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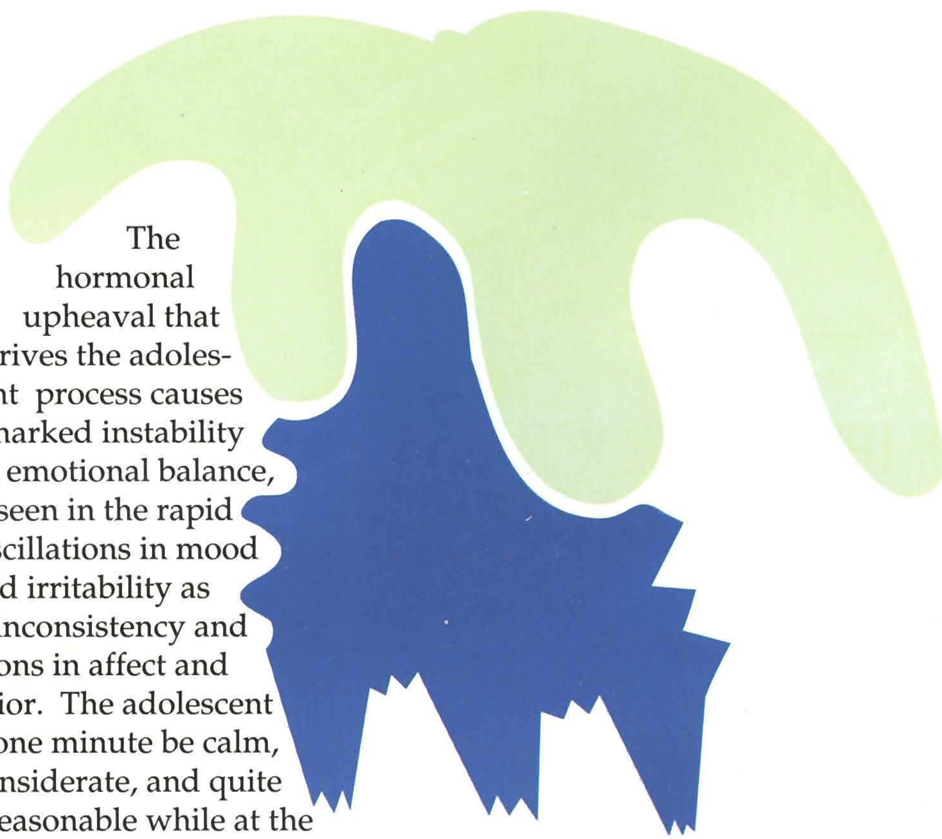
AMERICAN SOCIETY OF DENTISTRY FOR CHILDREN

JOURNAL OF DENTISTRY FOR CHILDREN

SEPTEMBER–OCTOBER 1991



The hormonal upheaval that drives the adolescent process causes marked instability in emotional balance, seen in the rapid oscillations in mood and irritability as well as inconsistency and vacillations in affect and behavior. The adolescent may one minute be calm, considerate, and quite reasonable while at the next moment one notes depression or preschool-like temper tantrums, tears, and screaming demands for his or her own way.

There is great individual variation in onset and timing of the physiological changes that mark the beginning of puberty. In girls, there is an average two-year lead over boys in physical development, and their cognitive advances usually far exceed those of boys during the early phases of adolescence.

—*Robert C. Prall, M.D.*

LIFE HAS NO MEANING EXCEPT IN TERMS OF RESPONSIBILITY.

—*Faith and History (1949)*

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One of the purposes of adolescence is to achieve mature modes of internal regulation. Character formation is an integrative process that aims at the elimination of conflict and arousal of anxiety.

Cover art and design by Sharlene Nowak-Stellmach

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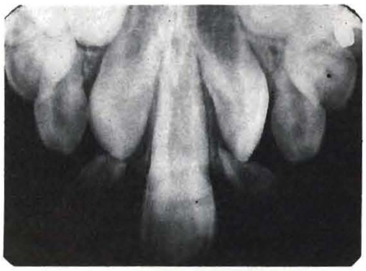

EPIDEMIOLOGY

FLUORIDATION

CASE REPORTS

For the busy reader

Child abuse: A survey of ASDC members and a diagnostic-data assessment for dentists—page 361

Child abuse is defined as the non-accidental physical injury, minimal to fatal, inflicted upon children by persons caring for them. The dentist's role: to observe and examine any suspicious evidence; to record such evidence, including statements; to remain objective; to treat any dental injuries; to maintain a therapeutic relationship with the family; and to hold the child whose life is in danger, and transfer that child for proper medical care.

Requests for reprints should be directed to Dr. Michael D. Saxe, Pediatric Dentistry, Bruce Medical Annex, Suite G, 2545 S. Bruce Street, Las VfWBs, NV 89109.

Pediatric dentists need to know about the changing economics of health care—page 367

During the 1980s, there were changes not only in the general structure of the health-care industry, with a shifting of services to other facilities, but also many innovations in payment mechanisms — new approaches to modify and control the marketplace. Dramatic increases in expenditures for health services are expected. There may even be increased funds for children's dental services.

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

Evaluation of treatment times for class II composite resin restorations—page 372

Despite preventive measures, restorative therapy continues to be the type of treatment most frequently provided by dentists. The time spent on each class II composite resin restoration was registered within the framework of a clinical study. The results, and their statistical inferences, based on the treatment times of a restricted number of restorations made by three dentists, obviously should not be considered as the sole basis on which the fee for restoration can be established. The learning effect has an influence as well.

Requests for reprints should be directed to Dr. C.M. Kreulen, Department of Pediatric Dentistry, Academic Centre for Dentistry Amsterdam (ACTA), Louwesweg 1, 1066 EA Amsterdam, The Netherlands.

The prevalence of postoperative sensitivity in teeth restored with class II composite resin restorations—page 378

There is much less consensus about the number of complaints of postoperative pain after composite resin restorations are placed, when compared with the widely recognized phenomenon of a short interval of pain after other restorations of teeth. This study involved 244 Class II restorations provided for fifty-six patients, who were between fifteen and thirty-five years of age. A fourth of the patients accounted for 60 percent of the complaints.

Requests for reprints should be directed to Dr. P.J. Borgmeijer, Department of Pediatric Dentistry, Academic Centre for Dentistry Amsterdam (ACTA), Louwesweg 1, 1066 EA Amsterdam The Netherlands.

Management of oral complications associated with tumor therapy in pediatric patients—page 384

Mucositis and ulceration are the most frequent oral complications of tumor therapy in children, seemingly developing as a secondary response to the toxicity of chemotherapeutic agents and radiation on rapidly dividing basal epithelial cells. Other acute manifestations of therapy include candidiasis, viral and bacterial infections, bleeding, pain, gingivitis, xerostomia, and dental caries. Abnormalities of the dentition include enamel hypoplasia, diminutive teeth, delay or failure of tooth development and eruption, and altered root formation.

Requests for reprints should be directed to Dr. Anne

R. Simon, Department of Pediatric Dentistry, School of Dentistry, CB# 7450, University of North Carolina, Chapel Hill, North Carolina 27599-7450.

Generalized juvenile periodontitis in a thirteen-year-old child—page 390

The generalized form of juvenile periodontitis (GJP) is diagnosed on the basis of the general pattern of alveolar bone loss and attachment loss around permanent teeth. This paper reports a case in which a thirteen-year-old patient with evidence of periodontitis around her primary teeth at a prepubertal age subsequently developed GJP that affected premolars, permanent incisors, and molars. Periodontitis in prepubescent children is of great concern, because periodontitis may be due to an underlying systemic disease not apparent in the patient's medical history.

Requests for reprints should be directed to Dr. Keiko Watanabe, Assistant Professor, Department of Periodontics, College of Dentistry, Box 6998, University of Illinois at Chicago, Chicago, IL 60680.

Radiographic features of the bones of the hand and wrist in achondroplasia: report of case—page 396

There is a lack of clear differentiation between achondroplasia and other forms of short-limbed dwarfism. The cardinal features of achondroplasia are easily recognized, with more than 80 percent of cases sporadic. Increased parental age at the time of conception is associated with such cases. The dental anomalies are tapered maxillary incisors, delayed eruption and malocclusion. The case of a thirty-nine-month-old Chinese boy is described in this report. Diagnosis was made on the basis of clinical appearance and radiographs of the left hand and wrist.

Requests for reprints should be directed to Dr. Lisa L.Y. So, Department of Children's Dentistry and Orthodontics, Prince Philip Dental Hospital, 34, Hospital Road, Hong Kong.

Increasing numbers of pediatric AIDS patients—page 400

Many pediatric and general dental practitioners may be unaware of the extent to which the AIDS epidemic has spread among children in the United States. As of October 1990, a total of 154,917 cases of AIDS had been reported in the general U.S. population; with 2,686 AIDS cases in children younger than 13 years of age, and 604 cases in adolescents between thirteen and nineteen years of age. More than three fourths of the pediatric cases are minority children; by contrast, 42 percent of adult cases are in minority populations.

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

Water purification systems and recommendations for fluoride supplementation—page 405

The recommended level of water fluoridation for optimal dental caries reduction is 0.7 ppm (mg/l) to 1.0 ppm, with 4.0 ppm being the maximum contaminant level allowed by the EPA. In communities that do not fluoridate water supplies, fluoride supplementation may be necessary. Guidelines are provided. This article also familiarizes the reader with the types of water purification systems available, including eight types of point-of-use systems that remove fluoride from water. Dentists should ask patients about their drinking water to determine whether fluoride supplementation is needed.

Requests for reprints should be directed to Dr. Ernest J. DeWald, Commander, USA Dentac, ATTN: HSBD-ED, Fort Campbell, Kentucky 42223-1498.

Premature extractions of primary molars and the crown/root ratio of their permanent successors—page 409

Little attention has been paid to the possible effect of premature extraction on the future development of the

Continued on page 357

BUSY READER *Continued from page 353*
individual successor. This study examined the ultimate root length and area (in cross-section) of the successors of fifty-three unilaterally extracted mandibular molars as compared with the intact antimeres, which served as individual controls. Radiographs were taken using the long-cone technique. Significantly larger area ratio in the extraction group was found, suggesting a smaller root area. All differences, although statistically significant, were of no clinical importance.

Requests for reprints should be directed to Dr. I. Brin, Dept. of Orthodontics, Hebrew Univ.-Hadassah SDM, P.O. Box 1172, Jerusalem, Israel.

Single maxillary central incisors in the midline—page 413

The appearance of a single tooth in place of two teeth can be related to fusion of two neighboring tooth buds or to agenesis of a tooth germ. An eight-year-old boy was examined; one tooth had the morphology of a maxillary left central incisor. An occlusal radiograph showed a single maxillary central incisor in the midline with a single root, a single pulp canal, and an open apex. Two other cases, both of which involved girls, are described here: one ten-year-old with a single, permanent, maxillary right central incisor; the other, age eleven, with a maxillary left central incisor. The evidence presented indicates that these are instances of a central incisor, in which the homologous tooth and its successor erupted in the midline.

Requests for reprints should be directed to Dr. Eliyahu Mass, Department of Pediatric Dentistry, The Maurice and Gabriela Goldschleger, School of Dental Medicine, Tel Aviv University, Ramat Aviv 69978, Tel Aviv, Israel.

Child abuse: A survey of ASDC members and a diagnostic-data-assessment for dentists

Michael D. Saxe, DMD
James W. McCourt, DMD

Child abuse is defined as the non-accidental physical injury, minimal to fatal, inflicted upon children by persons caring for them. Statistics of the U.S. Department of Health and Human Services show that the incidence of maltreatment increased by 66 percent between 1980 and 1986. Among the abuse cases, however, significant changes have occurred: physical abuse increased by 58 percent, and sexual abuse more than tripled its 1980 rate (this report indicated 25.2 children per 1,000 or a total of more than 1.5 million children, nationwide, experienced abuse or neglect).[1] Further, it is estimated that between 100,000 and 500,000 children are sexually molested in the United States annually.[2]

Health professionals are mandated and protected by law to report cases of suspected child abuse. Every state gives immunity from civil and criminal liability to all persons who make reports in good faith. In fact, most states have penalties that can be exacted against mandated persons who fail to file a child abuse report, with penalties ranging from twenty-five to thousands of dollars in fines and up to six months imprisonment.

Dr. Saxe at time of submission was a second year Chief Resident, Pediatric Dentistry, Bruce Medical Annex, Suite G, 2545 S. Bruce Street, Las Vegas, Nevada 89109.

Dr. McCourt is Assistant Professor, Dept. Pediatric Dentistry, University of Texas Health Science Center, San Antonio, Texas 78284-7888.

Reporting of suspected child abuse is a professional responsibility.

TABLE 1 □ SURVEY QUESTIONS AND RESULTS

Survey Questions	Answered Yes	Answered No	Improperly Answered
1. Have you ever suspected abuse of a child patient?	79/157 (50.3)	78/157 (49.7)	
2. If yes, did you report this case to the appropriate agency?	52/78 (66.7)	26/78 (33.3)	1/78
3. Have you avoided reporting a case of possible abuse, because you did not want to get involved?	9/145 (6.2)	136/145 (93.8)	12/157
4. Do you have the numbers of local agencies to report a child abuse case posted in a prominent area?	57/157 (36.3)	100/157 (63.7)	
5. Do you know the common signs of child abuse?	137/152 (90.1)	15/152 (9.9)	5/157
6. Do you know what you as a dentist should do in documenting a case of child abuse?	105/154 (68.2)	49/154 (31.8)	3/157
7. Did you know that you are mandated and protected by law to report suspected cases of child abuse?	136/157 (86.6)	21/157 (13.4)	
8. Does your office have a protocol for documenting suspected child abuse?	42/156 (26.9)	114/156 (73.1)	1/157

In spite of these laws, many health care professionals, including dentists, do not get involved.

Malecz found that 7 percent of the dentists responding to his survey stated that under no circumstances would they report child abuse. The most common reasons given were:

□ Uncertainty about the diagnosis (41 percent).
□ Fear of litigation involvement (26 percent).
□ Lack of knowledge of child abuse (19 percent).
□ Possible effects on their practice (6 percent).[3]

A survey in 1978 by Becker, Needleman and Kotelchuck reported that 77 percent of the pedodontists they surveyed were aware of their legal responsibilities to report child abuse.[4] Zellman in 1990 documented that people who report child abuse were influenced by their perception of benefit or harm to the child or family.[5]

Dentists, especially those catering to the child patient, have a unique opportunity to observe child abuse since 50-65 percent of all cases of child abuse involve injuries to the mouth and face.[6] A continuous pattern of abusive trauma can be difficult to follow. A parent or guardian who physically abuses a child rarely returns to the same physician or emergency room when the child needs treatment for the abuse. The parent or guardian may not have the same cautious attitude toward the child's dentist; in fact, the dentist may be the only health care practitioner to see the child with any regularity.[7] As dentists, we should be prepared to document correctly and report immediately a possible case of child abuse.

To gain a better understanding of the knowledge dentists have on the topic of child abuse, a postcard survey was mailed to 250 randomly selected members of the American Society of Dentistry for Children (every fifteenth member on a non-alphabetical, territorial list, of over 3500 members). This allowed us to survey dentists who treat children. Of the 250 surveys, 157 responses were received, producing a response rate of 62.8 percent. Some of the answers given were not used in tabulating the results, because some questions were left unanswered, and others were circled both "yes" and "no" (Table).

In general, the dentist's role in identifying and reporting child abuse is as follows:

□ To observe and examine any suspicious evidence that can be ascertained in the office.
□ To record, according to the law, any evidence that may be helpful in the case, including physical evidence and comments obtained from questioning or interviewing.
□ To remain objective toward all parties.
□ To treat any dental injuries.
□ To establish and maintain a professional therapeutic relationship with the family.

- ☐ To hold the child whose life is in danger, and transfer the child to a hospital or a physician for proper care.[8]

This paper will address these objectives and provide the dentist with a protocol to follow in documenting child abuse.

SURVEY RESULTS AND DISCUSSION

See Table for survey results. As compared to other surveys, the dentists sampled in this study were more aware of their legal responsibility to report child abuse.[4] Also, they are reporting more often without the fear of involvement. It is significant that of the seventy-nine dental practitioners who suspected abuse of a child patient, all fifty-two practitioners who reported knew the law. In contrast, this knowledge of the law was indicated by 19 percent of the practitioners who did not report abuse, (chi-square $p = < .001$).

Today these dentists have a better understanding of child abuse and the important role that they play in combatting it. Awareness of the signs of child abuse has increased with the increasing involvement and help of city, state and professional organizations. But with the increase in child abuse cases, only a small number of dentists established a protocol for reporting and documenting them.[1] This survey has addressed the need for continued exposure, for the dentist, to the subject of child abuse. And it also reflects the need of a reporting protocol for the dental office.

DOCUMENTATION

In preparing for the documentation process the dentist should know:

- ☐ The common signs of abuse.
- ☐ What information to gather legally from the child.
- ☐ Whom to call once child abuse has been determined.

Having a data-and-diagnostic-assessment (D.D.A.) form on file simplifies this technical process (see form 1-5). This D.D.A. form should include all of the information needed for the local child abuse agency to represent the child in court and to document the case legally. This form may not only limit the amount of involvement by the dentist, but will also provide the dentist and the authorities a useful reference.

DIAGNOSTIC ASSESSMENT

The significance of the information requested on the D.D.A. form is justified as follows. The child's full name, address, and date of birth are needed, because most states protect children to the age of 18. If the child is either physically or mentally handicapped, however, the protection age varies from state to state. Information regarding the parent or legal guardian may be important since certain aspects of a family situation can predispose a child to physical abuse. The natural parent tends to be the abuser 86.9 percent of the time, compared to step- or foster parents who constitute 8.2 percent of abusers.[8] Economic difficulty and unemployment can also predispose a child to abuse, since low family-income puts a child at significant risk for maltreatment.[1] Following the information on the child and the family situation, the name and telephone number of the child's physician should be obtained in case the physician is needed later for a phone consultation.

Physical indications

The skin is the most frequently involved organ system in child abuse, and many show evidence of bruises or welts in various stages of repair due to attacks on different occasions.[9] The children should be asked about certain unusual bruises, which may have satisfactory explanations. For example, the Vietnamese can induce symmetrical linear bruises on the back and chest from coin rubbing (Cao qio), the folk treatment for symptoms of fever, chills, or headache.[10] Small circular burns seemingly caused by a cigarette, or rope burns around the wrist may also be signs of abuse. Most bite marks associated with abuse or neglect (42.8 percent) are located on the head and neck.[11] Additionally, the specific individual details of bite marks may help identify the abuser. Patchy hair loss, known as traumatic alopecia, is caused by holding the child down during the attack (common in sexual abuse). All these are important physical indications of abuse that dentists should know.

The oral cavity

Damage to the lips and oral cavity is especially common since the attacker may use force to try and silence the child. Injuries frequently include fractured, missing, or discolored teeth; lip contusions; lacerated labial and lingual frenula; jaw fracture; tongue damage; and abrasions in the corner of the mouth. Unless sexual abuse results in oral conditions or is related to visible physical abuse, there are few signs and symptoms that can help the dentist recognize the problem.[7] Orofacial signs of sexual abuse may include gonorrheal lesions of the mouth and eyes; venereal warts (condyloma acuminatum); syphilis of the lips and mouth; and erythema and petechiae of the palate. All indicators of abuse should be carefully diagrammed and accurately described regarding color, stage of repair, size, pattern, and surface description.

Assessing the data

As data accumulate on the D.D.A. form, an explanation should be sought for the suspected signs of abuse. First, the parent or caretaker, in the absence of the child, should be asked to explain the suspicious bruises or trauma. Second, the child, away from the caretaker, should be asked to explain the injury. Common sense is called for during this communication process. For example, if a child states that he fell on the pavement and cut his ear plus the side of his face, his answer should be disregarded, if corresponding trauma is present on the other side of the face as well.

Certain behavioral characteristics are common to physically abused children.[12] They may react inappropriately to stimuli, speak in monosyllables, or seem unaware of their surroundings. Problems such as seductive behavior, aggression, guilt, ambivalence, and acting out may also be seen.[13] A male dentist may provoke fear in the sexually abused child, since most sexual abusers are men (only an estimated 5-20 percent of all reported cases are perpetrated by women).[14] When questioned, an abusive parent may hesitate to explain or may become hostile. Parents with vague answers regarding the cause of injury, or parents who describe a child as accident prone, are suspect. Evaluate the explanations by parent/guardian and child to see whether they are consistent with each other. Record your impressions for possible reference at a later date, since this becomes a legal document. Inconsistent answers between parent and child require continuation of the D.D.A. form.

Photographs and radiographs

Photographs may be necessary to document observations. In some states, the dentist must be authorized, by parental consent, to take photos. Dentists, therefore, need to be aware of the rulings regarding these issues in their respective states. Photos should show the child's name and the date of the examination in the photograph. By holding a measurement marker and a color scale next to the area, it's easier to date the injury, identify the object that caused the injury, and help identify the abuser (as in bite mark identification).

Radiographs are valuable in court because they can identify undetected fractures and can document the existence of previous injuries inflicted at different times. Diagnostic quality of x-rays are important and the films should be retained in the child's dental record. Parental consent is necessary, however, to obtain x-rays.

Care and consultation

A telephone consultation with the child's physician (again, parental consent to make the call may be needed) is often indicated. The physician can provide information on earlier, suspected traumatic abuse.

Once child abuse is suspected, the dentist must provide necessary dental treatment, and/or refer the child to a hospital for medical treatment. The child who seems in danger of further abuse, should be taken into protective custody. In many states, the health practitioner (usually the physician) may do so, if the child is believed to be in danger.[1] In 50 percent of known cases, severely abused children who return to the abuser will die of continued abuse. In non-fatal abuse, 35 percent of the victims are abused again within a year.[15]

Chart/File # ____________

DATA AND DIAGNOSTIC ASSESSMENT

____
Date of Report

Patient's Name: ____________

Address: ____________

Phone: ____________ Date of Birth: ____________

Childs School: ____________

Parents or Legal Guardians:

1. ____________
2. ____________

Occupations:

1. ____________
2. ____________

Physician's Name: ____________

Physicians' Phone: ____________

General Reason for Survey: ____________

Page 1 of Data and Diagnostic Assessment

Check Indication:

___ Unexplained bruises and/or welts (look for various stages of healing):

___ *Unilateral* ___ *Bilateral* ___ *Midline* ___ *Bony areas*

___ Burns and/or abrasions:

___ *Oral* ___ *Facial* ___ *Body*

___ Skin lesions

___ Bite Marks

___ Patchy hair loss

___ Blood clots:

___ *Ear* ___ *Eyes* ___ *Nose* ___ *Oral*

___ Oral lesions:

___ *Trauma* ___ *Sexually Transmitted (Lab test)*

___ Injury to the dentition/periodontium/maxillofacial complex:

___ *Maxilla*:

___ *Anterior* ___ *Posterior*

___ *Mandible*:

___ *Anterior* ___ *Posterior*

___ Inflammation

Location of Physical Indication:

____

____

____

Diagram Injuries Below:

Page 2

Description of Indicator:

Color: ______________________________

Stage: ______________________________

Size (measurement) ______________________________

Pattern: ______________________________

Surface description: ______________________________

Child's Explanation of Injury:

____

____

____

Child's Behavior:

___ Speaks in monosyllables ___ Unaware of surroundings

___ Over-affectionate ___ Dressed poorly

___ Disruptive behavior ___ Passive, withdrawn behavior

___ Overly protective of parent

Parental Explanation of Injury:

____

____

____

Parent's Attitude:

___ Hostile ___ Negative self-image

___ Very critical ___ Hesitant in answering

___ Overly protective of child

Are both the child and the parent/guardian explanations consistent?

___ Yes ___ No *(If "yes", Continue assessment)*

Page 3

Doctor's Overview of Child's and Parent's Explanation:

____

____

____

____

____

Documentation:

___ Photos (must have child's name, date, and measurement/color markings):

___ Number of photos

___ Color ___ Black and white

___ Radiographs (must have child's name and date):

___ Number of radiographs

___ Periapicals ___ Bitewings ___ Panogram

Radiographic Interpretation:

____

____

____

Medical Information From Patient's Physician *(may need consent)*:

____

____

____

Page 4

Current Treatment *(refer out trauma when needed)*:

____

____

____

Call authorities and have child taken into custody when needed.

