

NOVEMBER—DECEMBER 1997



The development in the child of aggression, rage, and destructiveness is a complicated matter. The exact origins of these clear feelings are still being debated by philosophers, theologians, and infant psychiatrists.

The weight of current psychiatric opinion falls on the side of an almost inborn natural kind of destructive energy in the human animal.

There is, as well, a developmental explanation for the appearance of anger in the baby's life. The infant, as he approaches and passes his first birthday, needs his parents desperately and understands his helplessness and his neediness. At the same time, because he is growing, he is pushing away from his mother's enclosing arms. He is looking away from her and walking away from her. This movement away frightens him and this fear evokes the anger that we see. He is torn between his need to be a part of the parent and to be separate. Anger is born out of this struggle.

—Herman and Anne Roiphe

WHAT'S BEGUN IN ANGER, ENDS IN SHAME.

—Benjamin Franklin



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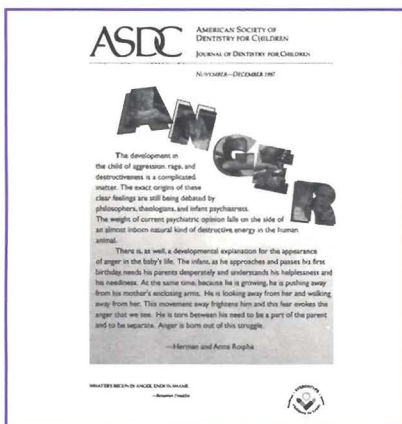
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The infant is torn between his need to be a part of the parent and to be separate. Anger is born out of this struggle.

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Sonia Viada, DDS, MS; Nancy Rivera, DDS, MS; Soraya Nava, DDS, MS; Nelson Hernández, DDS; Alexis Morón, DDS, MS; Jose Contreras, DDS, MS

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Michael P. Nedley, DDS and G. Kent Powers, DDS, MSD

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The authors developed a Confidence of Dental Hygienist in Child Management Scale, and evaluated the effect of the training for dental hygiene students.

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Calendar

1998

ASDC Educational Seminar, The American Club, Kohler, WI, April 25
Congress of the European Orthodontic Society/German Orthodontic Society, Mainz, Germany, June 2-7
International Congress on Dentistry for the Handicapped, Yokohama, Japan, September 3-6
ASDC Annual Meeting, Hyatt Regency, Beaver Creek, CO, October 14-18.

1999

ASDC Educational Seminar, Anchorage, AK, April 30-May 1
ASDC Annual Meeting, El Conquistador, Puerto Rico, September 29-October 3

DEMOGRAPHICS

Children with disabilities are aging out of dental care

H. Barry Waldman, BA, DDS, MPH, PhD
Steven P. Perlman, DDS, MSD

In the mid-1990s there were more than 5 million children between three and seventeen years of age with disabilities. Each year more than one hundred thousand of these children graduate into adulthood. In 1994-1995 there were more than a quarter of a million of these former children between the ages of eighteen and twenty-one years.¹ Each year tens of thousands "age out" of the Early and Periodic Screening, Diagnosis and Treatment (EPSDT) program of the Medicaid system, which guarantees dental care for poor children.^{1,2} Most states provide minimal if any dental services for adults within the Medicaid system. Question—what happens to the low income children with disabilities who "age out of dental care"?

DEFINING DISABILITIES

The term *disability* can be defined narrowly or broadly depending upon the need for and use of the data. The

program under the auspices of the *Individuals with Disabilities Education Act* uses a broad definition.* *The Social Security Disability Insurance Program* uses the narrow definition of persons with disabilities as individuals who are "unable to engage in substantial gainful activity". The broader definition in the *Americans with Disabilities Act of 1990* considers an individual to be disabled, if the person

- Has a physical or mental impairment that substantially limits one or more major life activities.
- Has a record of such an impairment.
- Is regarded as having such an impairment.

