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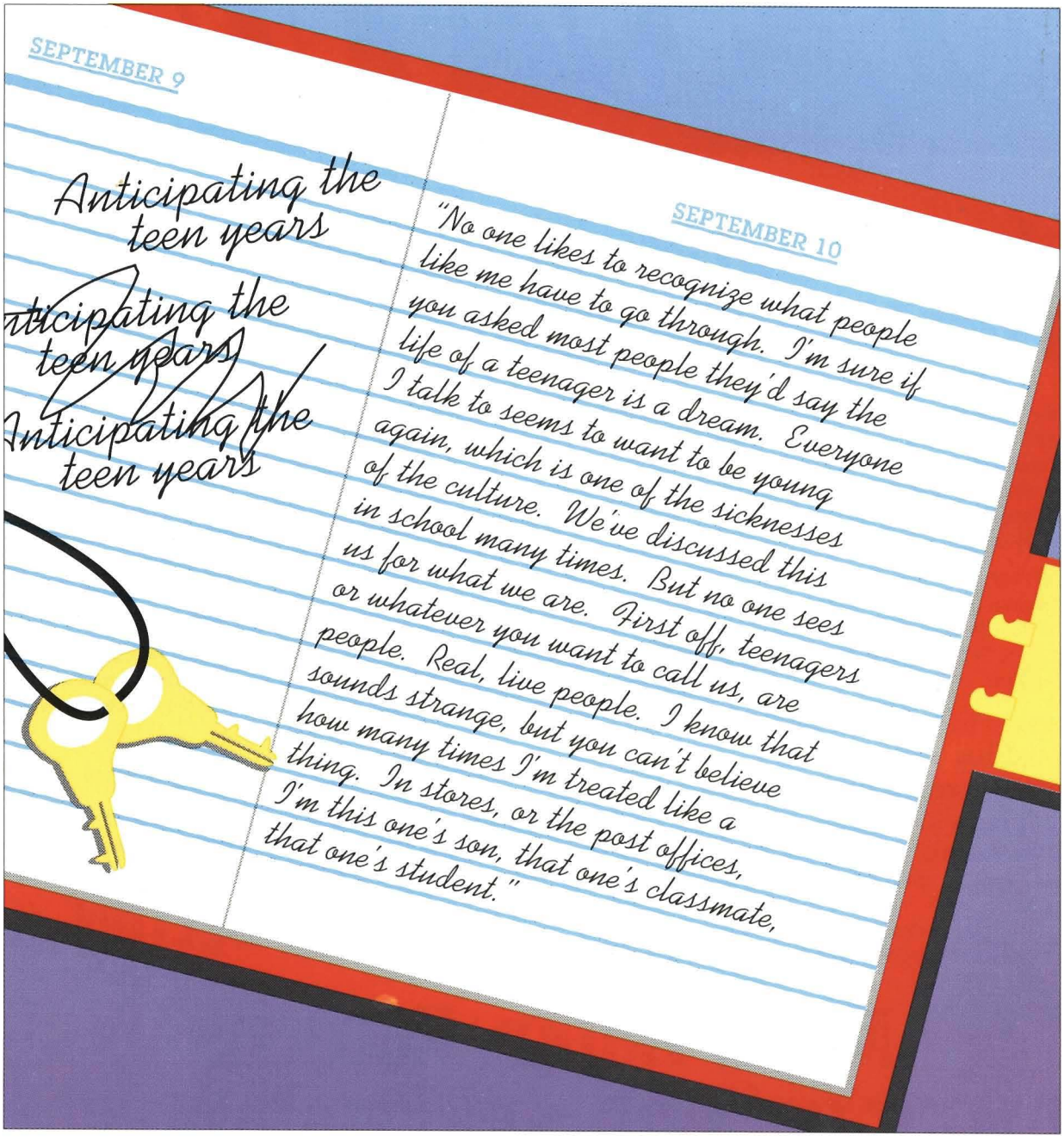
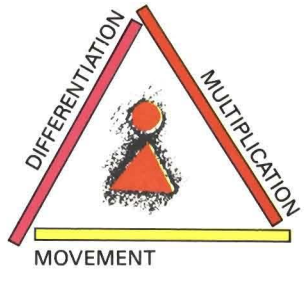


NO MATTER WHAT PHILOSOPHY OF LIFE WE ESPOUSE, IT IS IMPORTANT TO SEE CHILDHOOD AS A STAGE OF LIFE, NOT JUST AS THE ANTEROOM TO LIFE.

—David Elkind



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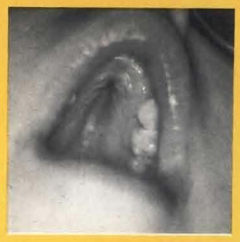

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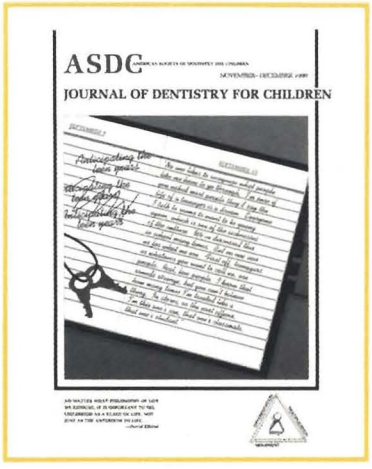
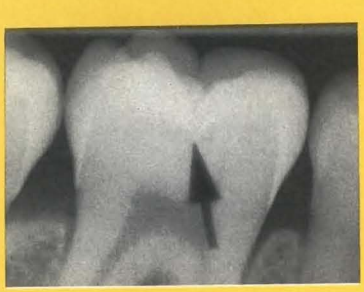


The adolescent years can be emotionally disturbing for the adolescent, parents, and others who must work with him. Deliberate preparations in the preadolescent years is very important.

DEMOGRAPHY

EPIDEMIOLOGY

CLINIC



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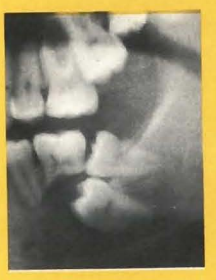

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CASE REPORTS

For the busy reader

Reaching more children with needed dental services—page 417

If we are to reach more children with needed dental services, needed financial support is essential, as well as an appreciation of the attitudes and motivation of children and their parents and guardians regarding their dental health and needed services. As children's perceptions can be modified, efforts to provide these services can be enhanced.

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.

Confirming the continuing potential for pediatric dental services in nonurban areas—page 421

Results from the 1986-87 NIH study of the oral health of U.S. schoolchildren indicate continued widespread improvement in dental decay rates. Higher rates of dental decay continue in nonurban area children. New data confirm the willingness of nonurban families to seek needed dental services for their children.

Request 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.

Occurrence of primary incisor traumatism in Brazilian children: a house-by-house survey—page 424

Subluxation was the most frequent primary tooth injury in this study, with 121 cases (38 percent) reporting this condition. Subluxation with enamel crown fracture (21 percent); crown fracture (18 percent); pulpal hyperemia (7.55 percent); root fracture (6 percent); intrusion (4 percent); and complete displacement (1.5 percent); were the other kinds of dental trauma.

Requests for reprints should be directed to Dr. Maria F.T.B. Bijella, Department Odontopediatria e Orthodontia, Faculdade de Odontologia de Bauru - USP, Caixa Postal, 73, 17001 - Bauru-SP, Brasil.

Occlusal hidden caries: a bacteriological profile—page 428

The bitewing radiographs of cases of hidden caries show a distinct radiolucency in the dentine beneath the occlusal surface; clinically, however, the tooth looks sound. The attempt of this study was to obtain insight into the bacteriological profile of hidden caries lesions. They contained mutans streptococci and lactobacilli combined with soft and light-colored dentine.

Requests for reprints should be directed to Dr. Willem E. van Amerongen, Department of Pediatric Dentistry, Academic Centre for Dentistry Amsterdam, Louwesweg 1, 1066 Amsterdam, The Netherlands.

In vitro assessment of the microleakage around preventive resin (laminate) restorations—page 433

When composite resin polymerizes, it contracts and this results in gaps at the tooth-restoration interface. These gaps allow the ingress of acids, enzymes, ions, bacteria and their products, a phenomenon known as microleakage, which may predispose a tooth to discoloration, recurrent decay and pulpal inflammation. In the first part of the study, the effect of etching and washing times on leakage was investigated. The second part examined the effect of the omission of bonding agent on leakage.

Requests for reprints should be directed to Dr. W.P. Saunders, Department of Conservative Dentistry, Glasgow Dental Hospital and School, 378 Sauchiehall Street, Glasgow G2 3JZ Scotland.

Regulation therapy by Castillo-Morales in children with Down syndrome: primary and secondary orofacial pathology—page 437

Given here is a synopsis of the most important primary and secondary orofacial disorders in children with Down syndrome. Included in the primary findings are lingual diastasis and "stair palate". Summed up are those findings that are primarily related to malfunction and can be influenced by Orofacial Regulation Therapy.

Requests for reprints should be directed to Dr. G.J. Limbrock, Kinderzentrum, Heiglhofstr. 63, D-8000 Munich 70, Germany.

Orofacial regulation therapy in children with Down syndrome, using the methods and appliances of Castillo-Morales—page 442

The results of a six-year study, using neuromuscular stimulation of the hypotonic orofacial area, are encouraging. Nearly two thirds of seventy-four children with Down syndrome, ranging in age from six weeks to eight years old, were successfully treated. Their symptoms were significantly reduced to allow improvement in mastication, deglutition, speech and facial expression.

Requests for reprints should be directed to Dr. G.J. Limbrock, Kinderzentrum, Heiglhofstr. 63, D-8000 Munich 70, Germany.

Drooling, chewing and swallowing dysfunctions in CP-children: treatment according to Castillo-Morales—page 445

Drooling in patients with cerebral palsy occurs frequently and is not due to hyperproduction of saliva, but rather to a dilatory and disordered pattern of swallowing. These children also suffer from eating, drinking and vocal disorders. The therapeutic concept of Orofacial Regulation Therapy includes functional diagnostics of oral sensorimotor dysfunctions; a special manual to encourage and facilitate a myofunctional program; and removable activating palatal plates and other orthodontic appliances.

Requests for reprints should be directed to Dr. G.J. Limbrock, Kinderzentrum, Heiglhofstr. 63, D-8000 Munich 70, Germany.

Permanent molar impactions and an odontogenic keratocyst: report of case—page 452

Requests for reprints should be directed to Dr. Russ L. Kendell, Department of Dental Therapy, Curtin University of Technology, Kent Street, Bentley 6102, Western Australia.

Management of cervical root perforation: report of a case—page 454

Requests for reprints should be directed to Dr. K-J Nordenvall, Department of Pedodontics, Folktandvarden, Fjarilen, Turingegatan 26, 151 34 Sodertalje, Sweden.

Regional odontodysplasia: report of case—page 459

Requests for reprints should be directed to Dr. W. O'Neill, Department of Pediatric Dentistry, UMKC School of Dentistry, 650 E. 25th Street, Kansas City, MO 64108.

Scleroderma in pediatric patients—page 462

Requests for reprints should be directed to Dr. Ruth A. Anderson, Department of Pediatric Dentistry, College of Dentistry, Howard University, 400 W Street, N.W., Washington, D.C. 20059.

Dentistry for the at-risk patient—page 466

Requests for reprints should be directed to Dr. Burton L. Nussbaum, 15 Forge Ln. Cherry Hill, NJ 08002.

Reaching more children with needed dental services

Demography

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

The improving environment for the practice of pediatric dentistry is the product of a wide-ranging series of interrelated developments in population demographics, increasing levels of education for the general population, improvements in third-party insurance programs, moderation in the production of health personnel and a host of other diverse factors. These many elements have resulted in the increased availability of dental services for the increasing numbers of families that are demanding needed dental treatment for their children. That was the message in a series of earlier presentations in the *Journal of Dentistry for Children*.[1-5]

But as pediatric dentists (and the dental profession in general) extend services to ever greater segments of the population, a shrinking "hard core" of underserved children (and adults) remains. No health program can expect to reach every single individual in our diversified communities. But at a time of reported marked decreases in dental caries rates, the continued financial and professional success of pediatric dentistry may well depend upon the ability to reach many of these remaining underserved "hard core" groups.

No doubt the allocation of additional government resources would improve the potential for increased services for the poor. But even if additional monies were

Dr. Waldman is Professor and Chairman, Department of Dental Health, School of Dental Medicine, State University of New York at Stony Brook, Stony Brook, NY 11794-8715.

made available, other factors must be considered, in any effort to reach increased numbers of children with needed preventive and restorative services. Specifically, we need an understanding of the family's perceptions and attitudes regarding their youngster's general oral health and needed dental care. But equally as important, and often overlooked by the dental practitioner, we must have a greater appreciation of

- ☐ Children's awareness and interest in the health (and even esthetics) of their mouths.
- ☐ Those factors that will motivate children to develop a concern for and attention to their dental needs.

An example (and indeed, a very unfortunate example) of misguided interest and motivation for a particular form of dental esthetics recently drew the attention of one school official. In a New York City high school, the principal felt it was necessary to ban decorative "gold caps" for teeth because he believed they do not convey the serious image his students need, to be successful in the business world.

"Most of the gold teeth are simply for cosmetic reasons, students say. The caps, which slip over one's real teeth, range in cost from $40 to over $100. Especially popular right now are gold caps over the two front teeth, sometimes etched with initials or dice or another emblem. Boys wear them more often than girls."[6]

It seems that the gold teeth first appeared in the mouths of drug dealers or others looking for ways to show their newfound wealth. But now it has become a fad (or is it fashion accessory?). "I just like gold teeth," said a fourteen-year-old. (If only we could focus such an intense interest in dental care in a more positive direction.)

KNOWLEDGE

Most adults (including parents and guardians) are aware of the importance of dental care. By the mid-1980s, there were indications that the general public was getting the message on the value of health promotion and disease prevention. More than 95 percent of a national sample (in all age-cohorts over seventeen years of age) reported that it was important to see a dentist on a regular basis; 98 percent recognized the importance of brushing and flossing teeth; and 96 percent recognized the primary causes of loss of teeth in children.[7]

ATTITUDES AND MOTIVATION

An extensive series of research reports on children's dental-health attitudes and motivation exists in the dental public health and general psychology literature. The need is to incorporate this information into the efforts to expand pediatric dental services to the remaining underserved populations in our communities. For example:

- ☐ One of the more disturbing findings in some study reports is the "...relatively minor degree to which health was salient..." for children (between seven and eighteen years of age).[8]
- ☐ Because health problems seem relatively unlikely for children, attempts to change levels of perceived vulnerability should begin at earlier ages. High-risk groups often contain disproportionate numbers of youngsters who see themselves as relatively invulnerable to a range of health problems.[9] Thus, rather than waiting until children reach "a more appropriate age," increasingly, health education on smoking, drugs and AIDS is being introduced in primary school grades.
- ☐ An individual component of a child's "system" of health perceptions, expectations and vulnerability may be resistant to change. Thus it may be necessary to aim educational efforts at the child's total "system" of perceptions in order to change specific health-problem expectations.[9,10] In this sense, dental health-education programs should be part of a comprehensive health-education program.[10,11] And because younger children have difficulty with the concept of "health," it would seem that a disease-oriented, health-education program would be more effective.[9]
- ☐ Efforts to modify beliefs and attitudes may not be enough to ensure the desired results. Because of a weak correlation between perceived vulnerability to illness and dental health behaviors, specific short- and long-term reward mechanisms may be needed.[12,13]
- ☐ There are differences in how innercity and noninnercity children rate important dental conditions in a series of "mouth appearance pictures." Younger, inner-city children indicate a preference for a healthy mouth (fewer cavities). Their noninner-city counterparts preferred more attractive teeth (straighter teeth). As the children age, however, the likelihood of inner-city children showing a preference for a healthier mouth decreases.[14]

INNER-CITY CHILDREN: AN EXAMPLE OF A POPULATION THAT USES FEWER DENTAL SERVICES

In 1986, there were more than forty-four million children between age two and seventeen years of age living

Funding alone will not ensure expansion of dental treatment.

in metropolitan statistical areas (MSA).† Almost sixteen million of these children lived in central city areas (Table 1). In each age-cohort, a smaller percent of central city children than noncentral city children had a dental visit in the past year (Table 2). Similarly, central city children had fewer dental visits per child than children in noncentral city areas (Table 3). The dental-visit pattern of central city children approximated the visit-pattern of children in nonmetropolitan areas (Tables 2 and 3).

Unfortunately, national surveys on decayed-missing-filled (DMF) experience and other aspects of oral health do not report data by central city and noncentral city subregions within the MSA.[15] General comparative patterns of dental disease and treatment for inner- and non-inner-city metropolitan children, however, can be developed by reviewing other available demographic data. Clark *et al*‡ and Kaufman *et al* extended the frequently reported findings of the relationships of dental needs and services with socioeconomic status (SES) by substituting the varying federal-government income-eligibility-guidelines for subsidized school lunch programs for the usual elements of poverty.[17,18] Based on subsidized lunch-program-eligibility, the authors reported that children in lower SES groups had higher caries prevalence, more untreated lesions, fewer caries-free mouths and less use of sealants. The next step was to couple these findings with general national data, which indicate that there are greater numbers of individuals below the poverty level living in inner-city areas of MSA (as compared to non-inner-city areas).[19] Thus, the reality is that despite general declines in caries rates, many inner-city children continue to be in particular need of dental services.[16]

IN GENERAL

Yes, additional government financial support for dental programs would increase the possibility of dental services for those in need of care. But the reality is that the funding alone may not ensure the expansion of dental treatment to the underserved population of children, if we are not able to modify the attitudes and perceptions of children and adults. In addition, if the health attitudes and motivational research provide any direction for health practitioners, it is that the expansion of pediatric dental services for inner-city children may well depend upon the ability to:

- ☐ Stress the need for care at earlier ages, while emphasizing the consequences of dental disease.

Table 1 ☐ Number of children in millions by place of residence: 1986.[16]

Age	Metropolitan statistical area			Nonmetropolitan statistical area
	Total	Central	Noncentral	
2-4 yrs.	8.3	3.2	5.1	2.5
5-11 yrs.	17.9	6.8	11.1	5.6
12-17 yrs.	16.1	5.8	10.3	5.3
Total	44.3	15.8	26.5	13.4

Note: The data presented in these tables appeared earlier in a more extended review, in *Journal of Dentistry for Children*, of the difference in the use of dental services by metropolitan and nonmetropolitan children.[3]

Table 2 ☐ Percent of children with a dental visit in the past year by place of residence: 1986.[16]

Age	Metropolitan statistical area			Nonmetropolitan statistical area
	Total	Central	Noncentral	
2-4 yrs.	32.0%	29.1%	33.8%	29.1%
5-11 yrs.	72.3	67.5	75.1	65.7
12-17 yrs.	71.2	65.1	74.6	66.1

Table 3 ☐ Number of visits per child by place of residence: 1986.[16]

Age	Metropolitan statistical area			Nonmetropolitan statistical area
	Total	Central	Noncentral	
2-4 yrs.	0.7	0.7	0.7	0.8*
5-11 yrs.	2.0	1.5	2.3	2.0
12-17 yrs.	2.9	2.4	3.2	2.4

*Relative standard error greater than 30 percent

†Generally speaking, a metropolitan statistical area consists of a county or group of counties containing at least one city (or twin cities) having a population of 50,000 or more plus adjacent counties that are metropolitan in character and economically and socially integrated with the central city. Towns and cities, rather than counties, are the units used in defining metropolitan statistical areas in New England.[15]

‡The authors provide a detailed referenced review of the changing patterns of dental caries experience and periodontal disease in children as related to various demographic characteristics (including, socioeconomic status, race, geographic regions and size of communities) and the availability of fluoridation.

- ☐ Develop an awareness and concern for a "total health service package."
- ☐ Provide a series of rewards which will offer an immediate feedback mechanism.

In the past, pediatric dentists provided dental services using these very same approaches; but for middle- and upper-class patient populations.[20] If we are now to expand our services to an underserved "hard core" patient population, it may well be necessary to rethink many of the "tried-and-proven" approaches to reach many of these children.

But the attempts to reach these underserved children may be occurring at a most opportune period; a time when members of the government are becoming aware of the need for increased attention to the services for children.

"Mr. Rostenkowski (Chairman of the House Ways and Means Committee) said that, 'the sad story of the 1980s was that the old have gotten more while the young have gotten less.' "[21]

If we are to reach more children with needed dental services, the lessons to be learned are that needed financial support is essential, but so too is

- ☐ An appreciation of the attitudes and motivation of children (and their parents and guardians) regarding their dental health and needed services.
- ☐ What steps can be taken to modify children's perceptions in an effort to enhance efforts to provide needed dental services.

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Confirming the continuing potential for pediatric dental services in nonurban areas

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

The conclusion of earlier writings (summarizing available data on dental decay rates [through 1979-80], numbers of practitioners and dental-visit patterns [through 1986]) was that there was a favorable potential for pediatric dentists in nonurban practice.[1,2]

"Individual pediatric practices must evolve to reflect the changing patterns of dental disease and the competitive realities in today's (and tomorrow's) world. Similarly, pediatric practitioners may need to consider practice arrangements in all geographic areas that continue to demonstrate an increasing demand for services."[2]

Recently published national Decayed-Missing-Filled-Surfaces (and Teeth) (DMFS and DMFT) rate-data for 1986-87 provide information that confirms the continuing potential for pediatric dental services in nonurban areas.

EARLIER REPORTS

Earlier reports confirm:

- ☐ There are 13.4 million children between two and seventeen years of age living in nonstandard metropolitan areas (nonMSAs).†
- ☐ A smaller percentage of children living in nonMSAs than their metropolitan counterparts reported a visit to the dentist in the previous year. Between the 1960s–1970s and 1970s–1980s, however, the percent of nonMSA children reporting a dental visit in the past year, as compared to MSA children, increased at a faster rate.
- ☐ MSA and nonMSA children between five and eleven years of age reported the same number of dental visits per child. NonMSA children between twelve and seventeen years, however, reported fewer visits than MSA children (but equal in number to central city MSA children).
- ☐ NonMSA children had lower rates of private dental insurance coverage than MSA children.
- ☐ The number of dentists per population was greater in MSA than nonMSA regions.
- ☐ In 1979-1980, children in MSA had 1) lower DMFS and DMFT rates, and 2) fewer unmet dental needs, than their counterparts in nonMSA regions.

RECENT FINDINGS

Nationally

Results from the 1986-87 National Institutes of Health (NIH) study of the oral health of U.S. schoolchildren indicate continued widespread improvement in dental decay rates.

Dr. Waldman is Professor and Chairman, Department of Dental Health, School of Dental Medicine, State University of New York at Stony Brook, Stony Brook, NY 11794-8715.

†Generally speaking, a metropolitan statistical area consists of a county or group of counties containing at least one city (or twin cities) having a population of 50,000 or more plus adjacent counties that are metropolitan in character and are economically and socially integrated with the central city. Towns and cities, rather than counties, are the units used in defining MSA in New England.[3]

Table 1 ☐ Average DMFS and DMFT rates for children age 5-17 years by gender and residence: 1979-80, 1986-87.[3,4]

	Metropolitan statistical area		Nonmetropolitan statistical area	
	1979-80	1986-87	1979-80	1986-87
	DMF – Surface			
Male	4.34	2.75	4.70	3.02
Female	5.00	3.31	5.40	3.35
	DMF – Teeth			
Male	2.65	1.78	2.85	1.95
Female	3.10	2.12	3.21	2.15

Table 2 ☐ Average percent filled (F) of DMFS and DMFT rates for children age 5-17 years by gender and residence: 1979-80, 1986-87.[3,4]

	Metropolitan statistical area		Nonmetropolitan statistical area	
	1979-80	1986-87	1979-80	1986-87
	Percent F/DMF – Surface			
Male	77.6%	82.3%	70.2%	80.1%
Female	77.9	83.3	74.1	81.3
	Percent F/DMF – Teeth			
Male	78.9%	83.5%	73.8%	80.6%
Female	80.6	84.6	78.0	82.4

- ☐ Between 1979-80 and 1986-87, there was a decrease in DMFS and DMFT rates for both MSA and nonMSA children.
- ☐ In both resident areas and in both examining periods, females had higher DMFS and DMFT rates than males (Table 1).
- ☐ Between the late-1970s and mid-1980s, the percent filled (F) of the DMFS and DMFT rates (i.e. a measure of the receipt of dental care) increased in both resident areas for males and females. The percent filled (F) was slightly greater, however, for females (Table 2).
- ☐ In 1986-87, despite the increase in services (based on the percent F/DMFS and F/DMFT rates) nonMSA children received fewer needed services than their MSA counterparts (Table 3).

Regionally

Although at the national average level, MSA children had more care for their dental decay needs than nonMSA children, (i.e. percent F/DMFS and F/DMFT) on a regional basis, this relationship was reversed for children in New England, the Northeast and the Northwest. NonMSA children, as compared to their urban counterparts, had higher percent F/DMFS rates. In the Midwest, there was no real difference (Table 4).

WHAT DOES THIS ALL MEAN?

"Despite lower third-party dental insurance coverage and lower expenditures for dental services, the general population in nonurban areas, as compared to the population in metropolitan areas, is continuing to increase their use of dental services at a faster rate."[2]

Previous reviews could compare visit pattern data to determine the potential for nonurban pediatric dental practice. But now, actual examination data from the second half of the 1980s, indicate that while dental decay rates decline, higher rates of dental decay continue in nonurban-area children. And most importantly, these new data confirm the willingness of nonurban families to seek needed dental services for their children. And in many regions of the nation, nonurban children are receiving more needed services than their urban counterparts. Indeed, the prospects for pe-

Use of dental services is increasing at a faster rate in nonurban areas than in metropolitan areas.

