maxillary anterior occlusal radiograph, six months postextraction, demonstrating lack of spontaneous eruption of the maxillary right permanent central incisor.

the maxillary right permanent central incisor had yet to erupt (Figure 3).

The medical condition of the patient improved steadily, so that by 7.8 years, he no longer took medication and had not experienced an asthmatic attack for over eight months. A joint decision was reached among the allergist, the oral surgeon, the orthodontist, and the pediatric dentist to intervene surgically. The patient was admitted by the oral surgeon to the short-stay surgical unit of the hospital. The overretained maxillary right primary central incisor and both impacted mesiodens were extracted, under general anesthesia. The patient tolerated the procedure well, the extractions were uncomplicated, and healing progressed normally.

Spontaneous eruption of the maxillary right permanent central incisor was anticipated, following the removal of the mesiodens. Six months postoperatively, however, the tooth had not erupted and was covered with newly formed bone (Figure 4). In preparation for the surgical exposure of this tooth, molar bands were placed by the orthodontist on the maxillary permanent first molars, brackets were bonded to the three erupted maxillary incisors, and an .0175 wire was positioned (Figure 5).

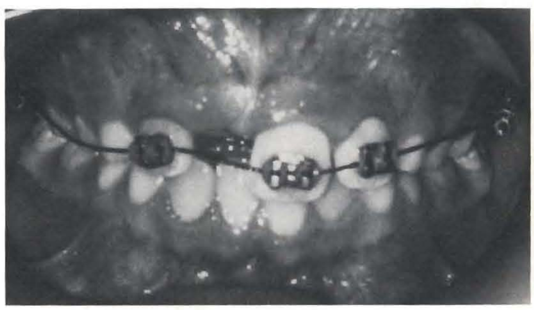

Figure 5. At age 8.8 years, patient prepared orthodontically for the surgical exposure of the maxillary right permanent central incisor.

Surgery to expose the crown was delayed, due to another traumatic injury experienced by the patient while playing. The bonded bracket on the maxillary left permanent central incisor had been dislodged and this tooth was extruded approximately 1 mm. The tooth was repositioned and the bonded bracket was replaced.

Figure 6. At age 9.1 years, maxillary right permanent central incisor being moved incisally.

At age nine years, the patient was admitted to the short-stay surgical unit of the hospital. Under intra-

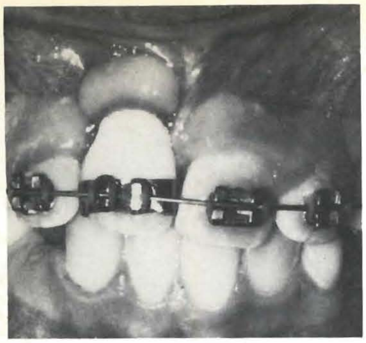

Figure 7. At age 9.4 years, maxillary right permanent central incisor alignment within the arch.

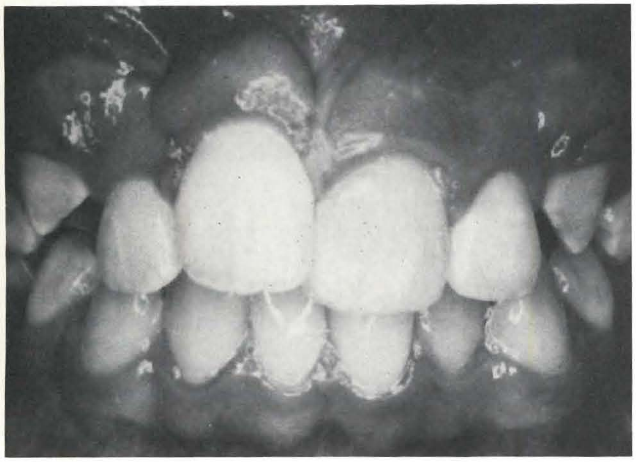

Figure 8. At age 10.8 years, phase one orthodontic treatment completed.