Other more extensive definitions include specific limitations in performing socially defined roles and tasks in such areas as personal relationships, family life, education, recreation, self-care and work. Thus it is necessary to distinguish between:

- Disability as the functional limitation within the individual caused by physical, mental or sensory impairments; and
- Handicap as the loss or limitation of opportunities to take part in the normal life of the community on

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*Students are reported by the following disability categories: specific learning disabilities, speech or language impairments, mental retardation, serious emotional disturbance, multiple disabilities, hearing impairments, other health impairments, visual impairments, autism, deaf-blindness, and traumatic brain injury.¹

an equal level with others, due to physical and social barriers.**.**.*.³

CHILDREN WITH DISABILITIES

The media so often has emphasized the special needs of the enlarging elderly population and their myriad of difficulties that we tend to overlook the reality that many of our children are disabled and severely disabled (5.8 percent and 1.3 percent, respectively, of children less than eighteen years of age).³ Between the 1987-1988 and 1994-1995 school years, the number of children with disabilities (age 3-21 years) increased from 4.5 to 5.4 million (Table 1). During the 1990s, except in the "multiple disabilities" and "deaf-blind" categories, the number of children increased in each of the disability categories (Table 2).

In 1994-1995, as defined within the jurisdiction of the *Individuals with Disabilities Education Act*, there were:

- More than half a million children with disabilities, three to five years of age.
- More than 4.6 million children between six and seventeen years of age.
- Almost a quarter of a million young adults between 18 and 21 years of age, including more than ten thousand in Florida, Illinois, Ohio, and Pennsylvania, and more than twenty thousand in California, New York and Texas (Table 3).
- More than 2.5 million children had specific learning disabilities.
- More than one million children had speech and language impairments.
- More than a half million children had mental retardation (Table 4).
- More than 3.3 million children with disabilities lived in non-inner-city areas (three quarters of this population) (Table 5).

In addition, the Supplemental Security Income program provided benefits to more than one million chil-

**The Social Security Administration also provides cash assistance for low-income aged, blind persons with disabilities under the Supplemental Security Income (SSI) program. For children under eighteen years of age, the disability must be of comparable severity to that of an adult.

***The Bureau of Census defines childhood disabilities to include the following health conditions: asthma, autism, blindness or vision problems, cancer, cerebral palsy, deafness or serious trouble hearing, diabetes, hay fever or other respiratory allergies, head or spinal cord injury, heart trouble, impairment or deformity of the back, side, foot or leg, impairment or deformity of fingers, hands or arms, learning disability, mental or emotional problem or disorder, mental retardation, missing legs, feet, toes, arms, hands or fingers, paralysis of any kind, speech problems, and repeated ear infections.^{†3}

Table 1 Number of students (ages 3-21 years) served under the Individuals with Disabilities Education Act: 1987-88 through 1994-95.¹

School Year	Number (in millions)
1987-88	4.5
1989-90	4.6
1991-92	4.9
1993-94	5.3
1994-95	5.4

Table 2 Number of children ages 6-21 served during the 1990-91 and 1994-95 school years by disability condition, and change during the period.¹

	1990-1991	1994-95	Change 1990-91 through 1994-95	
			Number	Percent
Numbers in 000s				
Specific learning disabilities	2,144.0	2,513.9	369.9	17.3%
Speech or language impairments	987.8	1,023.7	35.9	3.6
Mental retardation	551.5	570.9	19.4	3.5
Serious emotional disturbance	390.8	428.2	37.4	9.6
Multiple disabilities	97.6	89.6	-7.9	-8.2
Hearing impairments	59.2	65.6	6.4	10.7
Orthopedic impairments	49.3	60.6	11.2	22.8
Other health impairments	56.3	106.5	50.2	89.0
Visual impairments	23.7	24.9	1.2	5.0
Autism**	5.4*	22.8	17.4	320.6
Deaf-blindness	1.5	1.3	-0.2	-12.7
Traumatic brain injury**	.2*	7.2	6.9	283.3
All disabilities	4,367.4	4,915.2	547.8	12.5

*1991-92 data
 **New categories
 Note: totals differ due to rounding

dren with disabilities and 8,750 blind children at the end of 1996 (Table 3).

Note: In mid-1997, the federal government has ruled that it will eliminate disability benefits for more than 95,000 children (representing more than half of those cases that have been reviewed under strict new standards established by the 1996 welfare legislation). Most of the children losing benefits (78,000 of the 95,000) have "mental disorders". More than 75 percent of the cases reviewed in six states (Iowa, Kansas, Louisiana, Mississippi, Montana and Texas) will lose benefits.^{‡9}

Compounding the difficulties faced by children with disabilities is the reality that many, in particular many minority group children, live in families with incomes below the poverty level.