Table 3 □ Average percent filled (F) of DMFS and DMFT rates for children by age and residence: 1979-80, 1986-87.[3,4]

Age	1979-1980		1986-1987	
	MSA	nonMSA	MSA	nonMSA
	Percent F/DMF – Surface			
5 yrs.	40.0%	24.5%	32.2%	17.3%
10 yrs.	74.4	70.3	79.1	75.1
15 yrs.	79.5	71.9	83.7	81.4
	Percent F/DMF – Teeth			
5 yrs.	35.9%	21.3%	27.5	32.0%
10 yrs.	74.6	72.7	78.9	74.7
15 yrs.	81.7	76.4	84.6	82.8

Table 4 □ Average percent filled (F) of DMFS rates for children by region and residence: 1986-87.[4]

Region	1986-1987	
	MSA	nonMSA
I (New England)	83.1%	92.5%
II (Northeast)	77.9	84.3
III (Midwest)	89.5	89.4
IV (Southeast)	73.8	69.7
V (Southwest)	84.4	69.0
VI (Northwest)	87.4	90.3
VII (Pacific)	90.6	84.4
U.S. Total	82.9%	80.7%

diatric practice continues to be favorable – in many unexpected locales.

"From the practitioner's perspective, a favorable dentist-to-population ratio, a large backlog of needed services and an increasing demand for care would seem to be a most favorable environment for dental practice."[2]

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MEDICAID'S DRAIN ON STATE BUDGETS

Medicaid continues to erode state budgets and this year became the second largest spending category in the ledger books, supplanting higher education for the first time, says the National Association of State Budget Officers. States will spend $25.2 billion in 1990 to cover some 22 million Medicaid recipients; the federal share will total $35.1 billion. Between 1989-1990, state Medicaid expenditures grew 18.4 percent. The program's spending spurt is outpaced only by prison program spending, says NASBO. Besides inflation, states are spending more on Medicaid because of federal mandates enacted over the last several years. By next year, states will have spent at least $2.5 billion to comply with laws demanding improved nursing home care and wider health coverage for poor pregnant women and children. On average, states will devote 12 percent of their budget to Medicaid programs this year; that's up from 10.8 percent in 1988 and 11.2 percent in 1989, NASBO adds. Twenty one states will devote more than 12 percent of their budget to Medicaid, including New York, Maine, Rhode Island, and Massachusetts. Two states – Alaska and Wyoming – spent less than 4 percent of their budget on Medicaid. But these states cover less than 40,000 recipients.

In contrast, Oregon, which in 1990 will cover 213,222 recipients and is planning to seek a federal waiver so it can trim benefits and widen coverage to the state's uninsured, will spend only 5.7 percent of its budget on Medicaid. Oregon is among the bottom five states that spend the least on Medicaid.

The Nation's Health, September 1990.

Epidemiology

Occurrence of primary incisor traumatism in Brazilian children: a house-by-house survey

Maria Francisca Thereza Boro Bijella, DDS, DS
Fátima Nazaré Ferreira Gazel Yared, DDS, MS
Vitoriano Truvijo Bijella, DDS, DS
Eymar Sampaio Lopes, DDS, DS

Any injury of thermic, chemical or physical nature that affects a tooth is referred to as dental trauma. The dentist should manage injuries of primary incisors because they might not only affect appearance, chewing ability and development of the middle third of the face, but also could lead to undesirable dental habits.[1] It is also known that traumatic injury in primary teeth may interfere with development and eruption of the succeeding permanent teeth.

Several authors have written on this important subject.[2-15] Internal and external hypoplasia, hypocalcification, root and crown dilaceration and even fracture of the crown of a permanent tooth are among the sequelae described in the various reports. The majority of papers are based on reports of children seeking treatment in dental school clinics or in public hospital dental clinics.[4,8,9,16-21] Other reports are based on public

Dr. Maria Bijella is Associate Professor, Department of Pedodontics and Orthodontics, School of Dentistry of Bauru, University of São Paulo, Bauru, State of São Paulo, Brazil.

Dr. Yared is Assistant Professor, School of Dentistry, Federal University of Pará, Belém, State of Pará, Brazil.

Dr. Vitoriano Bijella is Associate Professor, Department of Social Dentistry, School of Dentistry of Bauru, University of São Paulo, Bauru, State of São Paulo, Brazil.

Dr. Lopes is Full Professor, Department of Social Dentistry, School of Dentistry of Bauru, University of São Paulo, Bauru, State of São Paulo, Brazil.

school reports, ages of the children ranging from three to seven years.[22-25] Ferguson and Ripa took their sample from children enrolled in a public health program.[26]

We found no published reports on injuries to the primary teeth that had been based on a house-by-house survey. We think that this approach is the only way to obtain reliable samples to study the prevalence of dental trauma in the younger age-groups.

MATERIALS AND METHODS

The study sample comprised 576 Brazilian children (295 male and 281 female), ages ten to seventy-two months. Sixty-six streets in an urban area in Bauru, a town of 200,000 people in the state of São Paulo, Brazil, were selected. All houses in the selected streets were visited and questionnaires were completed, showing the number and ages of the children, their sex, and the dental history related to dental trauma. There were 174 children with positive reports of injuries to the primary teeth. The parents were invited to bring the children to the Paedodontic Clinic of the Dental School of Bauru. The 157 children presented were subjected to a thorough clinical and radiographic examination. A complete dental history was obtained from the parents. The injuries were classified according to Vono *et al* and Bijella.[27] No other specific classification for injuries to the primary teeth was found in the dental literature.

RESULTS AND DISCUSSION

The house-by-house study showed a 30.2 percent occurrence of injury to primary teeth, which is close to that found in other investigations.[19,22,24-26,28,29] The comparison with earlier studies can hardly lead, however, to meaningful conclusions, due to the varied methodologies. Surveys based on patients of dental clinics, as done in most of the papers, may tend to show larger percentages of dental trauma. Several investigators used samples obtained in Schools of Dentistry.[8,9,16,17,19,20] Others used samples consisting of pedodontic clinic patients.[21,29,30] Preschool children three to seven years old, were studied by others.[24-26] It is generally observed that younger children are subjected to higher percentages of injury to the primary teeth.

Our data show that traumatic injury to primary teeth may occur as early as five months of age and that the ten-to-twenty-four- month-old group shows the highest rate (14 percent) (Table 1).

This agrees with the data of several other investigators, including Ravn, who worked with twelve-to-

Injuries occur most often between ten and twenty-four months of age.

thirty-six-month-old children.[7,9,12,16,19,21] They are not, however, close to the data of Andreasen and Ravn (28 to 48 months), Garcia Godoy *et al*, Jacobsen and Sangnes (7 to 67 months), Sanchez *et al* (48 months) or Selliseth (24 to 48 months).[17,20,22,25,30]

In agreement with others, we were not able to find significant differences between males and females.[19,24,25,30,31] Selliseth concluded, however, that starting with one year of age, boys show higher rates of dental trauma than girls.[17]

The maxillary arch is involved in a higher percentage of trauma cases (97.92) (Table 2). Others are in ac-

Table 1 ☐ Distribution of children according to sex and age at time of dental accident.*

Age group (in months)	Male	Female	Totals
5 |—— 10	8	6	14
10 |—— 24	44	34	78
24 |—— 36	15	12	27
36 |—— 48	12	8	20
48 |—— 60	10	5	15
60 |—— 72	1	2	3
Total	90	67	157

*Only children who attended to clinical and radiographic examination.

Table 2 ☐ Distribution of primary incisor accidents in children 10 to 72 months of age, according to tooth type and dental arch.

		Maxilla	Mandible	Totals
Central incisor	Number	266	4	270
	percent	92.36	1.39	93.75
Lateral incisor	Number	16	2	18
	percent	5.56	0.69	6.25
Totals	Number	282	6	288
	percent	97.92	2.08	100.00

Table 3 ☐ Occurrence of injuries to primary incisors in children ten to seventy-two months of age according to side of face and dental arch.

	Right		Left		Totals	
Dental arch	Number	percent	Number	percent	Number	percent
Upper	135	47.87	147	52.13	282	100.00
Lower	3	50.00	3	50.00	6	100.00
Totals	138	47.92	150	52.08	288	100.00

Table 4 ☐ Children's distribution according to side of injury.

INJURY		Left side	
		with trauma	no trauma
Right side	with trauma	113	17
	no trauma	27	419

$X^2_{McNemar}$ = 1.84 not significant

cord.[2,7,17,26,32] The upper central incisors are the most affected teeth, which agrees with the findings of several investigators.[2,8,9,19,20,24,25,29]

Our survey produced the following percentages of trauma; upper central incisor (92.36), upper lateral incisor (5.56), lower central incisor (1.39) and lower lateral incisor (0.69), the same order found by Ravn.[8]

The statistical analysis shows a small and insignificant difference between right and left sides (Tables 3, 4). The literature does not show general agreement on this point: some believe there is no difference, while others believe the right side to be the most involved.[8,9,19,22,25,29]

There is not general agreement about the most frequent type of injury in the primary dentition. According to some, dental intrusion occurs most frequently.[16,17,34,35] Others say dental luxation, with or without displacement, occurs more often.[5,22,31] Dental luxations with displacement were seen most often by Haavirkko and Rantanen and by Schreiber; however, dental fracture occurs most frequently in the data of Ferguson and Ripa, Rinderer and Zadik.[24,26,29] Garcia Godoy *et al* and Sanchez *et al* found pulpal bleeding as the most frequent primary tooth injury.[25,30] Jacobsen and Sangnes claim it is subluxation, which is also the most frequent in our data.[20] Subluxation affects the periodontal ligament, causing tooth mobility without displacement. The tooth also becomes very sensitive to physical stimulae. We had 121 (38.05 percent) cases with this condition (Table 5). This was also reported by Jacobsen and Sangnes.[20] The other kinds of dental trauma were: subluxation with enamel crown fracture (21.07 percent); crown fracture (18.23 percent); pulpal hyperemia (7.55 percent); root fracture (6.20 percent); intrusion (4.40 percent); and complete displacement (1.57 percent). There were no cases of extrusion or crown-root fracture, in agreement with others.[22,25] Still others found root fracture as the least frequent injury.[5,16,21,34,35]

Table 6 shows the distribution of crown fractures according to class of injury. Class I is a crown fracture limited to the enamel. Class II is a crown fracture with dentinal exposure, and Class III is crown fracture with pulp exposure. Class I was the most frequent fracture, in agreement with others.[25,26] From a total of fifty-eight fractured incisors, nine were Class III (15.52 percent), a very high percentage as compared with some others.[22,29]

When the sample was classified in accordance with the number of traumatized incisors, the highest per-

Table 5 ☐ Distribution of teeth according to the type of injury and children's age.*

Type of injury	Age group (in months)						Totals	
	5–10	10–24	24–36	36–48	48–60	60–72	Number	Percent
Crown fracture	7	20	15	3	3	–	48	18.23
Concussion	1	16	4	–	3	–	24	7.55
Subluxation	18	46	21	18	13	5	121	38.05
Subluxation with enamel fracture	4	41	9	6	5	2	67	21.07
Subluxation with lingual or labial displacement	–	–	2	1	1	–	4	1.25
Intrusion	3	6	3	2	–	–	14	4.40
Extrusion	–	–	–	–	–	–	0	–
Full displacement	–	5	–	–	–	–	5	1.57
Root fracture	–	9	3	5	2	1	20	6.20
Crown-root fracture	–	–	–	–	–	–	0	–
Alveolar bone fracture	–	1	1	–	–	–	2	0.62
Unerupted tooth	3	–	–	–	–	–	3	0.94

*Some teeth showed more than one type of injury.

Table 6 ☐ Distribution of teeth according to the type of crown fracture and children's sex.

Class		Male	Female	Totals
I	Number	22	10	32
	percent	59.46	47.62	55.17
II	Number	12	5	17
	percent	32.43	23.81	29.31
III	Number	3	6	9
	percent	8.11	28.57	15.52

Table 7 □ Distribution of children, according to age, and the number of traumatized teeth.

Age group (in months)	Number of traumatized teeth									
	1		2		3		4 or more		Totals	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent
5–10	6	31.58	12	63.15	0	–	1	5.26	19	100
10–24	21	28.76	48	65.75	2	2.74	2	2.74	73	100
24–36	5	18.52	18	66.66	2	7.41	2	7.41	27	100
36–48	9	45.00	11	55.00	0	–	0	–	20	100
48–60	4	26.66	10	66.66	1	6.67	0	–	15	100
60–72	1	33.33	1	33.33	0	–	1	33.33	3	100
Totals	46	29.30	100	63.69	5	3.19	6	3.82	157	100

centage was for children with two traumatized incisors (63.69 percent), followed by one traumatized incisor (29.30 percent) (Table 7). This was in agreement with the findings of Andreasen; but not with those of Ferguson and Ripa, Korns, Ravn, or Zadik.[9,24,26,28]

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Clinic

Occlusal hidden caries: a bacteriological profile

Karin L. Weerheijm, DDS
Johannes J. de Soet, PhD
Johannes de Graaff, PhD
Willem E. van Amerongen, DDS, PhD

Occlusal caries is difficult to diagnose, especially when we are dealing with a caries lesion beneath an apparently intact enamel surface.[1] These lesions are often called hidden or occult caries, later in this article to be referred to as hidden caries.[2-4] This phenomenon is observed more frequently in recent years.[5] Hidden caries lesions can be detected in bitewing radiographs. The bitewing radiographs of these cases show a distinct radiolucency in the dentine beneath the occlusal surface; clinically, however, the tooth looks sound. In most of the epidemiological surveys, using criteria from WHO, teeth with hidden caries are diagnosed as sound.[6] If we are dealing with lesions in cases of hidden caries, we have to consider the consequences this may have on the interpretation of the results of recent epidemiological surveys.

Little is known about the mechanisms involved in the development of hidden caries. Is it due to a special bacteriological profile or is the extensive use of topical fluorides the important factor?[2,4,5] Although the phenomenon was recognized only within the last few years, it is theoretically possible that these lesions could have

Drs. Weerheijm and van Amerongen are with the Department of Pediatric Dentistry; and Drs. de Soet and de Graaf are with the Department of Oral Microbiology, Academic Centre for Dentistry, Amsterdam.

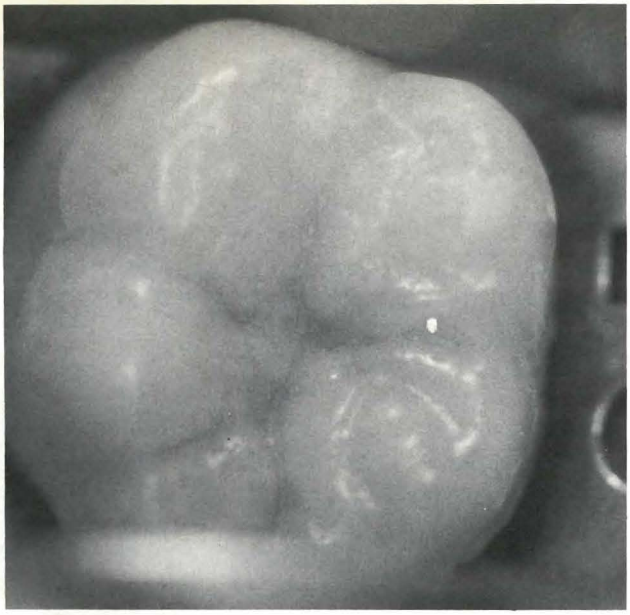

Figure 1a. Fissure, clinically not suspected (tooth 46, patient number 8, Table 2).

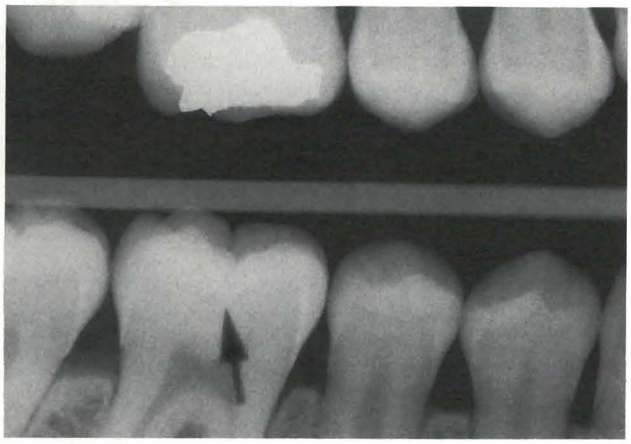

Figure 1b. Bitewing radiograph of the tooth in question.

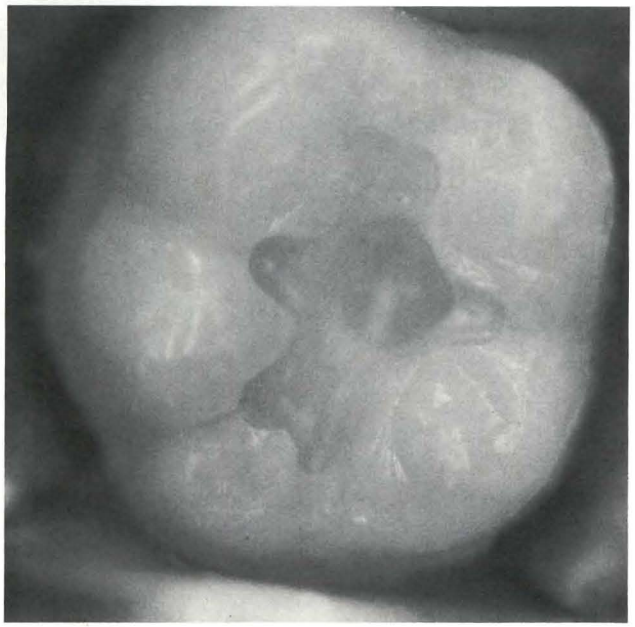

Figure 1c. Fissure, after sampling and standard preparation.

been found before the use of fluoride was generally accepted. It is just as conceivable as that the shape of the fissure could be the important factor in the development of hidden caries.

The aim of this study is to obtain insight into the bacteriological profile of hidden caries lesions. This bacteriological profile will be compared with the bacteria from the saliva of hidden-caries patients as well as from patients in a control group. Attempts will be made to interpret the results within the frame of a possible performance of a curative- restorative therapy.

MATERIAL AND METHODS

Nine patients with eleven teeth with hidden caries were selected from patients of the department of pediatric dentistry of the Academic Centre for Dentistry Amsterdam (ACTA). The teeth were selected by trained members of this department according to the following criteria:

- ☐ An apparently intact enamel surface, as diagnosed by using a mirror, excellent lighting, air syringe and careful use of a probe.
- ☐ A distinct radiolucency in the dentine visible on bitewing radiographs.

At a subsequent session, plaque was collected from the fissure and placed in reduced transport fluid (RTF).[7] Also the secretion- rate and buffer capacity of stimulated saliva were determined, using Dentobuff strips. Dentobuff strip uses an indicator system incorporated in the test strip. A drop of stimulated saliva was placed on the test pad and after five minutes reaction time, the color developed on the test pad was compared with the Dentobuff strip color chart. Yellowish-brown indicates a pH $\leq$ 4, which means that the buffer capacity is low; green indicates a pH 4.5-5.5, which means an intermediate buffer capacity; and blue indicates a pH $\geq$ 6, which means a high buffer capacity. In cases of mixed color reactions the lowest value was used. For bacteriological investigation, saliva samples were kept in well- sealed tubes without further additives. Subsequently the tooth was placed under rubber dam and cleaned with pumice. From this moment, the operator was allowed to use only sterile instruments. Interpretation of the radiolucency on the bitewing radiograph combined with the clinical observation enable the operator to decide where the teeth had to be opened (Figure 1). The teeth were opened at this site as far as the dentinoenamel junction, using a small sterile diamond bur cooled by sterile pyrogen-free water. A dentine sample was taken with an excavator and with a new round bur just under the dentinoenamel junction

and placed into 1 ml RTF. This sample was labeled A. With another round bur, caries was removed, and just before all caries was removed, a second dentine sample was taken in the same way as the first one. This sample was labeled B. The saliva, plaque and dentine samples were transported immediately to the Department of Oral Microbiology for culturing.

Meanwhile, the remaining caries was removed, the preparation finished, and the tooth restored with amalgam. It was no longer necessary to maintain aseptical conditions. A control group of ten patients with a comparable dental history and no detectable hidden caries was selected from the patient files of the pediatric department.

Bacteriological tests

As soon as the samples arrived in the laboratory (within one hour after sampling), the samples were exposed to sound waves (sonication) ten times for one second, and diluted in Todd Hewitt broth (Oxoid, Basingstoke, UK). Serially, dilutions of the samples were inoculated on blood agar, TYCSB, Rogosa agar and a nitrocellulose membrane (millipore, poresize 0.45μ) placed on blood agar. All plates were incubated anaerobically (80 percent N_2, 10 percent CO_2, 10 percent H_2) at 37°C for two days, except the TYCSB plates, which were incubated anaerobically for four days. Predominant colonies (at a 10 percent level) on Rogosa agar and blood agar were isolated for further identification. The colonies on TYCSB were identified by colony morphology. In order to count the numbers of *S. sobrinus* and *S. mutans*, the colonies on nitrocellulose membranes were identified according to the I.B.T.(immune blotting technique).[9] First, replicates of the membranes were made and then the membranes were blocked with 1 percent Bovine Serum Albumin in PBST (Phosphate buffered saline with 0.05 percent Tween and 0.001 percent Merthiolate). Then the membranes were incubated with horseradish peroxidase labeled goat anti-mouse immunoglobulins serum (Bio-Rad 170-6515). After detection of second antibody-crossreactive bacteria, by staining with chloronaphthol (150 mg in 50 ml ethanol, 250 ml 10 mM citrate buffer at pH = 5.5 and 125μl H_2O_2), the membranes were incubated with monoclonal antibodies against *S. sobrinus* at room temperature for sixteen hours. The antibodies that adhered to the *S. sobrinus* colonies were stained with labeled anti-mouse serum and chloronaphthol as described above. The same procedure was followed with monoclonal antibodies against *S. mutans*. Positive colonies were counted between incubation cycles. The salivary components of the control group were measured similarly as from the experimental group. Statistical analysis was performed by using the chi-square, the Kendall's Tau C and the student t- test.

RESULTS

From the hidden caries group, 55 percent of the saliva samples contained over 10^6 mutans streptococci per ml saliva. The salivary secretion rate varied from 0.2 to 1.88 ml saliva per minute, while the buffer capacity varied from low to high (Table 1A). Saliva from the control group (Table 1B) contained over 10^6 mutans streptococci per ml saliva in 50 percent of the saliva samples, while the salivary secretion rate varied from 0.4 to 1.84 ml saliva per minute and the buffer capacity from intermediate to high. Considering the buffer capacity, the groups did not differ significantly (chi-square = 3.06, df = 2, n.s.). The secretion rates showed no significant differences between the groups (t = -0.15, df = 17, n.s.), and no significant differences were found in the numbers of the mutans streptococci (chi-square = 1.53, df = 1, n.s.).

The results of the hidden-caries group are shown in Table 2. All the A samples contained between 10^2 - 10^5 colony-forming units (CFU) on blood agar per ml sample (with exception of the molar 47 of patient number

Table 1A ☐ Patient data and salivary characteristics collected from the hidden-caries group.

Patient	Age (y)	buf[1]	sec[2]	mut[3]
1	10	2	1.00	<6
2	8	2	0.20	<6
3	12	2	0.96	>6
4	14	3	0.72	>6
5	15	3	0.85	>6
6	18	2	1.32	<6
7	14	2	1.56	>6
8	14	1	0.72	<6
9	16	3	1.88	>6

Table 1B ☐ Salivary characteristics of the control group.

Patient	Age (y)	buf[1]	sec[2]	mut[3]
C1	11	3	1.02	>6
C2	17	2	1.26	>6
C3	6	3	0.40	>6
C4	18	3	1.58	>6
C5	9	3	0.52	ND
C6	15	2	0.84	ND
C7	16	2	1.56	<6
C8	6	3	0.62	<6
C9	11	3	1.84	>6
C10	12	3	0.94	<6

[1]Buffer capacity of the saliva measured with Dentobuff: 1:low; 2:intermediate; 3:high
[2]Salivary secretion rate in ml/min
[3]Colony forming units (log CFU / ml saliva) of *S. mutans*.
ND Not Detected

Table 2 ☐ Bacteriological data of the hidden-caries dentine samples.

Patient	Tooth	A sample				B sample			
		tot[1]	mut[2]	sob	lac	tot[1]	mut[2]	sob	lac
1	46	5.0	34	ND	<1	0	ND	ND	ND
2	36	4.6	10	3.5	43	2.9	5	36	7
3	37	4.6	1	ND	5	4.9	11	ND	<1
4	36	3.7	5	ND	3	1.7	99	ND	<1
5	47	4.5	18	ND	10	4.8	3	ND	<1
6	47	0	D	ND	ND	0	D	ND	ND
6	37	2.0	62	35	ND	1.8	5	ND	ND
7	47	4.3	38	2	7	4.3	22	<1	77
8	46	5.5	35	ND	28	3.0	8	1	17
9	25	2.3	100	ND	ND	2.5	35	ND	ND
9	34	3.5	7	ND	70	2.5	47	ND	5

[1]Total numbers CFU on blood agar (log CFU / sample).
[1]Percentages *S. mutans*, *S. sobrinus* and lactobacilli of the total CFU on blood agar (log CFU/sample).
D = sample contained 2 CFU of *S. mutans*
ND = Not Detected

6). The B samples contained between 10 - 10^4 CFU per ml sample, with exception of the B sample of patient number 1, where no microorganisms could be detected. Although the numbers of CFU on blood agar vary between the teeth and also between the A and B samples of one tooth, mutans streptococci could be detected in all A samples. In eight A samples lactobacilli were also found. The B samples showed the same microorganisms as the A samples, in most cases. In the A and B samples of molar 47 of patient number 6, only two colonies of *S. mutans* were found. Lactobacilli and *S. sobrinus* were not detected at all. The dentine of this tooth showed a dark black hard area, after opening with a diamond bur, while the dentine of all other lesions studied was soft and showed variations in color from white to brown.