National Child Abuse Hotline: 1-800-422-4453

Child/Parent Consultation:

a) Tape record consultation if possible

b) Inform parent(s) of your suspicions and your legal responsibilities.

c) Inform parent(s) of the agency contacted about their case

Final Notes of Case:

____

____

____

____

____
Dentist's Signature **Date**

____
Witness **Date**

Page 5

If warranted, the local child abuse authorities should be contacted, informed of the current situation, and assisted in determining whether the child should be placed in protective court custody. Three important pieces of information are required for the authorities to investigate the case:

☐ The child's location and home address.

☐ Evidence that the injury fits the definition of abuse or neglect.

☐ Ability of the local authorities to protect the child.

All reports and actions should occur within 48 hours.

The last step in the assessment involves a final consultation with the parent. The dentist (and the dental assistant, who serves as a witness) may meet with the parent alone, or in the presence of the local child abuse authorities, the police, or any of these professional combinations. It is best to tape record this consultation. On tape, the parent should be informed of the dentist's suspicions and legal responsibilities as a dentist to report possible child abuse victims. It is possible that the parent may be very surprised at the findings. They should be told that no blame is being assigned to them, but the authorities must determine whether any person (such as a daycare worker or a family friend) may be an abuser. If the parent/dentist consultation is conducted in private, the dentist should inform the parent/guardian that the local authorities have been contacted about the case. Once any remaining notes are made about the final consultation, the document should be signed and dated. The dental assistant, present during this entire process, should also sign and date the document.

CONCLUSION

The most important step in reporting and properly documenting child abuse is advance preparation. Child abuse is not usually a one-time occurrence. It is the dentist's professional and societal responsibility to prevent the potential continuation of pain and suffering in a child. Because child abuse is increasing in our society, the dentist, as well as other health-care professionals, must be educated in the signs of child abuse, their legal responsibilities in reporting child abuse, and whom to contact when child abuse is suspected. The survey results reflect the need for a documentation protocol for the dental office, as our knowledge on child abuse continues to grow. This diagnostic assessment form provides a guide through the documentation process and develops a step-by-step decision on the probability of child abuse.

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Pediatric dentists need to know about the changing economics of health care

Practice management

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

"We just don't know very much about the income of pediatric dentists!" was the conclusion of an earlier presentation on the economics of pediatric dental practice.[1] While more specific information is needed in this particular area, reports on the general income of dentists continue to be favorable. Nevertheless, the economics of dentistry (and health care in general) are continuing to undergo rapid changes and pediatric dental practitioners must continue to maintain currency with events that ultimately could affect individual practices. The following presentation will review the general economics of dentistry and health care services; with emphasis on some developments and future prospects in the individual states.

COMPENSATING FOR THE CHANGES

Dramatic changes in the general structure of the health-care industry occurred during the 1980s. Among these changes have been the shifting of many services from hospital inpatient settings to outpatient hospital and freestanding facilities, the dispensing of prescription drugs at sites other than community pharmacies (e.g. groceries, department stores, and mail-order pharmacies) and the delivery of dental services in department stores, "smile centers", and "one-day" care facilities.

In addition to these structural changes, there have

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been innovations in the payment mechanisms adopted both to finance and contain the costs. Health maintenance organizations, preferred provider organizations, utilization reviews, mandatory second opinions, required outpatient treatment for particular procedures, higher copayments and deductibles, and a host of company-sponsored, self-insurance schemes are but a few of the approaches to modify and control the system.[2]

While these many changes were occurring in the structure of our health system, there was "... deterioration of data upon which the historical methodology (for determining national health expenditures) had been based..."[2] Budget reductions in the Internal Revenue Service (IRS) limited the usefulness of the traditional estimates of national expenditures for health services, and the existing insurance industry measures could not keep up with the rapid changes in the health-insurance marketplace.

In the late 1980s, improved survey procedures conducted by the Bureau of the Census, the Bureau of Labor Statistics, and the American Dental Association replaced IRS based estimates. As a result, federal agencies had revised their estimates of national expenditures (during the 1980s) for dental care and other health services. While "(s)ignificant changes have been made to estimates of spending for professional services and to estimates of what consumers pay out of pocket for health care...," the general spending trends continued.[3]

By 1988, national health expenditures reached almost $540 billion ($478.3 for personal health expenditures or $1,882 on a per capita basis). Almost $30 billion were expended for dental services. Overall spending for health amounted to 11.1 percent of the gross national product; more than twice the share that it represented in 1960. Between 1987 and 1988, personal health expenditures increased by 10 percent; dental care expenditures increased by 8.5 percent.[3]

Between 1960 and 1988, constant dollar net income from dental practice increased by almost 50 percent

DENTAL EXPENDITURES AND PRACTITIONER INCOME

Current and constant dollar (i.e. removing the effects of inflation) national expenditures for dental services continued to increase through the late 1980s. Similarly,

Table 1 ☐ Current and constant dollar national expenditures for dental services, per capita expenditures, expenditures and net income per active dentists: 1960, 1970, 1980, 1985, 1988.[4-8]

	1960	1970	1980	1985	1988
National expenditures (in billions)					
Current dollar	$2.0	$4.7	$14.4	$23.3	$29.4
Dental price index (1982 = 100)	28.95	42.07	84.70	122.67	147.59
Constant dollar	$6.8	$11.1	$17.0	$19.0	$19.9
Per capita expenditures					
Current dollar	$10.52	$21.86	$61.27	$94.33	$115.74
Constant dollar	$35.79	$51.62	$72.34	$78.51	$78.35
Number of active dentists	90,120	102,220	126,240	134,201	138,749
Expenditures per dentist					
Current dollar	$22,192	$43,282	$106,932	$164,573	$211,893
Constant dollar	$75,454	$108,589	$134,664	$141,578	$143,424
Net income as a percent of gross income	55*	52	43.6**	34.4	33.7
Net income					
Current dollar	$12,205	$22,205	$46,622	$56,613	$71,407
CPI (1982-84 = 100)	29.5	38.8	82.4	107.6	118.3
Constant dollar	$41,372	$58,005	$56,580	$52,614	$60,360

*1958
**1981

Table 2 ☐ Per capita health expenditures and percent change: 1980, 1990, 2000.[10]

	1980	1990	2000
Per capita health expenditures	$1,016	$2,425	$5,515
Percent change from previous period	–	138.7	127.4

Table 4 ☐ Estimated range of per capita expenditures for health care: 1990.[10]

High:	Massachusetts	$3,031
	California	$2,894
	New York	$2,818
Low:	Utah	$1,784
	Wyoming	$1,756
	Mississippi	$1,751

Table 3 ☐ Estimated sources of payment for personal health national health expenditures: 1980, 1990, 2000.[10]

	1980	1990	2000
Out-of-pocket*	27.4%	26.7%	26.1%
Employer sponsored	28.5	28.7	27.9
Non-group insurance	5.6	4.5	4.3
Other private insurance	1.4	1.4	1.5
Medicaid			
State	4.6	4.5	4.2
Federal	5.7	5.7	5.3
Medicare	16.2	19.2	22.2
Other Public	10.5	9.4	8.4
Totals	100.0%	100.0%	100.0%

*Does not include employee share of premiums for employer-sponsored insurance. These payments are included in the "employer-sponsored" category.

Table 5 ☐ Political jurisdictions with the highest and lowest rates of increase of per capita health expenditures: 1980-1990, 1980-2000.[10]

		Per capita expenditures	Percent income	
			1980-1990	1980-2000
High:	Arizona	$848	160.7	490.3
	Alaska	$921	157.2	485.5
Low:	Wisconsin	$1,097	123.2	407.3
	Dist. of Col.	$1,241	108.4	374.1

Note: The per capita expenditures for these four jurisdictions do not represent the extremes for all states. The 1980 per capita health expenditures ranged from $708 and $711 for the States of Idaho and New Mexico, to $1,257 and $1,284 for the States of New York and Massachusetts.

per capita current dollar spending for dental care increased. Constant dollar per capita expenditures more than doubled between 1960 and the mid- and late 1980s. Despite an increase of almost fifty thousand professionally active dentists since 1960, constant dollar spending per dentist almost doubled during the past twenty-eight years (Table 1).

During the past thirty years, the mounting overhead costs of dental practice (particularly personnel costs) have had a dramatic impact on the return from dental practice activities. The ratio of net income to gross dental receipts decreased from 55 percent to approximately a third.[5,6] Nevertheless, current and constant dollar net income from practice has continued to increase. Between 1960 and 1988, constant dollar net income from dental practice increased by almost 50 percent (Table 1).

DENTAL INSURANCE

Approximately 97 percent of expenditures for dental services are derived from private sources (out-of-pocket and insurance). Medicaid accounts for 3 percent of dental expenditures. Between 1970 and the early 1980s, private health insurance coverage for dental services increased annually at an average rate of 33.9 percent.[3] Some of this expansion was the result of employers switching from fee-for-service arrangements to health maintenance organizations and preferred provider organizations. In an effort to attract employees to these plans, dental coverage was added to the package. In addition, employers adopted self-insurance schemes and used the money saved by these moves to increase the dental insurance package.

The increasing trend for dental insurance coverage may be changing as industries attempt to control the increasing costs of employee fringe benefits. In 1980, dental plan participation was available for 56 percent of full-time employees with health insurance, in medium and large firms. By 1984, the proportion increased to 77, but then fell to 71 percent in 1986 and 66 percent in 1988.[3,9]

As a result of these developments, the annual growth in dental insurance expenditures from 1982 through 1988 slowed to 9.9 percent. Reflecting the continuing increase in insurance coverage (albeit greatly slowed in the last years of the 1980s), out-of-pocket spending by consumers for dental services decreased from 97 percent of total expenditures in 1960 to 55 percent in 1988.[3]

And yet, despite these many changes, the 1966 through 1987 Health Interview Surveys carried out by the National Center for Health Statistics indicated that U.S. residents averaged 2.0 dental visits in 1986 compared to 1.6 in 1965. During the 1970s, the number of visits per person increased at the rate of 1.3 percent per year; 2.7 percent per year in the 1980s.[3]

OVERALL HEALTH EXPENDITURES

The continuing marked increases in overall health costs have been reported ad nauseam in the lay and professional literature. For example, in the years between 1980, 1990 and 2000, per capita health expenditures are expected to more than double in each of the respective decades: reaching $5,515 per person in the year 2000 (Table 2). But despite the expected dramatic increases in expenditures for health services, limited changes are projected during the next decade, in the distribution of the sources of payment for health services. Except for some increases in Medicare (from 19 percent to 22 percent of health care expenditures) most

Table 6 □ Rank ordering of states by estimated and projected per capita health expenditures, change in per capita health expenditures and percent of the population that is uninsured: 1980-2000.[10]

		Ranking ordering			
	Per capita health expenditures	Per capita health expenditures	Change in per capita health health expenditures	Percent of population that is uninsured	Percent of population that is uninsured
	(1990)	(1990)	(1980-2000)	(1988)	(1988)
Alabama	$2,286	27	13	15	15.1%
Alaska	2,367	22	2	13	15.8
Arizona	2,211	31	1	7	17.7
Arkansas	1,944	43	46	2	21.8
California	2,894	2	17	10	17.2
Colorado	2,415	21	20	22	13.0
Connecticut	2,699	6	40	51	5.8
Delaware	2,268	28	36	34	10.2
Dist. of Col.	2,586	9	51	14	15.7
Florida	2,427	20	4	4	18.4
Georgia	2,072	38	39	25	12.6
Hawaii	2,469	16	8	42	8.1
Idaho	1,726	50	16	11	16.4
Illinois	2,619	8	29	35	10.1
Indiana	2,201	32	32	21	13.6
Iowa	2,351	23	35	44	7.9
Kansas	2,548	12	23	33	10.4
Kentucky	1,875	44	43	16	14.9
Louisiana	2,185	34	44	9	17.3
Maine	2,175	35	5	45	7.8
Maryland	2,436	19	41	38	9.5
Massachusetts	3,031	1	38	48	7.3
Michigan	2,569	11	42	41	8.2
Minnesota	2,480	15	49	50	6.6
Mississippi	1,751	49	45	6	17.9
Missouri	2,568	10	10	32	10.5
Montana	2,059	40	27	12	15.9
Nebraska	2,452	17	21	31	10.5
Nevada	2,757	4	9	8	17.3
New Hampshire	1,981	41	19	36	9.9
New Jersey	2,224	30	30	40	8.3
New Mexico	1,792	46	3	1	22.8
New York	2,818	3	48	28	11.5
North Carolina	1,833	45	34	20	13.8
North Dakota	2,661	17	6	47	7.5
Ohio	$2,493	14	26	37	9.6
Oklahoma	2,139	36	37	5	18.0
Oregon	2,312	25	15	18	14.6
Pennsylvania	2,536	13	11	43	8.0
Rhode Island	2,707	5	47	49	7.2
South Carolina	1,689	51	31	26	11.9
South Dakota	2,322	24	18	17	14.7
Tennessee	2,262	29	33	19	14.2
Texas	2,192	33	28	3	21.4
Utah	1,784	47	22	27	11.7
Vermont	1,956	42	25	39	9.2
Virginia	2,076	39	24	30	10.8
Washington	2,311	26	7	24	12.8
West Virginia	2,088	37	12	23	12.9
Wisconsin	2,449	18	50	46	7.6
Wyoming	1,756	48	14	29	10.9
United States	$2,425				13.1%

other sources will change fractionally. Out-of-pocket and employer sponsored insurance arrangements will continue to constitute, respectively, somewhat more than a quarter of health care expenditures (26 percent and 28 percent) (Table 3).

BUT HEALTH EXPENDITURES ARE NOT DISTRIBUTED UNIFORMLY

Although the spending for health care increased throughout the country, by 1990 there were significant differences between the various states, based on estimates for 1) per capita expenditures and 2) the past and projected rates of increase of per capita expenditures.

In 1990, per capita expenditures for health care ranged from $3,031 in the State of Massachusetts to $1,751 in the State of Mississippi (Table 4). Between 1980, 1990 and 2000, the States of Arizona and Alaska had the highest, and the State of Wisconsin and the District of Columbia had the lowest rates of increase of per capita health expenditures (Tables 5 and 6).

The past increases in health care costs pale by comparison with the magnitude of the increases that will occur by the year 2000. By the turn of the century, we will spend an estimated $1.47 trillion annually for health care.

To pay this bill, between 1980 and 2000:

- ☐ Employers and employees will have to pay a 529 percent increase for employer-based health coverage (from $66 billion to $412 billion).
- ☐ Families will pay a 512 percent increase in out-of-pocket expenses (from $63 to $386 billion).
- ☐ State governments will have to increase expenditures by 480 percent (from $11 billion to $62 billion).[10]

Confounding the already complex fabric of expenditures for health services is the reality that in 1988, 13.1 percent of the population of the nation (31.8 million people) were uninsured for health care services; ranging from a high of 22.8 percent and 21.8 percent, respectively, in the States of New Mexico and Arkansas to 6.6 percent and 5.8 percent, respectively, in the States of Minnesota and Connecticut (Table 6).

PROJECTIONS FOR THE FUTURE OF DENTISTRY – SOME THOUGHTS

The dire reports in the lay and professional literature regarding the expected early demise of the dental profession, are increasingly being replaced by publications that describe the changes in available personnel, increased retention of teeth, increasing dental needs and demand for services: all of which augur well for the future of the profession. For example, (between 1980 and 2000) despite an increase in the dentist per 100,000 civilian population ratio from 53.5 to 59.0, the ratio of available dentists per one million "teeth at risk" will decrease from 43.4 to 35.5.[11,12]

But dental care in general, and pediatric dental services in particular, exist in the larger reality of health care services. There are no specific data relating expenditures for pediatric dental services to the wide range in current and projected statewide per capita health expenditures. (No doubt there are equal or even greater per capita expenditure variations within particular areas of individual states.) But surely, limited overall per capital expenditures for health services in a particular state or locale does not bode well for a willingness to expend funds for needed dental care.

But by using available interstate comparison data (Table 6), pediatric dental practitioners (especially those in states with particularly lower rank orderings of per capita expenditures and a high percent of the population with no health insurance) should be in a more favorable position to lobby for increased government, industry, union, and insurance company attention to general health needs in their communities. Who knows, there might even be increased funds for dental services; especially for the care of children.

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Clinic

Evaluation of treatment times for Class II composite resin restorations

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Despite preventive measures, restorative therapy continues to be the type of treatment most frequently provided by dentists.[1,2] In various countries payment for dental treatment is based on each individual operation.[3] With respect to restorative treatment, it is well known that the labor factor is the primary component of the cost of the restorative product, and that the material represents only a small portion of the ultimate cost.[4] In order to establish the fee for restorations on the basis of the individual operation, it is necessary to know how long placement of a restoration takes. Deviations in treatment time may occur as a result of the size or location of the restoration, various patient-related factors, and the treatment methods and skill of the individual practitioner.[5]

In the past, a few studies focused on the treatment times for amalgam restorations, and the various factors that affect the time required for the operation.[6] There has been very little research done on the treatment times required for Class II composite resin restorations.[3,4,7] In order to gain more insight into this question, the time spent on each such operation was registered within the framework of a clinical study on the behavior of posterior composite resin restorations.

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This article presents the results of these registrations.

MATERIAL AND METHODS

A total of fifty-six patients received 183 Class II composite resin restorations. Each patient was given a series of three restorations, made of Herculite XR[a], Clearfil Ray Posterior[b] and Visiomolar[c]; a few patients received a double series. The materials were randomly allocated to one of the teeth to be restored. As far as possible, the cavities to be made met the requirements for standard two-surface or three-surface Class II restorations. The selection of the patients was focused upon criteria to include patients with corresponding characteristics of the dental arches (see Kreulen *et al*).[8]

The operative phase of the study was performed by three dentists. Each patient was treated by only one of them, allocated at random. To ensure that all three of the dentists carried out the restorative procedure in the same way, each step of it was carefully depicted. During treatment all the dentists were assisted by one and the same dental assistant, who saw to it that this protocol was adhered to.

The operations involved in the placement of composite resin restorations in this study have been described by Kreulen *et al* and the materials were handled as recommended by the manufacturers.[8] Standard preparations according to Rodda were made, after which rubber dam was applied.[9] Following the removal of carious dentin and selective beveling of the outline, the removed dentin was replaced by a radiopaque glass ionomer lining cement[d]; in the case of particularly deep cavities, this was generally preceded by the application of a calcium hydroxide lining[e]. Plastic matrix bands and light-reflecting wedges were positioned[f]; this was followed by an enamel-etching procedure, after which the bonding agent was applied[g]. The box section of the preparation was filled in three horizontal layers of composite resin, each of which was light-cured separately. Additionally the step section was filled and cured, using one or two layers. After initial finishing, the rubber dam was removed and the occlusal height of the restoration was verified and adjusted if necessary. The restoration was polished two weeks later.

In this study, the use of the rubber dam was standard procedure.

During the restorative process, the assistant registered the time that was required, using a stopwatch, and each placement was recorded individually. The treatment time includes only those operations that are actually required for the placement of a restoration. Thus the time spent on procedures such as use of an anesthetic was not included in the registration. The treatment procedure may be divided into five stages, as shown in Table 1. Although the application of rubber dam took place during the preparation stage, it is recorded as part of the preliminary work. The time required for the separate stages of treatment was rounded off to the nearest whole minute.

The treatment time may be influenced by a number

Table 1 □ Stages in the restorative process of Class II composite resin restorations.

Treatment stage		Treatment procedure
1.	*Preparation*	Preparation of a standard Class II cavity, removal of carious dentin, selective beveling of the outline.
2.	*Preliminary work*	Application of rubber dam, nail polish, $Ca(OH)_2$, glass ionomer cement, matrix and wedges. Etching procedure. Application of bonding agent.[8]
3.	*Filling*	Application of composite resin and intermediate light-curing.
4.	*Finishing*	Removal of the excess of composite resin and contouring; removal of rubber dam and verification of the occlusal height.
5.	*Polishing, during a separate visit*	Verification of marginal adaptation/occlusal height and adjustment if necessary; polishing with the aid of extra fine diamond stones, rubbers and polishing paste.

[a]Kerr
[b]Cavex Holland/Kuraray
[c]ESPE
[d]Fuji lining cement (GC)
[e]Life (Kerr)
[f]Transparent (Pre)molarbands and Luciwedges (Hawe)
[g]As prescribed by the manufacturers

of factors. For this reason, following treatment, additional information was recorded, such as whether the restored tooth was a premolar or a molar (type of tooth), and whether the restoration included two, mesioclusal and distoclusal, or three, mesiodistoclusal, surfaces (type of restoration). This information provides an indication of the size of the restoration. Broadly speaking, it is relatively simple to prepare a cavity, if a tooth already contains an amalgam restoration. Consequently, the presence of an existing amalgam restoration in the tooth to be restored was recorded. The skill and experience of the dentist carrying out the procedure may also be a factor. Table 2 has been made to give an impression of these features in terms of the postgraduate years at the start of the operative phase of the study, and whether or not the dentist is active in a general dental practice. At the beginning of the operative phase, none of the three dentists had any experience with the placement of Class II composite resin restorations. Thus it was decided to investigate a possible learning effect on the treatment times. For this purpose, the group of restorations each dentist made (60,59[h], and 63 respectively) will be divided in half, on the assumption that the end of the period, during which the first half of the placements took place, marks the end of a learning phase.

STATISTICAL PROCEDURE

In the trial design selected, the influences of a few variables on the treatment times are to be studied and analysis of variance, ANOVA (software:SPSS), is most appropriate. ANOVA, however, requires more or less normal distributions in all cells, which was not met in this study. Placement of a particular type of restoration will take an average time to complete; it will not often be completed in a shorter length of time. More time will be needed, if problems arise during treatment. The latter situation may be expected to occur more frequently, leading to a distribution with a long tail on the right-hand side of the average time, due to the extreme instances. This is known as a positively skewed distribution.

In order to obtain a better distribution of the data, natural logarithmic transformation (ln) is applied to the results.[6,10] This has three side-effects:

1. After ln-transformation, the mean of each population can be calculated. The value of the ln-mean does not correspond to the actual average treatment time. Retransformation (exponential function) should be applied, therefore, and conversion of the ln- mean results by definition in the so called geometric mean. Thus ANOVA compares geometric means instead of ordinary means (= arithmetic means) to study possible differences and effects of the variables.

2. In case of a normal distribution after ln-transformation, the retransformed ln-mean (= geometric mean) coincides with the median of the raw data. In this study, the geometric mean may be read as the median of the raw treatment times. The geometric mean, however, is more efficient in estimating the median of the population than the sample median.

3. ANOVA is used to test the influence of the variables. The effects are expressed in relative differences (percent) with respect to the geometric means, rather than in time (minutes). This fact appeals more to the imaginative powers of the reader.

For clearness, the results are presented as geometric means with their 90 percent concentration intervals. The arithmetic means are also shown, in order to enhance the comparability of the present study and for practice-building purposes. Due to the skewed distribution, the value of the geometric mean will be smaller than the arithmetic mean.

RESULTS

Table 3 shows the average treatment time per stage, the geometric mean, and the 90 percent concentration interval of the geometric mean time. The overall av-

Table 2 □ Experience of the three dentists.

Dentist	1	2	3
Postgraduate years	11	2	4
General practice	part-time	negative	negative
Lecturer	part-time	full-time	full-time

Table 3 □ Average treatment times (arithmetic means), geometric means and their 90 percent concentration intervals for Class II composite resin (standard deviations in parentheses), in minutes (n = 182).

Treatment stage	Arit. mean (std. dev.)	Geom. mean	90 percent concentration interval
1. Preparation	9.1 (4.5)	8.1	3.8 – 17.5
2. Preliminary work	15.1 (3.6)	14.7	10.1 – 21.5
3. Filling	7.6 (2.8)	7.2	4.2 – 12.4
4. Finishing	9.2 (2.7)	8.8	5.5 – 14.1
5. Polishing	4.4 (1.7)	4.0	2.1 – 7.8
Totals	45.4 (11.9)	44.0	29.5 – 65.6

[h]The time registration of one restoration was excluded, because this recording appeared to be incorrect.