"...poverty, and not race or ethnicity, is the important factor influencing the disproportionate representation of

†See previous presentations in the *Journal of Dentistry for Children* for reviews of the numbers of special children and their need for care.⁴⁻⁷

‡For a detailed review of the new welfare legislation, see a previous review in the *Journal of Dentistry for Children*.¹⁰

Table 3 □ Number of children with disabilities served under the Individuals with Disabilities Education Act (1994-95 school year) and the Supplemental Security Insurance program (December 1996) by age, disability and state.^{1,8}

	Individuals with Disabilities Education Act				Supplemental Security Income	
	Age			Totals	Disabled children	Blind children
	3-5 years	6-17 years	18-21 years			
	(in 000s)					
Alabama	8.5	85.5	5.1	99.2	29.6	86
Alaska	2.1	14.9	.6	17.6	1.0	15
Arizona	7.3	62.4	2.8	72.5	12.7	152
Arkansas	6.9	43.5	2.2	52.6	18.8	121
California	52.0	471.8	20.6	544.4	79.0	1,857
Colorado	6.8	58.6	2.8	68.2	9.9	60
Connecticut	6.9	63.4	3.4	73.8	5.8	79
Delaware	2.0	12.8	.6	15.4	2.6	25
Dist. Columbia	.3	5.8	.5	6.6	3.1	15
Florida	25.2	258.9	10.6	294.6	62.9	295
Georgia	12.8	111.9	4.5	129.2	29.8	270
Hawaii	1.2	13.4	.5	15.1	1.0	13
Idaho	2.9	19.2	.7	22.9	3.8	35
Illinois	25.0	215.9	10.5	251.4	49.9	238
Indiana	11.1	111.6	5.9	128.6	20.5	154
Iowa	5.7	55.4	3.0	64.0	7.3	149
Kansas	5.9	43.8	1.9	51.7	8.7	69
Kentucky	14.0	63.6	3.1	80.8	24.2	123
Louisiana	9.7	74.7	4.4	88.7	39.7	203
Maine	3.3	26.0	1.3	30.6	2.8	35
Maryland	9.1	84.1	3.7	96.8	13.8	77
Massachusetts	14.3	134.4	7.9	156.7	16.8	609
Michigan	17.7	156.4	8.8	182.9	41.8	187
Minnesota	10.8	79.7	3.5	93.9	11.5	145
Mississippi	6.5	56.4	2.7	65.5	25.4	60
Missouri	7.9	104.1	4.8	116.8	21.6	122
Montana	1.6	15.3	.7	17.6	2.4	24
Nebraska	3.3	33.3	1.4	38.1	4.5	34
Nevada	2.9	22.6	.9	26.4	3.4	86
New Hampshire	1.9	20.6	1.1	23.8	1.9	19
New Jersey	15.9	167.5	8.4	191.9	23.1	105
New Mexico	4.1	39.6	1.7	45.4	6.9	61
New York	45.0	308.7	20.6	374.4	86.3	289
North Carolina	15.1	120.0	4.4	139.6	32.1	269
North Dakota	1.1	10.4	.6	12.2	1.3	15
Ohio	18.2	194.1	11.4	223.6	55.8	369
Oklahoma	4.9	62.7	3.1	70.8	12.1	156
Oregon	5.6	58.6	2.7	66.9	6.9	93
Pennsylvania	19.7	177.3	10.4	207.4	45.6	278
Puerto Rico	3.3	33.8	3.4	40.5	na	na
Rhode Island	2.1	20.5	1.1	23.7	3.0	25
South Carolina	9.9	69.8	2.9	82.6	18.8	203
South Dakota	2.2	12.9	.6	15.8	2.8	25
Tennessee	9.8	107.9	6.0	123.8	24.8	184
Texas	30.6	368.6	21.3	420.5	58.4	703
Utah	4.6	45.0	1.6	51.2	4.7	79
Vermont	1.2	9.0	.5	10.7	1.4	15
Virginia	12.7	117.8	5.7	136.2	24.2	197
Washington	12.8	87.4	4.3	104.5	12.8	107
West Virginia	4.5	38.7	2.2	45.3	8.9	85
Wisconsin	13.1	84.6	4.6	102.2	21.8	129
Wyoming	1.5	10.2	.5	12.2	1.2	8
Total US.	523.8	4,665.3	238.7	5,427.8	1,009.2	8,751

Note: totals differ due to rounding

Table 4 □ Number of students (ages 6-21 years) served under the Individuals with Disabilities Education Act by disability: 1994-95.¹