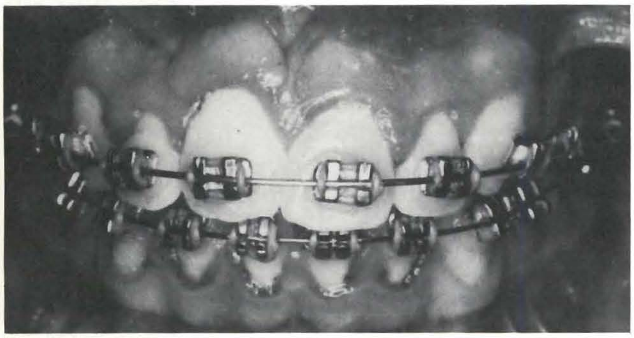

Figure 9. At age 13 years, during phase two orthodontic treatment.

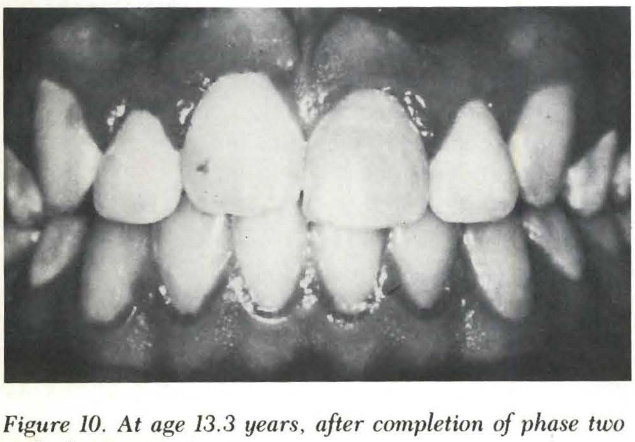

Figure 10. At age 13.3 years, after completion of phase two orthodontic treatment.

venous sedation, the crown of the maxillary right permanent central incisor was exposed, a sliding tissue flap was positioned, and a button bracket was bonded to the labial aspect of the crown. The surgical procedure was tolerated well by the patient and healing was uneventful.

The tooth was moved incisally using elastics (Figure 6). Three months postoperatively, the bonded button bracket was replaced with a banded bracket. A flex archwire with elastics was placed (Figure 7). At 10.8 years, this phase of the orthodontic treatment was completed and the patient was placed in retention (Figure 8).

At age 11.8 years, following the eruption of the permanent dentition, additional orthodontic treatment, utilizing a maxillary headgear for retraction in conjunction with alignment of both the maxillary and mandibular teeth, was required (Figure 9). Total treatment time for the second phase of orthodontic treatment was twenty months (Figure 10). The patient is currently fourteen years of age and in retention.

DISCUSSION

Under normal circumstances, surgical intervention for the extraction of the mesiodens in this case would have been initiated at an earlier age. The decision to delay treatment was based, however, upon the medical condition of the patient at that time. Eventually, at around eight years of age, the asthmatic attacks ceased and have not recurred. The ultimate long-term results of the dental treatment have been functional and esthetically satisfactory.

The crown of the maxillary left permanent central incisor exhibits a mild blue discoloration. Whether this discoloration is the result of the presence of the mesiodens, the surgical intervention, or the traumatic episodes is difficult to determine definitively. This tooth is asymptomatic, however, and registers a pulp vitality test response in the normal range, relative to the adjacent teeth.

The gingival tissue around the crown of the maxillary right permanent central incisor is healthy and contoured to the tooth. It is positioned at an esthetic level in relationship to the level of the gingival tissue surrounding the contralateral tooth.

This report has attempted to contribute to the existing knowledge for the treatment of mesiodens, to emphasize the need to individualize the treatment plan, and further, to demonstrate the importance of cooperation among dental and medical specialists to improve total patient care.