Table 4 ☐ Relative effects of the variable dentist on the geometric mean treatment time. The geometric mean treatment time is also shown (in minutes)

	Geom. mean	Dentist 1 n=60	Dentist 2a n=30	Dentist 2b n=33	Dentist 3 n=59	Sign[1]
Preparation	8.1	−9%	55%	−8%	−14%	*
Preliminary work	14.7	−5%	28%	−2%	−8%	*
Filling	7.2	−12%	19%	12%	−4%	*
Finishing	8.8	−5%	26%	−9%	−4%	*
Polishing	4.0	−40%	40%	19%	9%	*
Totals	44.0	−10%	32%	−1%	−6%	*

1) * = p<.001 (One-way ANOVA, dentist effects)

Table 5 ☐ Effects of three variables on the treatment times; the effect of each level in terms of percentage in respect to the other level (n = 182).

	Existing amalgam restoration (yes/no)	Type of restoration (two/three-surfaces)	Type of tooth (premolar/molar)
Preparation	17% p<.01	31% p<.001	18% p<.001
Preliminary work	n.a.[1]	4% not sign.	9% p<.01
Filling	n.a.	33% p<.001	23% p<.001
Finishing	n.a.	16% p<.001	13% p<.01
Polishing	n.a.	4% not sign.	10% p<.05
Totals	7% p<.01	17% p<.001	14% p<.001

1) n.a. = not applicable

Table 6 ☐ Average treatment times (arithmetic means), geometric means and their 90 percent concentration intervals for Class II composite resin restorations (standard deviations in parentheses), in minutes (n = 152).

Treatment stage	Arith. mean (std. dev.)	Geom. mean	90% concentration interval
1. Preparation	7.8 (3.2)	7.2	3.8−13.8
2. Preliminary work	14.2 (2.6)	13.9	10.2−19.1
3. Filling	7.2 (2.8)	6.9	4.1−11.6
4. Finishing	8.7 (2.5)	8.3	5.4−13.0
5. Polishing	3.9 (1.5)	3.7	2.0−6.7
Totals	41.8 (8.8)	41.0	29.8−56.3

Table 7 ☐ Total treatment times for the combination of the variables 'type of restoration' and 'type of tooth', in minutes (not including the treatment times of dentist 2a; n = 152).

Combination variable (restoration- + tooth type)	Geom. mean	90% concentration interval	n
Two-surface + premolar	36.8	30.0−44.4	76
Two-surface + molar	42.8	32.0−57.5	27
Three-surface + premolar	43.2	33.7−55.4	31
Three-surface + molar	55.1	40.5−74.8	18

erage treatment time for Class II composite resin restorations amounts to 45.4 minutes (SD 11.9). Notable is the considerable time needed to perform the preliminary work (15.1 minutes, SD 3.6). As stated, the geometric means are slightly smaller than the arithmetic means.

The results of the investigation into the learning effects showed that, particularly in the case of dentist 2, these effects may be expected to influence the treatment times of the dentists involved, unequally (interaction). As a percentage reduction in treatment times for Period 2 compared with Period 1, dentist 1 had a reduction of 10 percent; dentist 2, 33 percent; and dentist 3, 0 percent. These are the raw effects. In the following analyses dentist 2 was 'split' into dentist 2a (first period) and dentist 2b (second period). In this manner the interaction of the factor dentist with the learning effect, which is found to be significant ($p < .001$), can be reduced.

Table 4 shows the influences, in terms of percentage, of the dentists in relation to the geometric mean treatment times. These effects have been weighted, for example, that if one of the dentists made more three-surface restorations with respect to the others, a correction will be made. The influence of dentist 2a is obvious; during each stage this factor contributes to an increase in the treatment time (for example, a 55 percent increase during the preparation stage), while after the learning phase the difference is much less pronounced (a reduction of 8 percent during the preparation stage).

The difference in effect of the two levels of each of the variables *existing amalgam restoration*, *type of restoration* and *type of tooth* on the treatment times following ANOVA can be expressed in terms of percentages (Table 5). Each factor has been weighted. All three of them have a significant influence on the total treatment time (Table 5, bottom line). It is noteworthy that the preliminary work stage was not affected by the type of restoration. The presence or absence of an existing restoration in the tooth to be restored, on the other hand, results in a difference of 17 percent in the preparation stage ($p < 0.01$). Since this factor is not thought to be of influence for the subsequent stages, no percentage has been calculated in those cases.

If the learning phase of dentist 2, being dentist 2a, has been disregarded, Table 6 can be constructed. It is obvious that the arithmetic mean and the geometric mean do not differ as much as in Table 3, because the times of dentist 2a are responsible for most of the extreme instances.

As stated, the type of tooth and the type of restoration provide an indication of the size of the restoration that was made. The geometric mean treatment times for the combination of these variables, not including the treatment times of dentist 2a, are shown in Table 7. A small restoration (premolar + two-surface) takes 36.8 minutes, while the placement of a large one (molar + three-surface) requires 55.0 minutes ($p < .001$).

DISCUSSION

In the light of the times recorded, it could be discussed whether these are representative of a general dental practice. The three dentists may conceivably have worked somewhat faster or slower than the 'average' dentists. In the present study each patient also received a standard Class II amalgam restoration[i] and the treatment times recorded for these amalgam restorations provide some material for comparison with the treatment times recorded by Advokaat.[6] Although it is difficult to reach a firm conclusion, the average treatment times for amalgam restorations in this study roughly agree with those recorded by Advokaat (Advokaat: two-surface Class II restoration, 24.3 minutes; and three-surface Class II restoration, 30.0 minutes. In the present study these times are 22.4 ($n = 42$) and 30.3 ($n = 19$) minutes, respectively [data not shown in the results]).

As far as possible, the conditions under which the three dentists worked were standardized. This means that, in addition to the treatment protocol, every effort was made to ensure that all treatment was carried out in the same room, using the same equipment. Although it is often said that the use of rubber dam is not absolutely necessary in the case of composite resin restorations, in the present study its application was standard, especially as this procedure does not take as long as is generally thought.[6,11] Furthermore, the recording of the treatment time was not done with the aid of a video-registration, as described by Dilley, because this is extremely labor-intensive, where large numbers of restorations are involved.[7]

Several factors can affect the ultimate treatment time required. In addition to those already mentioned, the size of the tooth, the size of the cavity, and various patient-related factors may also be of influence. While the size of the tooth has been compensated for to some extent by the distinction between premolars and molars, a further distinction can be made between large and small teeth. In general, the larger the tooth, the more tooth tissue will actually be removed during the preparation stage. Although this increases the treatment time, the ample outline of the preparation will enable the dentist to evaluate the inner part of the cavity faster and better, which will in turn facilitate treatment. The time needed for filling will probably be increased, as the composite resin will have to be cured in several subsequent layers. Whether these influences result in substantially different treatment times is doubtful.

In order to establish the size of the cavity, Advokaat employed a clinical estimation of the size of the restoration with respect to the tooth size.[6] This format lacks, however, an objective measure and calibration of the operators. The size of a cavity can also be expressed in terms of whether or not a $Ca(OH)_2$ lining is necessity. This lining is used in the case of particularly deep cavities, and the necessary of removing the carious dentin will mean an increase in preparation time. The presence or absence of this lining did not prove, however, to be a clear factor in determining the size or depth of a cavity, since the necessity for such a lining was based on personal interpretation.

Similarly, no objective measures can be determined for patient-related factors, such as a small oral orifice. In addition, in preparing mesial or distal cavities, as well as in restoring upper or lower teeth, differences may occur in treatment time. These factors were not included in the analysis, however, because small numbers in all categories will result, if the number of variables was increased. The same is applicable to the three composite resin materials and a possible effect of their handling properties.

As in the studies conducted by Advokaat and Hendriks, the variable 'dentist' proved to be of considerable influence, which can be attributed to the skill of the dentist (see Table 1).[4,6] Dilley on the contrary, does not report such an effect.[7] In the present study, a sig-

The three dentists operated under standardized conditions.

[i]Tytin (Kerr)

nificant learning effect was also recorded for one of the dentists. In addition, the combination of the variables 'type of tooth' and 'type of restoration', as an indication of the size of the restoration, likewise led to significant differences in the treatment time.

The overall treatment time required for Class II composite resin restorations was longer than that reported by Hendriks (37 minutes).[4] Hendriks employed a less extensive clinical procedure (i.e. no bevels, no glass ionomer base) and this may be expected to take a shorter time. In a study on composite resin restorations in primary and permanent teeth, Dilley recorded a time of 14.59 minutes for filling and finishing.[7] The results of the present study with respect to filling and finishing (arithmetic mean, 15.2 minutes; dentist 2a excluded) show a reasonable agreement with those of Dilley.

The results of this study provide an indication of the treatment time needed for the placement of Class II composite resin restorations. The statistical inference, based on the treatment times of a restricted number of restorations made by three dentists, should obviously not be considered as the sole basis on which the fee for restoration can be established. In view of the influence of the learning effect, these results certainly have to be considered in that perspective.

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AIDS AND HIV INFECTIONS IN CHILDREN

HIV affects multiple organ systems. Manifestations are diverse and include nonspecific findings, progressive neurologic disease, lymphoid interstitial pneumonia, recurrent invasive bacterial infection, opportunistic infections, and specified malignancies. Other common clinical manifestations include generalized lymphadenopathy, hepatosplenomegaly, failure to thrive, recurrent diarrhea, and parotitis. Cardiomyopathy, hepatitis, and nephropathy can also occur. Craniofacial abnormalities in infants with prenatally acquired HIV infection have been reported, but whether these are caused by HIV infection or by other factors, such as maternal drug use or genetic characteristics, is controversial.

Pediatric AIDS patients often have recurrent serious infections caused by common bacteria such as *Streptococcus pneumoniae, Haemophilus influenzae* type b, *Staphylococcus aureus*, and *Salmonella* species. Lymphoid interstitial pneumonitis (LIP) occurs in 30 percent to 50 percent of children with AIDS. Developmental delay and failure to thrive are common, and a progressive or static encephalopathy develops in many HIV-infected children.

Report of the Committee on Infectious Diseases.
Twenty-second edition, 1991. Elk Grove Village, IL:
American Academy of Pediatrics, p 115.

The prevalence of postoperative sensitivity in teeth restored with Class II composite resin restorations

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It is a widely recognized phenomenon that after restoration of a tooth, the patient can experience pain, which in many cases disappears after a short time. This is also a familiar occurrence in composite resin applications. There is much less consensus, however, about the number of complaints. From various studies of the occurrence of sensitivity following the placing of Class II composite resin restorations, it can be concluded that the percentage of complaints received varies from 0-40 percent.[1-6] It is generally found that such complaints quickly diminish, however, after a week.[3,5,7] Only Qvist, who finds 25 percent of the restored teeth still sensitive a week after restoration, reports that this percentage remains at 6 percent after two years, and 3 percent after three years.[4]

Similarly, there is no unanimity of opinion about the cause of postoperative sensitivity, let alone about the solutions that are proposed to reduce these complaints. Brännström and Pashley state that as a result of etching the dentin, the pulp becomes accessible to bacteria and bacterial products.[8-10] Hence they also argue that care must be taken at least to ensure that the smearplugs in the entrances to the dentinal tubules remain intact. Qvist carries this approach still further: if the dentin is coated with a dentin-adhesive before commencement of etching, there will be a 50 percent reduction in postoperative sensitivity.[4] This opinion is diametrically opposed to that of Kurosaki and Fusayama, who report that the etching of the dentin does not cause any damage to the pulp, as long as a dentine adhesive is applied after etching.[11,12] In fact, they state that the application of resin that is directly applied to the dentine does not produce any evidence of inflammatory reactions in the pulp, an opinion that is shared by Cox.[13] Fuks, on the other hand, does find a reaction of the pulp, which diminishes, however, after a week, because reactive dentine is formed, and the permeability of the dentin should thus be reduced.[14] Nevertheless, it is usually considered to be safer to coat the dentin with an under-layer.[15] Because the objective of this procedure is to cover all the dentine, the application of a calcium hydroxide under-layer alone is not enough, because of the inferior properties of the material.[6] Using a glass ionomer cement as the only liner also does not appear, however, to be desirable, particularly in deeper cavi-

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It is not possible to prevent trauma from operative and restorative procedures completely.

ties. It is not so much the polyacrylic acid, but rather the added acids appear to be responsible for the toxic effects upon the pulp.[16,17] For this reason, the application of both cements is also recommended in deeper cavities.

A completely different cause for postoperative sensitivity is ascribed to the polymerization shrinkage of composite resin materials. As a result of this shrinkage, stress can develop in the tooth, microleakage can occur, and hydraulic forces can be produced within the fluid in the dentinal tubules.[9,18] To a certain extent, these effects can be amplified still further by the thermic expansion coefficients of composite resin materials, which can differ significantly from those of the teeth.[19]

The proposed solutions to the problem of shrinkage from polymerization also vary widely. Sometimes they are general in nature, such as limiting the indicated area in terms of the size of the restoration; but they can also be aimed more specifically at the restorative procedure to be followed.[20,21] Thus, among others, Eick reports that the effect of shrinkage from polymerization can be limited by applying and polymerizing the composite resin material in layers.[22]

All in all, no unanimous by accepted guidelines can be given yet for the avoidance of postoperative sensitivity after restoration with composite resin material. Furthermore, the reaction of the pulp to a combination of measures, each of which may possibly be able to contribute to the reduction of the complaints has not been investigated. Based upon the literature, the most important measures appear to be:

- ☐ Only "standard" Class II preparations should be eligible for restoration with composite resin material.
- ☐ Avoid etching of the dentine, as far as possible.
- ☐ Apply calcium hydroxide base to the deeper spots in the prepared cavity.
- ☐ Coat the remaining dentine with a glass ionomer cement.
- ☐ Apply the composite resin material in layers, followed by polymerization.

This article will describe the findings of the study of postoperative complaints, following the placing of Class II composite resin restorations. It will also evaluate how these complaints are influenced by the patient, the dentist, the choice of material, and the type of tooth.

MATERIAL AND METHODS

The study mentioned above forms part of a longitudinal clinical study into the behavior of Class II composite resin restorations in comparison with that of amalgam. For a detailed description of the design of this study, refer to the article by Kreulen *et al.*[23] Only a few details relevant to the study of postoperative sensitivity will be repeated here.

Two hundred forty-four Class II restorations were provided for fifty-six patients between fifteen and thirty-five years of age. For each patient, one or two series of four restorations were made: three of composite resin and one of amalgam. The amalgam restoration (Tytin*) should be regarded as a control in this context. For fifty-one patients, a single series of four restorations was made, and for five patients two series were made. Three dentists were involved in the study, two of whom made twenty series and one of whom made twenty-one series of four restorations. In every series, three different light-curing composite resin materials were used: Herculite XR**, Clearfil Ray Posterior†, and Visiomolar‡. All four restorative materials were applied in a randomly selected order. The selection of patients was made on the basis of a number of criteria, of which in particular, the size of the restorations to be provided is detailed here. These restorations were not permitted to be wider occlusally than approximately a third of the width between the tips of the cusps; and proximally,

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*Tytin-Kerr Sybron
**Herculite XR-Kerr Sybron
†Clearfil Ray Posterior-Cavex/Kuraray
‡Visiomolar-Espe

Table 1 □ Numbers of teeth with and without symptoms of postoperative sensitivity, classified by type of restorative material and duration of the sensitivity.

Restorative material	Herculite	Clearfil	Visiomolar	Tytin	Totals
no sensitivity	47	49	43	48	187
less than one week	5	6	11	6	28
one to two weeks	3	3	1	0	7
more than two weeks	4	0	5	7	16
intermittent	2	3	1	0	6
constant	0	0	0	0	0
more than ½ year	0	0	0	0	0
Totals	61	61	61	61	244

Table 2 □ Premolars and molars with and without postoperative sensitivity.

Restorative material	Herculite	Clearfil	Visiomolar	Tytin	Totals
no sensitivity	37	32	29	24	122
Premolar					
sensitivity	7	8	10	4	29
no sensitivity	10	17	14	24	65
Molar					
sensitivity	7	4	8	9	28
Totals	61	61	61	61	244

Table 3 □ Number of patients with 0, 1, 2, 3 or 4 sensitive teeth, classified by the total number of restorations studied per patient.

		Number of teeth with postoperative sensitivity					
		0	1	2	3	4	Totals
Restorations studied per patient	4	16	21	11	3	0	51
	8	1	3	1	0	0	5
Totals		17	24	12	3	0	56

Table 4 □ Number of restorations and number of teeth with postoperative sensitivity per dentist.

	Dentist A	Dentist B	Dentist C	Totals
Restorations studied	80	84	80	244
Teeth with postoperative sensitivity	18	23	16	57

the outline had to be just explorable. Cervically, the outline should not go past the cementoenamel junction. The second and third molars and the mandibular first premolar were not eligible for the study on principle (Kreulen *et al*).

The data obtained which could be of relevance to postoperative sensitivity can be divided into primary data and evaluation data. The primary data refer to the dentist giving treatment, the patient, the tooth and the type of restorative material. One week after fitting the restorations, as well as after half a year and after a full year, the patients were surveyed regarding the presence or absence of postoperative sensitivity. These data were recorded according to the following categories:

0 = not sensitive
1 = sensitive but diminishing, lasting no longer than a week
2 = sensitive but diminishing, lasting one to two weeks
3 = sensitive but diminishing, lasting more than two weeks
4 = intermittent sensitivity, not diminishing
5 = constant sensitivity, not diminishing
6 = lasting more than half a year.

The data were processed with the use of SPSS (Statistical Package for Social Sciences). Statistics were performed by means of the chi-square test.

RESULTS

The results have been calculated both for the total number of restorations and for the composite resin restorations alone. They are classified according to:

□ Type of restorative material,
□ Type of element (premolar/molar),
□ Patient,
□ Dentist.

Type of restoration material

In Table 1 the findings of the survey are given, classified according to restorative material and the duration of the postoperative sensitivity. Fifty teeth (23 percent) appeared to show some form of postoperative sensitivity. Half of these were sensitive for less than one week, while sixteen teeth (28 percent) were sensitive for longer than two weeks. None of the teeth, however, showed lasting sensitivity. After half a year, intermittent sensitivity was also no longer to be found in any of the patients.

The restorations in Visiomolar appear to lead to a greater tendency to postoperative sensitivity (30 percent) than the other restorative materials (20 percent for Clearfil and 23 percent for Herculite). There is no significant difference, however, between the different composite resin materials ($P > 0.05$). The teeth that were restored with amalgam (Tytin) showed a comparable degree of sensitivity (21 percent).

Type of tooth

In Table 2 a distinction is made between premolars and molars with and without postoperative sensitivity. Classifying the data according to duration of the sensitivity has been omitted here, since the contents per category would be too small in a detailed extension of Table 1. Postoperative sensitivity appears to occur more frequently in molars than in premolars (30 and 19 percent of the restored teeth, respectively). This difference remains virtually the same, if the teeth with an amalgam restoration are not taken into consideration (the percentages of sensitive teeth are then 32 and 20, respectively). The differences observed, however, are not significant (P > 0.05). Nevertheless, it is noticeable that Clearfil does not appear to contribute to the differences observed between premolars and molars.

Patient

Table 3 gives the number of patients having none, one, or more than one tooth with postoperative sensitivity. A distinction has also been made here between patients with one and with two series of four Class II restorations. Of the fifty-six patients, seventeen (30 percent) have no teeth showing postoperative sensitivity; twenty-four patients (43 percent) have one sensitive tooth; twelve patients (22 percent) have two sensitive teeth and three patients (5 percent) have three teeth showing postoperative sensitivity. Fifteen of the fifty-six patients thus have more than one sensitive tooth. In this group of patients, there is one for whom eight restorations were supplied. Since the chance of a sensitivity complaint can increase with the number of restorations, this patient in fact should not be included in this group. The remaining fourteen patients (25 percent) have a total of thirty-one teeth (11 x 2 + 3 x 3) with postoperative sensitivity. A quarter of the patient population is thus responsible for almost 60 percent of the complaints recorded.

Dentist

In Table 4 it can be seen how the restorations are divided among the three dentists and how many sensitive teeth were found per dentist. Although more teeth with postoperative sensitivity (27 percent) were found among dentist B's patients than among dentist A's patients (22 percent) and dentist C's patients (20 percent), the differences are not significant (P > 0.05).

DISCUSSION

Based upon the literature, a treatment procedure has been set up that makes the assumption that the likelihood of postoperative sensitivity will be small. In this respect, however, the results were somewhat disappointing. Some form of sensitivity was recorded for a mean 23 percent of the teeth treated. Numerous reasons may lie behind this. In particular, the following should be considered:

☐ The method of recording;
☐ The application of materials;
☐ The method of treatment.

> A fourth of the patients accounted for 60 percent of the complaints.

Method of recording

By conducting a survey among the patients, an attempt was made to gain an impression of the number of complaints. Completely subjective observations are thus involved here, which can only give a general impression of the criterion under measurement. For this reason, no attempts were made to obtain more detailed data about the nature of the complaints. The subjectivity of the method of recording is accentuated still further by the presence of what is known as the "patient effect" (Table 3). It is striking that 68 percent of the complaints were recorded for 27 percent of the total group of patients. Apparently the pain threshold value plays a not-insignificant role here: some patients simply complain more quickly than others. There is no guarantee that a patient who reports that he has had no complaints has also in reality felt nothing.

Application of materials

As stated in the introduction, a number of treatment principles were adopted, which together were expected to prevent the occurrence of postoperative sensitivity: the preparation was to be kept as small as possible, calcium hydroxide was to be applied at deeper spots in the cavity, glass ionomer cement was to replace the dentine removed, and the composite resin material was to be applied in layers. On the whole, these measures were aimed at protecting the pulp and limiting shrinkage by polymerization.

It is difficult to determine the protective effect of the calcium hydroxide and the glass ionomer cement. The calcium hydroxide was not applied in all cases, sometimes applied at a single spot in the cavity, and sometimes at a number of spots. Classifying the results according to whether or not a calcium hydroxide base was used showed no difference in sensitivity. It is not clear, however, how each of the dentists applied the criterion "at deeper spots". For this reason, accurate conclusions cannot be drawn from these findings.

The glass ionomer cement can certainly form a barrier against possible harmful effects of the composite resin material; but it can have, nevertheless, a toxic effect upon the pulp due to the free acids in the material itself. In addition, it is not impossible that this barrier function against the resin component of the composite and indeed against the etching material failed to work optimally. After the initial hardening of the cement, the preparation was given its final finishing. During this process, the surplus cement was removed. In practice, this meant that occasionally some dentine became apparent again here and there, with all the possible consequences of this.

The effect of the measures aimed at reducing shrinkage from polymerization is equally difficult to establish, in particular because a complex of measures is concerned, all of which are targeted toward the same objective. Although one of the basic principles was that the preparation should not exceed the "standard enlargement" too greatly, it is never possible to avoid completely some degree of variability in size, in a clinical situation. Although the observed difference between restorations in molars and premolars gives the impression that the volume of composite resin material has an influence upon postoperative sensitivity, this finding cannot yet be sufficiently supported. In principle, a comparable difference should have been observed, if composite restorations in two planes (MO and DO) could have been compared with those in three planes (MOD). Such a comparison is only meaningful, if this can be done by tooth type. The study group, however, was too small for this.

In addition, the difference found between premolars and molars appeared not only to have been observed with the composite resin restorations, but just as much with the amalgam restorations. Although it cannot be concluded from this that shrinkage from polymerization, therefore, had no influence, it is also not inconceivable that the degree of difficulty, and thus the related precision of the treatment, may have had an influence upon the complaints of sensitivity recorded. Nevertheless, because of comparable findings with amalgam, the relationship between complaints of sensitivity and volumetric shrinkage is not yet completely determined. In fact, during the clinical execution of this study, a further twenty-six small composite-resin restorations were made, for which only the preparation took a different form in comparison with the original treatment procedure. Here, preparations without a step were involved. For these twenty-six box restorations, a complaint of sensitivity only occurred in three cases (11.5 percent). Although the number of restorations is too small to attach much importance to the complaints observed, this could still be looked upon as giving support to the difference observed between premolars and molars; or, to put it more accurately, between smaller and larger restorations. Further research is necessary to obtain more clarity on this point.