Disability	Number (in 000s)	Percent
Specific learning disability	2,513.9	51.1
Speech or language impairments	1,023.7	20.8
Mental retardation	570.9	11.6
Serious emotional disturbance	428.2	8.7
Multiple disabilities	89.6	1.8
Hearing impairments	65.6	1.3
Orthopedic impairments	60.6	1.2
Other health impairments	106.5	2.2
Visual impairments	24.9	0.5
Autism	22.7	0.5
Deaf-blindness	1.3	<0.1
Traumatic brain injury	7.2	0.1
All disabilities	4,915.2	100%

Table 6 □ Race and ethnicity distribution by income category of secondary-school-age students with disabilities and those in the general population: 1992-93.¹

Income category	Students with disabilities	General student population	Adjusted population of students with disabilities
Lowest income category			
African-American	39.6%	37.4%	44.4%
Hispanic*	10.9	16.9	—
White	47.0	58.6	54.4
Middle income category			
African-American	21.5	20.5	23.7
Hispanic*	9.4	13.8	—
White	66.8	75.5	73.7
Highest income category			
African-American	10.4	9.2	10.7
Hispanic*	2.7	6.8	—
White	83.5	87.5	86.4

*Because individuals in the Census category "Hispanic" may be of any race, ethnic distributions are adjusted in this column to apportion the Hispanic population in each income category among the other categories in proportion equal to their representation in the population.

Table 5 □ Estimated number of students with disabilities in inner-city and non-inner-city school districts: 1992-93.¹

	Inner-city	Non-inner-city
	(in 000s)	
Specific learning disability	554.0	1,684.3
Speech or language impairments	232.9	847.6
Mental retardation	147.8	403.5
Serious emotional disturbance	89.3	205.3
Multiple disabilities	29.6	45.6
Hearing impairments	16.2	36.6
Orthopedic impairments	13.9	27.8
Other health impairments	23.3	58.0
Visual impairments	6.1	15.1
Autism	7.0	8.2
Deaf-blindness	0.7	1.1
Traumatic brain injury	0.5	2.7
All disabilities	1,121.5	3,335.7

Table 7 □ Average cost for an adult Medicaid dental claim in responding states: 1995.¹¹

State	Cost	State	Cost
Alaska	\$55	Nebraska	\$21
California	470	New Mexico	20
Florida	46	New York	30
Georgia	70	North Carolina	80
Hawaii	108	North Dakota	143
Idaho	31	Ohio	14
Illinois	39	Oklahoma	497
Indiana	99	South Carolina	133
Iowa	83	South Dakota	100
Louisiana	224	Texas	200
Massachusetts	32	Utah	83
Missouri	21	Wyoming	50

minority groups (amongst secondary school-age students with disabilities)"††¹ (Table 6).

These are the very children who will "age out of dental care" as they confront the very limited availability of adult dental services provided under the Medicaid program.

MEDICAID AND MEDICAID DENTISTRY

Medicaid (Title XIX) is a means-tested health program established as part of the Society Security Act of 1965 and is designed to provide access to health care for certain low income populations. The program is administered by states with federal government oversight

provided by the Health Care and Financing Administration. Medicaid is financed jointly by the federal and state governments; and within broad federal guidelines, states have flexibility in establishing income and asset requirements, benefit packages, and reimbursement fees.²

□ Early and Periodic Screening, Diagnostic and Treatment Program

Medicaid offers the nation's largest preventive care program through the Early and Periodic Screening, Diagnostic and Treatment (EPSDT) program. Included within the federal requirements are dental services which are to be furnished by direct referral to a dentist for diagnosis and treatment for children three years of age and older. The intent of the program is to provide eligible children from birth to age twenty-one with comprehensive and periodic screenings for any illnesses, abnormalities, or treatable conditions and referral for

††For a detailed review of children living in poverty, see a previous review in the *Journal of Dentistry for Children*.¹¹

treatment. Many states, however, had limited EPSDT programs.

The Federal Omnibus Budget Reconciliation Acts of 1989 and 1990 require "...that states provide any medically necessary services to eligible children to treat conditions discovered under a screen, regardless of whether that service is included as a part of the state's Medicaid plan... (for) all children born after September 20, 1983, and up to age nineteen whose families are below the (federal) poverty level."²

These expansions made coverage available to an additional three to four million children. Variations in state Medicaid policies, however, have left almost 40 percent of children living in poverty without access to basic primary and preventive care.² The unavailability of services often is related to limited provider participation in the Medicaid program due to low reimbursement fees (expenditure per recipient of dental care has decreased by more than 50 percent [in constant dollars—removing the effects of inflation] since the early 1970s), excessive administrative burdens and cumbersome forms and delays in payment.††

But each year "more than one hundred thousand children with disabilities graduate into adulthood. Each year tens of thousands age out of the EPSDT program and the 'guarantee' of dental care." What happens to them?