The ratio of the mutans streptococci and lactobacilli in the dentine samples (Table 2) differ from the ratio of these microorganisms in the collected plaque sample (Table 3).

When mutans streptococci were found they were *S. mutans* and *S. sobrinus* in accordance with the species found in the plaque samples. The lactobacilli from the carious dentine samples were identified using an API50 system. The most isolated species were *Lactobacillus casei* subspecies. Two *Lactobacillus acidophilus* strains were isolated from tooth 34 of patient 9. The dentine of patient 2 contained at least two types of lactobacilli: *Lactobacillus plantarum* and *L. casei*, according to the API50 system. The other microorganisms detected are unidentified Gram positive rods.

DISCUSSION

It is generally accepted that mutans streptococci are associated with the initial development of caries, and lactobacilli with the further development of the lesion.[10,11] These bacteria have been found also in hidden caries lesions. The problem with such studies is to exclude possible contamination of the dentine samples with microorganisms from the plaque. To overcome this problem a plaque sample was collected before opening the tooth. Subsequently the remaining plaque was removed with pumice, after which the tooth was opened and the dentine samples collected. The results show that the composition of the plaque flora differs from those of the dentine samples. The ratio of *S. mutans*, *S. sobrinus* and lactobacilli in the plaque do not correspond to those in the dentine. Considering the carefully used aseptic procedure, these results suggest that the bacteriological samples are not a contamination from the plaque but more likely reflected the composition of the dentinal flora itself. The observation that similar microorganisms have been in nonhidden caries lesions suggests that the process of demineralization might be the same in both caries processes.[11]

Generally, bacteria in dental plaque are metabolically active, because of the influx of substrates and outflux of several end products. It has been found that sugars can be carried through the enamel, which bacteria inside the teeth may use as a substrate for fermentation. This results in metabolically active bacteria that produce acids in the dentin. Whether these acids cause a caries lesion depends on the buffer capacity of the dentin. The results of this study suggest that dentine might become carious by this phenomenon.

Furthermore, it is known that an active process is characterized by demineralization of the dentine and a continuing destruction of the tooth structure. Which criteria are used to discriminate between inactive and active lesions? When the consistency of an opened lesion is soft (demineralized dentin) and with a light color, we clinically consider this an active caries lesion. So hardness and color of the dentine are factors for clinically judging a lesion. This suggests that we are dealing

Table 3 ☐ Plaque composition with respect to *S. mutans, S. sobrinus* and lactobacilli.

Patient	Tooth	*S. mutans*[1]	*S. sobrinus*[2]	Lactobacilli[1]
1	46	ND	ND	ND
2	36	6	10	ND
3	37	3	3	+
4	36	70	ND	7
5	47	ND	+	+
6	47	2	+	ND
	37	ND	ND	ND
7	47	83	ND	+
8	46	2	ND	ND
9	25	7	ND	ND
	34	17	ND	ND

ND = Not Detected
+Counts less than 1 percent.
[1]Percentage of total CFU on bloodagar.

with active caries processes. It is known that demineralized dentine can remineralize, however, under certain conditions, resulting in an arrest of the caries process. With the exception of tooth 47 of patient 6, where the lesion after opening appeared to be remineralized, all teeth contained soft and light-colored dentine. Furthermore, in contrast with the other teeth studied, tooth 47 contained low numbers of mutans streptococci and no lactobacilli. This suggests a relationship between the numbers of microorganisms and the clinical appearance of the caries process. It seems advisable at the moment, nevertheless, to consider hidden caries in general as an active process. For the moment, corrective treatment seems to be necessary. From the saliva levels of mutans streptococci it has been reported that patients who have more than 10^6 mutans streptococci per ml are considered at risk.[10] Fifty percent of both groups contained over 10^6 mutans streptococci per ml. As for the buffer capacity, no significant difference between the groups was found. Further analyses with Kendall's Tau C (Kendall's Tau C = 0.40, $p < 0.05$) revealed that a significant relationship was found between the group and the buffer capacity. The buffer capacity of the patients in the control group tended to be higher than the buffer capacity of the patients in the experimental group (Table 1A and B). The observation that the chi-square test showed no significant difference, could be attributed to the small number of patients in the group. Until now, hidden caries lesions are only diagnosed by the use of bitewing radiographs. This method of diagnosis is not commonly used in epidemiological surveys, conducted in accordance with WHO-criteria. Considering the fact these lesions are active caries lesions, it might be worth reconsidering these criteria.

CONCLUSION

The hidden caries lesions contained mutans streptococci and lactobacilli combined with soft and light-colored dentine. These lesions are considered, therefore, to be active caries lesions, which should be diagnosed and treated as such.

SUMMARY

Eleven teeth from nine patients with occlusal caries lesions beneath an apparently intact enamel surface were treated. Two dentine samples, one from the dentinoenamel junction (Sample A) and one just before all the caries appeared to be removed clinically (Sample B), were taken under aseptic conditions and examined for bacteria. In all cases, the A samples and in ten cases the B samples contained mutans streptococci, lactobacilli or both, while in ten cases the dentine after opening was soft with a light color. This suggests that we are dealing with active caries lesions, which should be treated as such.

The salivary data of the patients group were compared with the salivary data of the control group. Although the buffer capacity of the groups were not significantly different (chi-square), further analysis revealed that a significant relationship could be found between the group and the buffer capacity (Kendall's Tau C). While the other salivary components (numbers of mutans streptococci per ml saliva and the secretion rate) showed no significant differences. Unfortunately these occlusal caries lesions are not detected with the present criteria used in epidemiological surveys.

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In vitro assessment of the microleakage around preventive resin (laminate) restorations

William Philip Saunders, BDS, PhD, FDS, RCS Edin
Ronald Strang, BSc, PhD
Imtiaz Ahmad, BDS, MSc

A clinical procedure for restoring minimal occlusal caries and, at the same time, preventing caries in the remainder of the fissure pattern has been described.[1-4] The technique involves the investigation of the carious lesion with a bur, restoration of the cavity with composite resin, and the sealing of the remaining fissure pattern with a sealant, using the acid-etch technique. Micromechanical bonding of the resin to the enamel takes place, which improves the retention of the material.[5] Where the caries extends into dentine, the cavity may be lined with a calcium hydroxide-based material or a glass ionomer cement, depending on the depth. The use of a bonding agent has also been advocated.[6]

Simonsen has described three types of preventive resin restoration, depending on the extent and depth of the carious process.[7,8] The most extensive of these restorations is termed Type C and may extend into dentine. A calcium hydroxide lining is advocated before placement of the composite resin. More recently, a glass ionomer cement has been used as a substitute for the composite filling itself, or a lining of glass ionomer is placed under the composite resin restoration.[9] The latter have become known as *laminate* restorations. The advantage of the use of glass ionomer is that the material bonds to tooth structure and to composite resin.[10,11] In addition, glass ionomers release fluoride, which may reduce the incidence of secondary caries.[12,13]

When composite resin polymerizes, it contracts and this results in gaps at the tooth-restoration interface.[14] These gaps allow the ingress of acids, enzymes, ions, bacteria and their products, a phenomenon known as microleakage.[15] Microleakage may predispose a tooth to discoloration, recurrent decay and pulpal inflammation.[16] A number of studies have investigated marginal leakage associated with the various types of preventive resin restoration.[6,9,17,18] There have been no reports on the incidence of microleakage with the laminate preventive resin restoration.

The purpose of this study was to evaluate, *in vitro*, the microleakage of laminate restorations using two resin systems and the influence of etching and curing regimen on the leakage.

MATERIALS AND METHODS

One hundred and thirty-five extracted, human third molars were cleaned with a slurry of pumice and water on a bristle brush in a low-speed handpiece. Teeth were chosen with similar occlusal dimensions. Narrow cavities were then prepared in the central mesiodistal fissure using a dome-shaped, diamond fissure-bur in a high-speed handpiece. All cavities were extended into dentine. The teeth were then separated into nine groups of fifteen teeth each.

Dr. Saunders is with the Department of Conservative Dentistry, Glasgow Dental Hospital and School, 378 Sauchiehall Street, Glasgow G2 3JZ Scotland. Dr. Strang is Principal Physicist, Dental Physics Unit, Department of Clinical Physics and Bioengineering, West of Scotland Health Boards. Dr. Ahmad is Dall Research Fellow in Dental Materials.

Table 1 ☐ Materials used in the preventive resin restorations, with associated tooth groups.

Material	Tooth groups								
Glass ionomer	A	B	C	D	E	F	G	H	I
Vitrabond (3M, St Paul, MN)	+	−	+	−	+	−	+	−	+
XR Ionomer (Kerr, Romulus, MI)	−	+	−	+	−	+	−	+	−
Bonding Agent:									
Scotchbond Dual Cure (3M, St Paul, MN)	+	−	+	−	−	−	+	−	+
XR Bond (Kerr, Romulus, MI)	−	+	−	+	−	−	−	+	−
Composite:									
P50 (3M, St Paul, MN)	+	−	+	−	+	−	+	−	+
Herculite XR (Kerr, Romulus, MI)	−	+	−	+	−	+	−	+	−
Fissure sealant									
Delton (Johnson & Johnson, East Windsor, NJ)	+	−	+	−	+	−	+	−	+
Sealite (Kerr, Romulus, MI)	−	+	−	+	−	+	−	+	−
Fluoroshield (Caulk Dentsply Int, Milford, DE)	−	−	−	−	−	−	−	−	+
Etch/wash times									
60s/45s	+	+	−	−	−	−	−	−	−
15s/15s	−	−	+	+	+	+	+	+	+
Curing									
Composite and sealant together	−	−	−	−	−	−	+	+	−
Composite and sealant separate	+	+	+	+	+	+	−	−	+

In the first part of the study, the effect of etching and washing times on leakage was investigated. Four groups (A,B,C,D) of teeth were chosen randomly and the cavities restored with laminate restorations, using two combinations of materials as shown in Table 1. Each cavity was lined with the appropriate glass ionomer cement. These materials were applied according to the manufacturer's instructions. The occlusal enamel of the teeth in Groups A and B was then etched with 37 percent orthophosphoric-acid gel for 60 secs, rinsed with water from a three-in-one syringe for 45 secs and dried with oil-free air.

The teeth in Groups C and D were etched with the same acid, but the time of application was reduced to 15 secs and the rinse-time also reduced to 15 secs. The glass ionomers were not acid-etched in any of the groups. Following careful drying of the teeth with a three-in-one syringe, the appropriate bonding agent was applied to the walls of the cavities and the surface of the glass ionomer lining. Each agent was cured with a visible light source (Luxor, ICI Co. Ltd., Macclesfield, UK), Groups A and C for 20 sec and Groups B and D for 30 sec, as recommended by the manufacturers. The cavities were restored with posterior composite resin in one increment and polymerized from two positions above the filling, for 60 sec each. The light-cured fissure-sealants were placed over the composite and the remaining pits and fissures and cured from two positions above the occlusal surface, for 20 sec each.

The second part of the study examined the effect of the omission of bonding agent on leakage. Two additional groups of fifteen teeth, E and F, were restored with preventive resin restorations. The glass-ionomer lining was placed as before, but the enamel of all groups of teeth was etched and washed for 15 sec. No bonding agent was applied to the cavities and the preventive resin restorations completed, as for the previous groups.

The effect of polymerization technique was studied in the third experiment. In Groups G and H, the cavities were again lined with the appropriate glass ionomer cement, etched for 15 sec, washed for 15 sec and dried with oil-free air. Bonding agent was then applied to the cavities and polymerized. Composite resin was placed in the cavity, contoured, and the fissure sealant placed over the occlusal surface of each tooth. Unlike the previous investigation (Groups C and D) where the composites and sealants were cured separately, in this part, however, they were polymerized together in two positions, from above, for 60 sec each.

The final group of teeth, I, were restored with laminate restorations, using a new fluoride-releasing fissure sealant with a patented phosphate adhesion promoter (Table 1). Following placement of the glass ionomer, the teeth were acid-etched and washed for 15 sec each. Bonding resin was applied, cured, and the cavities filled with posterior composite resin. Polymerization of the resin was again done from two positions above the occlusal surface for 60 sec each. The com-

posite and remaining pit-and-fissure system was covered with the sealant and cured from two positions for 20 sec each, as recommended by the manufacturers.

The restored teeth were stored in deionized water for one month, at 37°C and, immediately before testing, thermocycled through 5°C, 37°C, and 55°C and 37°C, ten seconds at each temperature for 3000 cycles.

The microleakage associated with each restoration was assessed, using a dye-penetration technique. The teeth were dried, the apices of the roots sealed with cyanoacrylate cement, and coated with two layers of nail varnish so that only the restorations, and a band of surrounding tooth structure approximately 1 mm wide, were exposed. The teeth were immersed in a 2 percent solution of methylene blue dye for twenty-four hours at 37°C, rinsed thoroughly in tap water, and sectioned with a water-cooled, diamond disc in a buccolingual plane through the restoration.

Each specimen was examined under X6 magnification and the extent of leakage was scored according to the criteria shown in Table 2. The data were analyzed using the Kruskal Wallis test, corrected for ties, to determine whether there were any significant differences among the Groups A,B,C and D. A statistical analysis was carried out, using the Mann Whitney U test, to determine whether there were significant differences in leakage between pairs of groups.

Table 2 ☐ Score of leakage around restorations.

Degree of microleakage	Depth of penetration
0	No microleakage detected
1	Dye penetration up to composite resin-sealant junction
2	Dye penetration up to dentinoenamel junction
3	Dye penetration short of pulpal wall
4	Extensive dye penetration to and into pulpal floor

Table 3 ☐ Microleakage scores for preventive resin restorations.

Tooth group	Marginal leakage scores				
	0	1	2	3	4
A	10	4	1	0	0
B	11	3	1	0	0
C	11	3	1	0	0
D	12	2	0	0	1
E	12	1	0	1	1
F	8	7	0	0	0
G	12	3	0	0	0
H	13	2	0	0	0
I	15	0	0	0	0

Fifteen teeth in each group

RESULTS

The microleakage scores for each group of restored teeth are shown in Table 3. Statistical analysis of the effect of etching and washing time on the degree of leakage for the two sets of materials showed that there were no significant differences in leakage for the four Groups A-D (H^1 6.32, $p > 0.05$).

There were no significant differences in leakage between those groups that had been restored with and without bonding agent ($p > 0.05$), or between groups where the composite resin and sealant had been polymerized separately or together ($p > 0.05$). There were no statistically significant differences in leakage, when one series of materials was compared with another ($p > 0.05$), except for Groups I and F, where the group restored, using the fluoride releasing sealant, showed less leakage than the group restored with the Kerrs products, without bonding agent ($p < 0.05$).

DISCUSSION

The importance of thermocycling before microleakage testing has been discussed by Jensen and Handleman.[19] They regarded this process as a means of accelerating the effects of storage in water. It has been shown that the variables of water temperature and length of time to testing for thermocycling is not critical.[20]

In the present study, reducing the time of etching and rinsing of the enamel did not affect the incidence of microleakage. This is in agreement with Stephen *et al* and Eidelman *et al*, who found that reducing the etching-time of enamel from 60 sec to 20 sec did not affect the quality of retention of fissure sealants.[21,22] In addition, Fuks *et al* reported that the reduction in etch-time had no effect on the microleakage of a fissure sealant.[23] The reduction in chairside time required for etching and washing makes the placement of the laminate restoration a more cost-effective procedure.

Various surface treatments have been suggested for improving the composite/glass ionomer bond, including etching of the glass ionomer surface with phosphoric acid.[11] In the present study, however, the glass ionomers were not acid-etched because one of the manufacturers recommends specifically that this should not be done. Subrata and Davidson have shown that partial dehydration of glass ionomer, followed by the addition of a bonding agent, improved the bond strength of glass ionomer to composite resin, comparable to that achieved by acid-etching.[24] Their study was carried out on glass ionomers that contained no resin. Both glass ionomers used in the present investigation were resin-containing materials. Thus, it might be expected that some bonding could occur between the resin in the glass ionomer and the bonding and composite resins. The omission of the bonding agent, however, did not

affect the degree of microleakage adversely. There was no statistically significant difference between those groups with bonding agent and those without. If the use of the bonding agent was found to be unnecessary, the provision of the laminate preventive resin restoration would be less time-consuming. The lack of any significant differences in the microleakage between those groups in which the composite and sealant were polymerized together, as opposed to separately, would indicate that combined polymerization may also be useful in reducing treatment time.

Raadal reported that a control group of preventive resin restorations with unetched enamel showed extensive leakage compared with an etched group.[17] It has been shown that sealant retention is limited to the inclined cuspal planes and not to the recesses of the occlusal fissures.[25,26] In the present study, the whole of the occlusal surface of each tooth was etched and the fissure sealant extended across the included cuspal planes.

The incidence of microleakage in this study varied between no leakage and 46.7 percent. Leakage of preventive resin restorations was reported to occur in 16.7 percent to 25 percent of cases.[6] The fact that the variables tested in this study had no significant effect on the microleakage would suggest that the leakage of preventive resin restorations is dependent on that of the fissure sealant. There has been a wide spread of results for the incidence of leakage associated with fissure sealants, varying from no leakage, in one study to 100 percent.[27,28] The reason for this may be due to the fact that there is no standardized screening procedure for testing leakage of sealants *in vitro*.

One of the groups of teeth (I) tested in this study had been restored with a fissure sealant containing releasable fluoride and an adhesion promoter. This sealant is 50 percent filled and contains 2 percent sodium fluoride by weight, as well as a PENTA phosphate adhesion promoter. This group performed well in this study with none of the teeth showing leakage after one-month storage and thermocycling. The advantage of the use of this sealant is that some degree of chemical adhesion to enamel is achieved and the presence of releasable fluoride may reduce the possibility of secondary caries, should the restoration leak. The long-term effects of the use of this material is unknown with respect to the stability of the adhesion promoter and the prolonged release of fluoride ions.

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Regulation therapy by Castillo-Morales in children with Down syndrome: primary and secondary orofacial pathology

G. Johannes Limbrock, MD
H. Hoyer, DMD
H. Scheying, MD

Since the mid-1970s, when Castillo-Morales developed the Orofacial Regulation Therapy for Down syndrome children, constant and meticulous observation of orofacial symptoms in the growing child led to new findings.[2,3]

We now distinguish between primary orofacial signs present at birth and in the first year of life on the one hand, and secondary alterations that develop with untreated school-age children on the other hand (Figures 1 to 5).

Included in the primary findings are:

☐ Lingual diastasis

☐ "Stair palate".

Castillo-Morales compared the lingual diastasis to diastasis recti abdominis. In hypotonic babies, especially in those with Down syndrome, we often find a weak abdominal musculature and a deficiency of linea alba fibers crossing the median line. Thus, the abdominal median line presses forward in a roll-shape when the muscles contract.[3]

In the tongue one sees only the lingual diastasis during contraction in the sagittal direction: for example, when the tongue is extended (Figures 6,7).

This phenomenon occurs in 80 percent to 90 percent of the infants with Down syndrome. It is only rarely observed in other children, healthy or ill. Probably, the prominence of the median line of the tongue during sagittal contraction is due to an insufficiency in the number of transverse fibers of the genioglossus muscles as well as an insufficiency of the fibrous septum linguae.

Figure 8 shows the embryological development of a human tongue. In the 6th week of gestation, there are three lingual buds, of which the median one disappears. The junction between the two lateral buds will be the lingual septum, where in Down s. infants the diastasis phenomenon will arise.

In the 15th week, the genioglossus muscle fibers begin to decussate (Figure 9) in the midline, as do the transverse muscle fibers and the fibrous tissue.[10] In Down syndrome embryos the fiber decussation is probably not as great as in normal children. This is an assumption that until now is only supported by some histological sections of the tongues of Down s. newborns and nursing infants, by Castillo-Morales in Argentina.

At the gestational age of about thirteen to sixteen weeks, the rooting reflex, and the sucking and swallowing actions appear. From this time on, we can formulate the influence of functional tongue and lip movements on the growth and shape of maxilla and mandible.

The lingual diastasis is marked in the first three years of life and then is often concealed under a thicker tongue surface, and may disappear because of midline development of the tongue.

Dr. Limbrock is with the Institute for Social Pediatrics and Youth Medicine, University of Munich, in the Kinderzentrum (Professor Hellbruegge is Director of the Institute). Dr. Hoyer is with Pediatric Dentistry, Altona Children's Hospital, Hamburg (Professor Blunck is Director). Dr. Scheying is with the Pediatric Rehabilitation Center Werner-Otto Institute, Hamburg and is the Director.

Figure 1. Development of orofacial pathology in Down syndrome infants.

Primary characteristics	Secondary characteristics
Hypotonia of orbicularis, zygomatic, masseter, temporalis, and mimic muscles	Lower lip everted, becoming prominent
	Angle of the mouth is pulled down
Reduced tonus of ligamentary apparatus of mandibular joint	Upper lip is inactive and pulled up, with underdeveloped lateral parts and short frenulum
	Open mouth breathing, drying of mucous membranes
Disorders of the immune system	Chronic periodontitis; respiratory infections
	Drooling; chapping of lower lip and angles of the mouth
Hypotonic tongue protrusion and followed by active tongue protrusion	Tongue protrusion or thrust during drinking, sucking the pacifier, eating and speaking; tongue sucking
Diastasis linguae, concave lingual blade, and feeble frenulum linguae	Later: relative macroglossia, rarely true macroglossia; dry tongue surface with chapping;
	Protrusion of upper and lower front teeth;
	Indistinct pronunciation
Primarily reduced jaw angle	Progredient reduction of jaw angle, but less reduction in growth of length of mandible.
Primarily reduced total length of the mandible	Open bite with dentoalveolar components; protrusion of front teeth
	"Pseudoprognathism, ANGLE class III
	Habitual subluxation of the jaw
Hypoplastic middle face:	Tongue protrusion due to too small oral cavity
Hypoplasia of maxilla in sagittal and transversal directions	Maxillary sagittal growth and palatal height remain reduced
Reduced palatal height, but not a narrow palate, observable by cephalometry	Maxillary transversal growth reduces progrediently
"Stair palate"; eminent and remaining tectal prominences	Possibly formation of V-shaped palate, high in appearance
Hypotonic velum, sometimes submucous cleft of palate and/or velum	Velar insufficiency, occasional contraction to a concave shape
Retarded dentition, microdontia, anodontia, hypodontia; aberrant teeth	Retardation of bite function
	Oral stereotypes

(Modified from Castillo-Morales and other authors: 1,4,6,7,8,12,13,14)

The diastasis itself is not a functional problem and no reason for treatment, but it influences the shape of the stimulating button on the palatal plate: instead of a round-shaped button for the normal tongue, the diastasis requires an oval button. Both of them have a central hole and are placed at the dorsal edge of the plate.

The "stair palate" is a palatal form that is often found in the first years of life in Down syndrome children. But it also occurs in prematures and newborns, especially in those with muscle hypotonia and sucking problems. In normal children it disappears from the second year of life on.[9] With the name "stair palate", Castillo-Morales characterized the step-like transition of the often striking palatal prominences (Figure 10).[3]

The form of the palatal prominences in Down babies, between two and sixteen months of age, was measured by Fischer-Brandies: It is very prominent in 12 percent of the cases; less so in 68 percent; and barely distinguishable in approximately 20 percent (Figure 11).[7] In his cephalometric study, Fischer-Brandies also revealed that the vertical palatal height is reduced in relation to the transverse dimension, as well as in comparison with the vertical palatal height of a normal population.[6,7] This is in accordance with the findings of Westerman and Cohen.[4,14]

The often used description of a high and "gothic" palate, therefore, fits neither the absolute or relative dimensions nor the palatal form. Down s. children of school age, however, sometimes show a V-shaped palate, which leads to the impression of a high palate (Figure 12).

Other important findings are that the overall length of mandible is reduced and grows slightly less than normally. The jaw angle is smaller than in normal children and lags progressively behind the norm.[7] The open bite must be caused primarily, therefore, by dentoal-

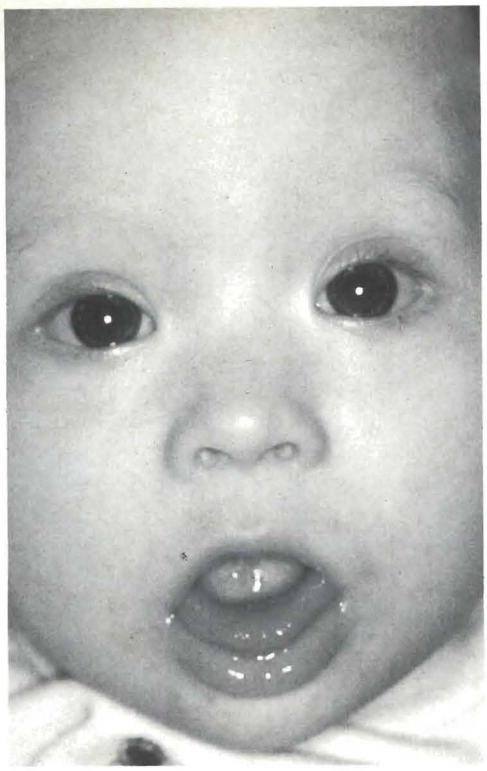

Figure 2. Six-month-old Down s. infant with hypotonic tongue protrusion; concave lingual blade; retracted, inactive upper lip; and everted lower lip.