In addition, yet another (admittedly not significant) difference in sensitivity was found concerning the three composite resin materials. The percentages for Clearfil Ray Posterior, Herculite XR and Visiomolar were 20, 23, and 30 percent, respectively. The shrinkage from polymerization of Clearfil is 2.3 vol. percent after 30 minutes; and that for Herculite 2.5 vol. percent (as measured by the Material Sciences Department of ACTA). Thus far, the relationship between shrinkage and sensitivity appears to be correct. The shrinkage from polymerization for Visiomolar, however, appeared to be far lower, i.e. 1.3 vol. percent. On the basis of these values, the use of Visiomolar should lead to fewer rather than more complaints. When the shrinkage is related to the Young modulus, however, a value can be calculated for the shear-stress [lit.]. For Visiomolar and Clearfil, this value appears to be virtually equal, but for Herculite it is almost three times smaller. This also does not agree with the clinical findings.

Taking into account, among other factors, the weakening of the composite resin materials as a result of water absorption, no relationship with postoperative sensitivity can be stated. It is known, however, that Visiomolar absorbs virtually no moisture, which should mean that complaints of sensitivity that occur should continue for a long time. This does not appear to be the case (see Table 1); with Visiomolar, the complaints also disappear in a relatively short time. The same is in fact true of the microleakage that can occur as a result of shrinkage from polymerization. This phenomenon is also more likely to give rise to long-lasting complaints.

In summary, the only conclusion that can be drawn from the above findings is that it is not so simple to trace the origin of complaints of sensitivity.

Method of treatment

Unfortunately, it is still not possible to prevent completely the occurrence of trauma, in most cases of a reversible nature, during the preparation and restoration of teeth. In particular, preparation with the bur, irrigating and especially drying come to mind here. Given the fact that no difference was found between the sensitivity complaints after restoration with amalgam and those after restoration with composite resin, leads to the suspicion that for the time being, it is the method of treatment that has the greatest influence on the results reported in this article.

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Management of oral complications associated with cancer therapy in pediatric patients

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Cancer is the second most common cause of mortality in childhood in the United States. It is predicted that in the current year, 6600 children will develop cancer and 1800 will die, predominantly from leukemia.[1] Treatment for childhood malignancies has improved markedly over the past forty years as reflected by decreased mortality rates.[2] Intensive and therapeutically successful treatment, however, is associated with increased morbidity, often involving the oral tissues. Morbidity from oral complications can be severe, requiring suspension of cancer therapy. Complications are a particular concern in children, because growth and development may be altered. Barring prevention, appropriate management of complications resulting from radiation and chemotherapy, therefore, is essential.

Recent studies involving combined radiation and chemotherapy reveal oral complications are three times more common in children than in adults. The increased mitotic index in children is thought to be responsible for their greater susceptibility to cancer therapy.[3,4] Oral complications in children are similar to those in adults, although children can acquire additional long-term sequelae. Acute manifestations of cancer therapy include mucositis and ulceration, candidiasis, viral and bacterial infections, bleeding, pain, gingivitis, and xerostomia, and dental caries. Abnormalities of the dentition include enamel hypoplasia, diminutive teeth, delay or failure of tooth development and eruption, and altered root formation (Figure 1). Factors influencing the severity and type of oral complications include type of malignancy and location, dose and protraction of radiation, degree of oral care before and during treatment, and developmental status of the patient.[3,5,6]

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MUCOSITIS

Mucositis and ulceration are the most frequent oral complications of cancer therapy in children and are thought to develop as a secondary response to the toxicity of chemotherapeutic agents and radiation on rapidly dividing basal epithelial cells (Figure 2). Changes in the oral mucosa begin at the nadir of the neutrophil count and resolve as the neutrophil count increases.[1] Initial symptoms include a burning sensation, dryness of the mouth, tingling of the lips, and pain. The oral mucosa becomes erythematous, particularly the soft palate, pharynx, buccal mucosa, and sublingual tissue. Discrete ulcers develop and coalesce, resulting in formation of large ulcerated areas. Chemotherapeutic agents frequently associated with mucositis include antimetabolites and in particular methotrexate, fluorouracil and cytarabine, anti-tumor antibiotics such as daunorubicin, dactinoymycin, doxorubicin, bleomycin, and hydroxyurea.[6-9] Chemotherapy in conjunction with radiation may result in more rapid onset of mucositis and increased morbidity.

Severe pain is often associated with mucositis and ulceration. Excessive discomfort can interfere with food

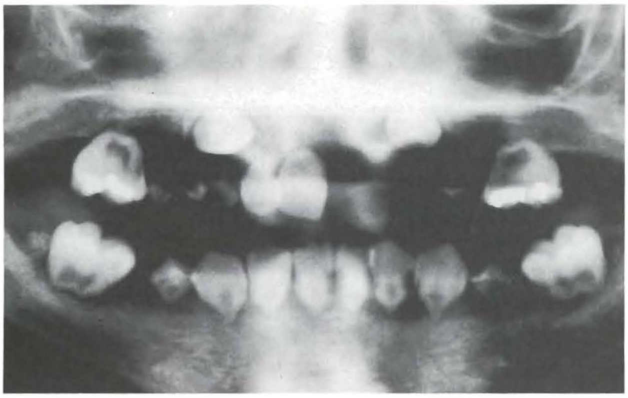


Figure 1. Panoramic radiograph reveals the absence of root formation on the permanent teeth as a result of radiation therapy for a rhabdomyosarcoma of the right middle ear at age two years.

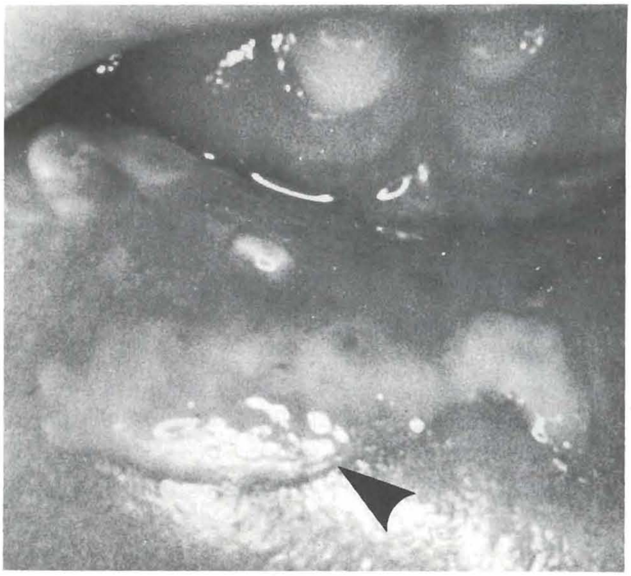


Figure 2. Mucositis and ulceration of the oral soft tissue in response to tumor chemotherapy. (Photograph courtesy of Dr. W. E. Wright)

and liquid intake requiring oral or intravenous administration of analgesics including morphine.[8,10] Postponement of cancer therapy may be necessary, when pain or complications persist. Routine management consists of topical mouthrinses containing palliative agents. Ingredients used in multi-agent topical mouthrinses can include aluminum, magnesium hydroxide, tetracycline, diphenhydramine hydrochloride, sucralfate, dyclonine hydrochloride, nystatin, hydrocortisone, sorbitol and lidocaine hydrochloride. The ingredients and formulae may vary, although most regimens have the same therapeutic goal.

The topical regimen known as "Mary's magic mouthwash" has been used extensively for topical relief of mucositis. Magic mouthwash was first prescribed at the M.D. Anderson Tumor Institute for treating the multifaceted pathogenesis of radiation mucositis.[11] The apparent clinical success of the medicaments contained in the mouthwash led to many institutions adopting the regimen. "Magic mouthwash" contains hydrocortisone as an anti-inflammatory agent, tetracycline as an antibacterial agent, nystatin as an antifungal agent, and diphenhydramine as an antihistamine and topical anesthetic (Table 1). Frequent formulation of the solution is needed due to a short shelf-life. Furthermore, tetracycline is unstable in solution with the other medicaments and must be dispensed separately. The formulae and medicaments included in "magic mouthwash" can vary depending on the prescribing institution.

A number of other mouthrinses have been formulated by various institutions. "Philadelphia mouth-

Bleeding of the gingiva and oral mucosa is commonly associated with chemotherapy.

wash" contains diphenhydramine, aluminum, and magnesium hydroxide suspension, 2 percent viscous lidocaine, and flavoring agents (Table 2).[12] Aluminum and magnesium hydroxide form a viscous layer to coat or protect the mucosa and promote healing. Diphenhydramine and lidocaine serve as topical anesthetics. Lidocaine should be prescribed with extreme caution, however, in pediatric patients. Systemic absorption occurs readily through ulcerated mucosa and toxicity can develop rapidly in children.[13]

Sucralfate, a complex of sucrose octasulfate and polyaluminum hydroxide, can be used as a topical oral suspension to manage mucositis. The complex was initially developed to treat duodenal ulcers by selectively binding to proteins of damaged mucosa and preventing acid diffusion.[14] In the oral cavity, sucralfate forms an adhesive paste that protects the mucosa and promotes healing.[15] Sucralfate has also been used in pretreatment regimens for oral mucositis.[16] Formulas and regimens may vary depending on the prescribing institution. Sucralfate suspension contains sucralfate and 70 percent sorbitol (Table 3). Stomafate suspension consists of sucralfate, diphenhydramine and pseudoephedrine syrup with aluminum and magnesium hydroxide suspension (Table 4).[12]

The prescription of multi-agent topical mouthrinses has been based primarily on subjective observation and clinical experience. Furthermore, long-term clinical studies to support the effectiveness of various topical agents are limited. Currently, use of multi-agent regimens is declining in favor of 0.12 percent chlorhexidine mouthrinse. Chlorhexidine has most of the properties of multi-agent regimens except analgesia. In children receiving bone marrow transplantation and chemotherapy, chlorhexidine has been effective in controlling mucositis and infection.[17-19] Pretreatment protocols have also been established using 0.12 percent chlorhexidine to prevent mucositis and infection. The versatility and effectiveness of chlorhexidine make it one of the most promising single agents for prevention and management of oral complications.

Table 1 □ Mary's magic mouthwash.

Hydrocortisone powder	100 mg
Tetracycline	500 mg
Nystatin suspension	1,200,000 units
Diphenhydramine elixir (12.5 mg/5ml)	240 ml

Table 2 □ Philadelphia mouthwash.

Aluminum and magnesium hydroxide suspension	90 ml
Diphenhydramine elixir (12.5 mg/5ml)	90 ml
Lidocaine 2%	90 ml
Distilled water	180 ml

Table 3 □ Sucralfate suspension.

Sucralfate	8 gm
Sorbitol 70%	40 ml
Sterile water	120 ml

Table 4 □ Stomafate suspension.

Sucralfate	12 gm
Aluminum and magnesium hydroxide suspension	180 ml
Diphenhydramine and pseudoephedrine syrup	60 ml
Sterile water	60 ml

Table 5 □ White blood cell levels: clinical significance.

Normal	>1500 μL
Some risk for sepsis	500-1000 μL
Moderate risk for sepsis	200-400 μL
High risk for sepsis	<100 μL

INFECTIONS

Oral fungal infection

Candidiasis is common in children receiving treatment for a tumor, particularly during periods of severe immunosuppression and neutropenia. Children are at higher risk for oral candidiasis because of extensive use of broad-spectrum antibiotics, chemotherapy-associated immunosuppression, inadequate oral hygiene and nutrition, and poor physical condition. Candidiasis in children occurs most frequently on the buccal mucosa, tongue, gingiva, and pharynx (Figure 3). Atrophic candidiasis involves the tongue and is associated primarily with extensive use of broad-spectrum antibiotics. Unresolved oral candidiasis can progress to disseminated candidiasis. Distinguishing oral candidiasis from other mucosal lesions such as radiation mucositis or viral and bacterial infection can be difficult. Cultures should be obtained for diagnosis and treatment of immunocompromised patients.

Clinical management of oral candidiasis in children is similar to that in adults and consists principally of antifungal agents. The medicament and route of administration is determined by severity of the infection. Nystatin and clotrimazole are used primarily as topical agents for oral candidiasis. Protocols for treating oral candidiasis using 0.12 percent chlorhexidine mouthrinse have also been established.[17,18] Nystatin and chlorhexidine, however, should not be used simultaneously. Combining these agents results in formation of a chlorhexidine-nystatin complex that appears to be

Unresponsive and disseminated candidiasis can be life-threatening.

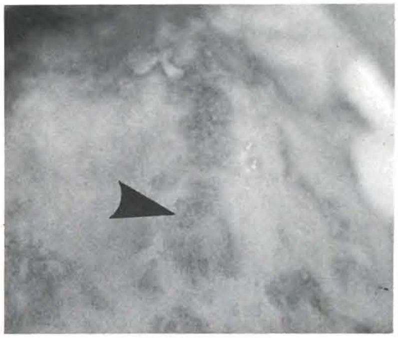
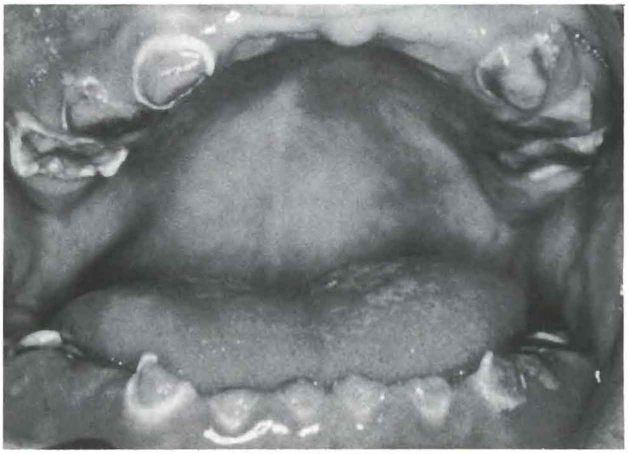


Figure 3. Candidiasis of the palate associated with head and neck radiation and chemotherapy. (Photograph courtesy of Dr. W. E. Wright)

Figure 4. Rampant caries can be the result of long-term use of oral medications high in sucrose.



ineffective against Candida.[20] Clotrimazole troches are generally more effective than nystatin for oral candidiasis.[21] Nystatin pastilles and oral suspension and clotrimazole troches can be cariogenic because of their sugar content (Figure 4). In pediatric patients, prescription of either agent should be accompanied by excellent oral hygiene.

Oral ketaconazole or fluconazole is recommended for chronic and more extensive infection such as esophageal candidiasis. Ketaconazole, however, is associated with hepatic toxicity which can lead to severe liver damage and fatal hepatic necrosis.[22] Fluconazole unlike other antifungal agents, can be administered orally or intravenously and has fewer side effects, the most common being gastrointestinal distress. The efficacy of fluconazole in comparison to amphotericin B has not been established.[23] Studies are being conducted to evaluate prophylactic use of fluconazole for oropharyngeal candidiasis in high-risk patients.[24]

Unresponsive and disseminated candidiasis can be life-threatening, requiring aggressive treatment with intravenous amphotericin B. Serious side effects frequently develop with amphotericin B, including azotemia or nephrotoxicity. The degree of azotemia and permanent damage to renal tubules is determined by dose and concomitant administration with other nephrotoxic agents.[25]

Oral bacterial infection

The majority of oral bacterial infections in children involve secondary involvement of mucosal ulcerations due to therapy-related neutropenia. The clinical appearance of inflammation can be reduced or absent in immunocompromised patients, making identification and appropriate management difficult. Lesions should be cultured before prescribing antibiotics, when possible. The presence of oral infection during treatment for a tumor is of particular concern, when the total peripheral white blood count is 500 ML or less (Table 5). During periods of severe neutropenia only conservative maintenance and emergency dental procedures should be considered in conjunction with antibiotic prophylaxis.[8,12]

The incidence of oral infection is reduced when all definitive dental treatment is completed before starting therapy. Caries lesions, abscesses, teeth with periodontal pockets of 6 mm or greater, and exfoliating primary teeth causing gingival irritation should be restored or extracted at least ten days before starting radiation or chemotherapy. Good oral hygiene before tumor treatment is essential to reduce morbidity associated with soft tissue infection. After tumor treatment has begun, dental and oral hygiene procedures can be performed without risk during periods of remission, or when blood counts are within normal limits.

Viral infection

Herpes simplex virus is the most common viral infection associated with chemotherapy and bone marrow transplantation. The virus may occur as a primary infection, or more frequently as reactivation of latent virus during periods of immunosuppression and intensive chemotherapy. The clinical presentation consists of severely ulcerated oral mucosa. Ulcerated lesions may be the portal for bacteria and fungi resulting in disseminated infection. Distinguishing between viral infection and nonspecific mucositis may be difficult. Viral cultures should be obtained for diagnosis and treatment of immunocompromised patients.

Treatment of herpes simplex infection consists of oral or intravenous acyclovir. Prophylactic acyclovir orally or intravenously should be considered in patients seropositive for herpes simplex virus and at high risk for reactivation.[26,27] Acyclovir is not indicated for patients at low risk for viral reactivation. Although the likelihood of developing acyclovir-resistance is low, potential side effects of intravenous administration include encephalopathy secondary to renal insufficiency and nephropathy.[28,29] Adverse effects encountered with oral administration include headaches and nausea.[30]

XEROSTOMIA

Radiation therapy involving the head and neck region can result in temporary or permanent destruction of salivary glands accompanied by pain, inflammation, and xerostomia. Altered salivary gland function also predisposes patients to severe radiation-associated dental caries. The severity of complications is related to the radiation dose and quantity of salivary tissue exposed. Chemotherapy in contrast to radiation, has fewer long-term effects on salivary glands. Patients receiving chemotherapy may experience temporary change in salivary quantity and quality.[31]

Managing xerostomia involves a combination of strategies including synthetic salivary substitutes, stimulation of remaining salivary tissue, maintenance of good oral hygiene and use of topical fluoride. Techniques are needed to sustain salivary gland function and prevent or minimize xerostomia. Investigations are being conducted to evaluate posttreatment administration of pilocarpine to maintain or augment salivary gland function following head and neck radiation.[32] Radioprotective agents to prevent xerostomia are also being examined.[33]

Table 6 □ Platelet levels: clinical significance.

Normal	200,000-400,000 µL
Some risk for bleeding	50,000-100,000 µL
Moderate risk for bleeding	20,000-40,000 µL
High risk for bleeding	<20,000 µL

BLEEDING COMPLICATIONS

Bleeding of the gingiva and oral mucosa is commonly associated with chemotherapy. The severity of bleeding reflects the degree of thrombocytopenia and is greatest during periods of neutropenia and immunosuppression. Gingival bleeding is exacerbated when oral hygiene is inadequate. Aggressive oral hygiene regimens are recommended, although modification may be necessary for patients with severe thrombocytopenia. In general, precautions should be taken when platelet counts are 50,000 per ML or less (Table 6).[12] Rinsing with chlorhexidine and use of disposable sponge tipped Toothettes* can replace toothbrushing during periods of severe neutropenia and thrombocytopenia.

Management of bleeding may require topical thrombin, aminocaproic acid and microfibrillar collagen hemostat. Platelet infusion is undertaken when local measures are unsuccessful. Techniques to restore neutrophil count and function following chemotherapy or bone marrow transplantation are being evaluated. In recent studies recombinant human granulocyte colony stimulating factor and granulocyte-macrophage factors have accelerated neutrophil recovery following cancer treatment.[34-36]

CONCLUSION

Cancer chemotherapy and radiation to the head and neck are associated with a high incidence of oral complications. The dentist must be prepared to diagnose and treat adverse effects and sequelae resulting from cytotoxic agents. Initiation of pretreatment oral care is also essential to minimize morbidity and improve the overall health of patients before and during cancer treatment.

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Generalized juvenile periodontitis in a thirteen-year-old child

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Periodontitis that affects children and adolescents is currently classified as follows: *Prepubertal periodontitis* (PP), *localized juvenile periodontitis* (LJP), *generalized juvenile periodontitis* (GJP), and/or *rapidly progressive periodontitis* (RPP). Certain systemic diseases such as *neutropenia, agranulocytosis, aplastic anemia,* or other "traditional blood dyscrasias", as well as *hypophosphatasia* and *Papillon-Lefevre syndrome* (PLS) have been known to manifest advanced periodontitis in the primary dentitions of prepubescent children. Periodontitis that affects the primary teeth in children who do not present with one of the aforementioned diseases is called PP[1]. It is suggested that other diseases such as *juvenile diabetes* and *histiocytosis X* should be excluded before a diagnosis of PP is made.[2] PP was first clinically characterized and defined as a disease entity in 1983 by Page and classified as a localized form (L-PP) and a generalized form (G-PP).[3] L-PP affects some but not all primary teeth and there are minimal signs of gingival inflammation. G-PP affects all the primary teeth and the gingiva exhibits severe inflammation with proliferation and cleft formation. G-PP was subsequently determined to be an oral manifestation of a systemic disease called *leukocyte adhesion deficiency* (LAD).[4,5] *Leukocyte adhesion deficiency* is a recently recognized autosomal recessive disease in which expression of the adhesion molecules Mo 1 (CD11b/CD18), LFA-1 (CD11a/CD18), and p150,95 (CD11c/CD18) glycoprotein is severely depressed. These adhesion molecules constitute a family of glycoproteins each of which is composed of noncovalently associated alpha and beta subunits.[6] It is believed that the defect is in the beta chain, which is necessary for cell surface expression of the alpha subunit.[7]

Localized juvenile periodontitis (LJP) is characterized by a pattern of attachment loss and alveolar bone resorption around permanent first molars and incisors or only around permanent first molars. The generalized form of *juvenile periodontitis* (GJP) is diagnosed on the basis of the general pattern of alveolar bone loss and attachment loss around permanent teeth.[8] It has been reported that GJP patients may have defective neutrophil chemotaxis (CX) and a suppressed autologous mixed lymphocyte response.[9,10] *Rapidly progressive periodontitis* was clinically characterized and defined as a clinical entity by Page.[11] Whether GJP and RPP are separate disease entities is still uncertain.

It is not known whether L-PP leads to GJP. In some retrospective studies it has been reported that development of LJP, periodontitis around premolars and molars, and GJP have occurred in patients who possibly had had L-PP.[12-17]

This paper reports a case in which a thirteen-year-old patient with evidence of periodontitis around her primary teeth at a prepubertal age subsequently developed GJP that affected premolars and permanent incisors, and molars.

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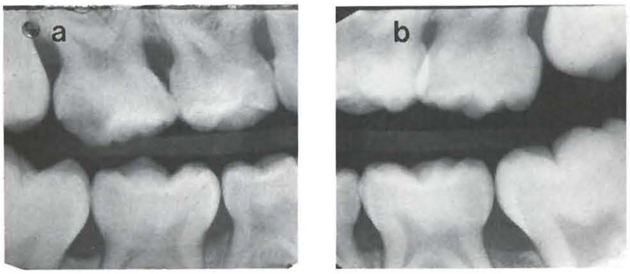


Figure 1 a,b. Radiographs of the patient taken at the age of 6.5 years. a: patient's right posterior segment. b: patient's left posterior segment. Alveolar bone resorption is evident around tooth-numbers A, B, and L.

CASE PRESENTATION

Medical history

The patient is a thirteen-year-old Hispanic female. The patient's medical history revealed that she was allergic to penicillin. She had asthma and had been using a Ventoin inhaler when necessary. According to the patient and her mother, she had had several asthma attacks in the past. Her last asthma attack occurred a year before being seen in the clinic and at that time she was admitted to the hospital. Since that episode, she has been using an inhaler once or twice a week. The patient has no history of recurrent infections. She was slightly overweight for her height. Her puberty was at 13.5 years or approximately four months before the initial appointment. She has two younger sisters (ages 1 month and 11 years). No detailed dental history was available for her eleven-year-old sister. She presented, however, with gingival inflammation and subgingival calculus. The patient's blood pressure was 120/84 at the first appointment. There was no evidence of hyperkeratosis of her palms and soles. The patient's mother stated that there was no history of diabetes or other systemic diseases in her family. No information was available about the patient's father.

Dental/periodontal findings

The patient was referred to the Department of Periodontics at the University of Illinois in April 1988, in order to obtain a second opinion on her gingival condition and treatment.