Adult Medicaid Dentistry

Results from the American Dental Association Council on Dental Benefit Programs' 1995 survey of state Medicaid agencies and constituent societies (thirty-eight states and District of Columbia responding) provide the latest insight into the difficulties faced by practitioners for the delivery of services to low-income, adult patients.

- Twenty-seven states reported that their states have an adult dental program.
- The average percent of dentists within a state with at least some Medicaid patients (children and adults) is 35 percent, ranging as low as less than 1 percent.
- The monthly average of eligible adults ranged from more than 4,500 in Missouri to almost 2.3 million in California. Thirty percent of eligible adults, however, actually receive dental care (ranging from 10 percent to 75 percent).
- Ten states have limited or only emergency care for adults.

- The average dental claim for an adult Medicaid patient ranged from less than fifty dollars in nine states (Florida, Idaho, Illinois, Massachusetts, Missouri, Nebraska, New Mexico, New York and Ohio) to more than 200 dollars in California and Louisiana (Table 7).

Comments attached to the responses included:

- "Poor—dentistry received .05 percent of the Medicaid budget." (Alabama)
- "Very poor—low fees and very limited adult services." (Georgia)
- "...lowest reimbursement in the country." (Maryland)
- "...fee schedules are around 55 percent of UCR..." (Nebraska)
- "...pay 25 cents on the dollar for UCR." (District of Columbia)
- "...reimbursement is so bad (23.75 percent) that patients cannot access services because of low numbers of providers." (Missouri)
- "(Reform is needed)...to separate out handicapped/elderly from the 'adult' population to provide services to them..." (West Virginia).¹³

NEED FOR DENTAL CARE

Although no national studies provide a general view of the dental needs of people with disabilities (National Center for Health Statistics—personal communication, September 1997) local studies of Independent Living Centers do indicate the need for services.

- Twenty-four to 30 percent of adults with cerebral palsy reported dental problems.
- Fourteen percent of adults with spinal cord injury, 30 percent with head injuries and 17 percent who were deaf reported dental problems.¹⁴

Persons with disabilities experience the same general health problems as people who are not disabled, but for many of them these conditions serve as added impediments to an already difficult life. In addition there are "secondary conditions" that

- Affect the life of the person with a disability.
- Can result in morbidity or premature death.
- Are causally related to the disability or for which the disability may increase a risk (e.g. urinary and renal complications, skin breakdown and decubitus ulcers, physical deconditioning and medication side effects).¹⁴

To this list must be added the impact of limited or total lack of dental care. While young dental patients with disabilities may "age out" of a pediatric dental prac-

††For a detailed review of Medicaid and Medicaid dentistry, particularly for children, see a previous review in the *Journal of Dentistry for Children*.¹²

tice, they just don't disappear. They continue to need dental services—which all too often are unavailable because of limited personal financial resources and unavailable government support programs.

“While there is increased awareness of the need to improve access to quality medical services (for persons with disabilities) dentistry has largely been ignored.”—1997¹⁵

Commentaries more than thirty years ago (1965) noted,

- Private dental offices often are not equipped to handle persons with developmental disabilities.
- Dentists traditionally have not been part of the health planning team, resulting in the omission of these services from many programs.
- Dental school curricula frequently do not include adequate experiences in the management of the people with disabilities.¹⁶

Thirty years later many of these same problems remain. For example, the passage of the Americans with Disabilities Act and other civil rights legislation guaranteed persons with disabilities that they no longer would be discriminated against in areas of employment, housing, travel and other aspects of daily living, including access to health care. Yet in a study conducted by the Academy of Dentistry for the Handicapped to determine the training being provided to predoctoral students in the area of dental management of persons with disabilities, it was found that, of the forty-nine responding schools, most dental schools in the 1990s provide minimal predoctoral curricular time to present the special circumstances and needs associated with the care of persons with disabilities.