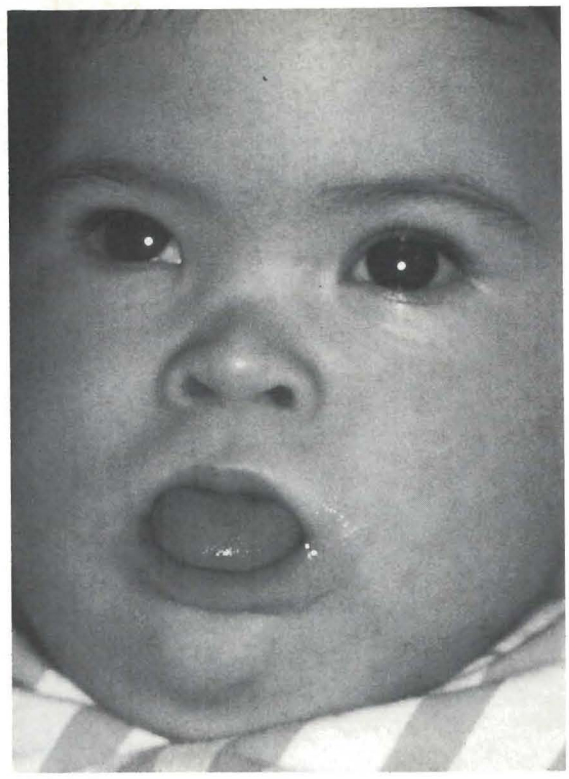

Figure 3. Ten-month-old Down s. infant with a protruding tongue that has an augmented tissue consistency and is regarded, therefore, as macroglottic (one of the five patients we found among 400 Down s. infants).

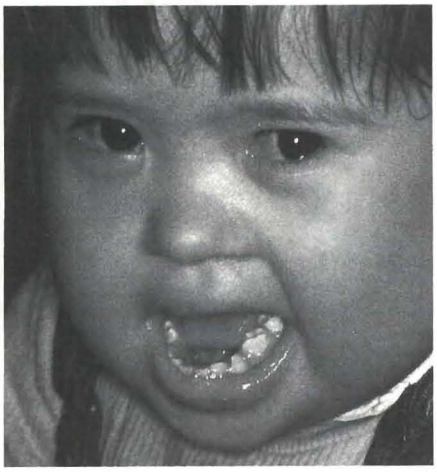

Figure 4. Five-year-old Down s. child with a beginning jaw protrusion ("pseudoprognathism").

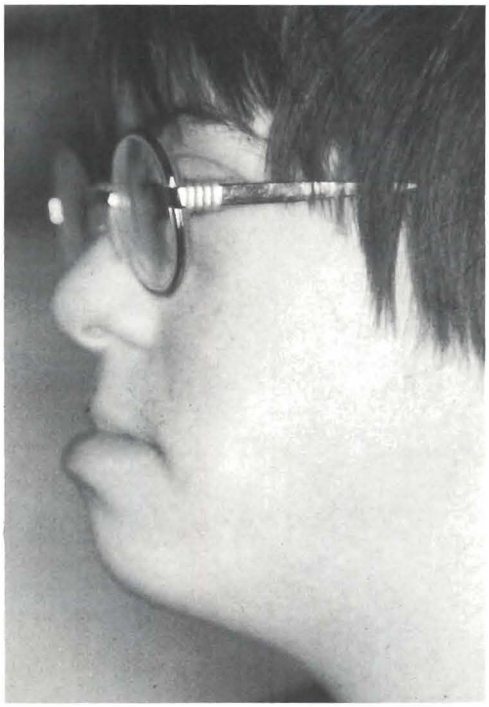

Figure 5. Thirteen-year-old Down s. child with a marked "pseudoprognathism" and thick and somewhat everted lower lip.

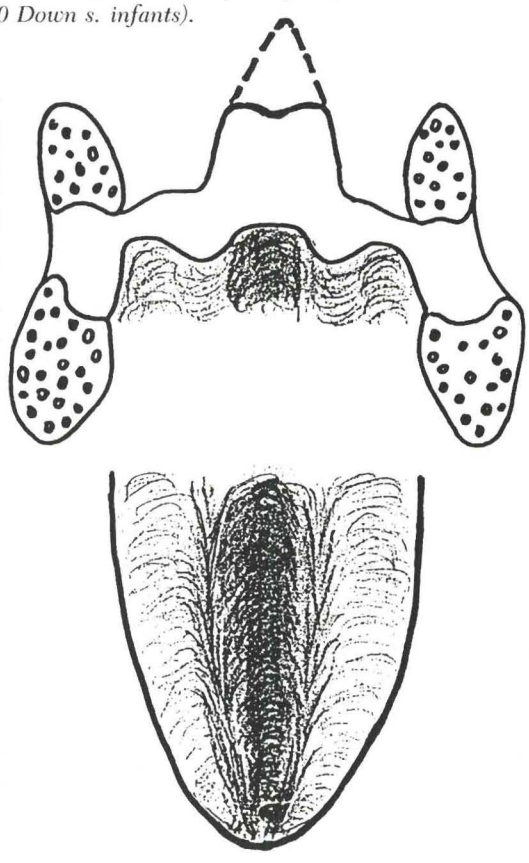

Figure 6. Lingual diastasis and stair palate. Above: Cross-section of the oral cavity of a Down s. infant. The dotted line shows the possibly later, pointed form of the palate. Below: View of the lingual diastasis when the tongue is extended. The middle line is arched, the sides are furrowed.

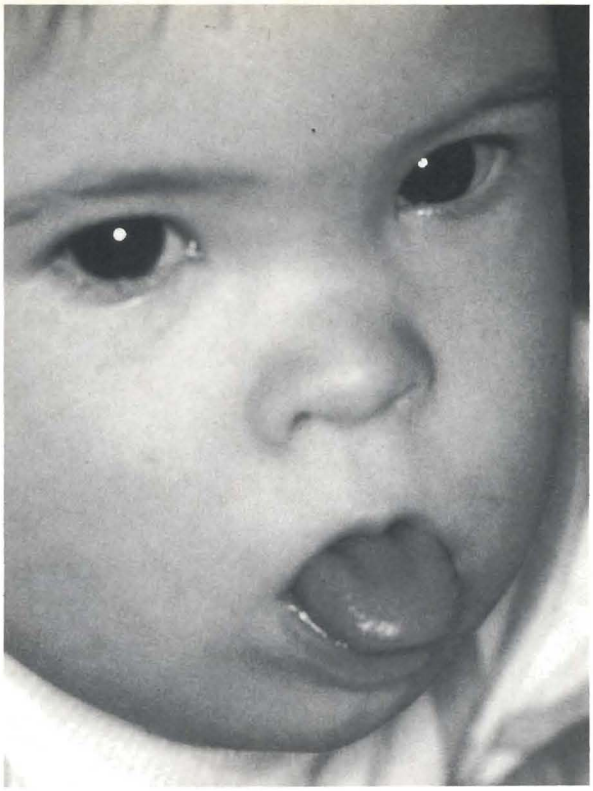

Figure 7. Ten-month-old Down s. infant (same patient as in Figure 3) with a lingual diastasis that appears when the tongue is extended.

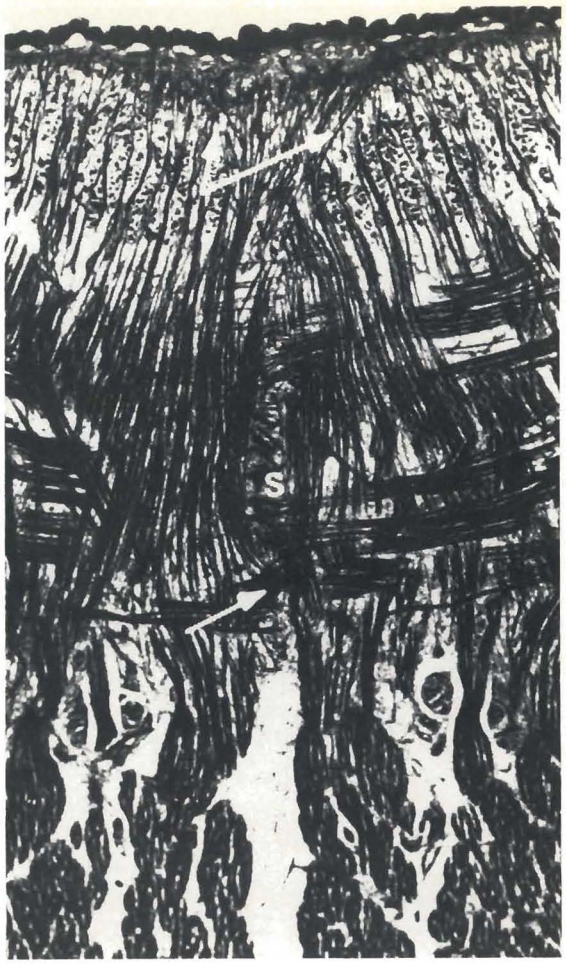

Figure 8. Lingual buds of a six-week-old embryo. Horizontal section of pharynx.

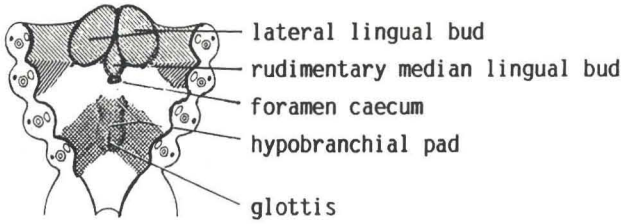

Figure 9. Coronal section through the tongue of a 15-week human fetus. Genioglossal decussations begin in the median line (septum = S) (arrows). From: Langdon et al 1979.

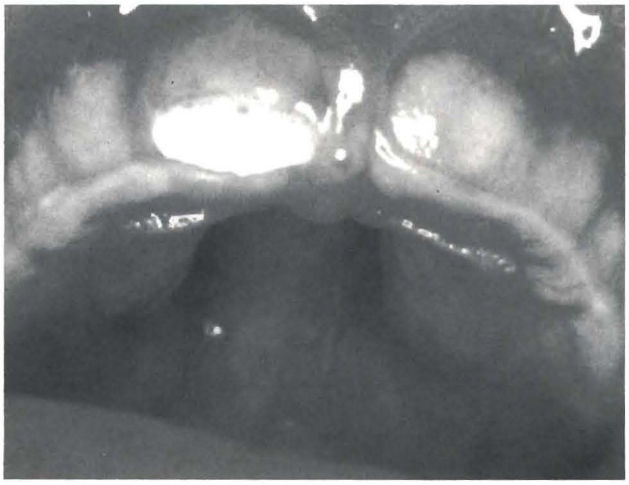

Figure 10. Thirteen-month-old Down s. infant with a typical "stair palate": the prominent eminences have a step-like transition to the palatal roof.

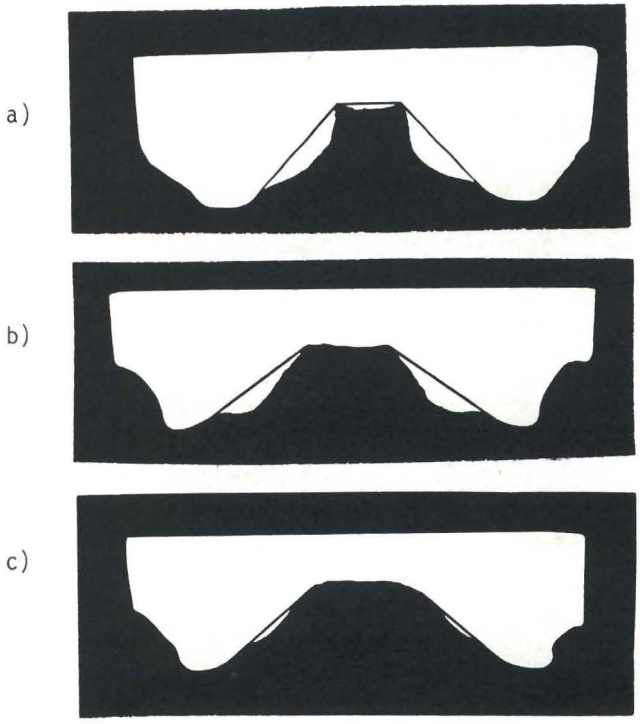

Figure 11. Palatal or tectorial prominences in Down s. babies, modified from Fischer-Brandies: a) very eminent, b) eminent and c) just visible prominences.[7]

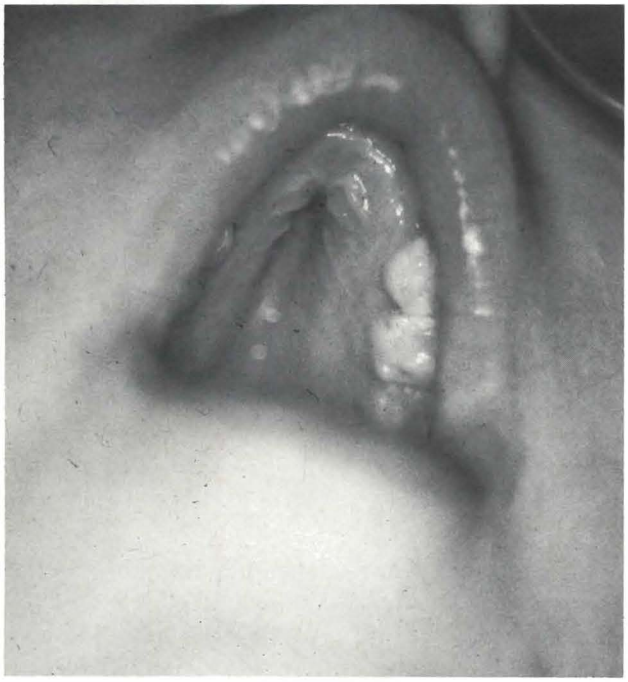

Figure 12. Nine-year-old Down s. child with a V-shaped palate, which seems to be high because of the marked palatal eminences.

veolar components. And the malocclusion of Angle class III is not due to the size of mandible, but to its protrusion and to the size of the maxilla. A hypoplastic maxilla is also part of a real prognathism. This is why the term of "pseudoprognathism" is not ideal for this situation.

Figure 1 shows a synopsis of the most important primary and secondary orofacial disorders in Down syndrome children.

A true macroglossia is very rare in Down s. infants. We found only five out of four hundred patients in Hamburg and Munich.[12] We regard a tongue as macroglottic when its size apparently is so big, that the patient can only with effort hold it in the mouth, and when the tissue of the tongue is augmented by digital palpation. This is the most important criterion, which is also part of the definition of macroglossia in other syndromes, for example, Wiedemann-Beckwith.

There are a number of other orofacial problems in Down s. children that are classified as primary or secondary findings: for example, caries or hypoplasia of the nasal cavity and of the sinuses. We have summed up those findings that are primarily related to malfunction and can be influenced, therefore, by Orofacial Regulation Therapy (Figure 1). This refers to the paper by Dr. Hoyer, which shows the three principles of the

The length of the mandible is reduced.

therapy: the functional diagnostic assessment, a special manual program of stimulation and, only in combination with the manual treatment, the palatal stimulatory plate.

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Orofacial regulation therapy in children with Down syndrome, using the methods and appliances of Castillo-Morales

H. Hoyer, DMD
G. Johannes Limbrock, MD

The varying hypotonia in Down syndrome (DS), is one of its most impressive signs. In particular, the facial expression and oral dysfunctions of these handicapped persons are uniquely characteristic. The more or less permanently open mouth; the prolapse of the tongue, exposed on the everted lower lip; and a lack of mastication, deglutition and speech are primarily caused by the hypotonic orofacial muscles.

Active facial muscular is evident in normal prenatal development. Even orofacial influence by thumb-sucking starts as soon as four to five months before birth. In DS children, however, this habit seldom develops even after birth, because hypotonic lips seem not to encourage interest in thumb- sucking.

The size of the hypotonic intrinsic muscles of the tongue is disproportionate to the size of the small oral cavity. The tongue appears too large for the mouth. We call this *relative macroglossia* and recommend careful examination before attempting surgical intervention. In about 400 DS infants, we found a real *macroglossia* in only five cases; the majority showed a hypotonic and more and more protruding tongue, referred to as *relative macroglossia* (Figure 1).

Also breathing through the mouth leads to a dehydration of bacteria and plaque on gums and teeth, and finally, therefore, to premature destruction of the dentition.

Dr. Hoyer is with the Altona Children's Hospital in Hamburg, Germany. Dr. Limbrock is with the Kinderzentrum in Munich, Germany.

This devastating development likewise indicates the need for early functional training of the orofacial muscles. A six-week-old baby with Down syndrome who has to be fed through a tube because of muscular weakness is certainly not too young to begin sensomotor therapy with newly developed dental devices.

The therapy employed is an expanded program of neuromuscular stimulation, including the orofacial area.

Castillo-Morales follows a dual concept and calls it Orofacial Regulation Therapy. He expanded his program of neuromuscular stimulation of the whole hypotonic body in Down syndrome, including all the orofacial area; in addition he used an acrylic palatal plate that stimulates the tongue and upper lip with

The method is based on the unconscious probing of a foreign object by the tongue.

special devices (Figures 2,3). The stimulators are marked in different colors. Those sections of the dorsal side of the tongue and upper lip that can respond are thus constantly exposed to tactile stimulation. The idea behind this technique is a simple but ingenious one, based on the same reasoning that prompts the tongue to probe the sharp edge on a tooth.

The following figures demonstrate the desired process in a six-month-old baby before and after the application of the device: the tongue is spontaneously withdrawn when its dorsal surface comes in contact with the oval suction-knob in the dorsal section of the plate: the mouth closes, deglutition results by elevating and withdrawing the tongue. Also the perioral muscles are activated by the corrugated texture in the vestibular front part of the plate (Figure 4 a,b). This process is explained in the sketch (Figure 5).

In this way, the plate acts as a stimulating device, which—in the mouth of the relaxed child—complements to maximum effect the usual manual therapy, without ever causing damage, if careful observation is maintained.

A nearly two-year-old girl was consequently treated with the Orofacial Regulation Therapy technique (Figure 6a). The following photograph shows the result after two years of treatment (Figure 6b).

In conclusion, some of the limitations of these studies are presented: because of permanent open-mouth, the risk of establishing habitual mouthbreathing is evident. This habit occasionally complicates treatment, but does not influence the main results. In any case, adenoidal growth should be removed for better nasal air-passage. Another fact that hinders a perfect analysis is the dual concept of the Orofacial Regulation Therapy, which makes it difficult to determine the grade of

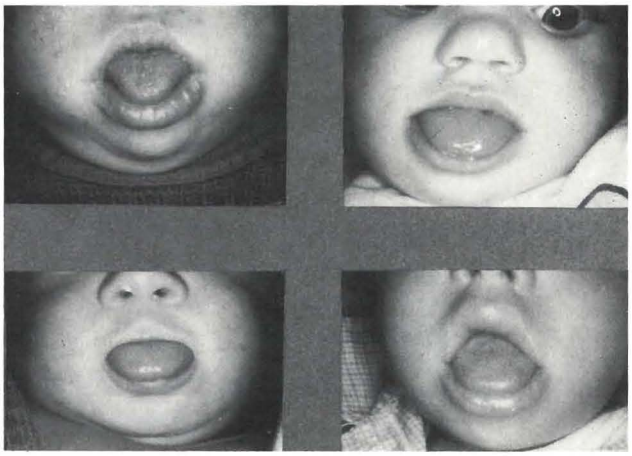

Figure 1. Relative macroglossia in Down syndrome.

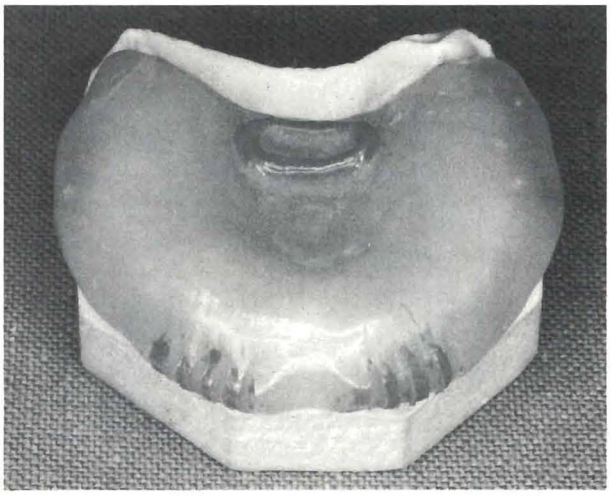

Figure 2. Placa de Memoria according to Castillo-Morales.

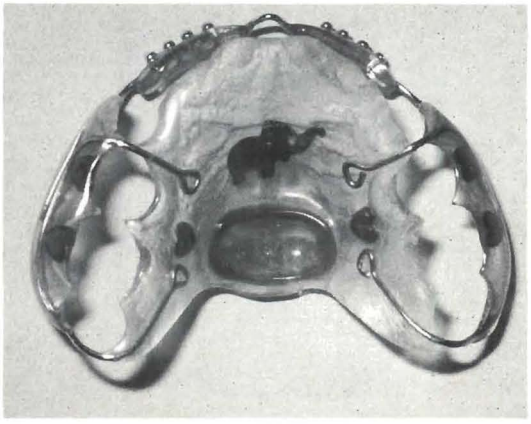

Figure 3. A palatal plate for the first dentition.

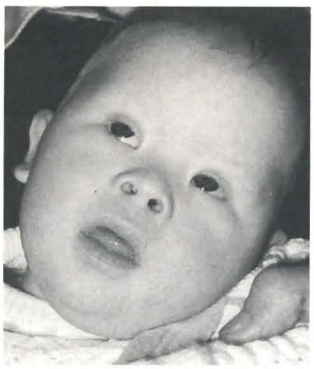

Figure 4a. Six-month-old infant with Down syndrome before use of the device.

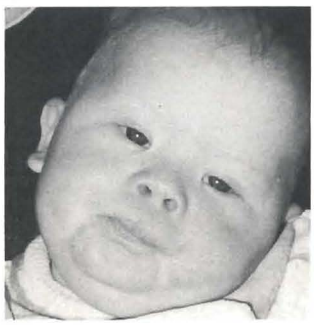

Figure 4b. The same infant after the application of the device.

Figure 6a. Hypotonic orofacial condition with relative macroglossia of a two-year-old girl with Down syndrome before regulation therapy.

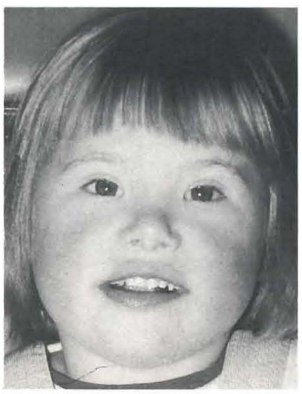

Figure 6b. The same patient after two years of functional treatment.

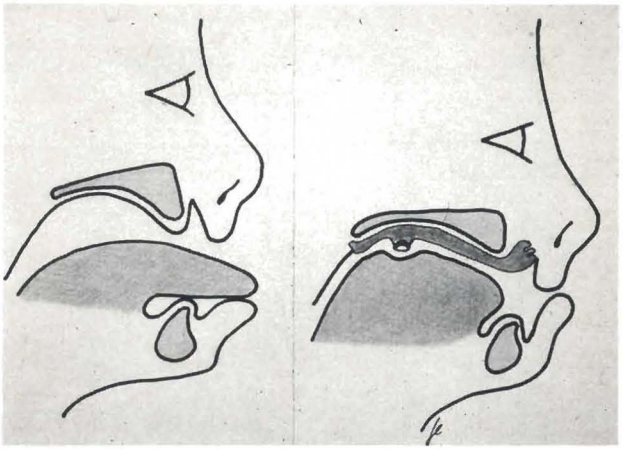

Figure 5. The schema illustrates the procedure in the oral cavity.

efficiency of only the intraoral appliances. Standardized experimental norms are not available, but are being developed. But the functional efficiency of the device was always proven, when it was omitted for a period of time in the early stages of treatment, resulting in a return to the initial pathological condition. This demonstrates that a conscientiously applied and sufficiently long-lasting treatment (between one and two years, at least four hours per day) will achieve the desired permanent results, when interdisciplinary support is provided by the medical team (dentist or orthodontist, pediatrician and therapist).

The results of the study are encouraging: approximately two thirds of seventy-four children with Down syndrome, ages between six weeks and eight years, were successfully treated. The orofacial hypotonic symptoms were significantly reduced to allow improvement in mastication, deglutition, speech and facial expression. These observations are in accordance with the recommendations of Castillo-Morales and with those of Avalle and Fischer-Brandies.

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Drooling, chewing and swallowing dysfunctions in children with cerebral palsy: treatment according to Castillo-Morales

G. Johannes Limbrock, MD
H. Hoyer, DMD
H. Scheying, MD

For many cerebral-palsied children, orofacial dysfunctions are a severe health problem, as well as a deterrent to normal acceptance by peers in school and society. These children suffer from drooling and eating, drinking and articulation disorders (Figure 1). Drooling in cerebral palsy occurs frequently and is not due to hyperproduction of saliva, but to a dilatory and disordered swallowing pattern.[5,14] The abnormal coordination of tongue, lips, and cheeks leads to long-term consequences of dysgnathia and periodontopathy.[4,6,7,11-13] Because of mouth-breathing, these children often suffer from respiratory infections.