In February 1988 the patient had experienced bleeding from the gingiva and her mother sought periodontal treatment for her daughter at a private periodontist's office. At that time, according to the patient and her mother, cleaning was done under local anesthesia in two appointments. Her mother was then told that her daughter would need extensive periodontal surgery. A second opinion was requested at the University of Illinois.

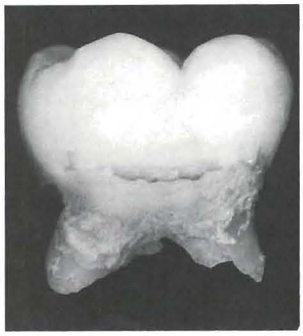


Figure 2. Photograph of exfoliated primary tooth with subgingival calculus formation. Representative of all exfoliated teeth.

The conversation with the patient's mother revealed that the patient was seen by her general dentist, when she was 6.5 years old, for discomfort due to mobile teeth (patient does not recall which teeth were involved) and spontaneous bleeding of her gums. Two sets of bitewing radiographs were taken at that time, and tooth A was extracted because of its carious condition. The bitewings showed moderate bone loss around teeth A, B and C, and slight bone loss around L (Figure 1a, b). Calculus spicules were evident on radiographs. The exfoliated primary teeth (A,B,C,E,F,I,J,L,M,R, and T), which were kept frozen by her mother, all exhibited unusually heavy calculus accumulation below the cementoenamel junctions (Figure 2).

The patient's gingiva presented with severe granulomatous inflammation around tooth numbers 9,10,12,14 (Figures 3,4). Most interdental papillae were soft and edematous. Bleeding on probing occurred at most of the sites. The Plaque Index was 90 percent, and the

Figure 3. Anterior photograph of the patient at the first appointment, showing granulomatous gingival inflammation.

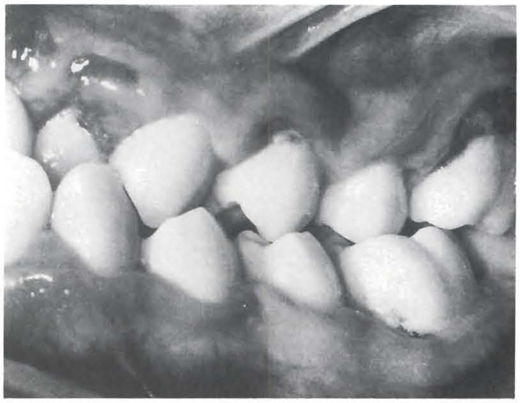
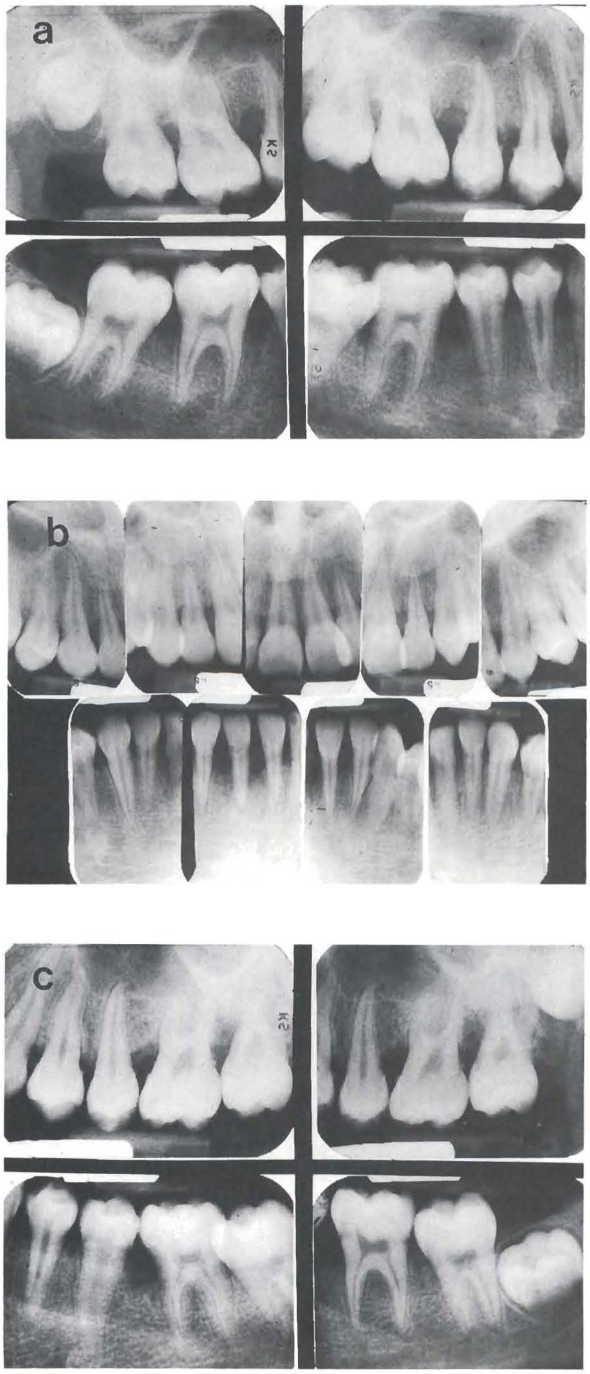


Figure 4. Left posterior photograph of the patient at the first appointment, showing severely inflamed gingival tissue.

Figure 5 a,b,c. Radiographs of the patient taken at the age of thirteen years. a: patient's right posterior region. b: patient's incisor region. c: patient's left posterior region.



Gingival Index was 2.0.[18,19] Pocket depths ranged from 4-7 mm interproximally in the maxillary arch and 2-5 mm interproximally in the mandibular arch. Furcation involvements were present on the distal surfaces of #3 and #14 (both Class II). Attachment losses of greater than 4 mm were present around several of the posterior teeth. Subgingival calculus was present throughout.

Radiographic examination of the permanent dentition revealed moderate to severe bone loss around premolars, several molars and a lower central incisor (Figure 5a,b,c). Calculus spicules were also evident on many teeth (Figure 5a,b,c).

The patient had anterior and posterior cross bite relationships except for #2/#30, #3/#30, #7/#26 & #27, #14/#18 and #15/#18. Fremitus was present on #25. There were no balancing interferences in either right or left lateral excursions.

Laboratory findings

The findings of laboratory tests of peripheral blood taken before the initiation of treatment were all within normal limits and were as follows: RBCs 4.62 million/μl, WBCs 7000/μl with the following differential: lymphocytes 49.4 percent, monocytes 4.7 percent, granulocytes 45.9 percent with normal morphology. The value for hemoglobin was 13.2 g/dl, HCT 38.5 percent, platelet 400 k/μl, fasting glucose level 90 mg/dl, and serum alkaline phosphatase 180 U/l. The calcium level was 9.7 mg/dl and the phosphorus level was 3.6 mg/dl.

These results excluded the possibility of her having *neutropenia*, *Chediak-Higashi syndrome*, *juvenile diabetes*, and *hypophosphatasia*. A gingival biopsy taken from #9 showed a dense infiltration of lymphocytes and plasma cells, and no indication of *histiocytosis* X. Neutrophil chemotaxis (CX) and CD11b/CD18 assays indicated that they were within normal range for normal healthy subjects in this age-category.[20,21]

Treatment and follow-ups

The initial therapy included oral hygiene instructions (OHI), scaling and root planing (S&R) using local anesthesia, and the re-evaluation of periodontal tissue response to the treatment. The gingival tissue response to the S&R was better in the mandibular than the maxillary arch. Most of the granulomatous gingival tissue except for the labial of #9 resolved. Bleeding on probing was present interproximally throughout the maxillary arch, however, and between several mandibular posterior teeth (total of fifty-two sites). The patient developed new supra- and subgingival calculus in the maxillary posterior area within a two-month period. There was no significant (greater than 2 mm) improvement in clinical attachment measurements. Interproximal probing depths ranged from 1-7 mm in the maxillary arch and 2-5 mm in the mandibular arch. The PI was 30 percent and the GI was 1.2. Occlusal adjustment was performed on tooth #25 to eliminate the fremitus. A Modified Widman Flap procedure was done in the maxillary left and right quadrants to gain access for more thorough root planing and debridement of the lesion. A free gingival graft was done on the labial of #25 to create a zone of keratinized gingiva. The patient has been monitored for a year following the surgical phase and has shown an improvement of her periodontal condition (PD < 5mm, GI = 0.5, PI = 25 percent). BOP was present at nine interproximal sites as opposed to fifty-two sites at the re-evaluation time (Figures 6 and 7).

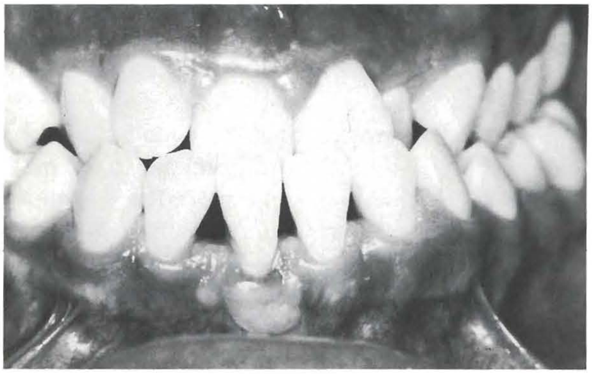


Figure 6. Anterior photograph of the patient a year following active phase of periodontal therapy.

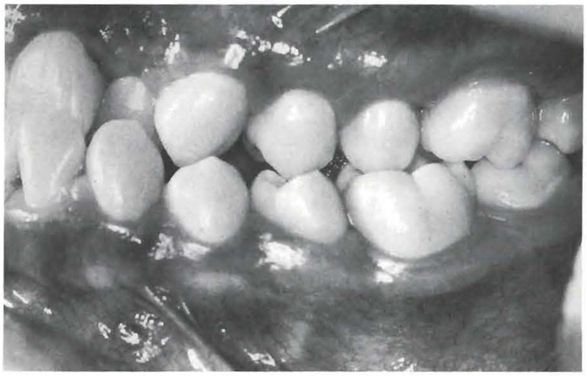


Figure 7. Left posterior segment of the patient's dentition a year following active phase of periodontal therapy is shown.

DISCUSSION

The term *periodontosis* (now referred to as *juvenile periodontitis*) was coined by Orban and Weinmann and clinical characterization was reported by Baer in 1971.[22,23] It was described as a disease of the periodontium occurring in an otherwise healthy adolescent. The onset is sometime during the circumpubertal pe-

Diagnosis of generalized juvenile periodontitis is based on the general pattern of bone and attachment loss around the permanent teeth.

riod (between eleven to thirteen years old). Baer recognized that there are two forms of juvenile periodontitis (JP): the classical and generalized forms. In the classical form, arc-shaped alveolar bone resorption occurs from the distal surface of the second premolar to the mesial surface of the second molar and the lesion is bilaterally symmetrical. In contrast, the generalized form of JP affects all teeth. Recently the American Academy of Periodontology defined GJP as a periodontitis that occurs at an early age (twelve to thirty years) and is characterized by a rapid and severe periodontal destruction around most of the teeth.[24] It was suggested that the GJP may be the terminal stage of periodontitis that had started as the localized form of JP.[2]3 In support of this idea was the result of the study by Gursolley *et al.*[25] In their study, the level of antibody against *Haemophilus actinomycetemcomitans* (Ha) Y4 and *Porphyromonas gingivalis* (Pg) were compared for patients with LJP and SP (generalized early onset severe periodontitis). It was found that the subjects who lacked antibody to both Ha and Pg were among the most severely involved subjects and that no LJP subjects lacked antibody to both pathogens. Thus, it has been suggested that those patients with LJP who cannot mount an antibody response to Pg or Ha Y4 may later develop GJP. In contrast, a thirteen-year-old patient with advanced GJP was reported to have a markedly elevated IgG antibody against Pg.[26] Further studies will be necessary to clarify the relationship between GJP and the presence of Pg antibody.

A further possible association between GJP and Pg has been suggested by Wilson *et al.*[26] In their microbiological study, Bg constituted 8-16 percent of the cultivable microflora and 13-20 percent of the total cell-count in subgingival plaque samples obtained from five of five lesions in a thirteen-year-old female with GJP; Pg was not found in a healthy site.[26]

It is not known whether L-PP evolves to LJP or GJP (or RPP). Among the case reports, there are two reports in which children with a possible history of PP developed GJP around circumpubescent age. In these two cases, however, the only history pertaining to the primary dentitions was premature exfoliation of the primary teeth.[16,17] In this report, alveolar bone resorption around primary molars was evident on the previous radiographs, and unusually heavy subgingival calculus was found around exfoliated primary teeth. Even though reports by Sjodin *et al* and others are retrospective in nature, their reports suggest that patients with L-PP may develop periodontitis around erupting permanent teeth.[12-17] This case report provides another example.

> In prepubescent children, periodontitis may be associated with a systemic disease.

There is strong evidence that PMNs play a critical role in the defense of the periodontium.[27,28] Van Dyke *et al* reported that nine of seventeen patients with GJP exhibited reduced PMN chemotaxis, compared to those of age- and sex-matched healthy controls.[29] In this report, the patient had normal chemotaxis as compared to the age-matched control-subject and minor control-group.

Periodontitis in prepubescent children is of great concern, because periodontitis may be due to an underlying systemic disease not apparent in the patient's medical history.[2] In this respect, proper laboratory tests and examinations must be performed to exclude such possibilities. When a child does not have such systemic diseases, the next major concern is reduction or elimination of the inflammation, if present, and prevention of further development of periodontitis around erupting permanent teeth. Whether the possible pathogens of L-PP such as Pg continue to infect the tissues adjoining erupting permanent teeth to initiate LJP or GJP is not clear, and also difficult to prove. Other factors such as the host immune-response may also play a major role in whether a subject with L-PP later develops periodontitis in the permanent dentition. In spite of these ambiguities, several case reports do suggest the possibility of development of periodontitis in circumpubescent children who also had periodontitis in their primary dentitions. It is critical, therefore, to identify periodontitis in the primary dentition and closely mon-

I would like to express my appreciation to Dr. Philip M. Hoag in the Department of Periodontics for reviewing this manuscript.

itor the periodontium of a child through the transitions from the primary, to the mixed, to the permanent dentitions.

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Radiographic features of the bones of the hand and wrist in achondroplasia: Report of case

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The cardinal features of achondroplasia are easily recognized: rhizomelic form of short-limbed dwarfism; enlarged calvaria with hydrocephaly and frontal bossing; midface deficiency; relative mandibular prognathism; depressed nasal bridge; short, stubby and trident hands; lumbar lordosis; prominent buttocks and protuberant abdomen, due to a pelvic tilt.[1] Fewer than 20 percent of reported cases are familial with an autosomal dominant mode of transmission. More than 80 percent of the cases are sporadic, representing point mutations.[2] Increased parental age at the time of conception is often associated with the sporadic cases.[3] The frequency of achondroplasia ranges from 0.00004 to 0.00014 with no sexual predilection. Gross overestimates of figures are possible, however, because of the lack of a clear differentiation between achondroplasia and other forms of short-limbed dwarfism.[4]

Development is relatively slow; especially the acquisition of motor skills associated with the large head and short extremities. The intelligence and the ultimate development are, however, almost normal. The dental anomalies said to be associated with achondroplasia are tapered maxillary incisors, delayed eruption, and malocclusion.[5-8] The characteristics of the malocclusion are anterior open-bite, due to the restricted base of the skull, the recessive maxilla, the upwardly tilted anterior palate, and the relatively protrusive mandible.[9]

Many of the features of achondroplasia closely resemble those of acrodysostosis, which includes peripheral dysostosis, nasal hypoplasia, and mental retardation.[10] Although acrodysostosis can occur sporadically, increased paternal age at the time of conception is evident in some cases. The most marked features include a short flat nose with a broad dimpled tip, anteverted nostrils and a depressed nasal bridge. The fingers and toes are short and stubby. Radiographs show shortening of the metacarpals and phalanges and evidence of premature fusion of the cone-shaped epiphyses and diaphyses of the phalanges.[1]

CASE REPORT

The patient was a Chinese boy, thirty-nine months old, whose thirty-nine-year-old father, twenty-seven-year-old mother, eleven-year-old sister and one-year-old brother were all normal. The pregnancy went to term without any complications and the birth weight of 3.3 kilograms was considered to be unremarkable. Delivery was complicated, because of the large size of the head. In addition to the big head, the shortness of the limbs was noticed during the neonatal period. Subsequently, additional features were noted, they were dilated veins on the forehead, separation of the cranial sutures, frontal bossing, maxillary hypoplasia, flat nasal bridge, trident hand, shortened proximal segment of

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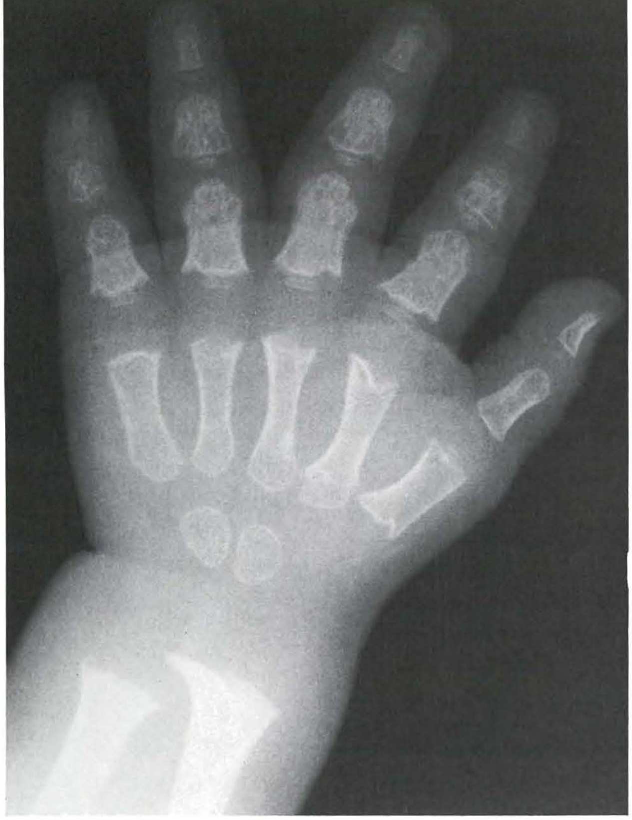

Figure 1. The proband's hand and wrist appearance at the age of thirty-nine months.

Achondroplasia is frequently confused with other chondrodystrophies.

the upper and lower limbs, marked lumbar lordosis and positive laxity of the joints. Intraorally, all of the maxillary incisor teeth and the mandibular lateral incisor teeth were small and conical in shape. The maxillary canines were peg-shaped, while the mandibular left central incisor was missing. The other erupted primary teeth were normal, although the clinical crowns were relatively short. Habitually, the mouth was open at rest time.

Delayed development was evident, especially in respect of gross motor control. The intelligence quotient was 68 according to the Griffith's Mental Development scale, and the mental age was estimated to be 26.6 months. There was a communicating hydrocephalus for which shunting procedures had been provided. The condition was diagnosed at the age of thirty-six months as achondroplasia with communicating hydrocephalus. It was considered that it was probably a spot mutation, and that the delayed development was due to environmental deprivation. The stature and body weight at the age of three years were 77 cm and 9 kg, both of which fell below the third percentile for Southern Chinese children in Hong Kong. The head circumference at 56.5 cm was, however, above the 97th percentile.

The posteroanterior radiograph taken at the age of thirty-nine months (Figure 1) of the left hand and wrist revealed abnormal development. The proximal and middle phalanges were broader and shorter than the other phalanges of the hand. The epiphyseal growing ends of the metacarpals, proximal and middle phalanges of digits II, III, IV and V, as well as the proximal end of metacarpal I, were deformed with roughened edges. Similarly, the ulnar surface at the distal end of

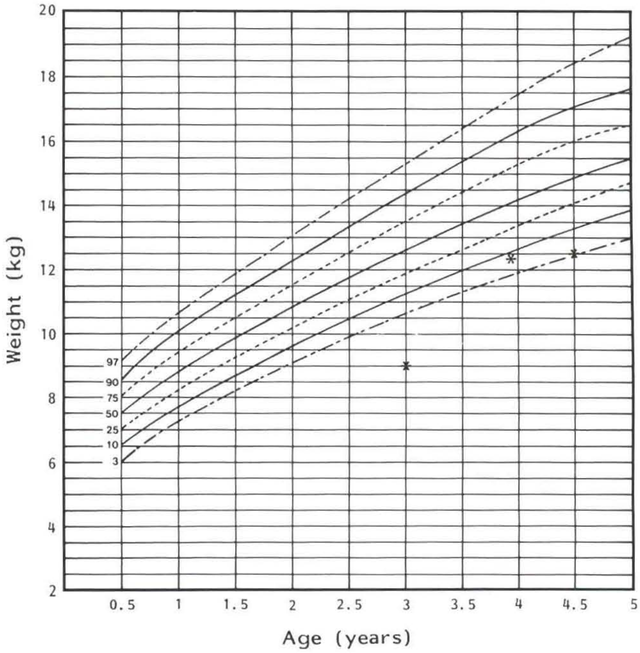

Figure 2. The body weight of the proband (*), when compared to normal peers was always below the 10th percentile.

the radius was developing abnormally. The bone trabeculation of the phalanges appeared abnormal. The skeletal age assessed from the Greulich and Pyle Atlas method was 16.1 months.[12]

DISCUSSION

The observed physical growth pattern resembled that seen in both achondroplasia and acrodysostosis. The nose was similar to that described in acrodysostosis; anteverted nostrils with a dimpled tip.

The growth in body weight (Figure 2) and standing height (Figure 3) were noted to be very slow, both below the respective tenth and third percentile levels for the comparable age-group.[11]

Analysis of the skeletal maturation of the hand and wrist region showed marked delay in development. The skeletal age was 16.1 months as compared to the mean of 30.22 months (± 6.52 months) for three-year-old Chinese boys in Hong Kong (Low, 1969). A secular trend of early skeletal maturation was recently demonstrated for Hong Kong, this may be attributed to improved socioeconomic and nutritional status; hence, the retarded skeletal maturation in the proband was extremely marked.[13] This agreed with the skeletal age being almost two years behind the chronological age.[14]

A comparison with the hand and wrist radiograph of a 21-month-old boy (Figure 4) taken from the study of Low, and the film (Figure 1) of the proband taken when he was thirty-nine months old showed a disproportionate widening and abnormal bone trabeculation of the diaphyses of the proximal and middle phalanges of digits II to V.[15] The broad and short proximal and middle phalanges resemble those reported by Langer and coworkers, who also remarked that the metacarpals were not strikingly abnormal.[16] The metacarpals of the proband were definitely abnormal. Although the growth ends of the metacarpals were abnormal, no extra epiphyses were detected as mentioned by Nehme and coworkers.[14] The carpal bones exhibited the most delay. In addition, the radius was definitely bowed (Figure 5). The abnormal development of the epiphyseal ends and the premature fusion of the phalanges, and the bowed radius were features previously documented as occurring in acrodysostosis; relatively few reports mentioned that these characteristics occur in achondroplasia.[1,4]

Although dental anomalies that include an anterior open-bite and tapered incisors have been found in achondroplasia and were evident in the proband, delayed eruption was not; he had a complete primary dentition by the age of thirty-nine months, except for the congenitally missing mandibular left central incisor. It is possible that the short clinical crowns were not an anatomical feature, but the result of delayed passive eruption.

The diagnosis of achondroplasia was made for the present case from both the clinical appearance and features observed on the radiographs of the left hand and

Figure 3. The standing height of the proband (*), when compared to normal peers was always below the 3rd percentile.