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INFANT MORTALITY

Of the 3,760,561 infants born in the United States in 1985, 40,030 died before reaching their first birthday. Of these, 26,179 died during the neonatal period (the first twenty-eight days of life), a rate of 7.0 deaths per 1,000 live births. During the postneonatal period (twenty-nine days to one year of life), 13,851 died, a rate of 3.7 deaths per 1,000 live births.

Between 1984 and 1985, the overall infant mortality rate declined from 10.8 to 10.6 deaths per 1,000 live births. (Neonatal rates added to postneonatal rates do not exactly equal the overall infant mortality rates due to rounding.) According to the National Center for Health Statistics (NCHS) of the U.S. Department of Health and Human Services, this decline was "not statistically significant." This means that the amount of decline was so small it could be attributed to random chance.

☐ According to NCHS, overall infant mortality rate decreases among each racial classification—whites, blacks, and nonwhites—also were not statistically significant.

☐ The 1985 stagnation in overall national infant mortality followed the slowdown in progress in reducing infant mortality that had been occurring since 1981. The 1985 stagnation was predicted a year ago by NCHS based on preliminary 1985 data and at that time was felt to be "consistent with the slowing in the rate of decline in infant mortality since 1981, following two decades of sustained decline."

Hughes, D.; Johnson, K.; Rosenbaum, S. *et al; The Health of America's Children.* Washington, D.C.: Children's Defense Fund, 1988, p 3.

ABSTRACTS

Harrington, Marilyn S.; Eberhart, Adele B.; Knapp, Jane F.; Dentofacial trauma in children. J Dent Child, 55: 334-338, September-October, 1988. This study identified dental and facial injuries. Of the 501 children reviewed here, 60 percent were boys and 70 percent were six years old or younger. The most frequent injuries were lacerations, intrusion/ extrusion, tooth fracture, and fracture of the mandible or maxilla. The leading causes of injury were fall, being struck by an object, bicycle accidents, assaults, and motor vehicle accidents. Risk-taking and aggressive behaviors, lack of adult supervision, and pre-existing handicaps related to injury.

Trauma, dentofacial; Fractures; Injuries, pediatric

Whitehead, B, Gene; Durr, David P.; Adair, Steven M.; Proskin, Howard M.; Monitoring of sedated pediatric dental patients. J Dent Child, 55:329-333, September-October, 1988. Pulse oximetry is a noninvasive technique used to monitor the oxygen saturation of hemoglobin (SaO_2) in sedated patients. It provides an earlier warning of respiratory distress than do traditional methods of monitoring blood pressure, heart rate, respiratory rate, and skin color. In this study of two of twelve children, sedated with 50 mg/kg body weight chloral hydrate, 25 mg hydroxyzine, and 40 percent nitrous oxide: 60 percent oxygen, had respiratory difficulties during dental treatment

Continued on page 381

from the cementoenamel junction to the alveolar bone, probably due to continuous eruption. This information should be taken into consideration for the assessment of the periodontal condition of the primary dentition.

Periodontal disease; Alveolar bone crest, interproximal; Molars, primary; Pediatric dentistry

Bimstein, Enrique; Machtei, Eli; Becker, Adrian: The attached gingiva in children: diagnostic, developmental and orthodontic considerations for its treatment. J Dent Child, 55:351-356, September-October, 1988. The diagnosis and treatment of gingival recession, at the permanent mandibular incisor area in children, should take into consideration normal development of the entire gingival unit and arch form. During the transition from the primary to the permanent dentition, areas of less than 1 mm in width of attached gingiva are normal. Tooth position has a definite correlation with the width of the attached gingiva. Incisor tooth alignment may be achieved as the result of intercanine growth, slicing or extraction of the primary canines, orthodontic treatment, or a combination of these, with an improvement of the gingival contour. A surgical approach should be considered only after a conservative one proves to be insufficent and/or when the recession is beyond the cementoenamel junction. Placque control appears to be the most important factor in limiting—and possibly reversing—an otherwise progressive gingival recession.