Because classic physiotherapy and motor speech therapy have a limited influence on these problems, Castillo-Morales and the authors modified and expanded the *Orofacial Regulation Therapy*, which was designed in the 1970s for children with Down syndrome, and was developed by Castillo-Morales in his rehabilitation center in Cordoba, Argentina.[1,2,8]

The therapeutic concept includes:

- ☐ Functional diagnostics of oral sensorimotor dysfunctions.
- ☐ A special manual *stimulation and facilitation program*.
- ☐ Removable activating palatal plates and other orthodontic appliances.

Dr. Limbrock is with the Institute for Social Pediatrics and Youth Medicine, University of Munich, Kinderzentrum in Munich, Germany. Dr. Hoyer is with the Altona Children's Hospital in Hamburg, Germany. Dr. Scheying is Director of the Pediatric Rehabilitation Center Werner-Otto Institute, Hamburg.

This paper presents the basic concepts of the therapy and the results of a six-year-study with sixty-eight cerebral palsy children, treated in the Pediatric Rehabilitation Center in Hamburg, the Werner-Otto-Institute. About 23 percent of the patients suffered from a spastic tetraparesis, about 19 percent from an athetosis and about 20 percent from a hypotonia, including three with cerebellar ataxia. The remainder of the group, in the main, had minimal sequelae of cerebral palsy; their mental problems were predominant. The age when en-

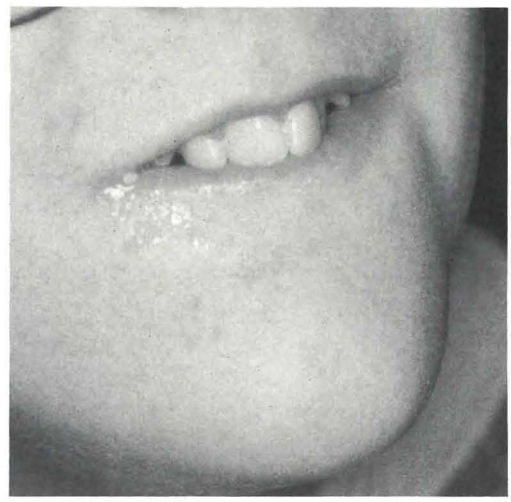


Figure 1. Fourteen year-old-boy with spastic teraparesis, who cannot walk. He suffers from drooling, and eating and articulation disorders.

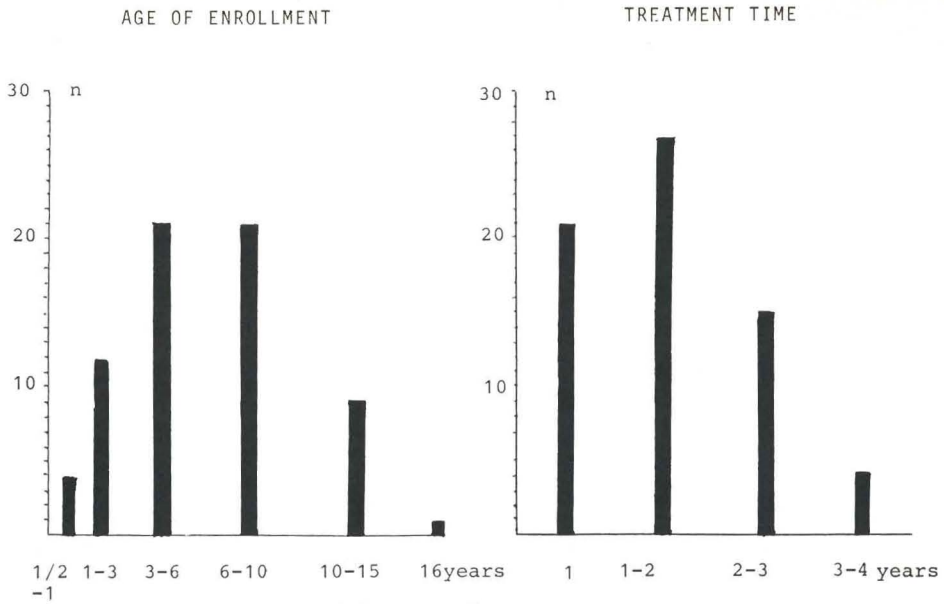


Figure 2. Orofacial therapy with sixty-eight cerebral-palsy children.

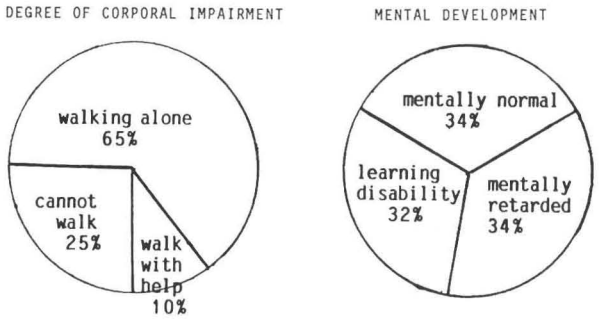


Figure 3. Orofacial regulation therapy in sixty-eight cerebral-palsy children.

rolled, the treatment time, and the degrees of physical and mental handicap are shown in Figures 2 and 3.

TYPE OF CEREBRAL PALSY AND ADEQUATE THERAPY

Although the orofacial symptoms depend on the neurological type of cerebral palsy, they are individually distinctive. Frequent examinations before and during therapy, therefore, are required. This is the task of a team comprising a neuropediatrician, a dentist or orthodontist, and a speech therapist or physiotherapist. To work with activating orthodontic appliances without this team and without the indispensable special manual, therapy would cause these children with cerebral palsy to suffer a severe neurologic deterioration in their oral and physical conditions.[9]

For demonstration purposes it is useful to present a simplified schema of cerebral palsy (Figure 4) together with adequate therapeutic principles:

- ☐ Spasticity,
- ☐ Athetosis,
- ☐ Hypotonia.

Figure 4.

SPASTICITY

- tense reclination of the head
- open mouth, tense and rigid facial and lip movements
- hypertonic tongue, cigar-shaped, often immobile and asymmetric
- tongue thrust during swallowing and speaking

ATHETOSIS

- involuntary writhing movements of head and limbs
- synkinesis of tongue, jaw and face
- spontaneous wave-like tongue movements
- abrupt wide mouth-opening until subluxation

HYPOTONIA

- flabby head reclination
- weak and poor facial movements, accompanied by an elevated inactive upper lip
- protruded large and flat tongue, sometimes atrophic

Spasticity

Spastic cerebral-palsy children, for example, with tetraparesis, diparesis, hemiparesis and mixed forms, often hold the head in a tense, reclined posture. The mouth is open, facial and lip muscles are hypertonic and their movements are sometimes exaggerated. The upper lip is underdeveloped and retracted and does not exert enough labial pressure on the anterior teeth. The tongue, hump-backed in a cigar-shape, is often rigid; it retracts while the mouth is open, or thrusts forward forcefully. The voluntary mobility of the tongue is restricted in every respect and often superposed by involuntary movements: for example, as seen in fibrillations or tongue thrust. These may occur especially while speaking and swallowing (Figures 5 and 6). The frequency

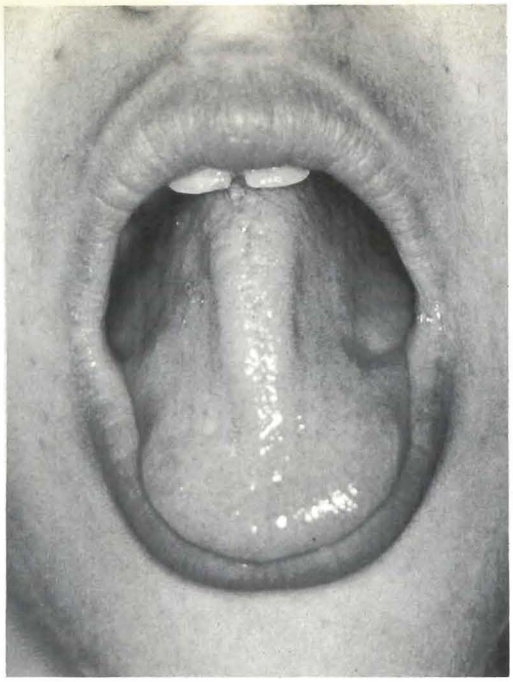
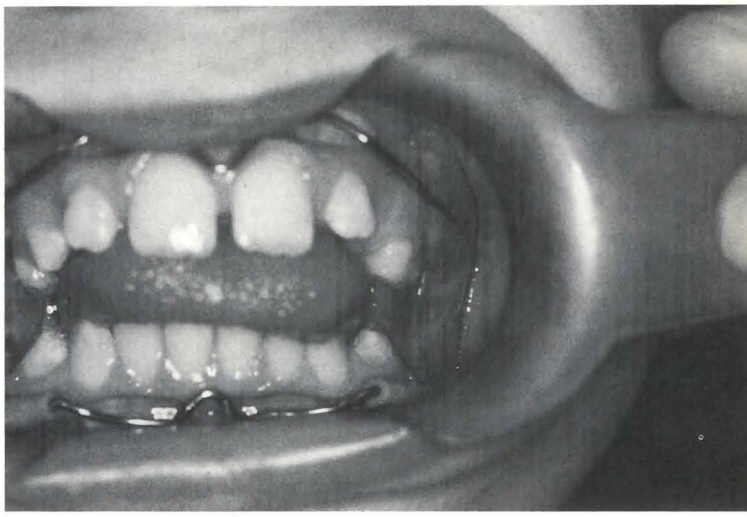


Figure 5. Seventeen-year-old-boy with spastic teraparesis, who can walk alone. He suffers from a strong tongue thrust that severely disturbs eating and speaking. The tongue shows the typical humped back and the lateral uncoordinated movements when thrust forward.

of orofacial disorders in our patients with spastic tetraparesis is shown in Figure 16.

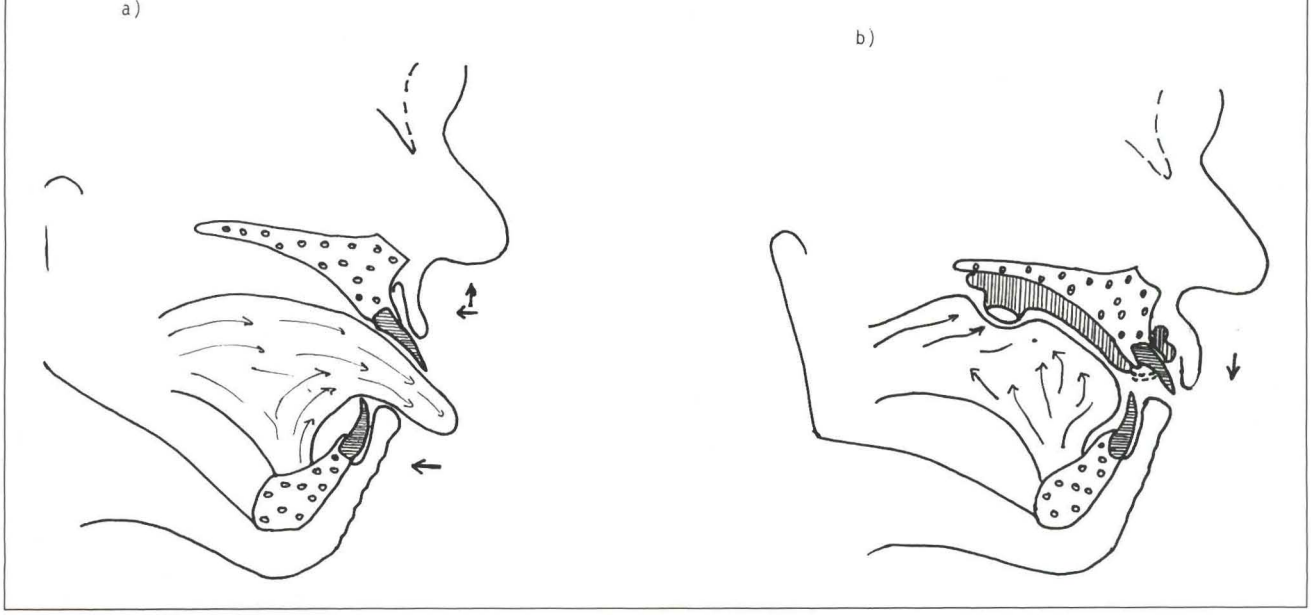
Functional therapy offers a variety of stimulating and facilitating elements for these problems. All require that the posture be carefully corrected and spasticity inhibited. To accomplish this generally requires working on a foam-rubber mattress. Shoulders and neck must be relaxed first, and then the occiput pulled constantly to stretch the neck during the exercise. With a large variety of movements like reclining, flexion, turning to the sides and returning, partly against resistance; as well as in swallowing or phonation, new intraoral sensory experiences and movements of the tongue, velum, and cheeks are achieved.[8]

Figure 6. Nine-year-old-girl with a mild tetraparesis ("diparesis in tetra-syndrome"). Her tongue presses into the open bite during swallowing and speaking.



The stimulating palatal plate (Figure 7) will help to reduce tongue thrust and provide the tongue with a contrary impulse to move backwards and upwards. The back of the tongue will elevate, which is essential for

Figure 7. Schematic effect of the palatal plate: a) The protruding and pressing tongue contributed to an open bite with the front teeth protruding, b) The palatal plate stimulates the tongue to move backwards and press upwards against the button with the central hole. The upper lip is stimulated to move from its retracted position (a) forward and (b) downwards.



swallowing and for the third articulation zone: *Gf;1 and K*. Cerebral-palsy children often cannot accomplish this movement.

The best result is achieved by a button-like stimulator at the dorsal edge of the plate, which must have a deep central hole in order to induce a sucking effect of the back of the tongue while it is pressed against the button (Figure 8).

In order to correct an asymmetry of the tongue or a restricted lateral mobility, the stimulator is adjusted to the left or right end of the dorsal edge. If both lateral movements need to be induced, two plates should be used, one with a right and one with a left button (Figure 9): Worn alternately daily, an hour or half hour at a time for four hours, not during sleep or feeding. This intermittent wearing prevents adaptation to the foreign body stimulus too quickly. This stimulatory reaction has been observed in newborns by J. Weiffenbach in California and is called the Weiffenbach reflex.[15]

To activate the upper-lip movement, the plate has deeply grooved lip stimulators or mobile beads mounted on a vestibular wire. The treatment results are shown in Figure 15.

Athetosis

Athetosis is characterized by a fluctuating muscle tone and an instability of head and body posture. The involuntary twisting limb and trunk movements are associated with permanent synkinesis of face, jaw, and tongue (Figure 10). This does not seem to stop involuntary "wave-like" movements of the tongue; in sleep, often superposed by a fine "worm-like" mobility of the tongue surface. The frequency with which orofacial dis-

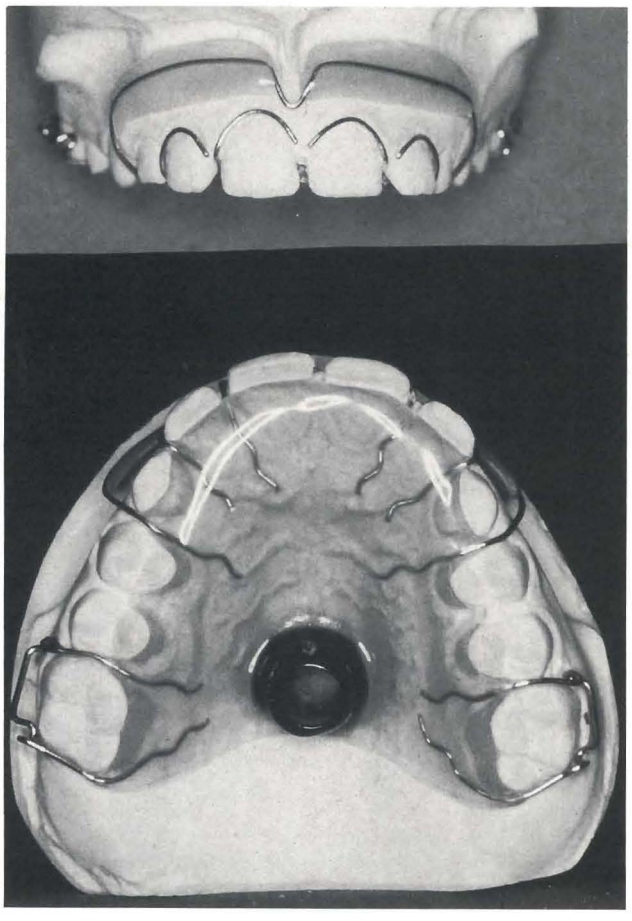


Figure 8. Palatal plate with a median button-like stimulator at the dorsal edge. The button has a central hole, to induce a sucking effect at the back of the tongue. The vestibular stimulation area is plain, in order not to exert too many stimuli simultaneously, because the child was hypersensitive.

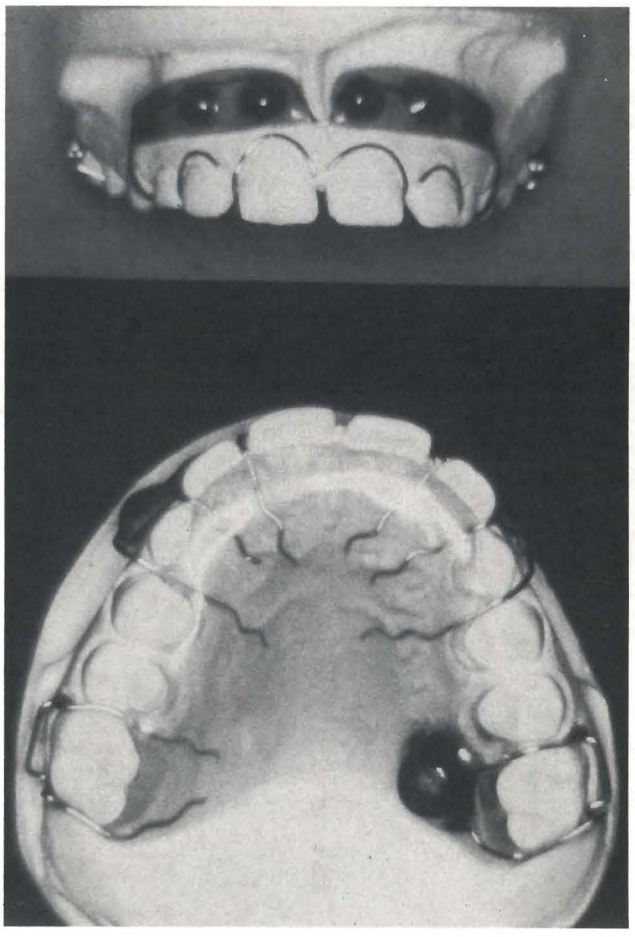


Figure 9. Palatal plate with a lateral button-like stimulator, also with a central hole. It is worn alternately with an identical plate with a button on the other side. The upper lip is stimulated in this case with bumpers built on the appliance.

Best results are achieved with a button-like stimulator.

orders appear in our athetotic patients is shown in Figure 16.

The special functional therapy requires a stabile posture that normalizes the muscle tone with adequate elements of inhibition and facilitation during the manual work in the orofacial complex.

Stimulation by plates must be timed carefully so as not to augment the dyskinetic movements. It tends to stabilize and orient the tongue: for example, with a small button and its central hole, or with a ridge at one side.

Mostly the stimulation must be confined to one element only, first tongue or first lip element. The treatment results are shown in Figure 17.

Hypotonia

The following symptoms of hypotonia are not only characteristic of a group of cerebral-palsy children, but also for most ataxias, some myopathies and congenital syndromes, like Moebius syndrome.

The hypotonic child shows a limp head reclination and trunk posture, and a limp facial expression, sad in appearance. The upper lip is often pulled up in a triangle shape; the lower lip is everted; and the corners of the mouth are pulled down. The tongue is bulky and protruded, often lying on the lower lip or beyond. In some cases, one or both sides of the tongue are atrophic (Figures 11,12). The frequency of the orofacial disorders in hypotonia is shown in Figure 16.

The Castillo-Morales' functional therapy uses intermittent traction and pressure to build up the muscle tone of the body and orofacial complex. At the same time, the tone is therapeutically normalized—in those parts that were tense—through compensation. This occurs, for example, in the muscles of the floor of the mouth. The hypotonic facial muscles are stimulated by

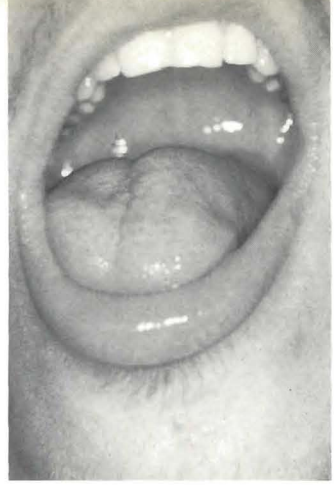


Figure 10. Fifteen-year-old-boy with a mixed athetosis. Together with the permanent involuntary trunk and limb movements, his face, jaw, and tongue movements are of a twisting nature.

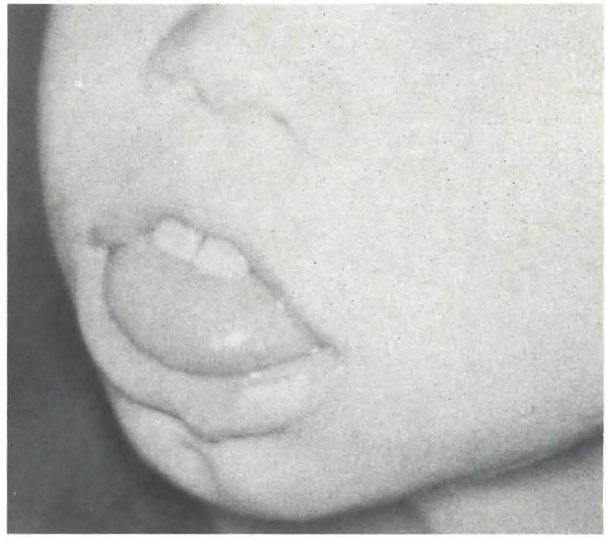


Figure 11. Nine-year-old-boy with a hypotonic cerebral palsy and a bulky, protruding tongue. He suffers from drooling, eating and drinking problems, and is severely mentally retarded.

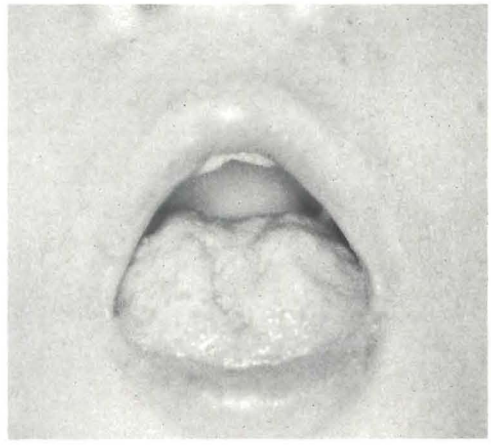


Figure 12. Ten-year-old-boy with Moebius syndrome (congenital aplasia of cranial nerves) and a lingual atrophy. After three years of treatment he could move the tongue and the mandible laterally and could chew and speak intelligibly.

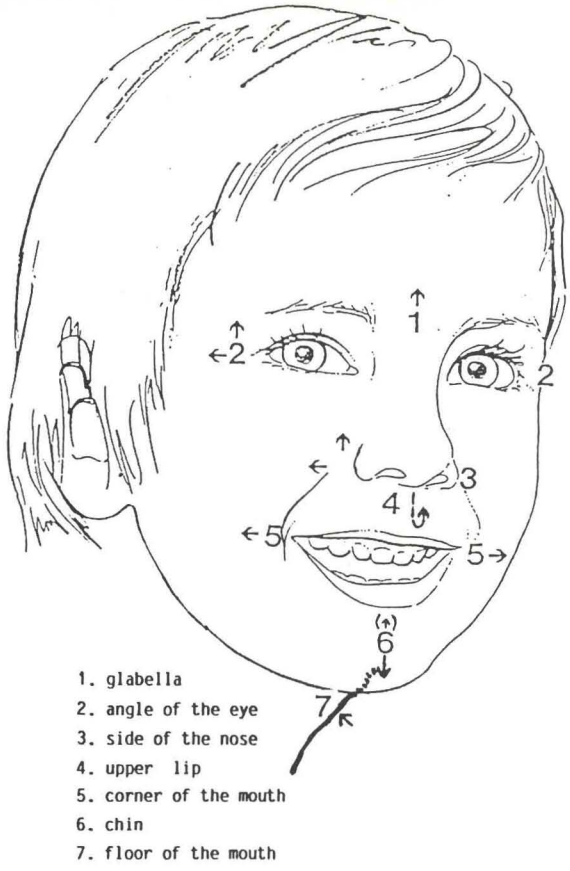


Figure 13. Facial motor points of Castillo-Morales neuro-developmental therapy.