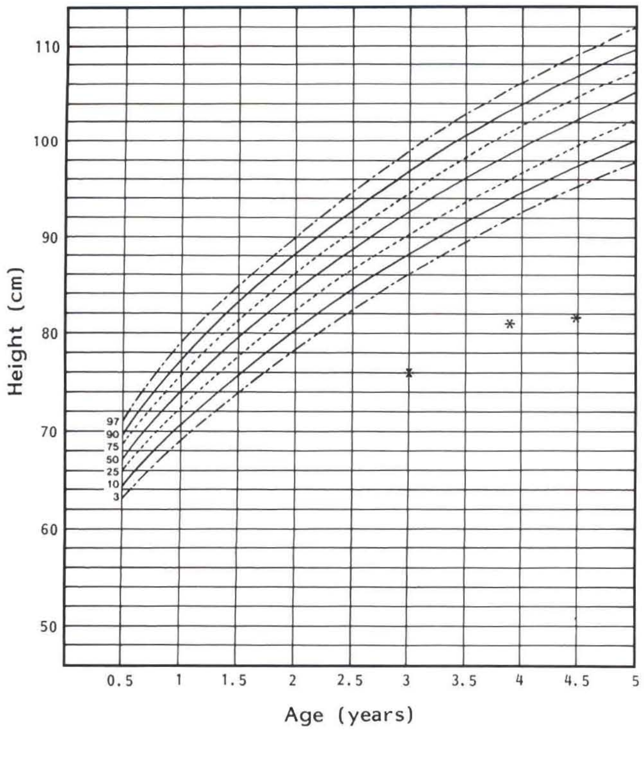

Figure 4. The hand and wrist appearance of an unaffected twenty-one-month-old Chinese boy.

Figure 5. The proband's bowed radius at the age of fifty-one months.

The clinical crowns were relatively short.

wrist. As achondroplasia is frequently confused with other chondrodystrophies, careful analyses of the radiographs are crucial to the confirmation of the diagnosis. The previously infrequently reported delay in ossification of carpal bones and abnormal trabeculation pattern of the metacarpals and phalanges were conspicuous features in this achondroplastic boy. The characteristics of the bones of the hand and wrist may not be isolated chance findings. All suspected cases of achondroplasia, therefore, should have hand and wrist radiographs taken to establish the true occurrence of these characteristics of the bones in achondroplasia.

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Epidemiology

Increasing numbers of pediatric AIDS patients

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The Acquired Immune Deficiency Syndrome (AIDS) epidemic that now is spreading throughout the world has received wide attention in the lay and professional press. Few, if any dental practitioners, are unaware of the increasing changes that are occurring in dental and medical practices as a result of this deadly menace. In the past, most epidemiologic emphasis of the disease was on the distribution and spread of AIDS in particularly high-risk groups in the population. More recently, it was realized that the AIDS epidemic increasingly is spreading beyond the boundaries of specific groups and reaching a widening swath of the general population.

Many pediatric and general dental practitioners, however, may be unaware of the extent to which the AIDS epidemic has spread among children in the United States. In an effort to describe the distribution and (where known) the source of the AIDS infection in children and adolescents, the following presentation will review available data from HIV/AIDS Surveillance Reports that are issued by the Centers for Disease Control (CDC) of the U.S. Public Health Service, and other federal reports.[1,2]

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Table 1 ☐ Number of pediatric* AIDS cases reported by type of exposure: 1981-84 to 1989-90.[1,2]

							Cumulative total	
Type of exposure	1981-84	1985-86	1986-87	1987-88	1988-89	1989-90	Number	Percent
Mother with/at-risk for AIDS/HIV infections	75	126	181	332	440	598	2,233	83.1%
Transfusion	14	23	25	59	57	43	250	9.2
Hemophilia/Coagulation disorder	6	8	13	28	33	28	136	5.0
Undetermined	na	2	6	14	27	28	67	2.5
Other	10						10	0.3
Pediatric subtotal*	105	159	225	433	557	697	2,686	100.1%
Adult/adolescent subtotal	8,840	9,410	14,799	24,008	32,360	36,857	152,231	
Total	8,945	9,569	15,024	24,441	32,917	37,554	154,917	

*Under 13 years of age at the time diagnosis of AIDS.
Note: Except for 1981-84, data are based on the 12-month period from April to March of each year. Cumulative data are through October 1990.

Table 2 ☐ Total U.S. child and adolescent AIDS cases reported through October 1990 by age-group, sex and race/Hispanic origin.[1]

Age-group	Non-Hispanic White	Non-Hispanic Black	Hispanic	Asian	American Indian	Total
Less than 5 yrs.						
Male	196	619	302	3	2	1,126
Female	185	595	280	1	3	1,065
5–12 yrs.						
Male	155	91	69	5		322
Female	42	77	50	3		173
Pediatric total	578	1,382	701	12	5	2,686
13–19 yrs.						
Male	216	140	88	6	5	456
Female	40	82	24	1	1	148
Total pediatric and adolescent	834	1,604	813	19	11	3,290

Note: Totals include individuals whose race/ethnicity were unknown.

Table 3 ☐ Pediatric* AIDS cases and deaths by year, through October 1990.[1]

Year	Cases diagnosed during interval	Death occurring during interval
Before 1981	6	1
1981	14	8
1982	29	14
1983	74	29
1984	111	49
1985	220	112
1986	309	148
1987	464	269
1988	552	279
1989	592	313
1990 (thru Oct.)	315	175
Total	2,686	1,399**

*Under 13 years of age at the time diagnosis of AIDS.
**Includes 2 children whose dates of death are unknown.

NUMBER OF CASES AND SOURCE OF INFECTION

As of October 1990, a total of 154,917 AIDS cases had been diagnosed and reported in the general U.S. population; 2,686 AIDS cases in children less than thirteen years of age (Table 1); 2,191 cases in children less than five years of age; 495 cases in children between five and twelve years of age; and 604 cases in adolescents between thirteen and nineteen years of age (Table 2). In addition, 94,375 adults and adolescents had died (a mortality rate of 62 percent.[1] By the same time, 1,399 children younger than thirteen years of age had died, a mortality rate of 52 percent (Table 3).

The overwhelming number of pediatric cases (2,233 or 83.1 percent) were associated with a mother with or at risk of AIDS infections (i.e. an intravenous [IV] drug user, had sex with an IV drug user, or was born in particular areas of the world [areas of Africa and some Caribbean countries where most of the reported cases occur in heterosexuals]) (Table 1).

INCREASES OVER TIME

Since the early 1980s, there has been an annual increase in the numbers of pediatric AIDS cases and related deaths. The number of cases reported in 1989-90 was more than six and half times the number reported between 1981 and 1984. The number of cases in all exposure types has increased, with the exception of transfusions and hemophilia/coagulation disorders, which appear to have stabilized (Tables 1 and 3; Figure 1).

PERSONAL CHARACTERISTICS

More than three quarters (78.5 percent) of the pediatric cases are among minority children. By contrast, 42 percent of adult cases are in minority populations.[1,2] Transmission from mothers with or at risk of AIDS/HIV infection accounts for the large majority of pediatric AIDS cases among all ethnic groups. For children younger than five years of age, however, there were almost seven times the number of cases of this type (i.e. transmission from mothers) among black than among white children.[2] Despite the availability and use of a reliable screening process for blood, transfusions remain the second leading source of HIV exposure. These latter cases are more prevalent among white children than minority children (Table 4).[2]

In all child and adolescent age-groups, particularly the adolescent group, there were more male than female reported cases of AIDS (Table 2).

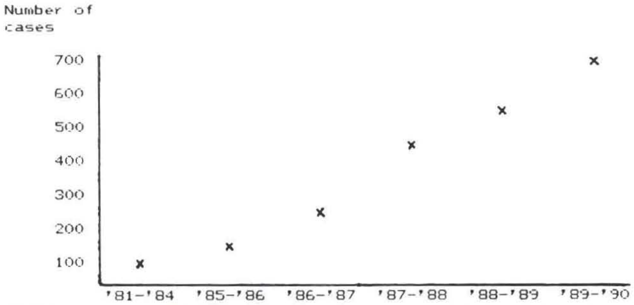
PLACE OF RESIDENCE

By state

The pediatric AIDS epidemic is not a disease limited to a single city or state of our country. Indeed, only four states (Montana, North Dakota, South Dakota and Wyoming) have had no reported cases of AIDS for children younger than thirteen years of age. Pediatric AIDS cases occurred in all other states with each of six political jurisdictions (California, District of Columbia, New Jersey, New York, Texas and Puerto Rico) reporting more than a hundred cases (Table 5).

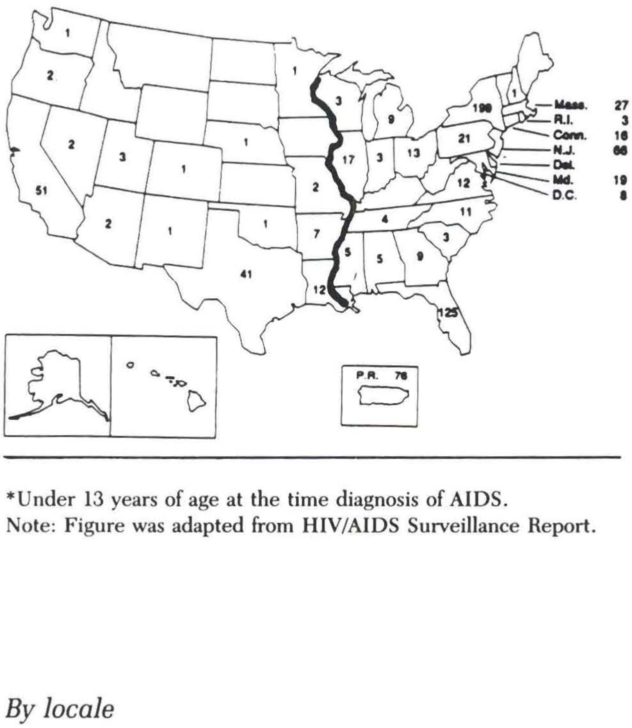
Although pediatric AIDS cases have been occurring throughout the country, during the past year (between November 1989 and October 1990), except for the states of Arkansas, California, Louisiana and Texas, almost all new cases have been in states that are east of the Mississippi River. During the past year, most states west of the Mississippi River have reported either one to three cases or no new cases of pediatric AIDS (Figure 2).

Figure 1. Number of pediatric AIDS cases 1981-84 to 1989-90.*[1,2]



*Under 13 years of age at the time diagnosis of AIDS.
Note: Except for 1981-84, data are based on the 12-month period from April to March of each year.

Figure 2. Pediatric AIDS cases reported between November 1989 and October 1990.*[1]



*Under 13 years of age at the time diagnosis of AIDS.
Note: Figure was adapted from HIV/AIDS Surveillance Report.

Table 4 ☐ Total pediatric* AIDS cases by type of exposure and race/Hispanic origin through October 1990.[1]

Type of exposure	Non-Hispanic White	Non-Hispanic Black	Hispanic	Asian	American Indian	Total
Mother with/at risk for AIDS infection						
IV drug use	172	627	309	1	2	1,115
Sex with IV drug user	74	205	187	1		468
Born in particular areas of the world**	2	204	2			209
Unspecified	34	139	44	1	2	220
Other**	65	97	55	1	1	219
Transfusion	132	55	58	5		250
Hemophilia/coagulation disorder	92	18	23	3		136
Undetermined	7	37	23			67
Total	578	1,382	701	12	5	2,686

*Under 13 years of age at the time diagnosis of AIDS.
**Areas of Africa and some Caribbean countries where most of the reported cases occur in heterosexuals.
***Include mothers who had sex with bisexual male, person with hemophilia, persons born in "particular areas" of the world or transfusion recipient.
Note: Totals include individuals whose race/ethnicity were unknown.

By locale

Pediatric AIDS cases are being reported beyond the limits of "*that city*." (Note: no doubt "*that city*" would be defined differently by individuals in various parts of our country.) By October 1990, five or more cases of pediatric AIDS were reported in fifty-eight metropolitan areas throughout the country); twenty-five or more cases were reported in nineteen metropolitan areas. In addition, 433 pediatric AIDS cases were reported in smaller metropolitan and nonmetropolitan areas (Table 6).

It is against the law to refuse to treat an AIDS or HIV-Positive patient.

It should be noted that, between February 1989 (the first period covered in the HIV/AIDS Surveillance Report series) and October 1990, the number of reported pediatric HIV cases in metropolitan areas increased by 85.6 percent. During the same period the number of cases in smaller metropolitan and nonmetropolitan areas increased by 91.6 percent.[1]

FROM THE PERSPECTIVE OF THE PEDIATRIC DENTIST

"You'd better not refuse to treat an AIDS or HIV-Positive Patient!"[3] The Americans with Disabilities Act signed into law on July 26, 1990, reversed almost one hundred years of case law precedent that held that a dentist's and physician's office was not a "place of public accommodation." The courts consistently had ruled that because a dentist's office was not a place of public accommodation, it was not subject to the regulations of any agency having jurisdiction to enforce antidiscrimination laws. Thus, in the past, practitioners could refuse to treat patients who were handicapped.[3]

The new federal law clearly defines "public accommodations" to include the "professional office of a health care provider, hospital, or other service establishment" (along with a list of other facilities, including: a laundromat, dry-cleaner, bank, barber shop, beauty parlor,

Table 5 □ Cumulative total of pediatric* AIDS cases through October 1990, by state.[1]

State	Cases	State	Cases
Alabama	20	Montana	–
Alaska	2	Nebraska	2
Arizona	7	Nevada	5
Arkansas	10	New Hampshire	5
California	204	New Jersey	275
Colorado	8	New Mexico	2
Connecticut	63	New York	777
Delaware	4	North Carolina	33
Dist. of Col.	34	North Dakota	–
Florida	369	Ohio	36
Georgia	44	Oklahoma	10
Hawaii	2	Oregon	5
Idaho	2	Pennsylvania	74
Illinois	64	Rhode Island	9
Indiana	10	South Carolina	21
Iowa	3	South Dakota	–
Kansas	3	Tennessee	14
Kentucky	5	Texas	106
Louisiana	36	Utah	8
Maine	2	Vermont	1
Maryland	65	Virginia	34
Massachusetts	69	Washington	12
Michigan	30	West Virginia	2
Minnesota	8	Wisconsin	4
Mississippi	10	Wyoming	–
Missouri	12		
		Sub Total	2,521
		Puerto Rico	161
		Virgin Islands	4
		Total	2,686

*Under 13 years of age at the time diagnosis of AIDS.

Table 6 □ Cumulative total of pediatric AIDS cases through October 1990 by metropolitan areas with 500,000 population or more and five or more cases.[1]

Area	Cases	Area	Cases
Anahein, CA	9	Monmouth-Ocean City, NJ	23
Atlanta, GA	25	Nashville, TN	6
Austin, TX	6	Nassau-Suffolk, NY	38
Baltimore, MD	47	New Haven, CT	27
Bergen-Passaic, NJ	27	New Orleans, LA	18
Birmingham, AL	6	New York, NY	706
Boston, MA	51	Newark, NJ	124
Bridgeport, CT	20	Norfolk, VA	7
Charlotte, NC	6	Oakland, CA	14
Chicago, IL	49	Orlando, FL	7
Cleveland, OH	9	Philadelphia, PA	52
Dallas, TX	14	Providence, RI	8
Dayton, OH	5	Raleigh-Durham, NC	9
Denver, CO	5	Richmond, VA	7
Detroit, MI	21	Riverside-San Bernardino, CA	15
Ft. Lauderdale, FL	46		
Ft. Worth, TX	7	Sacramento, CA	5
Greensboro, NC	6	St. Louis, MO	7
Hartford, CT	15	Salt Lake City, UT	5
Houston, TX	45	San Antonio, TX	11
Jacksonville, FL	23	San Diego, CA	19
Jersey City, NJ	40	San Francisco, CA	17
Las Vegas, NV	5	San Jose, CA	8
Little Rock, AR	5	San Juan, PR	107
Los Angeles, CA	90	Seattle, WA	10
Memphis, TN	6	Springfield, MA	9
Miami, FL	157	Syracuse, NY	5
Middlesex, NJ	25	Tampa-St. Petersburg, FL	24
Minneapolis-		Washington, DC	62
St. Paul, MN	6	W. Palm Beach, FL	53
All Metropolitan areas with population of 500,000 or more (including those with less than five cases)			2,253
All other areas			433
Total			2,686

Many pediatric and general dental practitioners may be unaware of the extent to which the AIDS epidemic has spread among children.

gas station, office of an accountant or lawyer, pharmacy and insurance office").[3]

As a result of the inclusion of AIDS patients and those testing HIV positive within the definition of "handicapped", it would indicate that a dentist who denies care to these individuals, or discriminates against them in any manner, could be subject to prosecution in a federal court.[3]

But pediatric dental practitioners have a long record of providing dental services to special populations of children. In addition, while changes in federal legislation may have clarified the environment for the general practice of dentistry, the reality is that the pediatric dental practitioner increasingly has been called upon to provide services to a growing and more diverse spectrum of special population groups of children.[4,5]

As a result of new forms of treatment, the life expectancy of AIDS and HIV positive children (and adults) will be extended (it is hoped until such time that preventive and curative measures are developed). Pediatric dental practitioners will be planning and providing services on an increasingly longer term basis for youngsters who are HIV positive and/or have AIDS.

The continuing spread of the AIDS pediatric (and adult) epidemic to more regions of the nation, surely will involve most, if not all, dental practices in the care of these individuals. In addition, there is the fact that hundreds of thousands (or is it millions?) of youngsters, adolescents and adults are, or soon may be, HIV positive and unaware of their status. And any effort to test patients to determine their HIV status is entangled in a nightmare of court rulings and legislative acts. The reality is that dentists and all other health professionals will need to come to terms with a communicable and fatal disease that has been transformed into a legal, political, and social anathema.

This review of the demographic developments in the United States indicates that there has been a continuing growth in the numbers of pediatric AIDS patients. The pediatric dental practitioner increasingly will be called upon to provide the needed care!

"... for every child who meets the CDC definition of AIDS, another two to ten are infected with HIV, and that by 1991 there will be at least 10,000 to 20,000 HIV-infected children in the United States."[2]

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Water purification systems and recommendations for fluoride supplementation

Fluoridation

Robert G. Glass, DDS

Fluoridating public water supplies is an idea that began with H. Trendly Dean. Dr. Dean, a dental surgeon in the U.S. Public Health Service, was correlating the occurrence of dental mottling with fluoride concentrations in certain communities; unexpectedly he found a lower incidence of dental caries in the patients living in the fluoridated areas. Dr. Dean concluded that the fluoride content of the drinking water caused a lower rate of dental caries.

In 1945, fluoride was introduced into drinking water in Grand Rapids, Michigan, the beginning of a study to be conducted over a period of ten years, comparing dental caries incidence in fluoridated versus nonfluoridated communities. Results after only five years of investigation revealed a sharp reduction of dental caries in the cities with fluoridated community water supplies.

MODERN FLUORIDATION

Today it is estimated that 250 million people in the world drink fluoridated water; 120 million of those live in the United States.[1]

The recommended level of water fluoridation for optimal dental caries reduction is 0.7 ppm (mg/l) to 1.0 ppm, with 4.0 ppm being the maximum contaminant

The author was a captain in the U.S. Army Dental Corps at the time this paper was written. It was written as a result of his participation in the Advanced Educational Program in General Dentistry at Fort Campbell, Kentucky.

level allowed by the Environmental Protection Agency (EPA).

Fluoride is added to our water supply in several different forms, depending on the size of the community. Hydrofluorosilic acid, sodium silicofluoride, and sodium fluoride are commonly used for fluoridating water supplies. Hydrofluorosilic acid is a by-product of phosphate fertilizer manufacture. Sodium silicofluoride and sodium fluoride are made through different methods of neutralizing hydrofluorosilic acid. Every year, 143,000 tons of fluoride are consumed.

In the United States, water fluoridation is endorsed almost universally by medical and dental associations and many scientific bodies. The AMA, ADA, PHS, and the Surgeon General agree water fluoridation is a cost-effective and safe way to reduce dental caries by 50-65 percent.[1]

Fluoride is incorporated into the developing tooth as a fluorapatite, which replaces the hydroxylapatite normally found in tooth structure. This can be written chemically as $Ca_{1}0\,(PO_4)_6\,(OH)_2 + 2F - > Ca_{1}0\,(PO_4)F_2 + 20H$.[1] The fluorapatite is less easily dissolved by mouth acids, and is therefore, more resistant to decay.

Various aspects of fluoridation have been studied, including factors such as cost-effectiveness; patient compliance; and caries reduction; and some of the methods of fluoride delivery such as water fluoridation; professionally applied fluoride; dietary supplements; and self-applied fluoride. These methods showed an average caries reduction of 50-65 percent, 30-40 percent, 50-65 percent and 20-50 percent, respectively.[2] Patient compliance is a major factor in all of the methods tested, except water fluoridation; "water fluoridation, therefore, is the most efficient caries preventive method in developed countries with municipal water supplies, and because individual compliance is not a factor, it is probably the most effective as well."[2] Water fluoridation is also the least costly method of fluoridation. According to the National Institute of Dental Research (NIDR), water fluoridation cost only 20 to 50 cents per person per year. The other methods cost two to four times as much.

In communities that do not fluoridate water supplies, or homes that have fluoride removed from their water, fluoride supplementation may be necessary. In order to recommend fluoride supplements, it is necessary first to determine how much fluoride is being ingested. The actual amount would be difficult to ascertain, but it can be reasonably assumed that if the fluoride content is below 0.7 ppm, people in that particular area will be fluoride deficient. The EPA or the local water treatment plant should be contacted to see what level of fluoridation they maintain. Home water should then be tested to determine the fluoridation level in the home. Most of the companies that sell water purification systems will test drinking water. Also, local health departments and dental schools may test water samples.

The American Academy of Pediatric Dentistry (AAPD) recommends the following as a guide for fluoride supplementation.[3] (Table)

Vehicles for delivering fluoride supplements are: so-

In the United States, there is widespread agreement among professional associations that water-fluoridation is a safe and cost-effective way to reduce dental caries by 50-65 percent.

Table □ A guide for fluoride supplementation.

Age (Years)	<0.3 ppm	0.3-0.7 ppm	>0.7 ppm
0-2	0.25 mg F	0.00 mg F	0 mg F
2-3	0.50 mg F	0.25 mg F	0 mg F
3-16	1.00 mg F	0.50 mg F	0 mg F

lutions that dispense 0.125 mg F per drop for infants; and tablets containing 0.25 mg F or 0.5 mg F for older children. Full-strength tablets or fluoride rinse containing 1.0 mg F can be prescribed at appropriate ages. The AAPD also recommends prescribing no more than 120 mg F at any one time in order to prevent accidental overdose. Children with active caries should be considered for supplementation, even if they live in optimally fluoridated communities: usually a fluoride mouthrinse (0.5 NaF) may be supplied. Dentists can also apply 1.23 percent APF solutions in the office.

WHAT ABOUT OVER-FLUORIDATION?

If an individual is ingesting water containing maximum fluoridation and is prescribed a fluoride supplement, mild dental fluorosis may develop. Dental fluorosis, a form of enamel and dentin hypomineralization, is produced in permanent teeth by excessive fluoride intake during a child's first five years of life; it is one of the earliest, most sensitive indicators of fluoride toxicity, and is associated with intakes that are approximately two times those found in optimally fluoridated water.

To date there is no generally accepted evidence that anyone has been harmed by drinking artificially fluoridated water. Studies in Texas and Colorado (8 ppm and 2.4 ppm) revealed no harmful effects other than dental fluorosis.[2] In 1968, the Society of Toxicology stated that, "Under controlled conditions as recommended by qualified public health authorities, the Society of Toxicology finds water fluoridation to be a safe measure."[4] The possibility exists, however, that some people are more sensitive to fluoride than others. The effects on susceptible individuals and the level at which these effects begin to occur should be determined. Unless the patient presents with active caries, fluorides should not be prescribed by the dentist in areas where fluoride concentration is greater than 0.7 ppm or where the concentration is unknown.

The opinions and views of this article are those of the author, and do not reflect the official policy or position of the Department of the Army or the Department of Defense.

WATER PURIFICATION SYSTEMS

Water purification systems have been available for many years, and are becoming more popular as people become concerned with pollutants in public water supplies. These systems can be bought from private companies or from many department stores. Whether they are used at the tap or for whole house treatment, the terms *point-of-use* (POU) and *point-of-entry* (POE) refer to home water purification systems.