Gingival attachment; Diagnosis; Oral pathology; Orthodontics

Bimstein, Enrique and Eidelman, Eliezer: The statistical unit for analysis of developmental changes in the attached gingiva in children. J Dent Child, 55:357-358, September-October 1988. The issue of an adequate statistical unit in periodontal studies, sites or mean per patient, has been the subject of discussion among researchers and clinicians. The attached gingival width is a measurement subject to developmental changes throughout life. It appears inappropriate to obtain a mean unit per patient.

Clinical measurements; Statistical analysis; Gingiva, attached

Hagman, F. Thomas: Anomalies of form and number, fused primary teeth, a correlation of the dentitions. J Dent Child, 55:359-361, September-October, 1988. Fusion is the most common anomaly found in the primary dentition. This review looks at the frequency of missing, fused, and geminated teeth, and the effect of fusion as it relates to the permanent dentition. Fusion occurs primarily in the lower anterior area of the primary dentition, and its location has a predictive effect on the developing teeth. Aplasia of the succedaneous lateral incisor is a 75 percent chance when primary canines and laterals are fused, and a less than 20 percent chance when the primary incisors are fused.

Fused teeth, primary; Anomalies, dental

Surmont, Paul A.; Martens, Luc C.; De Craene, Luc G.: A complete fusion in the primary human dentition: a histological approach. J Dent Child, 55:362-367, September-October, 1988. After the description of a case of an extracted, complete fusion, the literature dealing with the phenomenon of fusion and gemination is briefly reviewed. In this report, clinical and roentgenological information were compared to the histological data which had revealed an unexpectedly complex internal structure. Endodontic treatment would probably have been inopportune in this case. This histological examination did not give more information about the origin of double teeth. A preventive approach to this phenomenon—sealing the remaining fissures and fluoridating the smooth surfaces—is presented.

Fused teeth, primary; Histology

Oliver, Richard G. and Hodges, Christopher G.L.: Delayed eruption of a maxillary central incisor associated with an odontome: report of case. J Dent Child, 55:368-371, September-October, 1988, A maxillary left central incisor was found to be delayed in its eruption, with no apparent etiological factor. Two years later, an intraoral radiograph revealed a substantial odontome, which was removed surgically. Orthodontic traction was applied to the unerupted tooth. Histological examination confirmed the diagnosis of complex odontome.

Odontomes, compound (or) complex

Brandt, Stephen K.; Mason, Michael H.; Barkley, Randall: Ameloblastic fibrodentinoma: report of case. J Dent Child, 55:372-375, September-October, 1988. Investigation into the delayed eruption of a primary central incisor in a two-year-old child led to the discovery of an unerupted tooth and associated radiolucency. Histological examination identified the lesion as an ameloblastic fibrodentinoma, a rarely reported mixed odontogenic tumor similar to the ameloblastic fibroma. The maxilla, especially the anterior region, is an unusual location for both the ameloblastic fibrodentinoma and ameloblastic fibroma. Timely evaluation of delayed eruption of primary teeth can lead to early recognition and management of expansile neoplasms.

Ameloblastic fibrodentinoma: Neoplasms, oral

Ranalli, Dennis N,; Buzzato, John F; Braun, Thomas W.; Murphy, Steven M.: Long-term interdisciplinary management of multiple mesiodens and delayed eruption: report of case. J Dent Child, 55:376-380, September-October, 1988. A patient, followed over a ten-year period, had two impacted mesiodens accompanied by delayed eruption of the maxillary right permanent central incisor. The initial surgical intervention was delayed due to periodic episodes of childhood asthma.

The patient required two surgical procedures: one to extract the two mesiodens; the second to expose the crown of the unerupted permanent central incisor. Two phases of orthodontic treatment were required: the initial phase to move the impacted central incisor into the arch; and to align the permanent teeth within the arches. This report emphasizes the need for patient individuality and interdisciplinary cooperation in patient care.