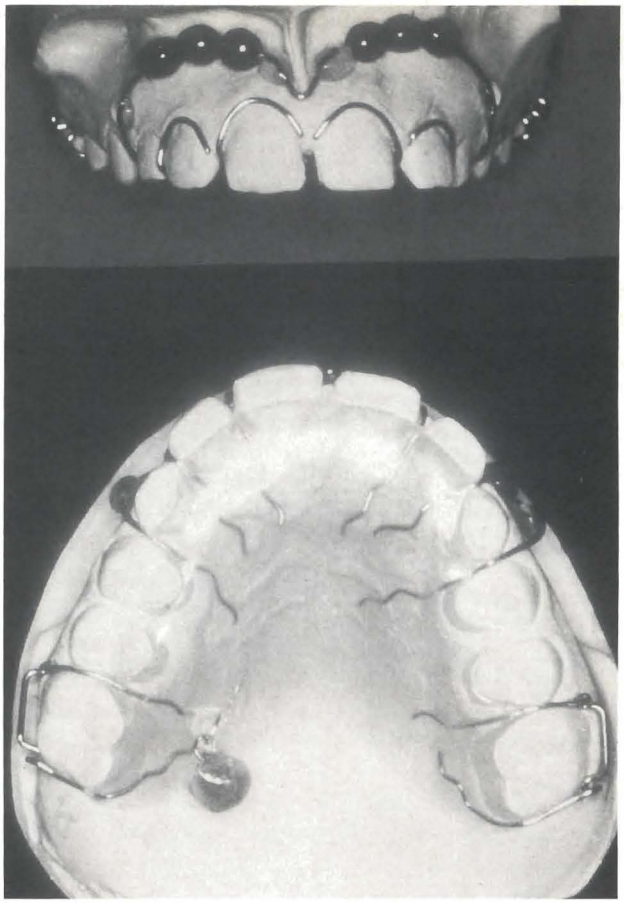


Figure 14. Palatal plate with a lateral pendulum stimulator to train the tongue to move laterally, and vestibular mobile beads on a labial wire. Both are strong stimuli for hyposensitive children.

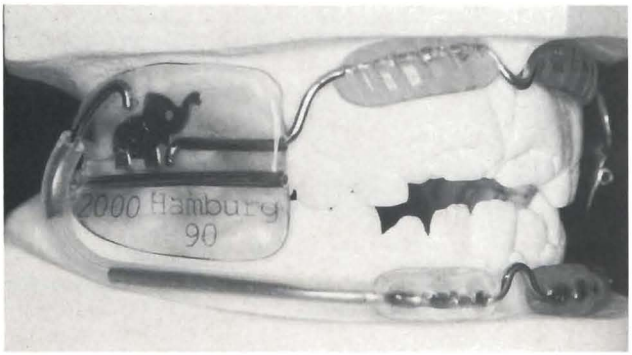


Figure 15. Mouth vestibular brace (MVS), a newly shaped vestibular dental brace. It is a strong stimulus for both lips, shown here with grooves for the upper lip and bumpers for the lower lip. It moves with the speaking movements of the jaw and can be worn for many hours daily, because foreign body adaptation is poor.

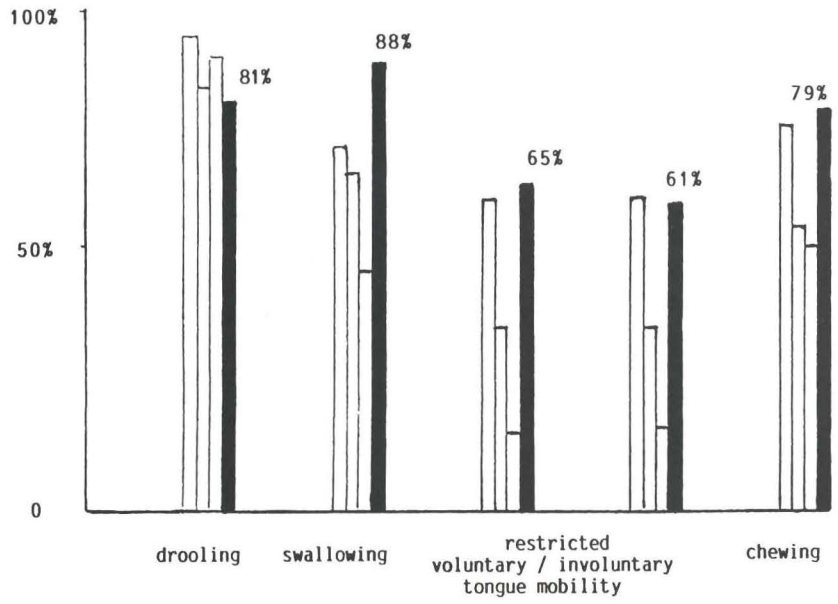


Figure 16. Frequency of orofacial dysfunctions in sixty-eight cerebral-palsy children with spastic tetraparesis/athetosis/hypotonia and the sum of all cerebral palsy groups (white column and percent).

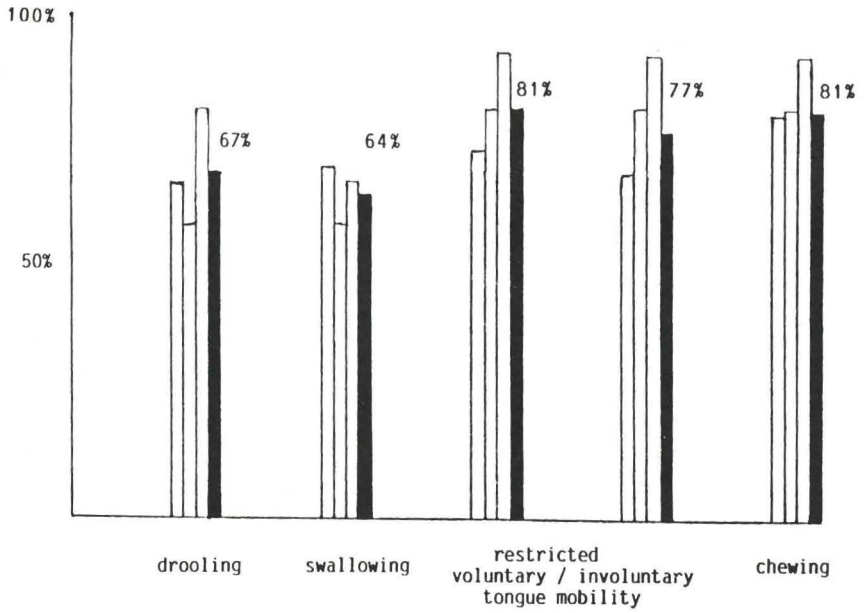


Figure 17. Improvements of orofacial dysfunctions in sixty- eight cerebral-palsy children with spastic tetraparesis/athetosis/hypotonia and the sum of all cerebral-palsy groups (white column and percent).

Castillo-Morales facial motor points, which are part of his neurodevelopmental therapy (Figure 13).[1]

The orthodontic devices must deliver a strong stimulus: for example, with broad oval buttons, broad lateral ridges, or finally with pendulums of different size, shape and material. For the upper lip we begin with bumpers and later, mobile beads on a vestibular wire may be used (Figure 14).

As lip function is difficult to influence in hypotonia, and the lateral tongue movement is not often disrupted, we developed a newly shaped, vestibular dental brace, called MVS (Mundvorhofspange or mouth vestibular brace) (Figure 15).[10] It consists of two vestibular supporting elements, lying in the buccal pouches, with fixed upper-lip stimulators and mobile lower-lip stimulators. Bumpers or mobile beads can be adapted to them. This mobile brace allows the patient to speak. The mobility prevents a quick adaptation to the stimulus, which easily occurs in hypotonic children.

A synopsis of the results in the three major types of cerebral palsy and the results of all sixty-eight patients are given in Figure 17. In case of severe drooling, the improvement rate was 72 percent.

A major limitation of the study was the lack of a control group. Parents would not present their children for control examination when no treatment was offered, and the great variety of individual cerebral-palsy and orofacial dysfunctions makes any comparison difficult.[5] The criteria used in the study, however, are reliable and clinical observations are reproducible.

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Case reports

Permanent molar impactions and an odontogenic keratocyst: report of case

Russ Leslie Kendell, BDSc

This report details a case of severe impaction of maxillary and mandibular permanent molars. An odontogenic keratocyst was also present in the lower left molar region.

REPORT OF CASE

The patient, a seventeen-year-old Caucasian male presented for routine dental examination. On oral examination there was a mesially impacted tooth in the region of the left mandibular second molar and the left maxillary second molar was not present in the mouth. The third molars were not evident and the first premolars had been extracted for orthodontic reasons.

A panoramic radiograph showed that it was the left mandibular third molar that was impacted under the distal of the first permanent molar and that the third molar was lying over the second molar (Figure 1,2). There was a 12 mm diameter radiolucent area mesial to the crown of the second molar and almost total root resorption of the first molar. There was also root resorption of the left mandibular second premolar. The left maxillary second molar was found to be impacted under the distal of the first permanent molar, the crown and root of which showed signs of resorption. In turn the third molar was impacted under the second. The right mandibular third molar was unerupted and mesially impacted. The right maxillary third molar was unerupted.

Dr. Kendell is with the Department of Dental Therapy, Curtin University of Technology, Bentley 6102, Western Australia.

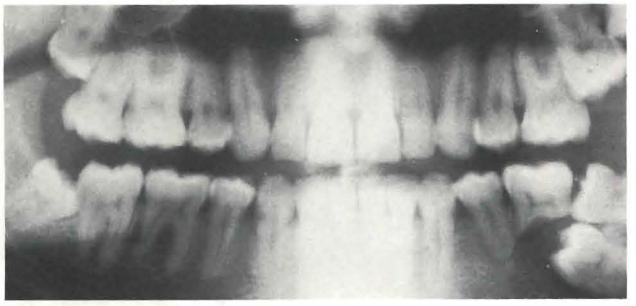

Figure 1. Panaramic film showing multiple impacted permanent molars.

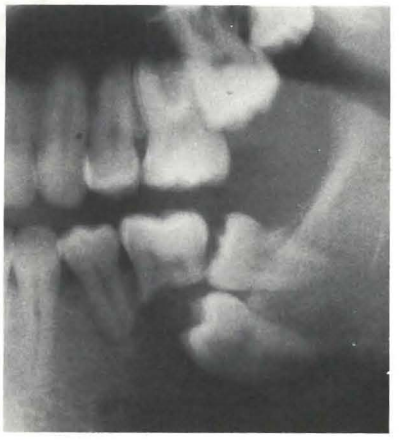

Figure 2. Panoramic film showing impacted left permanent molars, odontogenic keratocyst and root resorption of adjacent teeth.

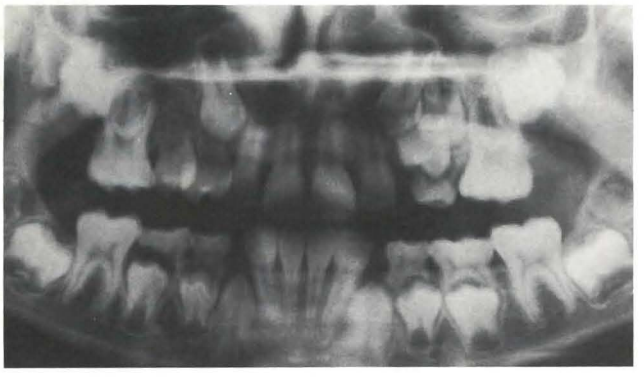

Figure 3. Panoramic film showing unerupted second permanent molars, and third permanent molars developing in positions mesial of the norm.

The patient was sent for oral surgery consultation and the following treatment subsequently performed. The four third molars and the second molars on the left were surgically extracted and the cystic lesion treated by enucleation. Histopathological assessment reported a diagnosis of an odontogenic keratocyst.

The vitality of the left maxillary first molar and the left mandibular second premolar and first permanent molar will be reviewed periodically and follow-up radiographs will be taken to check for recurrence of the cystic lesion.

A panoramic radiograph taken nine years previously, before orthodontic treatment, showed nothing abnormal in the positions of the second permanent molars (Figure 3). The positions of the developing third permanent molars appeared, however, to be mesial of the norm. Radiographic evidence of a cystic lesion was not apparent.

DISCUSSION

In this case the tendency for the third molars to develop in a more mesial position may have hindered the eruption of the left second permanent molars.

Huddy and Bimstein have detailed case reports of mandibular second permanent molar impaction.[1,2] Possible causes suggested are crowding and displaced tooth crypts.[1-3] Varpio and Wellfelt reported on the incidence of disturbed eruption of lower second molars and also noted crowding to be a frequent feature.[4] There was a significantly higher incidence in males and a higher incidence on the right side for unilateral impactions. The incidence of total impaction is estimated by Varpio and Wellfelt to be 0.7/1000.[4]

The odontogenic keratocyst is reported to be more common in males and in the second and third decades of life.[3] Occurrence in the mandible is more common than in the maxilla, and the first and second molar region is the most common location after the ramus-third molar area.[3]

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Management of cervical root perforation: report of a case

Karl-Johan Nordenvall, DDS, PhD
Karin Holm, DDS

Cervical root perforations and crown root fractures are serious dental conditions that are difficult to handle, if the tooth is to be retained. A surgical approach, such as intra-alveolar root transplantation, has been reported to be successful, provided the operating technique includes a minimum of trauma.[1-4]

Another approach is to effect orthodontic extrusion of the compromised tooth using an orthodontic technique. This is the preferred technique, if the pulp is vital and unexposed. Under these conditions, "slow" orthodontic extrusion over a period of three to six months is recommended. On the other hand, if endodontic treatment has been performed and the root canal walls are very thin (usually as the result of early loss of pulpal vitality), "rapid" orthodontic extrusion for three to six weeks might be the treatment of choice.[5-11]

The present case concerns a patient with a central incisor exhibiting iatrogenic injuries such as periapical infection due to unsuccessful endodontic treatment, weakened root due to extensive drilling during efforts to remove the root filling material, followed by cervical root perforation, and subsequent crown-root fracture.

CASE REPORT

The patient was a healthy Swedish boy sixteen years, two months old. He was referred to the pedodontic clinic in Södertälje by his school dentist, who unfortunately had perforated the root of the maxillary right central incisor. The radiograph (Figure 1) shows apical rarefaction and slight apical external inflammatory root resorption. Furthermore, the guide file pointed away from the root, indicating root perforation. Fortunately, the patient had no discomfort.

Status

Clinically, tooth mobility was slightly increased compared with the contralateral tooth. Percussion did not suggest any pathologic processes. Probing of the gingival pocket failed to reveal any communication between the perforation and the oral cavity, though there was some gingival retraction (Figure 2). Lateral x-ray examination established that the root perforation was located at the crest of the alveolar process on the buccal aspect of the tooth (Figure 3).

Diagnoses

The following main diagnoses were recorded:
Osteitis periapicalis resorptiva chronica, 522.61
Root perforation, 998.21

Therapy

After having consulted with members of the departments of Oral Surgery and Orthodontics, the following treatment plan was set up.

Dr. Nordenvall is Head of Pedodontic Department, Folktandvården, Fjärilen, Södertälje, Sweden. Dr. Holm is with the Orthodontic Department of the same institution.

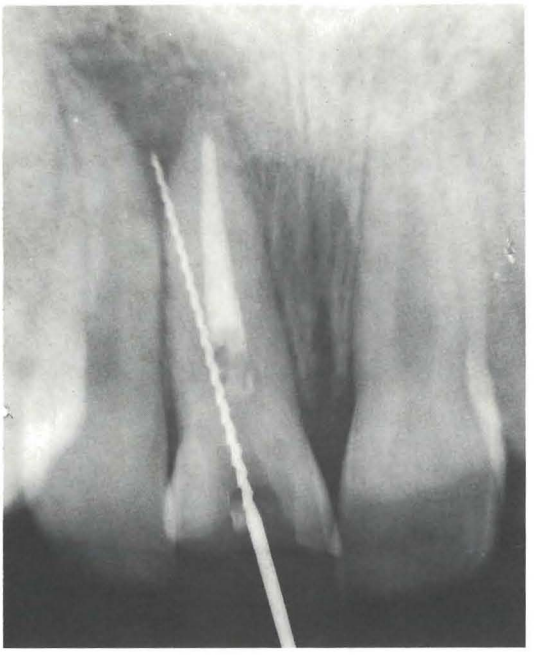

Figure 1. The right central incisor exhibits periapical bone destruction and slight inflammatory external root resorption. The file indicates root perforation during efforts to remove the gutta-percha filling material.

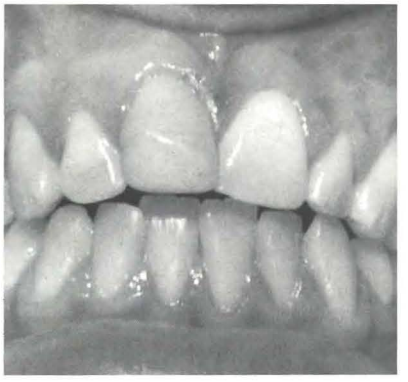

Figure 2. The clinical situation at the start of treatment. The right central incisor exhibits some gingival retraction.

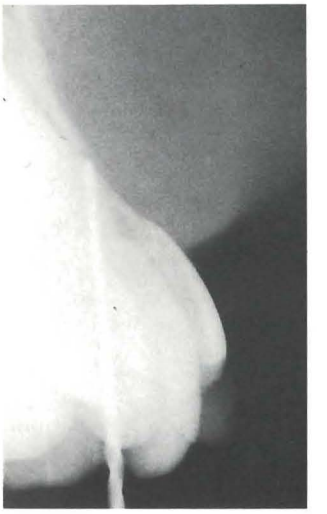

Figure 3. The site of the root perforation is localized in a lateral x-ray examination.

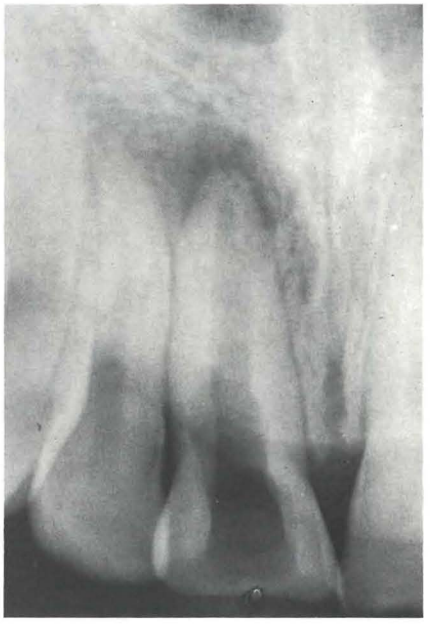

Figure 4. The situation after removal of the root filling material and before application of Calasept pulpal dressing.

- ☐ Removal of the gutta-percha root filling.
- ☐ Medicinal treatment of the root canal and the root perforation, using calcium hydroxide.
- ☐ Following hard tissue formation in the apical region and evident healing of the periapical tissue, obturation of the root canal including the perforation site, using gutta-percha technique.
- ☐ Clinical and radiographic follow-up.
- ☐ Further consideration of "rapid" orthodontic extrusion followed by prosthetic therapy.

The pedodontist (K-J.N.) started treatment according to the treatment plan. When the gutta-percha filling material was detected lingual to the perforation, poor adaptation to the walls made it easy to remove (Figure 4). Infection was probably present in a space between the root canal walls and the filling material. The apical foramen was found to be wide open. Calasept† was densely packed in the root canal and the pulp chamber. The access cavity was sealed off with IRM‡. The pulpal

†Scania Dental AB, Box 5 S-741 00 Knivsta, Sweden.
‡L.D. Caulk, Co., P.O. Box 359, Milford, DE, U.S.A.

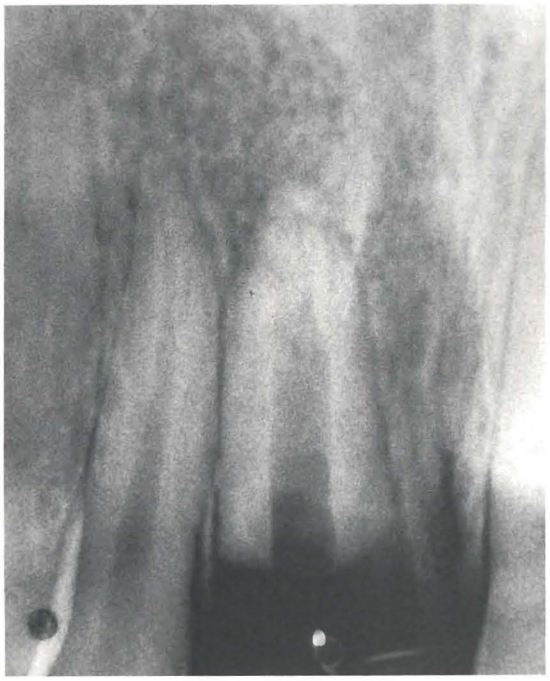

Figure 5. The tooth has been exposed to trauma. A horizontal crown-root fracture at the level of the root perforation is evident.

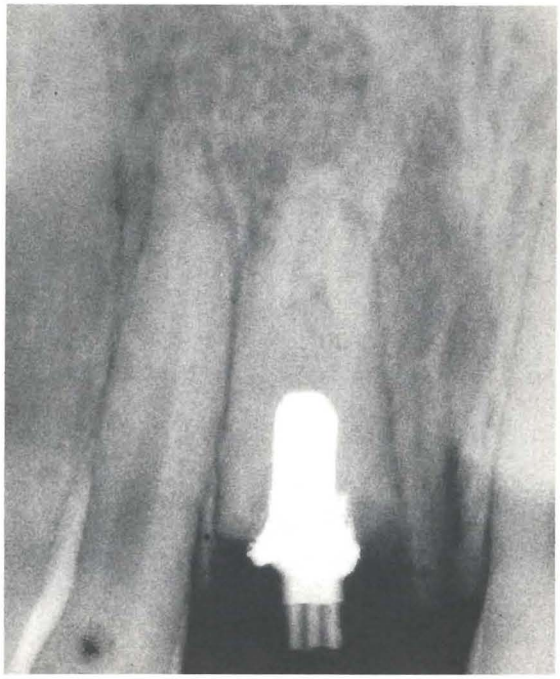

Figure 6. The situation after installation of a screw post, using Ketac-S as luting cement. Calasept is used as pulpal dressing.

dressing was changed after three months. After another four months, the radiograph showed signs of healing occurring. Clinically, hard tissue formation was found in the apical region and at the perforation. The operator planned to obturate the root canal after another three months, but when only one month had passed, the tooth was fractured by a relatively mild traumatic experience. The tooth fractured horizontally, the fracture running through the perforation (Figure 5). In agreement with the orthodontist (K.H.) , "rapid" orthodontic extrusion was chosen, although final endodontic treatment had to be postponed for technical reasons. After irrigation of the reinfected root canal and application of Calasept, a short screw post was cemented with Ketac-S* (Figure 6). A fixed orthodontic appliance with a long flexible "rod" was constructed (K.H.). The "rod" was ligated to the screw post. In this way the root was submitted to a uniform axial extrusive force.

The patient was seen once a week. After 3.5 weeks, the radiograph showed that the root had been extruded 4-5 mm (Figure 7). This was considered sufficient and the "rod" was inactivated. After two weeks a gingivectomy was performed on the tooth and root canal treat-

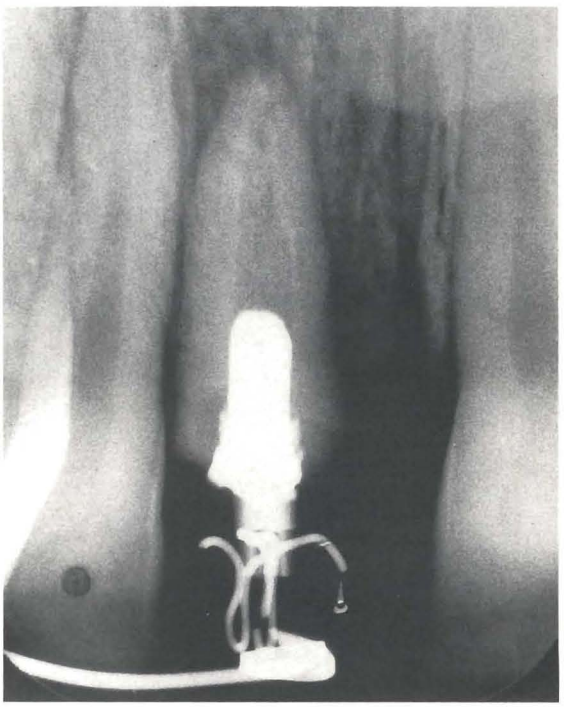

Figure 7. The situation after "rapid" orthodontic extrusion for 3.5 weeks. The root has been extruded 4-5 mm compared with Figure 6. Note the "rod".

*ESPE D-8031 Seefeld/Oberbay, West Germany

Teamwork
in action.

ment continued. When the pulpal dressing was changed once again, the root canal was filled with gutta-percha. The root was prepared for a cast, enveloping gold core to prevent further fracturing (Figure 8). Using a conventional technique, a core with an individual temporary acrylic crown was made (Figures 9,10). An acrylic crown was made, because the authors expected the need for some repetition of the orthodontic treatment. Recurrence was not evident, however, at the one-year control examination and is not likely to happen.

A uniform periodontal ligament is visible and exists around the entire root. The periapical destruction has healed (Figure 11). The tooth functions well, and clinically there is no evidence of replacement resorption or any other pathologic condition (Figure 12).

DISCUSSION

The planning and treatment of this patient are a good example of teamwork between different dental specialties. In this particular case, orthodontic extrusion was preferred to intra-alveolar root transplantation, be-

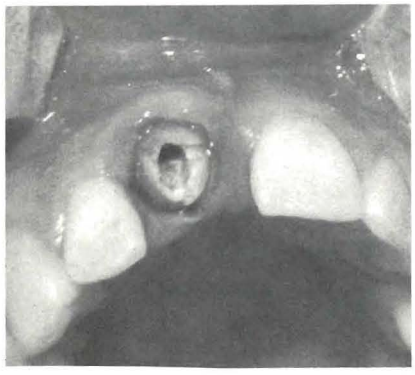

Figure 8. The clinical situation before cementing of a cast, enveloping gold core combined with an acrylic crown. A gingivectomy was done at the time of preparation.