POU systems must meet regulations set forth by the EPA. The Water Quality Association (WQA) is a nonprofit, international trade association representing firms or individuals engaged in the sale or production of designs and services for providing quality water for specific uses. Membership is voluntary. The WQA's purpose is to promote the individual right to quality water, the dissemination of water quality information, and the growth of the water quality improvement industry. The WQA publishes the *Validated Water Treatment Equipment Directory*. Equipment which is validated receives the WQA's *Gold Seal*. Validation means that a manufacturer-selected representative sample of a production line water purification system was tested at the WQA laboratory and was found to have met the requirements for total dissolved solids (TDS) reduction, hydrostatic tests, cycle tests, and nontoxicity of components set out in Industry Standard 5-322-84.[5]

The National Sanitation Foundation (NSF) also tests POU systems. The NSF is also a nonprofit organization that provides voluntary product testing and certification. They provide registered *Marks* for products that met specifications of the NSF. Their publication *NSF Listings - Drinking Water Treatment Units*, lists systems that have been certified.[6] To receive these publications, write to the WQA or the NSF. Certification by either of the above mentioned organizations shows that the equipment performs at the levels it claims.

SYSTEMS THAT REMOVE FLUORIDE

There are eight basic types of POU systems available on the market today. They are:

- Absorption through activated carbon filters.
- Anion exchange.
- Cation exchange softening.
- Disinfection.
- Distillation.
- Filtration.
- Oxidation.
- Reverse osmosis (RO).

These systems can be used alone or in combination. They reduce or rid the water of inorganic and organic contaminates. They eliminate foul odor, taste, color, and cloudiness. They produce water that is crisp, clear, clean, and taste free.

The RO, distillation, and absorption systems will remove fluoride from water. Depending on the size and type of system, it will remove between 90-99 percent of the fluoride in the water.

RO systems work by using the water pressure in the home to force water through a semipermeable membrane. This membrane has micropores that permit the passage of water, but reject most things dissolved in it. The water moves across a gradient from an area of high concentration to an area of low concentration; hence the name *reverse osmosis.* To operate maximally the filter or screen must be kept clean, and manufacturer's guidelines should be followed. Also, the system may give different results from home to home, due to differing levels of pressure in the lines.[7]

The distillation system is very similar to nature's way of producing water. Water goes into a vaporizing chamber and is brought to a boil. The steam rises, leaving behind most of the contaminants and chemicals of the source water. These are then flushed to a drain. The steam moves into a condensing chamber, where it is cooled and condensed to become distilled water. Manufacturer's recommendations for proper use should be followed.[7]

Absorption through activated alumina is a special application of the process of absorption through activated carbon filters. Absorption through alumina is used in fluoride reduction. The process employs an absorbent material, usually solid, capable of adhering gases, liquids, and/or suspended matter on its surface and in its exposed pores.[7]

Of these three methods, the two most commonly employed are distillation and RO. The use of these systems and their growth can be demonstrated by taking a look at the market data published by the WQA. They estimate the total market for water purification equipment to be 30 billion dollars. The WQA estimates that over 175 million dollars was spent on residential drinking water systems in 1985. Reverse osmosis is one of the largest components of the U.S. market, with an estimated 50,000 units sold in 1985. Another large component of the U.S. market is distillation. Sales of distillation water products in the U.S. reached approximately ten million dollars in 1985. According to the WQA, distilled water products are becoming an increasingly accepted method for drinking water quality improvement.[8] Activated alumina is not a major contender in the market for home use.

With RO and distillation making up a major position of the water purification market and sales projections showing a growing trend in purification systems in general, it becomes evident that a rising number of people are drinking water that is below the optimal level recommended to prevent tooth decay.

Because of this, it is important for the dentist and other health care personnel to discern whether patients are receiving optimally fluoridated water. Since many people who buy purification systems either do not realize or do not care that they may be removing fluoride from their drinking water, it is the dentist's and/or hygienist's responsibility to ask patients about their home water supplies.

CONCLUSION

This article will familiarize the reader with purification systems that remove fluoride from water, and encourage them to ask patients about their drinking water. It should also give them several organizations that provide water testing for fluoride content. Once the dentist knows the fluoride level of his patient's drinking water, he can use the recommendations of the AAPD to determine whether fluoride supplementation is needed.

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Premature extractions of primary molars and the crown/root ratio of their permanent successors

Case reports

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Shmuel Einy, DMD
Edith Koyoumdjisky-Kaye, DMD

Premature extractions of primary teeth affect the future development of the permanent dentition. This problem was dealt with previously on the level of the individual tooth with reference to the timing of eruption of the successor; on the arch-level with reference to loss of space; and on the interarch level as a local etiologic factor of malocclusion.[1-9] Little attention has been paid, however, to the possible effect of premature extraction on future development of the individual successor. If the permanent tooth presents with a complete crown and a partially formed root at the time of extraction of the predecessor, its root development could still be possibly affected, thus modifying the crown/root ratio. An indication that this ratio may be affected under certain circumstances following premature extractions of their predecessors was suggested by Brin and Kaye.[10] The purpose of this study was to examine the possible effect of premature extraction on the ultimate root length and area (in cross-section) of the successor as compared with the intact antimeres, which served as individual controls.

Dr. Brin is a senior lecturer and Dr. Ben-Bassat is a lecturer in orthodontics; Dr. Koyoumdjisky-Kaye is professor in orthodontics at the Hebrew University Hadassah School of Dental Medicine, Jerusalem. Dr. Einy is a dentist in the Israel Defense Forces.

MATERIALS AND METHODS

Forty-six children (seventeen boys and twenty-nine girls) who underwent unilateral extraction of at least one of their primary mandibular molars, were examined clinically and radiographically after the permanent successors completed root development. The mean age at the time of extraction was seven years, while the mean age at the present examination was fifteen years, six months.

A total of fifty-three pairs of teeth were examined, thirty-four pairs of first premolars (primary molars extracted at the mean age of seven years, two months) and nineteen pairs of second premolars (primary molars extracted at the mean age of six years, ten months). Only teeth with completed roots at the time of the present examination were included in the study. All the teeth were divided into two subgroups: very early and early extractions. Thus, thirty teeth were successors to primary molars extracted before the age of eight years, and twenty-three were successors to teeth extracted after that age.

The long-cone technique was used for the radiographic examination of the succeeding premolars and their antimeres. Only lower premolars were included in the sample, because of the more favorable conditions for obtaining orthoradial radiographs in the mandibular region. The radiographic image was transferred to acetate paper with a 0.3 mm tracing pencil. In order to avoid the error arising from possible radiographic distortion, the measured absolute values of crown and root length were related to each other, using the method described by Jacobsen and Lind.[11] The crown/root (C/R) ratio was measured by the following modification (Figure): the mesial and distal cementoenamel junctions (X and X') were connected by a straight line; the tip of the buccal cusp (T) was connected to the apex (A) by a vertical line. Root length (R) represented the distance A-I (I = intersection of X-X' and A-T) and crown height (C) represented the distance T-I. These measurements were considered appropriate as no attrition of the crown tips or root resorption were noticed.

In addition, tracings of the projected radiographs (magnified x 4.5) were performed and digitized. From these the crown and root areas (in cross-section) were computed. Further, the length and area C/R ratios were calculated for the successors of the prematurely extracted molars (the experimental group) and their intact antimeres.

An attempt was made to record the dental maturational status of the premolars at the time of extraction of their predecessors. Unfortunately, only a few patients (n = 20) in this study had radiographs preceding extraction; the radiographs permitted assessment of the root development of the permanent bud. Thus, the chronological age and sex of the examined patients were considered in the statistical evaluation. Accordingly, the mean C/R ratios were subgrouped by sex, age at the time of extraction (younger or older than eight years) and type of tooth (first or second premolar). Comparisons with controls within these subgroups were performed, using the Wilcoxon matched-paired, rank-signed test.

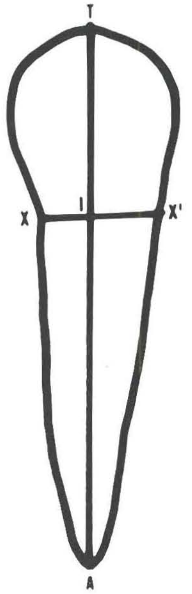

Figure. Tracing of lower premolar. X and X' = mesial and distal cementoenamel junctions; T = tip of the buccal cusp; A = apex; I = intersection of X-X' and A-T.

RESULTS

Length measurements

The mean crown and root length measurements are presented in Table 1. Note that the crown dimensions were found to be less variable than the roots, for both the first and second premolars, in most of the cases.

Length ratio

The mean C/R ratio for the total extraction group as well as among the boys and girls separately was almost equal to that of the control group (Table 2). A signifi-

Table 1 □ Mean ± SD of crown and root length (in mm).

	Extraction group		Control group	
	Boys	Girls	Boys	Girls
	n=11	n=23	n=11	n=23
1st premolar crown	7.77+0.70	7.65+0.87	7.66+0.68	7.09+1.65
1st premolar root	16.31+0.61	15.39+2.68	16.96+0.80	14.96+1.80
	n=9	n=10	n=9	n=10
2nd premolar crown	7.60+0.51	7.69+1.11	7.86+0.80	7.53+0.70
2nd premolar root	15.23+2.02	15.38+1.20	14.94+1.50	14.65+1.40

Table 2 □ C/R ratio by sex.

	C/R length ratio			C/R area ratio		
		Extraction	Control		Extraction	Control
	No	Mean + SD	Mean + SD	No	Mean + SD	Mean + SD
Totals	53	0.500+0.070	0.499+0.065	52	1.1470+0.407*	1.0815+0.347
Boys	20	0.492+0.062	0.490+0.076	20	1.1063+0.442*	0.9647+0.277
Girls	33	0.505+0.075	0.504+0.058	32	1.1720+0.388	1.1545+0.369

*p<0.05 for comparison with controls.

Table 3 □ C/R ratio by tooth and age at the time of extraction (in yrs).

		C/R length ratio			C/R area ratio		
			Extraction gr.	Control gr.		Extraction gr.	Control gr.
Tooth	Age	No	Mean + SD	Mean + SD	No	Mean + SD	Mean + SD
1st prem.	8−	18	0.4950+0.011	0.4952+0.047	19	1.1288+0.349	1.1128+0.363
1st prem.	8+	16	0.5018+0.048*	0.4738+0.072	14	1.0876+0.417	1.1256+0.415
2nd prem.	8−	12	0.4962+0.068	0.5018+0.043	12	1.1361+0.460	0.9743+0.169
2nd prem.	8+	7	0.5161+0.073*	0.5612+0.089	7	1.3340+0.473*	1.0920+0.406

*p<0.05 for comparison with controls.

cant difference ($p < 0.5$) between the extraction and control subgroups for the first and second premolars was found, however, when the extractions were performed after the age of eight years (Table 3).

Area ratio in cross-section

The mean C/R ratio for the total extraction group and the boys' subgroup was significantly larger ($p < 0.5$) than their respective controls (Table 1). Subdivision by both, the type of tooth and age at the time of extraction, indicated a significant difference for second premolars, successors to teeth lost after the age of eight years (Table 3).

DISCUSSION

Although the influence of premature extraction of primary molars has been tested in relation to the age of eruption of their permanent successors, its effect on root development has been given little attention.[1-10] Premature extractions may lead to acceleration or delay in the eruption time of the premolars. In case of acceleration, root length may not reach 2/3 or 3/4 of its ultimate length as expected in normal eruption.[12,13] The question of a possible effect of this occurrence on the mechanism of further root formation is addressed in this study.

The absolute length dimensions measured in the present study (Table 1) differed somewhat from those presented by Weeler, mainly for root length.[14] The high variability obtained could not be compared to data in the literature, because the standard deviations of the measurements do not appear in the dental anatomy texts. The differences may result, however, from the fact that our data were collected from radiographs, while the morphological studies used dried specimens. In order to overcome this problem, ratios were utilized in the present study. In addition, ethnic variability could account for some of the differences mentioned. For example, the root length of the first premolar as measured by us falls within the range of such measurements for a population of similar origin.[15]

The marginally larger dimensions found in boys (except for the second premolar in the extraction group) are congruent with the known sexual dimorphism.[16]

The dimensions of the second premolar that, in our study, did not conform to this rule, are acceptable, because this tooth is considered as variable in its development.

Judging from the higher area ratio and unchanged length ratio in the total extraction group (Table 2), it seems that the permanent root was more slender following the extraction of the predecessor. It is interesting to note, however, that only extractions performed at a later age affected root development (Table 3). No valid explanation for this phenomenon could be found in the literature. It could be conjectured, however, that the bone overlying the permanent bud at this later stage of its development was scarce or lacking, due to extensive caries lesions and periapical changes as their sequelae. Thus the relatively undeveloped bud was put under excessive environmental stress. The accelerated vertical movement of the bud might also have affected adversely the process of root formation. On the other hand, the deeper position of the developing bud in the younger age-group would account for more protective environment for its further, more normal development.

The contrasting results obtained for the first and second premolar length ratios (Table 3) are surprising. The difference in the mean time of extraction in these two subgroups may explain this phenomenon. The mean time of extraction of the first primary molar was by four months later than that of the second primary molar. This difference might have caused a more significant acceleration of the first premolar bud, which was also in a more advanced developmental stage. Thus, the first premolars developed a shorter root, when compared to their controls; whereas the second premolars presented with longer, but more slender roots (Table 3).

In view of the small number of teeth in the second premolar subgroup and the known variability encountered in the course of its development, the difference ($p < 0.5$) found for this tooth should be considered with caution. Also, it should be kept in mind that the statistically significant differences demonstrated in root lengths are of no clinical importance (mean difference of about 0.5 mm). Their existence, however, may suggest a possible trend in the continuing root-development process of the premolars affected by environmental factors, like premature extractions.

This study was supported in part by a grant from the joint Research Fund of the Hebrew University-Hadassah School of Dental Medicine founded by the Alpha Omega Fraternity and the Hadassah Medical Organization.
The authors wish to thank Mr. I. Einot for his help with the statistical workup.

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Single maxillary central incisors in the midline

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A congenitally missing maxillary central incisor is a phenomenon.[1,2] The appearance of a single tooth in place of two teeth can be related to fusion of two neighboring tooth buds or to agenesis of a tooth germ. Ellisdon and Marshall adopted the term "connation", suggested by Tomes in 1859, to describe the clinical appearance of a union of two teeth of the normal series, or union of a tooth of the normal series with a supernumerary tooth.[3] The term "double tooth" has also been used to describe a single tooth that was formed from an unseparated tooth mass.[1,4,5] Few descriptions of a single primary maxillary central incisor in the midline, followed by a single permanent central incisor can be found in the literature. One was related to "connation", another to "fusion across the midline", and one case was presented as a roentgeno-oddity without discussion.[3,6,7]

Case 1

An eight-year-old boy with a complaint of "unesthetic appearance of his teeth" was examined. His skeletal age was six years and his medical history was uneventful.

Clinical examination revealed a tooth with the morphology of a maxillary left central incisor. The tooth was slightly rotated mesiolabially, and its mesiodistal width was 9 mm. A simple, uncomplicated crown fracture involving enamel and dentine, was observed on the mesioincisal tip of the tooth (Figures 1,2). An occlusal radiograph revealed a single maxillary central incisor in the midline with a single root, a single pulp canal, and an open apex. The primary and permanent lateral incisors could be seen on both sides of the tooth (Figure 3). A photograph of the child at age three showed a single primary maxillary central incisor, not determinable as right or left (Figure 4).

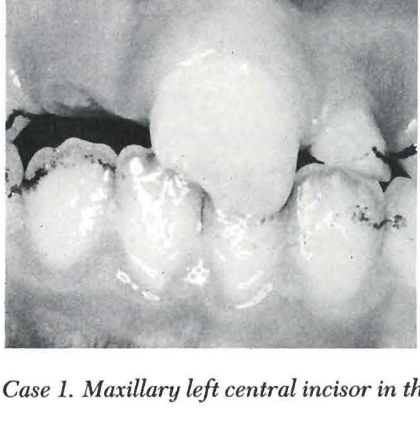

Figure 1. Case 1. Maxillary left central incisor in the midline.

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Case 2

A single permanent maxillary right central incisor located in the midline was observed in a healthy ten-year-old girl (Figures 5,6). She had two older brothers, one with a mild cleft lip. A history of trauma to the girl's teeth was denied by the mother. She stated that the child had only one primary maxillary central incisor. The mesiodistal width of the permanent incisor was 8.5 mm. The two lateral incisors were symmetrically positioned. A periapical radiograph showed a central maxillary incisor in the midline with a single root, a single pulp canal and a closed apex (Figure 7).

Case 3

A girl, eleven years, two months of age was examined. Her medical and dental history were noncontributory. Trauma to the anterior teeth was denied. Clinical examination revealed a tooth with the morphology of a maxillary left central incisor (Figure 8). The position of the tooth was in the midline, in front of the incisive papilla (Figure 9); the mesiodistal width was 8.5 mm. On the incisal edge of the right lateral incisor, a notch and a mild indentation running down the labial surface could be observed. A periapical radiograph revealed a central incisor in the midline with a single root, a single pulp canal, and a closed apex. Photograph of the child's primary dentition (Figure 10) showed a single primary maxillary central incisor, not related to either the right or left.

Panoramic radiographs of the three children matched their chronological ages and showed no morbidity. No changes in tooth number or morphology were found in other members of the immediate families including the sibling with the mild cleft.

DISCUSSION

The etiology of the condition is probably a congenitally missing bud of a central maxillary incisor. The primary tooth could have been a double tooth with one permanent successor. This concurs with the explanation suggested by Grahnen and Granath.[1] According to Osborn and Ten Cate the formation of one tooth instead of two could result from a disturbance in the mitotic potential of the incisor group.[8] The first bud of a group (incisor, canine, molar) lies in the center of the early group clone and the clone grows posteriorly. This theory is more suited to explaining missing lateral incisors or second premolars, rather than a central incisor.

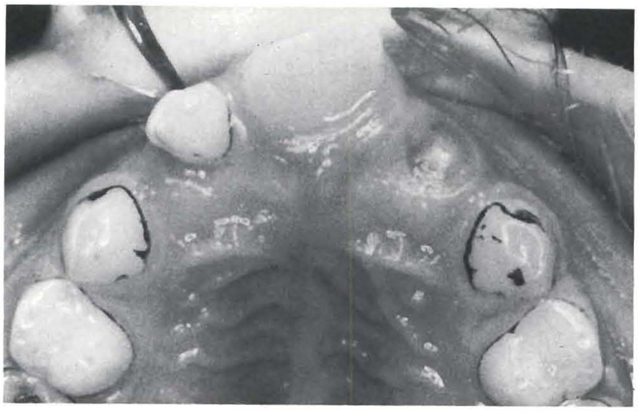

Figure 2. Same as Figure 1, palatal mirror view. The incisor is located exactly in front of the incisive papilla.

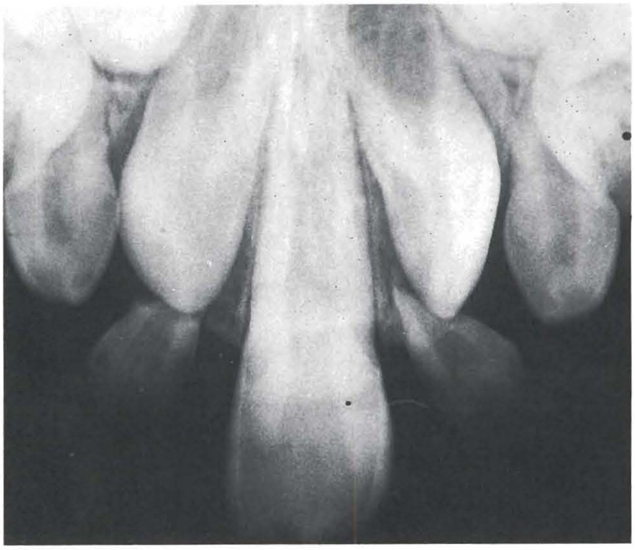

Figure 3. Case 1 periapical radiograph taken before shedding of tooth 52. Note normal shape of a left single incisor with an open apex in the midline.

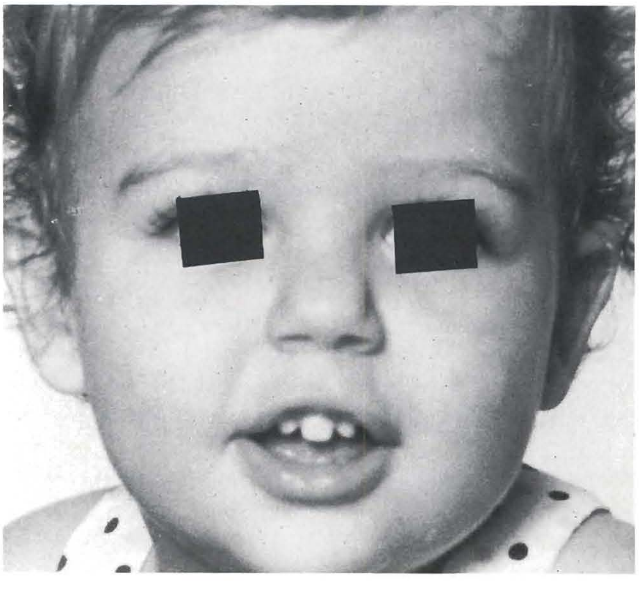

Figure 4. Photograph of the same child showing his primary teeth with a single central incisor.

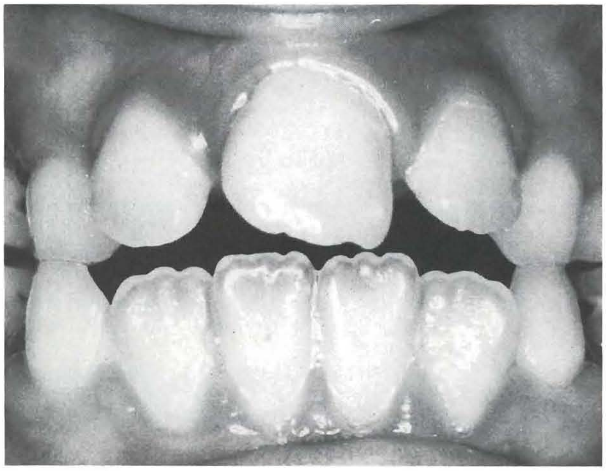

Figure 5. Case 2. Maxillary right central incisor in the midline.

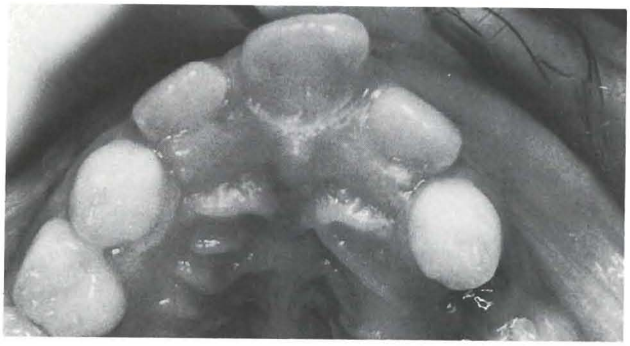

Figure 6. Same as Figure 5, palatal mirror view of the incisor located in front of the incisive papilla.

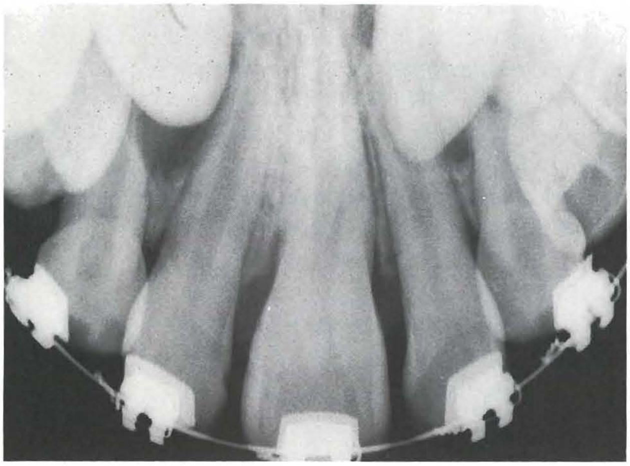

Figure 7. Case 2 periapical radiograph. Normal shape of a single-rooted right incisor.