Mesiodens, multiple; Eruption, delayed

Evaluation of Wear by Application of a Surface Sealant. G. Dickinson; K.F. Leinfelder; and C.M. Russell. University of Alabama at Birmingham, School of Dentistry, Birmingham, AL. It has been theorized that the phenomenon of surface wear on composite resins may be due in part to surface and subsurface cracks that are initiated during the finishing process. The purpose of this study is to investigate the effects of applying a surface sealant to posterior composite resins after conventional finishing.

Sixty-two samples of a proprietary composite resin (BIS-FIL) were inserted into Class I and II cavity preparations. In each case, conservative amalgam type preparations were employed. The occlusal cavosurface margins were not beveled. The cavity preparations were restored and cured in increments. All restorations were surfaced by conventional techniques, using twelve-fluted finishing burs, white stones and polishing points. On a randomized basis, 50 percent of the completed samples were treated with a specially formulated surface sealant. The sealant essentially consists of low viscosity resin composed of Bis-GMA, TEGMA, and THFMA. All restorations were impressed with a poly vinyl siloxane impression material, and cast in improved stone. All restorations were evaluated in accordance to the USPHS System, M-L Scale and Calibrated Cast System. At the end of six months, the loss of composite from the occlusal surface, when no surface sealant was employed, averaged thirty-one microns. By comparison, those that were sealed exhibited an average loss of only twenty microns or 35 percent less. On the basis of this phase of the study, it would appear that an appropriate surface sealant at the time of insertion can reduce appreciably the wear of posterior composite resins.

Resins, posterior composite; Sealant surface

NEWS

DECLINE IN CHILDREN'S CARIES REPORTED

Half of the schoolchildren in the U.S. have no tooth decay, according to a new government study conducted by the National Institute of Dental Research (NIDR).

Almost 40,000 children nationwide were surveyed by the NIDR during the 1986-87 school year. Institute epidemiologists compared the survey's results to those of a 1979-80 survey.

American children have 36 percent less dental caries than they did at the beginning of the 1980s, researchers found. That decline follows a similar drop in the prevalence of tooth decay during the 1970s.

"What we are seeing is the beginning of the end for a disease that has plagued mankind throughout history," said Dr. Harald Löe, director of NIDR.

Not only are fewer children getting cavities today, but those who do are getting fewer of them. In 1980, children had an average of almost five decayed, missing or filled surfaces on their permanent teeth. In 1987, children had an average of only three decayed, missing or filled tooth surfaces.

The average number of decayed or filled surfaces on the primary teeth of five-to-nine-year olds also dropped from more than five in 1980 to less than four in 1987.

Institute officials believe the widespread use of fluoride is mainly responsible for the decline in dental caries.

Decay on the smooth surfaces of teeth is disappearing. Today, two-thirds of existing caries is found on the occlusal surfaces of teeth. Decay on these tooth surfaces could be eliminated, NIDR officials say, by the combined use of fluorides and adhesive sealants.

Interproximal caries is also approaching eradication, according to survey findings. The prevalence of interproximal caries dropped 54 percent between 1980 and 1987, while the prevalence of decay on the chewing surfaces and the exposed smooth surfaces of teeth dropped 32 percent.

Children in the Northeast and Pacific Coast regions of the country continue to have the highest prevalence of tooth decay, while youngsters in the Southwest have the least caries, the survey found. Females have slightly more decay than do males—a pattern also seen in earlier surveys.

NIDR, one of the federal government's National Institutes of Health, celebrated its 40th anniversary on June 24, 1988. With the addition of the results of the children's survey, the dental institute now has reliable data on the oral health of some 150 million Americans aged five to eighty-five plus.

Two years ago NIDR conducted a national survey of oral health in adults. The overall results from that survey, added to the children's survey results, show continuing improvement in the