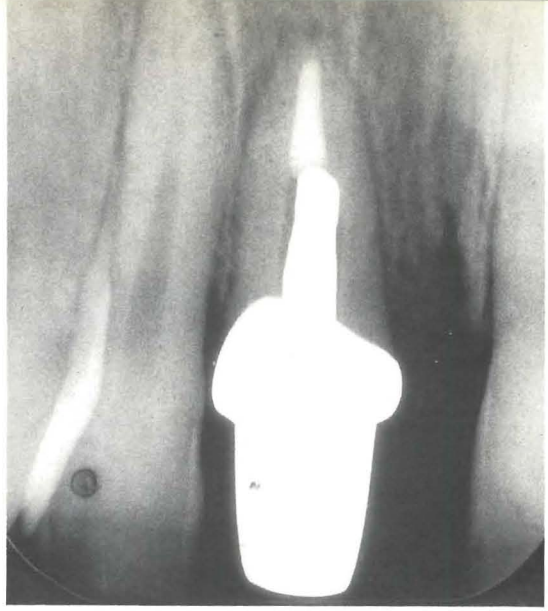

Figure 9. The radiographic situation at cementation of the restoration.

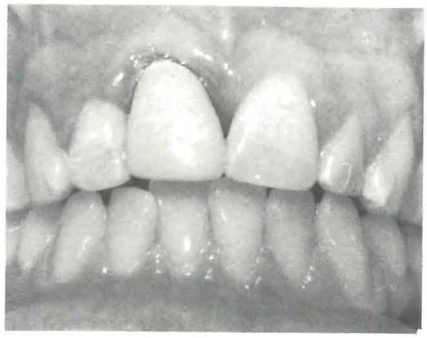

Figure 10. The clinical situation at cementation of the restoration.

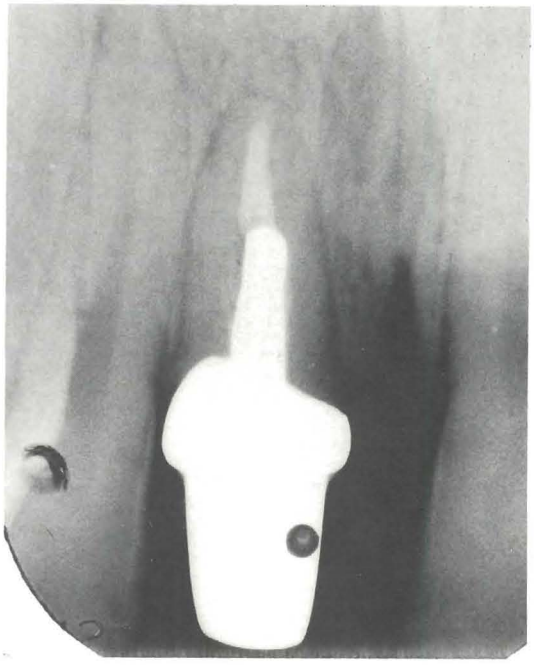

Figure 11. The one-year control. Periapical healing is completed. The periodontal ligament was intact around the entire root.

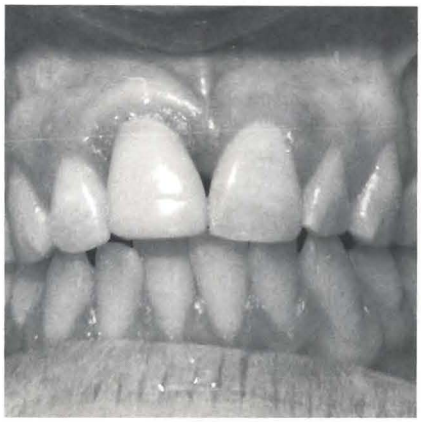

Figure 12. The one-year control. Clinically there is no sign of recurrence after the "rapid" orthodontic extrusion. The incisal edges of the centrals are in harmony. The gingival situation is acceptable.

cause the root was judged to be too fragile for a surgical approach. The tooth did in fact fracture, due to minimal trauma, even before the completion of the endodontic treatment. Fortunately the fracture was of the horizontal type, running at the level of the root perforation. With a surgical approach it is possible to rotate a root 180° when a fracture runs obliquely up under the bone margin on the palatal side. Thus a more suitable situation could be created from a prosthetic point of view. Another advantage of intra-alveolar transplantation is that fractures and cracks may be revealed that are not accessible with conventional x-ray examination. "Rapid" rather than "slow" orthodontic extrusion was chosen because the tooth was pulpless and also straight and slightly conical. It was postulated that application of an axial extrusive force would result in traction forces to the root cementum and surrounding bone. The risk of replacement resorption, due to pressure damage to the root cementum, was judged to be small. The endodontic treatment followed modern concepts concerning the handling of young traumatized, nonvital permanent teeth.[12]

The prosthesis allowed for compensation of the reduction in the marginal circumference of the root following extrusion. For this reason the crown converged more than usual in a cervical direction.

In the original treatment plan, endodontic treatment was followed by a period of observation before the final extrusion of the root and subsequent prosthetic therapy. Due to fracturing of the root, the plan had to be altered. It seems hazardous not to evaluate the result of endodontic treatment before obstruction of the root canal with a luted enveloping gold core. On the other hand, one has to consider the consequences of leaving the root unrestored for some time: difficulties in preparing and taking an impression of the root, because of possible apical shifting of the root and gingival overgrowth; the risk of microbial leakage; and further fracturing of the root, when using an ill-fitting crown and post. We decided, therefore, to complete prosthetic treatment immediately after finishing the endodontic procedures.

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Regional odontodysplasia: report of case

Durl W. O'Neil, DDS
Mark G. Koch, DDS
James W. Lowe, DDS, MS

Regional odontodysplasia, otherwise known by its synonyms, odontodysplasia, odontogenic dysplasia, odontogenic imperfecta, or ghost teeth is characterized by a localized arrest of tooth development. It may involve an isolated tooth or several adjacent teeth of the same quadrant. These teeth have an altered shape that ranges from irregular to peg-shaped in appearance. Pulp chambers are large with thin shells of enamel and dentin. Radiographically, they have decreased radiodensity, thus attributing to their ghost-like appearance. These teeth may exhibit a delayed eruption or total lack of eruption.[1]

Although both arches may be affected, the maxillary arch is involved twice as frequently as the mandibular arch.[2] In the maxilla, and mandible, the central incisors, lateral incisors, and canines are usually involved; and primary and permanent dentitions may be involved. When the primary teeth are dysplastic, the permanent dentition is usually affected also.

Etiology of this condition is unknown, and apparently trauma or systemic conditions may or may not be contributory.[3,4] Odontodysplasia seems to occur early during odontomorphogenesis.[5]

Dr. O'Neil is Associate Professor; Dr. Koch is Senior Resident; and Dr. Lowe is Professor and Chairman, Department of Pediatric Dentistry, University of Missouri-Kansas City.

LITERATURE REVIEW

This anomaly was first described and recognized in 1947.[6] The term odontodysplasia was first used by Zegarelli to define this rarely observed developmental disturbance of odontomorphogenesis.[7] Most cases of this defect involve only one quadrant of the dental arch. Except in cases of trauma, all quadrants should exhibit this malformation, if the etiological factor is of genetic influence or systemic imbalance. The first case involving all quadrants was reported by Herman. Any of the proposed etiological theories could explain this case.[8] After examining all of the evidence, the etiology must still be considered unknown.

CASE REPORT

A nine-year-old white female presented self-referred to the Dental Clinic at Children's Mercy Hospital in Kansas City, Missouri. Her chief complaint was missing teeth in the right maxillary quadrant. Examination verified her complaint and showed all teeth missing, from the quadrant identified by the patient (Figures 1,2). She was missing her permanent central and lateral incisors, primary canine, first and second primary molars, and permanent molar. A panoramic radiograph was obtained, and several "ghost teeth" were observed in this quadrant (Figure 3).

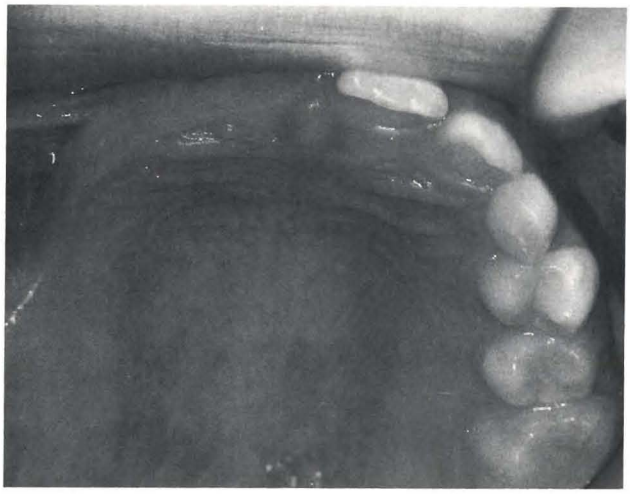

Figure 1. Clinical examination.

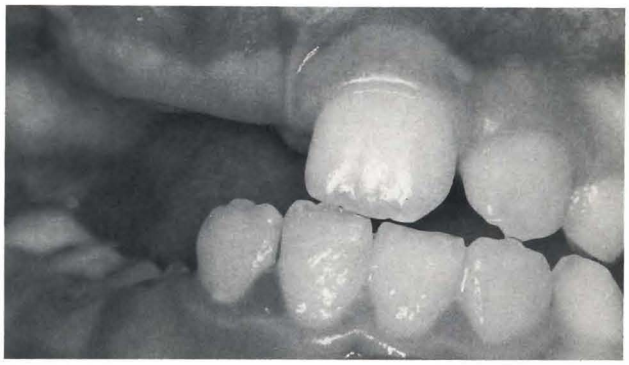

Figure 2. Clinical examination.

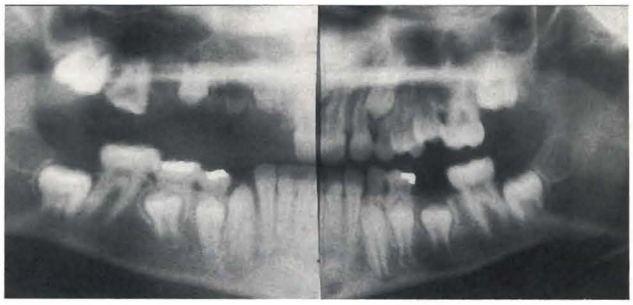

Figure 3. Panoramic radiograph.

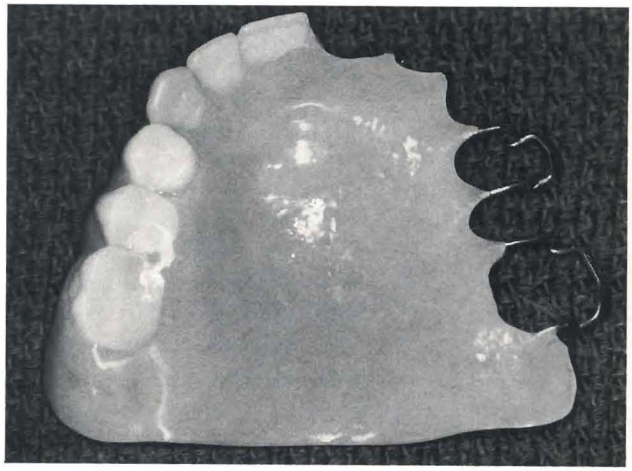

Figure 4. Partial denture.

The "ghost teeth" were located in the positions of the permanent incisors, canines, and first premolars. The crown of the second premolar was present, as were the first and second permanent molars. The incisors, canines, and first premolar had a diminished radiodensity with large pulp chambers and thin shells of enamel and dentin. Crowns of the second premolar and second molar appeared normal, while the first molar had thin, hypoplastic enamel. None of these teeth seemed to be making progress toward eruption.

The medical history was noncontributory with no family history of missing or hypoplastic teeth. No history of trauma or radiation therapy to the head or neck was noted. The affected side revealed a decreased vertical dimension.

Treatment for this patient consisted of fabricating a temporary acrylic maxillary partial denture replacing the "ghost teeth" with acrylic teeth to provide function and to increase vertical dimension (Figures 4, 5). The patient was placed on periodic recall to observe progressive eruptive changes of the "ghost teeth" and to monitor the growth and development of the maxillary and mandibular dental arches. In the future, a decision must be made on the final disposition of these "ghost teeth" to avoid the sequela of retained epithelial tissue.

DISCUSSION

This case demonstrates some of the bizarre and rarely observed disturbances of odontogenesis that can go unrecognized or undiagnosed for years. This patient will need to be closely observed and her growth and development monitored until a permanent prosthesis can be constructed to correct this defect.

Ghost teeth

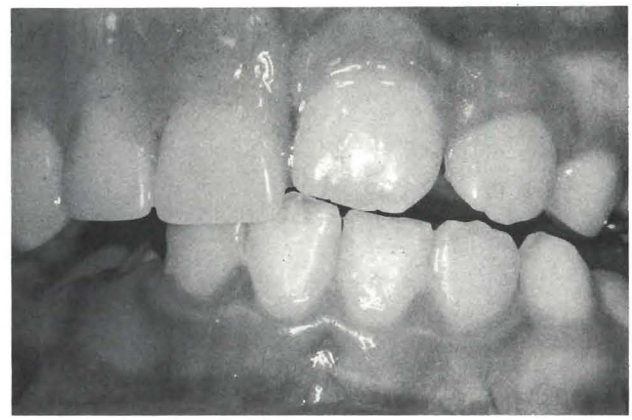

Figure 5. Partial denture.

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TOBACCO SMOKE EXPOSURE DURING INFANCY

Breast feeding has already been reported to influence infant urine cotinine levels among mothers who smoke, and the present study provides further documentation of that effect. Breast milk from mothers who smoke contains high levels of cotinine, and it is likely that ingestion and absorption of cotinine (and also nicotine) explains why breast fed infants of smoking mothers have higher urine cotinine levels than when the mother does not breast feed. Cotinine from this source carries different implications from cotinine produced from inhaled tobacco smoke and needs to be taken into account when using cotinine results to estimate ETS absorption. In the present study household ETS was not reflected in raised infant urine cotinine levels via ingestion in breast fed infants, when the mother did not smoke.

Chilmonczyk, B.A. *et al:* Environmental Tobacco smoke exposure during infancy. J Pub Health, 80:1205–1208, October, 1990.

Scleroderma in pediatric patients

Ruth A. Anderson, DDS
Tamara Ewell-Jackson, DDS

Scleroderma is a connective tissue disease of unknown etiology; no significant hereditary factors have been noted. The primary feature of the disease is excessive deposits of collagen and other connective tissue components in skin and internal organs.[1] Its development in children is often unrecognized because it is so rare. It may have a sudden onset and progress rapidly; the usual clinical picture, however, is a long chronic illness. Systemic sclerosis has been included in the group of autoimmune diseases. Evidence from recent studies indicated a marked increase in the rate of collagen synthesis in numerous organ systems of the body. This led Goetz in 1945 to coin the term "progressive systemic sclerosis" (scleroderma), resulting from a somatic mutation of sclerodermatous fibroblasts.[2] These immunologic factors can be implicated because lymphocytes seem capable of regulating connective tissue synthesis from fibroblasts.[3] Scleroderma is relatively uncommon; four to twelve new cases per one million population are diagnosed in the United States each year. Females are affected three to four times more frequently than males, and there is no racial predilection.[4] The classic description of the disorder usually appears in the third to fifth decade of life. The authors of these case studies have found the signs of the disease to appear in early childhood in some children.

In approximately 30 percent of the cases, skin ulcerations with subsequent atrophy can be seen, usually localized in areas of pressure, such as the fingertips. The second most frequent manifestation of this disorder, present in more than 85 percent of the patients, is Raynaud's phenomenon. Initial vasoconstriction and blanching are followed by a "cyanosis," resulting in ischemic necrosis of extremities and/or internal organs. Scleroderma of the face is observed in approximately 80 percent of patients suffering from generalized scleroderma.[5]

A form of systemic sclerosis that tends to pursue a slow progressive course is the CREST syndrome.[6] This syndrome is an acronym of the five major findings: Calcinosis cutis, Raynaud's phenomenon, Esophageal dysfunction, Sclerodactyly and Telangiectasia. Juvenile onset of CREST syndrome is extremely rare. When found, it is much more common, however, in females. The CREST syndrome can be separated serologically from other forms of scleroderma by its frequent association with anticentromere antibodies. The significance of the centromere antibody is unknown. Possibly its an epiphenomenon, but probably more closely related to disease pathogenesis, implying disease activity. These autoantibodies are recognized to be a prominent feature of Progressive Systemic Scleroderma (PSS). In 97 percent of the sera of these patients, antibodies for either anticentromere or anti-Sci-70 were present. The anticentromere antibody is as-

Dr. Anderson is Associate Professor, Pediatric Dentistry Department, Howard University, College of Dentistry, Washington, D.C. 20059. Dr. Jackson is Clinical Instructor, Oral Medicine Department, Temple University, College of Dentistry, Philadelphia, PA 19140.

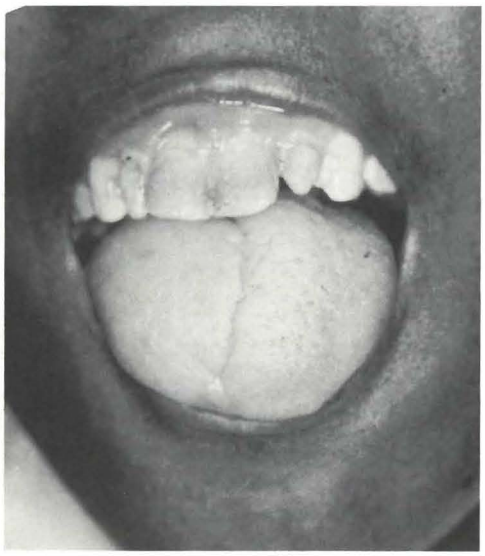

Figure 1. Hemiatrophy of the tongue musculature.

sociated with a longer duration of disease, a significant absence of diffuse skin involvement and decreased arthritic and pulmonary disease symptoms. On the other hand, anti-Sci-70 is associated with a significant pulmonary involvement.[7] Further characterization of these antigens is necessary to show whether they are due to a single immunological system. Cardiopulmonary findings are frequent in young people with progressive systemic sclerosis.[8] There have been only occasional reports on small numbers of children with this disorder. In most cases, initial manifestations were Raynaud's phenomenon, cutaneous induration of the hands and discolored skin patches.

Some authors reported finding that the average course of the disorder in the children in their studies was six to ten years. The immediate cause of death was congestive heart failure and acute pulmonary edema.[9,10] It has been concluded that survivorship in children with scleroderma was poor, but difficult to determine accurately because of the small number of cases reported. The poorest survival has been seen in older patients. Males have an even poorer prognosis than females. It has been found on occasion to improve spontaneously in children as well as in adults.[11,12] Even though prognosis is difficult to recognize early in the course of the illness, certain characteristics in children are probably indicative, however, of a poor prognosis such as the presence of Raynaud's phenomenon, antinuclear factor, LE cells and a raised estimated sedimentation rate.

Work was done in the departments of Oral Pathology and Oral Medicine, Temple University, College of Dentistry.

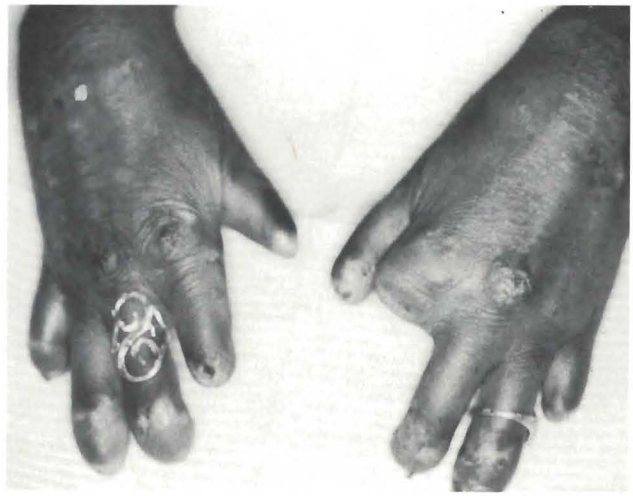

Figure 2. The forefinger of the left hand amputated due to gangrene; fingertips on the other fingers have all atrophied.

CASE REPORT 1

A twenty-year-old black female presented herself to the dental emergency room at Temple University, College of Dentistry, with the chief complaint of pain in the right mandible. Her medical history showed that a diagnosis of systemic scleroderma was made at the age of six years. The follow-up medical treatment is sketchy, because the patient relocated several times and was treated at numerous medical facilities. The patient states her paternal grandmother also has an advanced phase of the same disorder. The clinical appearance of this patient is one of an ongoing chronic illness. She walks with a gait due to amputation of her left leg in 1984 and the use of a prosthesis. Her facial skin is taut and shiny with oral constrictions, both vertically and horizontally. Her ear lobes are atrophied; there is some atrophy of the tongue musculature with an obvious hemiatrophy (Figure 1). The patient's hands are the most obvious sign, with the forefinger of her left hand amputated due to gangrene. The fingertips on the other fingers have all atrophied (Figure 2).

Systemically, the patient has a heart murmur and suffers with bouts of stomach tightness due to bowel obstruction. All other systems seem to be functioning adequately at this point. At the present time, she is not taking any medications. Because of the heart murmur, premedication with the use of antibiotics is indicated, however, before instituting any type of dental treatment.

CASE REPORT 2

This twenty-one-year-old black female presented herself to the dental hygiene clinic at Temple University,

Table ☐ Comparison of stages of two patients with scleroderma.

	Case report #1	Case report #2
Age disease first diagnosed	Four years	Sixteen years
Initial characteristic symptoms	Discolored spots on stomach Discolored spots on commissure of mouth Raynaud's syndrome in fingers	Discolored spots at commissure of mouth Indurations on dorsal surface of hand (Figure 4) Raynaud's syndrome in fingers and hands (1984)
Present clinical features of illness	Taut and shiny skin on face Stretch skin across malar prominence Tight skin at oral aperture Atrophied finger tips (Figure 2) Hemiatrophied tongue (Figure 1)	Rash across malar prominence Small ulcer on left middle finger Loss of elasticity at fissure of mouth; problem with opening mouth fully (4cm) Occasional rigidity on lateral surfaces of tongue (Figure 5)
Systemic symptoms	Heart murmur Bowel obstruction Restricted opening of oral cavity Gangrene set in left leg (amputated in 1984) Muscle rigidity in left eyelid (treated in 1984) Gangrene set in in left forefinger (amputated in 1983)	Heart murmur Infrequent esophageal spasms
Present age	Twenty years old	Twenty-one years old
Dental features (x-ray)	Xerostomia Gingival oozing Some bone loss in molar area	Over-retained primary molar Moderate bone loss in molar area
Family history	Paternal grandmother	Twin sister

College of Dentistry, to have her teeth cleaned. She stated she was being treated for scleroderma, which was diagnosed at age sixteen. The patient is a paternal twin, and her twin sister was diagnosed as having scleroderma at the age of six. Descriptive evidence indicated the twin sister has probably progressed to the most severe stage of this illness. Clinically, the patient in this case is still in the first stage of the disorder. Facially, her skin has shown no evidence of fibrosis appearing in the muscles. There is a light reddish rash that is limited to the cheek bone and nasal bridge area (Figure 3). Some lighter pigmentation is noticed at the commissures of her mouth (arrow). There are also signs of dryness of the skin and loss of skin folds around the orifice.

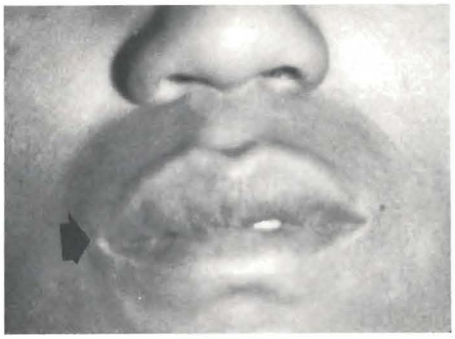

Figure 3. Rash that is limited to the cheek bone and nasal bridge area. Discolored pigmentation is noticed at the commissures of her mouth.

DISCUSSION

Systemic scleroderma presents several problems unique to dentistry (Table). First, since the oral aperture may be narrowed and the face rigid, access to the teeth and periodontium may be impaired as in Case 1. The scope of treatment is limited in this particular case, because of narrowing of the oral aperture; access for extraction of the impacted third molar was difficult. Restriction of mandibular movement occurs, when the muscles of mastication are affected by the disease and become rigid. Other manifestations of disease in the head and neck area include dysphagia because of esophageal involvement and a rigidity of the tongue (Figure 5). A non-acidic diet is recommended for these patients, as less painful to the cracked and dry lips.

Since these patients frequently suffer sclerotic "claw-

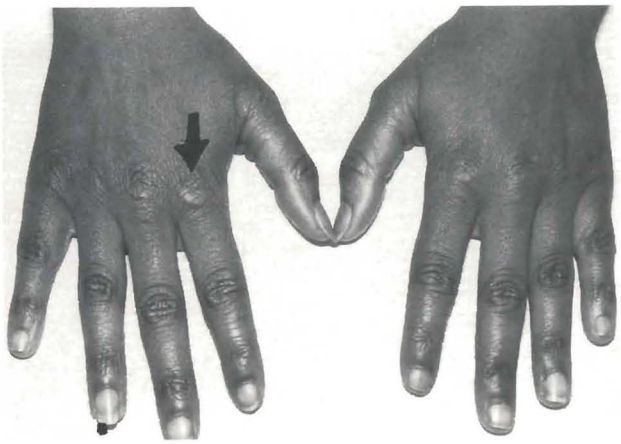

Figure 4. Indurations on dorsal surface of hand.