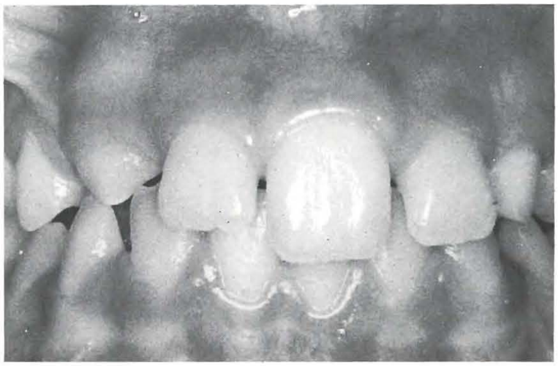

Figure 8. Case 3. Maxillary left central incisor in the midline.

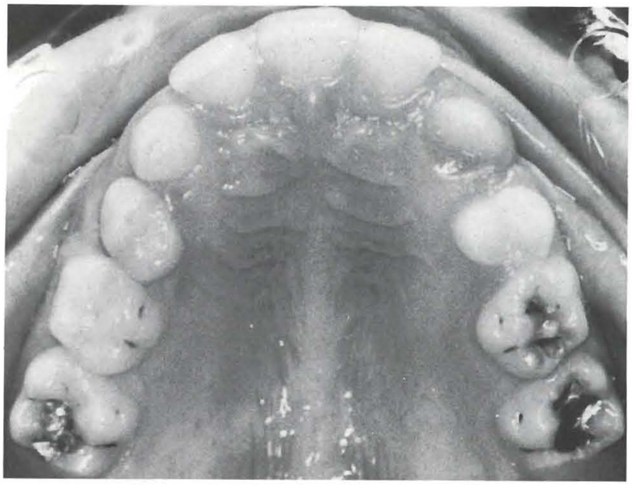

Figure 9. Same as Figure 8, palatal mirror view. The incisor is located in front of the incisive papilla.

Figure 10. Photograph of the same child showing her primary teeth with a single central incisor.

Table ☐ Mesiodistal crown width of maxillary permanent teeth in children with a single central incisor (in mm).

Teeth	Gender	Central incisor	Lateral incisor	First premolar	First molar
M-D width according	M	7.9–10.0	4.5–8.2	6.1–8.2	9.9–12.4
Moorrees[10]	F	7.1– 9.8	4.5–8.5	5.8–7.8	9.4–11.9
Case 1	M	9.5			10.0
Case 2	F	8.5	7.0	6.5	10.0
Case 3	F	9.0	6.5		11.0

Application of the unifying model suggested by Brook necessitated measuring the other teeth (Table 1).[9] The measurement of representative teeth from each group showed the teeth to be within the normal range suggested by Moorrees.[10] No indications of a more generalized disturbance in number or size were noted. There were no family findings of irregularities in size or shape in the three presented cases.

All permanent teeth were clinically and radiographically specific teeth, maxillary right or left central incisors, and all were positioned in the exact midline (Figures 2,6,9). The question of the primary teeth was more difficult since their shape did not allow finite diagnosis.

The evidence presented indicates that these are instances of agenesis of a central incisor, where the homologous tooth and its successor erupted in the midline.

The authors thank Dr. L. Belostoky and Dr. R. Shani for referral of patients and to Mrs. R. Lazar for her editorial assistance and preparation of the manuscript.

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FLUORIDE CONCENTRATION AND CARIES PROGRESSION

The increasing experimental evidence showing the ability of fluoride to arrest or even repair enamel lesions has been supported to date by relatively few clinical studies [Hollender and Koch, 1969; Hyde, 1973; Powell et al., 1981; Axelsson et al., 1987].

The results of this study indicate that fluoride levels in dentifrices have no effect on progression of radiographically detectable enamel lesions.

The occasionally extremely slow progression and the high numbers of apparently arrested approximal lesions as well as the trend towards lower progression rates in populations receiving fluoride supplements [Pitts, 1983; Gröndahl et al., 1984] imply that it should be possible to contain the caries development using a noninvasive method. However, while there are clinical observations which support the claim that small enamel lesions can regress [Backer Dirks, 1966; Axelsson and Lindhe, 1974; Pitts and Renson, 1987], the factors promoting the repair of more advanced caries on surfaces where constant plaque control is virtually impossible merit more attention. Thus, while fluoride has been singled out beyond doubt as a most important factor in the widely observed decline in caries prevalence, further longitudinal clinical studies are needed before specific fluoride agents and procedures can be recommended with qualified certainty as a measure to achieve the arrest or regression of advanced approximal caries.

Bjarnason, S. and Finnbogason, S.Y.: Effect of different fluoride levels in dentifrice on the development of approximal caries. Caries Res, 25:207–212, May-June 1991.

ABSTRACTS

Saxe, Michael D. and McCourt, James W.: Child abuse: A survey of ASDC members and a diagnostic-data assessment for dentists. J Dent child, 58:361-366, September-October 1991.

Survey results on the subject of child abuse from the American Society of Dentistry for Children membership are reported. The survey results reflected the need of a documentation protocol for the dental office. A protocol is proposed which reviews the need of thorough assessment of orofacial lesions before concluding that child abuse exists. A data- and diagnostic-assessment form has been made using guidelines of the American Dental Association and other related referenced articles. The diagnostic-assessment form is presented to guide the practitioner through the documentation process and to develop a step-by-step decision on the probability of child abuse in any individual case.

Child abuse; Pediatric dentistry; American Society of Dentistry for Children (ASDC); Documentation; Guidelines Role, dentists'; Indications, physical; Survey results

Waldman, H. Barry: Pediatric dentists need to know about the changing economics of health care. J Dent Child, 58:367-371, September-October, 1991.

A review is carried out of the general economics of dentistry and health-care services. Emphasis is placed on some developments and future prospects in the individual states, with consideration of the impact on pediatric dentistry.

Economics, health-care; Pediatric dentistry; Outpatient [and] free-standing facilities; Payment mechanisms; Health insurance; Income, dental practice

Kreulen, C.M.; van Amerongen, W.E.; Akerboom, H.B.M.; Borgmeijer, P.J.; van't Hof, M.A.: Evaluation of treatment times for class II composite resin restorations. J Dent Child, 58:372-377, September-October, 1991.

This article describes the results of an investigation into the treatment time required for the placement of Class II composite resin restorations. For purposes of this study, three dentists made 183 standard Class II composite resin restorations, both two-surface and three-surface, according to a detailed treatment protocol. Time registrations were carried out during the treatments, and each treatment was divided into five sections, each representing one stage in the restorative process. Of the various factors which may affect treatment time, the following were studied: the dentist; the type of tooth; the type of restoration; and the presence of an existing amalgam restoration in the tooth to be restored. Following ln transformation of the treatment times obtained, an analysis of variance was employed. In this study the average treatment time for Class II composite resin restoration was 41.8 minutes. The results indicated that the factors mentioned above significantly affect the treatment time. In addition, the treatment time of one of the dentists was influenced by a marked learning effect.

Restorations, composite resin, Class II; Treatment time; Labor; Fees; Protocol, treatment

Borgmeijer, P.J.; Kreulen, C.M.; van Amerongen, W.E.; Akerboom, H.B.M.; Gruythuysen, R.J.M.: The prevalence of postoperative sensitivity in teeth restored with class II composite resin restorations. J Dent Child, 58:378-383, September-October 1991.

Postoperative sensitivity is one of the problems a dentist can encounter after restoration of a tooth with composite resin. To reduce the possible causes of these complaints, the operative procedure has been adapted to some suggestions of other investigators. In this study the postoperative sensitivity was evaluated in a comparison between composite resin and amalgam: 244 (standard) class II restorations were made by three dentists in fifty-six patients. Each patient received one or two series of four experimental restorations: three of composite resin (Herculite XR, Clearfil Ray Posterior, Visiomolar) and one of amalgam (Tytin). All restorations were made according to a fixed protocol. The occurrence of postoperative sensitivity was recorded. Fifty-seven restorations showed a varying period of postoperative sensitivity; no case lasted longer than half a year. Molars have more postoperative sensitivity than premolars; the difference, however, is not significant ($P > 0.05$). The study shows further that there is no difference in postoperative sensitivity between restorations of composite resin and those of amalgam. The occurrence of postoperative sensitivity does not seem to be influenced by the choice of treatment procedure, type of tooth (premolar/molar), or by the patient or the dentist.

Sensitivity, postoperative; Restoration, composite resin, class II; Pain; Molars [and] premolars; Amalgam vs composite resin

Simon, Anne R. and Roberts, Michael W.: Management of oral complications associated with cancer therapy in pediatric patients. J Dent Child, 58:384-389, September-October 1991.

Treatment for childhood malignancies has improved markedly in recent years. However, radiation and chemotherapy

are often associated with morbidity involving oral tissues. Complications include mucositis, infection, xerostomia, and gingival bleeding. Children often acquire additional long-term sequelae due to potential impairment of growth and development. Management of oral complications has routinely consisted of multi-agent topical mouthrinses; oral and intravenous agents are also prescribed depending on the nature and severity of complications. The efficacy of multi-agent topical regimens is difficult to assess and long-term studies supporting such treatment are not available. Consequently, use of multi-agent mouthrinses is declining in favor of 0.12 percent chlorhexidine rinse. In children, appropriate management and prevention of oral complications are essential and require that dental care be integrated with cancer treatment.
Cancer; Oral tissues; Mouthwash; Chlorhexidine; Mucositis [and] ulceration; Pain; Development, dental

Watanabe, Keiko: Generalized juvenile periodontitis in a thirteen-year-old boy. J Dent Child, 58:390-395, September-October 1991.
There have been two previous cases reported in which children with a possible history of Prepubertal Periodontitis (PP) developed Generalized Juvenile Periodontitis (GJP) in their permanent dentitions at circumpubescent ages. This paper reports a case in which an apparently healthy 13-year-old girl, whose radiographs at 6 1/2 years of age showed horizontal bone loss around the primary molars, developed GJP. Blood tests (CBC, WBC differential, fasting glucose level, serum alkaline phosphatase) and a gingival biopsy were performed to exclude possible systemic diseases that might have been associated with alveolar bone resorption. Neutrophil (PMN) chemotaxis (CX) and adhesion molecule CD11b levels were also examined. The results of these tests were all within the normal range. This case report illustrates that an apparently healthy patient with PP may develop advanced periodontitis at a circumpubescent age.
Periodontitis, prepubertal [or] localized juvenile [or] generalized juvenile [and/or] rapidly progressive; Dentition, primary [and] permanent; Disease, systemic; Gingiva; Bone loss; Attachment; Puberty

So, Lisa L.Y. and King, Nigel M.: Radiographic features of the bones of the hand and wrist in achondroplasia: report of case. J Dent Child, 58:396-399, September-October 1991.
Achondroplasia is a well-established and documented medical condition. Most of the diagnostic features are discernible from a clinical examination, which are complemented by radiographic findings of the bones of the skeleton. The findings from the hand-and-wrist radiograph of an affected Southern Chinese male infant, age thirty-nine-months, are reported. The findings from this case indicated an infrequently reported abnormality in the development of the hand in achondroplasia.
Achondroplasia; Development, skeletal; Anomalies, dental; Pediatric dentistry; Radiography

Waldman, H. Barry and Waldman, Michael E.: Increasing numbers of AIDS patients. J Dent Child, 58:400-404, September-October 1991.
A description is provided of the distribution and the source of the AIDS infection in children and adolescents in the United States.
AIDS; Pediatric dentistry

Glass, Robert G.: Water purification systems and recommendations for fluoride supplementation. J Dent Child, 58:405-408, September-October 1991.
In communities that do not fluoridate water supplies, or homes that have fluoride removed from their water, fluoride supplementation may be necessary. The AAPD guide is reviewed, with dosage recommendations provided. Water purifications are de-

Continued on page 417

ABSTRACTS *Continued from page 359*

scribed and reviewed. Dentists should ask their patients about their drinking water.

Fluoride; Water, fluoridated; Supplementation; Dentistry; Water purification systems

Brin, Ilana; Ben-Bassat, Yocheved; Einy, Shmuel; Koyoumdjisky- Kaye, Edith: Premature extractions of primary molars and the crown/root ratio of their permanent successors. J Dent Child, 58:409-412, September-October 1991.

The effect of premature extractions of primary molars on further development of their successors was studied. A group of forty-six children with fifty-three unilaterally extracted mandibular primary molars was examined when the successors completed their root development. Radiographs of the successors to the extracted molars and their antimeres, which served as internal controls, were taken using the long-cone technique. Crown and root length and area in cross-section, were measured from tracings of the studied teeth and crown/root ratios were calculated. Significantly ($p < 0.05$) larger area ratio in the extraction group was found suggesting a smaller root area. In addition, root foreshortening for the first premolars and diminished root area for the second premolars, both successors to teeth extracted after the age of eight years, were found. Although statistically significant, all of the differences were of no clinical importance.

Primary molars, premature extraction; Succedaneous teeth, crown/root ratios

Mass, Eliyahu and Sarnat, Haim: Single maxillary central incisors in the midline. J Dent Child, 58:413-416, September-October 1991.

A single maxillary central incisor in both the primary and the permanent dentition is a rare phenomenon. Attempts to explain the etiology of this condition included "fusion across the midline", "conation" and "double tooth". Three cases are presented with morphologically normal single incisors in the permanent dentition, and evidence of single central incisors in the primary dentition. Agenesis seems to be the preferred etiologic explanation.

Agenesis; Incisor, central; Midline; tooth, single

Kreulen, C.M.; van Amerongen, W.E.; Akerboom, H.B.M.; Borgmeijer, P.J.; Kemp-Scholte, Ch.M.: A clinical study on direct and indirect class II posterior composite resin restorations. J Dent Child, 58:281-288, July-August, 1991.

Although posterior composite resin restorations are frequently used in dental practice, the clinical durability of these restorations is questionable when compared with amalgam restorations. The design of a clinical trial into the behavior of Class II posterior composite resin restorations is described here. In this study standard Class II restorations made of three posterior composite resins are tested and compared with amalgam restorations. The study is divided in two parts: in the first part (study one) all composite resin restorations were applied in a direct way; whereas in study two, the indirect inlay technique was used. An alternative box-cavity design has also been incorporated into these studies. Evaluation of the behavior of the restorations is divided in a clinical (modified Ryge criteria) and a nonclinical assessment. The latter consists of, among others, indirect examination of the marginal adaptation by means of impressions and evaluation of bitewing radiographs. To assess the influences of patient factors, every patient in study one or two received each restorative material. Moreover, the restorations were made by three dentists, to explore any possible influence of the operator.

Restorations, composite resin, posterior, class II; Study, longitudinal; Evaluation, clinical; Materials, restorative

Reid, James S.; Simpson, Matthew S.; Taylor, Geoffrey, S.: The treatment of erosion using porcelain veneers. J Dent Child, 58:289-292, July-August 1991.

The treatment of lost tooth tissue, due to erosion, in young patients is demonstrated. Lingual porcelain veneers using a nonetching, dentine bonding system are discussed. They are presented as a nondestructive, reversible process used to alleviate the patients' pain and restore appearance. Two cases are described demonstrating a combined approach between orthodontics and conservation. The technique is simple and straightforward, and offers advantages over existing techniques.

Enamel, tooth; Erosion; Dissolution; Incisors, maxillary; Lingual surfaces; Veneers, porcelain; Pain; Esthetics

Barak, Shlomo and Kaplan, Isaac: The CO_2 laser in surgery of vascular tumors of the oral cavity in children. J Dent Child, 58:293-296, July-August 1991.

Three cases of surgery performed with the CO_2 laser are presented to demonstrate its value in the excision of vascular lesions of the oral cavity in infants and children. The advantages of this type of surgery are: hemostasis; rapid healing; and lack of postoperative pain, discomfort and swelling. In the cases presented here, blood transfusions were not necessary.

CO_2 laser; Lesions, vascular; Oral cavity

Spear, Carol Sherrill and Savisky, Laura Anne: A study of children's taste and visual preferences in dentifrices. J Dent Child, 58:300-302, July-August 1991.

A study of dentifrice choices was conducted with four hundred thirty-five (435) fourth-grade children in Monongalia and Preston Counties, in West Virginia. The objective of this study was to determine whether children actually prefer those dentifrices that are de-

signed and flavored specifically for them. There were no statistically significant differences in taste preference between the regular and children's-flavored dentifrice of the same brand. Statistically significant differences existed, however, in visual preference, in which the child-oriented dentifrices and containers were preferred. Chi-square analysis indicated no significant differences in responses of children attending schools in rural and urban areas. It can be concluded that children do not prefer the taste of a dentifrice flavored specially for them over that of a regular-flavored dentifrice of the same brand. Children do visually prefer the dentifrices and dentifrice containers that are designed specifically for them.
Dentifrices; Toothpaste, children's; Flavor; Preferences

Nainar, S.M. Hashim and Crall, James J.: Child patient behavior: a new perspective. J Dent Child, 58:303-305, July-August 1991.
Recent observations regarding persistent physical changes within neuronal systems following series of temporally associated stimuli are reviewed in the context of behavioral conditioning. Repeated temporally-linked stimuli are thought to produce alternate pathways, which facilitate the storage and recall of learned associations. Empirical observations regarding child behavior in dental situations are examined on the basis of these new findings.
Behavior; Neurobiology; Conditioning; Pediatric dentistry

Waldman, H. Barry: Pediatric dentistry demographics: more than just numbers of children. J Dent Child, 58:306-309, July-August 1991.
A review is provided of the increasing numbers of minority group children and the relative stabilization in the numbers of non-minority children. The future of pediatric dentistry could depend on the ability to develop methods to increase services to minority group children.
Pediatric dentistry; Children, minority [and] non-minority; Demography

Waldman, H. Barry: There is no such thing as a typical family. J Dent Child, 58:310-313, July-August 1991.
A review of the changing family structure in which children are being raised is provided. Pediatric dentists need to recognize these developments, if they are to provide effective services to their patient populations.
Demography; Pediatric dentistry; Families, traditional vs. modern; Economics; Labor force, trends in

Rapp, Robert and Galletta, Dennis: Management of a departmental budget using an electronic spreadsheet. J Dent Child, 58:314-319, July-August 1991.
The phenomenon of a changing pattern of dental disease, accompanied by a reduction of the enrollment of students in dental schools, has created a declining economic basis on which dental schools must operate. Subsequently, the management of departmental budgets, in times of declining financial resources, is demanding more sophisticated approaches. Use of a computer-operated electronic spreadsheet can provide the departmental manager with a budget-management tool designed to achieve enhanced accuracy and efficiency. This article describes how an electronic spreadsheet program can be introduced into departmental budget management to achieve improved financial regulation.
Budget administration; Software, computer; Electronic spreadsheets

Harn, Stanton D. and Kuster, Curtis G.: A model for a children's dental health carnival. J Dent Child, 58:320-327, July-August 1991.
National Children's Dental Health Month has been called the dental profession's "most widely supported and publicly recognized event." Sponsoring and hosting a children's dental health carnival is an effective means for promoting the dental health of children. Such a carnival has been conducted successfully at the University of Nebraska Medical Center College of Dentistry for the past three years. Provided here is an outline and guide to the organization and logistics of a children's carnival. Descriptions of dental screenings, educational booths, educational games, dentally-related fun, games, and entertainment are discussed.
Dental health month; Carnival, dental health; Health promotion [and] education; Community activity

Larmas, Markku; Hietala, Eeva-Liisa; Similä, Seppo; Pajari, Ulla: Oral manifestations of familial hypophosphatemic rickets after phosphate supplement therapy: a review of the literature and report of case. J Dent Child, 58:328-334, July-August 1991.
Knowledge of inherited rickets also known as X-linked familial hypophosphatemic (XLH) or vitamin D-resistant rickets (HVDRR) with typical skeletal and oral symptoms, is updated. Histology and electron-probe microanalyzer showed globular dentin with pulpal horns extending to the dentinoenamel junction. A 160-μm-wide band of normally arranged dentin was found under the enamel and root cement. More sodium and a higher Ca/P ratio were observed in the affected teeth compared with the controls. Oral supplementation with phosphate solution cures the bone structure. Minor alterations, probably determined genetically, remained in the dentin.
Rickets; Pulpal horns; Dental manifestations; Heredity; Supplementation, phosphate

Boon, Lian Chin and Esa, Rashidah: Impeded eruption of a permanent maxillary incisor by a denticle and a cyst. J Dent Child, 58:335-336, July-August 1991.
A case of delayed eruption of the left

maxillary central incisor is presented. Combined surgical and orthodontic treatment were advocated as the preferred treatment in this case. The possible pathogenesis is discussed.
Eruption; Cysts; Tumors; Odontomas; Denticles

Goho, Curt: Ingestion of dental mirror fragments: report of case. J Dent Child, 58:337-339, July-August 1991.
Ingestion or aspiration of foreign objects is a potential complication of pediatric dentistry. Diagnosis and treatment are difficult, if the object is of low radiopacity, as routine radiographs may not discern the object. This case reports the possible ingestion of glass fragments from a disposable dental mirror and the resulting diagnostic difficulties. Procedures to be followed in such an event are discussed. Prevention is emphasized.
Pediatric dentistry; Ingestion; Dental mirror; X-rays; Radiopacity; Glass fragments; Liability

News

DENTISTS' CPR TRAINING SAVES LIVES

Knowing how to administer CPR has made heroes out of two ADA member dentists.

Dr. Stephen A. Brooks of Chattanooga, TN, and Dr. Tom Gallivan of Rutland, VT, are credited with saving the lives of two strangers to whom they administered CPR.

Dr. Brooks and his wife had gone to a local restaurant for dinner. As they were finishing the main course, they saw a man being helped to the floor. Brooks immediately went over to help.

"I was scared to death," he said, "especially when I realized he was not breathing and his heart wasn't pumping and there was nothing caught in his throat."

Brooks said he had never performed CPR on a "real" person before. Paramedics arrived after about a half hour and relieved Brooks and a nurse, also in the restaurant, who helped. Brooks said the man has fully recovered with no ill effects.

Brooks is in private practice in Chattanooga and is editor of the *Journal of the Tennessee Dental Association*.

Dr. Gallivan, a general practitioner in Pittsford, VT, was shopping in nearby Rutland when he saw a man collapsed in the parking lot. The man had stopped breathing and was turning blue.

"So I did chest compressions and breathed for him for maybe a minute," says Dr. Gallivan, "and then I heard the sirens."

An Advanced Life Support team from the fire department was on the scene and took over care of the man who had suffered from a rare condition called septal hypertrophy which impairs the ability of the heart to pump and causes problems with the heart's electrical pathways.

The physician who later treated the man said if the patient had not had immediate CPR, he would probably be dead. The man has fully recovered.

Both Brooks and Gallivan practice dentistry in states that require CPR training and certification for licensure renewal.

HIV INFECTION AND AIDS IN CHILDREN INCREASE

The problem of HIV infection and AIDS in children is increasing as more children become infected each year, according to an item in the July-August *Journal of Dentistry for Children.*

With an estimated 1,500 to 2,000 HIV-infected children born in 1989, the impact of HIV on mortality in children will become more severe, reports S.Y. Chu in "The Impact of the Human Immunodeficiency Virus Epidemic on Mortality in Children."

While certain therapies under evaluation may slow the course of HIV disease, HIV infection is likely to remain a highly fatal condition. Prevention of most AIDS cases in children requires prevention of infection in women.

The report notes that counseling and testing programs for women should be offered, and readily available, in health facilities that serve reproductive-age women at high risk of HIV infection.

Additionally, the report says that women who are already infected should be advised to consider the risk of perinatal HIV transmission in making reproductive choices.

NEW TAX CREDIT AVAILABLE TO DENTISTS

Dentists are eligible for a new tax credit, even in the current year, if they renovate their offices to increase access for disabled people, writes Phillip Bonner, DDS, in the August *Dentistry Today.*

The "disabled access credit" allows a credit for half of the costs of eligible renovations exceeding $250, but not more than $10,250 during any tax year.

The law defines eligible expenditures to include amounts paid or incurred for architectural modifications and materials or equipment used to remove barriers preventing a business from being accessible to or usable by people with disabilities. The law also requires the Internal Revenue Service to issue regulations.

The law applies to costs of providing access to facilities and services for people with physical or mental disabilities covered by the recently enacted Americans for Disabilities Act. New construction is not eligible.