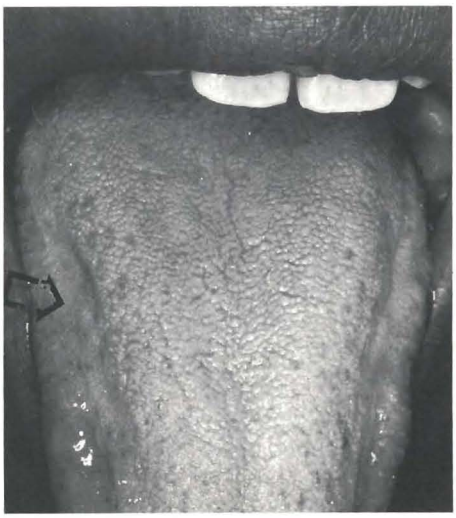

Figure 5. Occasional rigidity on lateral surfaces of tongue.

Improvement is on occasion spontaneous.

ing" of the hands or sclerodactyly (Figure 2), their manual dexterity is decreased. Toothbrushing and flossing, therefore, may be difficult or impossible. They should be placed on a preventive prophylaxis schedule, wherein they will have their teeth cleaned every three months professionally.

Dental radiographs demonstrate that there is a thickening of the periodontal membrane space, especially around the posterior teeth. In each case reported, there is some bone loss in the posterior area as well as a widening of the periodontal membrane space (Figures 6,7). Microscopically, this widening of the periodontal ligament is due to an increase of collagen and oxytalan fibers as well as an appearance of hyalinization and sclerosis of collagen with a decrease in the number of connective tissue cells.[13] Bone resorption of the angle of the mandibular ramus, bilaterally, is a frequent occurrence in this disease. This is probably a contributing factor in Case 1, who presented with the chief complaint of "pain in the lower left jaw" (impacted 3rd molar). The xerostomia can be attributed to a generalized fibrosis of the submaxillary glands and a thickening and hyalinization of the collagen fibers in the mucous membrane.

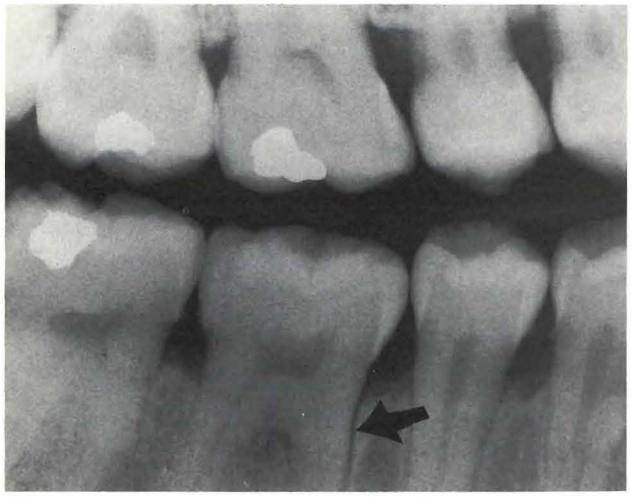

Figure 7. Progressive bone loss with widening of the periodontal membrane space (arrows) in patient 1.

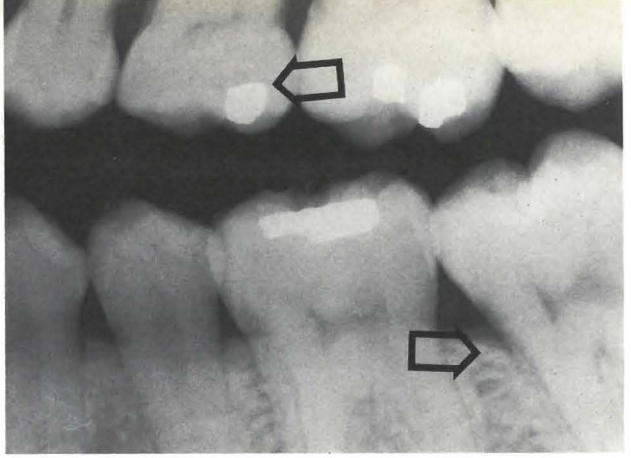

Figure 6. Moderate bone loss in molar area and over-retained primary molar (arrows) in patient 2.

Psychologically, both patients appeared to be well adjusted, amiable individuals. Even though Case 1 is in the severe stage of the disorder, her spirit is high and her outlook on life does not correspond with the horrifying solidification of her body tissues.

There is no adequate treatment for progressive systemic sclerosis, although partial remissions have been reported following cortisone therapy. Localized scleroderma (skin only) has an excellent prognosis, since spontaneous remission has been known to occur.

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Dentistry for the at-risk patient—mucopolysaccharidosis III (Sanfilippo syndrome): A nine-year case study

Burton L. Nussbaum, DDS

The identity of mucopolysaccharidosis (MPS) III was established in 1958 by Meyer and Grumbach and later by Meyer and Hoffman, Harris and Sanfilippo *et al.* Severe mental retardation, neurologic degeneration, and a Hurler' like clinical appearance characterize the condition. Pindborg and Gorlin feel that this is the most frequent type of mucopolysaccharidosis.[1,2]

"MPS III is caused by defective degradation and subsequent storage of heparin sulfate. Among the enzymes involved in the degradation of heparin sulfate are a specific heparin sulfate-N-sulfatase and a 2-glucosaminidase. Either enzyme has been shown to be involved in the pathogenesis of MPS III; in MPS IIIA, there is deficient activity of N acetyl-a-D-glucosaminidase. The two genetically different forms of MPS III cannot be differentiated clinically. They are the result of nonallelic mutations of genes whose defect becomes phenotypically manifest in the homozygous state. Sanfilippo syndrome has been diagnosed in utero."[1]

MPS III is autosomal recessive. It is thought to occur more often than the estimated 1 in 100,000 to 200,000 people. It has been reported in American Blacks.[2,3]

Facial features of these patients are very coarse. They can have a depressed nasal bridge, wide nails, heavy eyebrows and eyelashes, and thick lips. They also exhibit a mouth that is frequently held open accompanied by noisy mouthbreathing and common hearing loss; there are no corneal opacities.[2,3]

These patients have average height until ten to twelve years of age, and their growth slows. Sanfilippo patients usually have short necks. The severe mental retardation is not always apparent in early childhood. Psychomotor development lowers or plateaus from ages one to three, especially in the area of speech. Patients are easily agitated and can sometimes become aggressive. Some patients show stiffness of joints with a claw hand deformity. Hepatosplenomegaly is only slight or moderate. These people commonly have excessive body hair and occasionally have inguinal or umbilical hernias.[3]

The clinical features are present in infancy, but they are not pronounced until after one year. It is possible to perform prenatal diagnosis of MPS III. Radiographs of ribs, hands, skull and spine suggest thick and dense calvaria with poor pneumatization. ovoid thoracic vertebral bodies, flaring of the iliac wings, hypoplasia of lateral portions of acetabula, and finally, undermodeling of metacarpals. The patients excrete heparin sulfate excessively. Finally, peripheral lymphocytes, cultured fibroblasts, bone marrow cells and also cells of spinal fluid usually contain tachromatic granules..[2,3]

Webman, Hirsch, *et al* reported a case of Sanfilippo syndrome with obliterated pulp chambers and canals. The child was five years old. The authors felt that the heparin sulfate caused the obliteration.[4] According to other authors, there seem to be no remarkable oral

Dr. Nussbaum is in the private practice of Pediatric Dentistry. He is a Clinical Assistant Professor of Pediatrics at the University of Medicine and Dentistry of New Jersey, School of Osteopathic Medicine, Stratford, New Jersey. He is also an instructor in Otolaryngology at the Thomas Jefferson University School of Medicine. Dr. Nussbaum is an attending Pediatric Dentist at the University Medical Center - John F. Kennedy Memorial Hospitals, Stratford, New Jersey. He is also an attending at Thomas Jefferson University Hospital and The Children's Hospital of Philadelphia.

Patients with this syndrome are easily agitated.

manifestations. Tooth abscesses are a major concern in the final stage.[1,2]

Prognosis and treatment of these people are not favorable. Rarely do they survive to the third decade. Management is difficult due to their retardation and marked strength. Behavioral problems such as restlessness, aggressiveness, diminished attention span, and sleep disturbances become visible from the 2nd to the 5th year, and many times are the reason the parent seeks medical care. There is progressive loss of environmental contact and motor skills. Eventually this leads to a vegetative state with spastic diplegia. Supportive care is done through the use of tranquilizers.[2]

CASE REPORT

John was examined March 22, 1977 in the dental office. He was a six-year-old white male with diagnosed Sanfilippo syndrome. The health history stated that he could ambulate with difficulty. His coordination was "not perfect", and he had some emotional problems. He took no medications. A clinical examination revealed a depressed nasal bridge, a wide forehead, thick eyebrows, thick eyelashes, and thick pouting lips. The behavior was aggressive and poor. John had one carious lesion and needed an oral prophylaxis. His physician was called and the treatment plan was discussed. Because of John's poor behavior, it was felt that he would be best treated under sedation. The physician informed the office that John would be best treated with secobarbital sodium (Seconal). He further stated that chloral hydrate gave John an idiosyncratic reaction. He thus was treated using 100 mg of secobarbital sodium as prescribed by the physician. The procedures, restorations and preventive measures, were tolerated well. John had been NPO since midnight and the medication was administered in the office, after a pulse and blood pressure check.

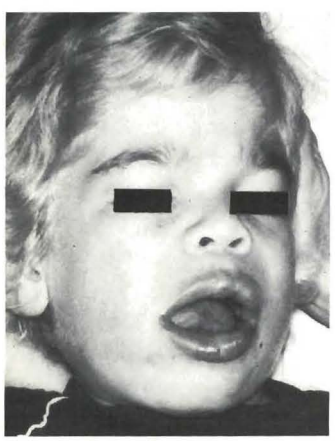

Figure 1. Photograph of patient showing coarse facial features of MPS III.

In 1978 John returned for two recall appointments, in which no new morbidity was found. For these appointments, 100 mg of Seconal were administered as a sedative. He returned in August 1979 for another recall. The parent indicated the following changes in John's health care. A neurologist was now consulted on a regular basis. The child took 45 mg of phenobarbital daily. He had bladder problems and convulsions, and had developed painful joints. This examination revealed tooth #D was very loose and a threat to John by way of aspiration. It was extracted at that visit using ethyl chloride to anesthetize the gingiva. The procedure was completed without premedication. Upon arriving at home, the patient had a seizure. Unfortunately the pediatric dentist was not told of this incident, because the seizures occurred so frequently that the parent did not see the relationship between the dental appointment and the seizure.

The patient returned in six months for another recall. He had grown in size and showed tremendous pooling of saliva in the throat due to lack of coordination of the throat muscles. He was still taking 45 mg of phenobarbital a day. The prophylaxis and fluoride applications were very difficult to accomplish. The combination of prophylaxis paste and saliva frightened him more, and made him struggle more violently. One carious lesion was found, and it was decided that to bring him back would traumatize him more than treating him immediately. The restoration was subsequently completed. The patient went home and several seizures occurred that day. The mother consulted with the physician and it was concluded that the dental appointment may have been the cause.

At eight years of age (1980) John's joints were very stiff. The pressure of the dental assistant's restraining

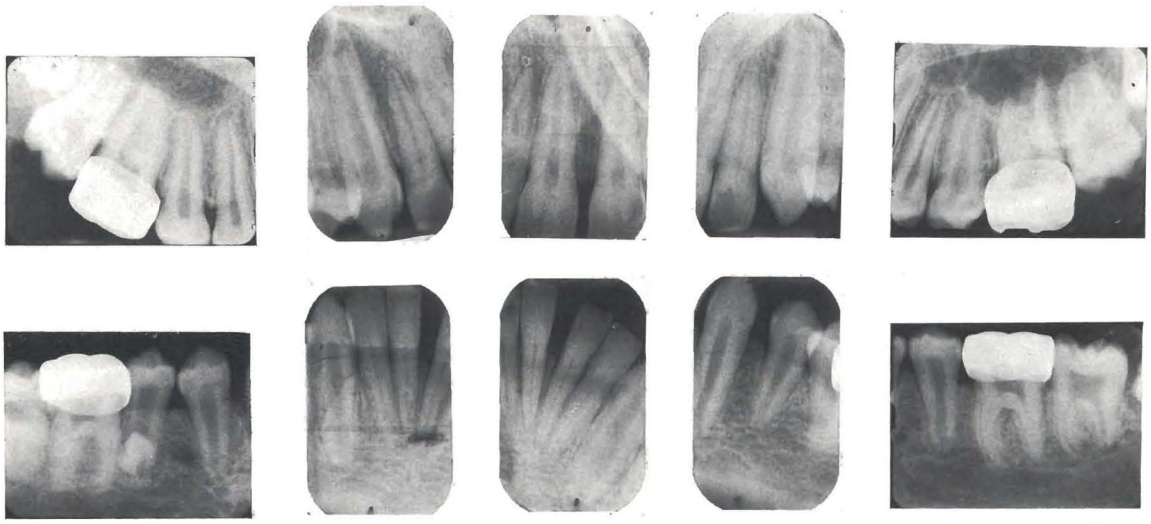

Figure 2. Radiographs showing normal dental anatomy of patient at age twelve.

and supporting him hurt him severely. Being unable to speak, he could not tell anyone about this and so the result was more seizures. For the next examination, the parent requested a consultation between the pediatric dentist and the neurologist. We decided to premedicate him with Seconal because of the success previously. The patient did not take his phenobarbital that morning, but took 100 mg of Seconal. The patient was now wheelchair-bound and was visibly stiffer and less mobile. The saliva problem was worse, but he was quieter. At this appointment, examination, prophylaxis and fluoride therapy, eight radiographs and one restoration were completed. The patient did not experience a seizure after the visit.

1981 was a good year for John's oral health. He needed only routine recall appointments. In January of 1982 an examination revealed hypoplastic first permanent molar teeth. The role of metabolic disturbances was explained to the parent regarding tooth development. The necessity of restoration was discussed. Since he could not talk, there was no way of knowing whether he ever had any discomfort from the hypoplastic areas. John's latest health history showed a decline in status at this time. A consultation with the physician led to the conclusion that he best be treated in a hospital, for several reasons: First, due to inadequate lung usage, John could be monitored before, during, and after surgery; all the teeth could be treated at the one appointment and so cause less trauma to the patient; local anesthesia would be unnecessary, thereby avoiding any postoperative problems. John was admitted to The Children's Hospital of Philadelphia. He was examined and a history was taken before surgery. He was treated in the Same Day Surgery Unit. The four first permanent molars were prepared and crowns were cemented. Finally, an oral prophylaxis was done and acidulated fluoride was applied topically.

Over the period of the next four years, John was seen on recall several times: at each visit he received an oral prophylaxis and topical fluoride. During that time, he had no tooth decay. His health, however, had deteriorated. The bladder problems had grown worse. His seizure activity increased, and be became more debilitated and fragile. Through the four years the sedation was raised to 200 mg of Seconal for a recall visit. Also, during this time his parents experienced increased difficulty in caring for him. In April 1986, John returned for a recall visit. At this examination, numerous carious lesions were found on facial surfaces of his maxillary and mandibular teeth, and on the interproximal surfaces of the maxillary anterior teeth. Finally the second permanent molars had erupted with areas of hypocalcification, similar to those that occurred in the first permanent molars. In all, there were sixteen decayed teeth. Radiographs were attempted, but to no avail. Because of John's fragile condition, a conference was held between the physician, neurologist, and the pediatric dentist. It was determined to hospitalize him because of his very diminished lung capacity, enlarged heart, active seizure disorder, and his profound retardation. To treat him in an office with local anesthesia and sedation would have been too dangerous. Finally, all dental care could be performed at one time under monitored conditions. Because of John's age, he was

taken to Thomas Jefferson University Hospital in Philadelphia, PA. Because of the volatile nature of his current health status, he was admitted and discharged through the Pediatric Intensive Care Unit. This provided superior nursing care during his brief stay in the hospital. John was seen for a postoperative visit. At this time the parents were again shown a prevention technique for two people to aid their child in home care.

John did not appear for his next recall. He developed pneumonia in December of 1986. He died that month.

CONCLUSIONS

1. Dental care for the severely disabled at-risk patient is necessary. It requires time and coordination of health care with any co-managers. These people have the same oral complaints as normal people, even if they cannot speak.

2. Most recall visits for the at-risk patient can be accomplished in an office environment.

3. Prevention and oral health care instruction are the most important parts of the recall appointment.

4. The radiographs showed normal dental anatomy.

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CHILDREN, ADOLESCENTS, AND TELEVISION

In 1984 the American Academy of Pediatrics Task Force on Children and Television issued a statement[1] that cautioned pediatricians and parents about the potential for television to promote violent and/or aggressive behavior and obesity. The influence of television on early sexual activity, drug and alcohol use and abuse, school performance, and perpetuation of ethnic stereotypes was also stressed. Advances in our understanding of the effects of television on children have prompted this update of the Academy's policy.

In 1989 the average child in the United States still spent more time watching television than performing any other activity except sleeping. According to recent Nielsen data, children aged 2 to 5 years view approximately 25 hours of television per week, children aged 6 to 11 years watch more than 22 hours per week, and adolescents 12 to 17 years watch 23 hours of television per week. Although the amount of commercial television viewed by children has declined since 1980, the most recent estimates of television viewing do not include the use of video cassette recorders. Therefore, the amount of time that children in our country spend in front of the television set has probably not decreased significantly in the past 8 years.

Television's influence on children is a function of the length of time they spend watching and the cumulative effect of what they see. By the time today's child reaches age 70, he or she will have spent approximately 7 years watching television. Therefore, television may displace more active experience of the world. For some children, the world shown on television becomes the real world.

AAP, Committee on Communications:

Children, adolescents, and television.
Pediatrics, 85:1119, June 1990.

ABSTRACTS

Waldman, H. Barry: Reaching more children with needed dental services. J Dent Child, 57:417-420, November-December, 1990.

If pediatric dentists are to expand dental services to underserved populations, then in addition to needed finances, attention must be directed to the concerns and motivation of the children in need of these preventive and restorative services.

Dental practice, pediatric; Location; Demography; Underserved groups, dental

Waldman, H. Barry: Confirming the continuing potential for pediatric dental services in nonurban areas. J Dent Child, 57:421-423, November-December, 1990.

National survey decayed-missing-filled data for the second half of the 1980s confirm the increasing use of dental services by nonurban children. Practice alternatives for pediatric dentists continue to exist, in many unexpected locales. Higher rates of decay continue in nonurban-area children.

Dental practice, pediatric; Demography; Location; Metropolitan versus non-urban areas

Bijella, Maria F.T.B.; Yared, Fatima N.F.G.; Bijella, Vitoriano Truvijo; Lopes, Eymar Sampaio. Occurrence of primary incisor traumatism in Brazilian children: house-by-house survey. J Dent Child, 57:424-427, November-December, 1990.

This paper presents the results of a house-by-house survey to learn the occurrence of dental injury to the primary teeth in Brazilian children from ten to seventy-two months of age. The study sample comprised 576 children, 295 boys and 281 girls living on sixty-six streets selected in an urban area (Bauru) in the state of Sao Paul, Brazil. There were 174 children (30.2 percent) with positive reports of injuries to the primary teeth. The results show the need of an educational program in order to prevent the occurrence of dental trauma.

Trauma, dental; Examination, clinical [and] radiographic; Subluxation; Fracture, crown

Weerheijm, Karin L.; de Soet, Johannes J.; de Graaff, Johannes; van Amerongen, Willem E.: Occlusal hidden caries: a bacteriological profile. J Dent Child, 57:428-432, November- December, 1990.

The aim of this study was to obtain insight into the bacteriological profile of hidden caries lesions. Eleven teeth were selected from a total of nine patients according to the following criteria: an apparently intact enamel surface, and a distinct radiolucency in the dentine visible on bitewing radiographs. Two dentine samples, one from the dentinoenamel junction (Sample A) and one just before all the caries appeared to be removed clinically (Sample B), were taken under aseptic conditions and examined for bacteria. In all cases, the A samples and in ten cases the B samples contained mutans streptococci, lactobacilli or both, while in ten cases the dentine after opening had soft, light-colored characteristics suggestive of active caries lesions.

Caries, occlusal [and] hidden; Bacteriology; Radiographs, bitewing; Streptococci; Lactobacilli

Saunders, W.F.; Strang, Ronald; Ahmad, Imtiaz: In vitro assessment of the microleakage around preventive resin (laminate) restorations. J Dent Child, 57:433-436, November-December, 1990.

The purpose of this study was to evaluate *in vitro*, the microleakage of laminate restorations using two resin systems and the influence of etching and curing regimen on the leakage. A total of 135 extracted, human third molars with similar occlusal dimensions were chosen. Cavities were prepared and the teeth were then separated into nine groups of fifteen teeth each. The incidence of microleakage in this study varied between no leakage and 46.7 percent. Leakage of preventive resin restorations was reported to occur in 16.7 percent to 25 percent of cases. The fact that the variables tested in this study had no significant effect on the microleakage would suggest that the leakage is dependent on that of the fissure sealant. The group restored with a fissure sealant containing releasable fluoride and an adhesion promoter performed well in this study.

Microleakage; Restorations, preventive resin; Restorations, laminate; Sealants, fissure

Limbrock, G. Johannes; Hoyer, H.; Scheying, H.: Regulation therapy by Castillo-Morales in children with Down syndrome: primary and secondary orofacial pathology. J Dent Child, 57:437-441, November-December, 1990.

Since Castillo-Morales developed the Orofacial Regulation Therapy for children with Down syndrome in the mid-1970s, close observation of orofacial symptoms in the growing child has led to new findings. Primary orofacial signs are present at birth through age one; secondary alterations develop with untreated school-age children. A synopsis of the most important disorders in children with Down syndrome is given. Findings that relate to malfunction are summed up; these findings can be influenced by Orofacial Regulation Therapy.

Down syndrome; Orofacial pathology; Regulation therapy

Limbrock, G. Johannes; Hoyer, H.; Scheying, H.: Drooling, chewing and swallowing dysfunctions in CP-children: treatment according to Castillo-Morales. J Dent Child, 57:442-448, November- December, 1990.

About 23 percent of the sixty-eight children with cerebral palsy, studied at the Werner-Otto-Institute's Pediatric Rehabilitation Center, suffered from a spastic tetraparesis. About 19 percent suffered from an athetosis; about 20 percent from a hypotonia, including three with cerebellar ataxia. The remainder of the group had minimal sequelae of cerebral palsy; mental problems were predominant. Their orofacial dysfunctions were treated according to the therapeutic concepts of Castillo-Morales.

Cerebral palsy; Dysfunction, orofacial; Drooling; Regulation therapy

Hoyer, H. and Limbrock, G.J.: Orofacial regulation therapy in children with Down syndrome, using the

Continued on page 410

ABSTRACTS *continued from page 406*

methods and appliances of Castillo-Morales. J Dent Child, 57:449-451, November-December, 1990.

The varying hypotonia in Down syndrome is one of its most dramatic signs. In particular, the facial expression and oral dysfunctions of these handicapped persons are uniquely characteristic. The more-or-less permanently open mouth; the prolapse of the tongue, exposed on the everted lower lip; and a lack of mastication, deglutition and speech are primarily caused by the hypotonic orofacial muscles. Breathing through the mouth leads to a dehydration of bacteria and plaque on gums and teeth, and ultimately to premature destruction of the dentition. This developmental syndrome indicates the need for early functional training of the orofacial muscles. Oral Regulation Therapy as described by Castillo-Morales was applied to seventy-four children here, with encouraging results.

Down syndrome; Hypotonia; Dysfunction, orofacial; Regulation therapy

Kendell, Russ L.: Permanent molar impactions and an odontogenic keratocyst: report of case. J Dent Child, 57:452-453, November- December, 1990.

A case of permanent molar impactions involving maxillary and mandibular second and third molars is reported. An odontogenic keratocyst was present in conjunction with root resorption of adjacent teeth. Treatment involved surgical removal of the impacted teeth and enucleation of the cystic lesion.

Impaction; Molars; Keratocyst, odontogenic; Oral surgery

Nordenvall, Karl-Johan and Holm, Karin. Management of cervical root perforation: report of a case. J Dent Child, 57:454-458, November-December, 1990.

The case reported here demonstrates, in the planning and treatment of this patient, a good example of teamwork among different dental specialties. The clinical examination of a healthy, sixteen-year- old Swedish boy produced the diagnoses of osteitis periapicalis resorptiva chronica and root perforation, after unsuccessful treatment by the school dentist who had referred the patient. Orthodontic extrusion was preferred to a surgical approach. The prosthetic treatment was completed immediately after finishing the endodontic procedures.

Root perforation, cervical; Fracture, crown root; Endodontics; Extrusion, orthodontic; Gutta-percha

O'Neil, Durl, W.; Koch, Mark G.; Lowe, James W.: Regional odontodysplasia: report of case. J Dent Child, 57:459-461, November-December, 1990.

A case is reported of a nine-year-old white female with regional odontodysplasia involving the maxillary right quadrant and the construction of a temporary acrylic removable prosthesis, to provide function and to increase vertical dimension.

Odontodysplasia; Odontogenic imperfecta; "Ghost teeth"; Odontomorphogenesis

Anderson, Ruth A. and Ewell-Jackson, Tamara: Scleroderma in pediatric patients: J Dent Child, 57:462-465, November-December, 1990.

Two cases of scleroderma are reported. Manifestations of the disease in the head-and-neck region present problems unique to dentistry. Patients should be placed on a preventive prophylaxis schedule—a professional cleaning every three months.

Scleroderma; Prophylaxis; CREST syndrome; Collagen

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