- Twenty-three schools (47 percent) reported eight or fewer didactic hours in the care of persons with disabilities; fourteen schools (29 percent) reported five or fewer hours.
- “...clinical activity ranged from zero to 120 hours, thirty-two (65 percent) reported ten or fewer hours, representing, on average, fewer than five treatment appointments.”¹⁷

In addition, persons with disabilities now are confronted by the added complexities of “managed care programs” (or is it “managed not to care programs”?). The emphasis on the economic aspects of these programs often runs counter to the economic realities of providing services for those with disabilities.

In some states which have established managed care arrangements for Medicaid dentistry (e.g. Arizona and Tennessee) there have been particular difficulties in attracting dentists to provide services for the disabled due to inadequate reimbursement. By contrast in Oregon, “The reimbursement (rates) for people with disabilities is 10-times the base rate, serving as an incentive to attract practitioners.”¹⁵

The need is to increase dental services for adults with disabilities. Is it not time we considered the difficulties of our former special pediatric patients who have grown to adulthood?

REFERENCES

1. Eighteenth Annual Report to Congress on the Implementation of The Individuals with Disabilities Education Act. Washington, D.C.: Department of Education, 1996.
2. Solloway, M.R.: An overview of health insurance coverage and access to child health supervision services, pp 5-40; in Solloway, M.R. and Budetti, P.P. (eds) *Child Health Supervision*. Arlington, VA: National Center for Education and Child Health, 1995.
3. Department of Commerce, Bureau of the Census: Americans with Disabilities: 1991-92. Current Population Reports, No. P70-33. Washington, D.C.: Government Printing Office, 1993.
4. Waldman, H.B.: Special pediatric population groups and their use of dental services. *J Dent Child*, 56:211-215, May-June 1989.
5. Waldman, H.B.: Almost eleven million special children. *J Dent Child*, 58:237-240, May-June 1991.
6. Waldman, H.B.: Almost twenty million chronically ill children. *J Dent Child*, 61:129-133, March-April 1994.
7. Waldman, H.B.: Almost four million children with disabilities. *J Dent Child*, 62:205-209, May-June 1995.
8. Office of Research, Evaluation and Statistics. '96 SSI Recipients by State and County. Washington, D.C.: Social Security Administration, 1997.
9. Pear, R.: After a review, 95,000 children will lose cash disability benefits. *New York Times*, August 15, 1997, pA1,28.
10. Waldman, H.B.: Changing welfare as we know it: Some thoughts about the impact on children. *J Dent Child*, 64:135-140, March-April 1997.
11. Waldman, H.B.: Child poverty vs Medicare and Social Security. *J Dent Child*, 64:282-286, July-August 1997.
12. Waldman, H.B.: Mid-1990s review of Medicaid and Medicaid dentistry. *J Dent Child*, 64:141-148, March-April 1997.
13. Council on Dental Benefit Programs; *1995 Survey results and Fact Sheet of State Dental Programs in Medicaid*. Chicago: American Dental Association, nd.
14. Arnett, H.: *First round results of the access to health survey selected disabilities and secondary conditions*. Boston: Independent Living Centers, 1994.
15. Dougherty, N.; Romer, M.; Birenbaum, A.: Protecting dental services for people with development disabilities. *New York State Dent J*, 63:12-14, June-July 1997.
16. Miller, S.L.: Dental care for the mentally retarded: a challenge for the profession. *J Public Health Dent*, 25:111-115, Summer 1965.
17. Fenton, S.J.: Universal access: are we ready? (Editorial) *Spec Care in Dent*, 13:94, May-June 1993.

Homeless children

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

“Historically, the homeless population has consisted largely of men, middle-aged or older, many of whom were disaffiliated, chronic alcoholics residing in the ‘skid row’ areas of large cities.”¹

In the 1970s, the homeless population increasingly consisted of younger men and women suffering from mental illness who were no longer institutionalized. Since 1980, however, the stereotype of the homeless “bag person” no longer accurately reflects the diverse population, an increasing number of whom are members of racial minorities, families and children.¹

While there are no nationwide studies of the homeless, estimates on the number of homeless range from 400,000 to three million persons in this country.² According to a 1994 study of the homeless in thirty cities:

- Fifty-three percent are African-American, 31 percent are white, 12 percent are Hispanic, 3 percent are Native American and 1 percent are Asian-American.
- Thirty-five percent are families with children.
- Twenty-five percent are children.
- Three percent are unaccompanied minors.
- Forty-eight percent of the homeless are single men.

- Eleven percent are single women.
- Forty-three percent are substance abusers.
- Twenty-six percent are mentally ill.
- Nineteen percent are employed full or part-time.
- Eight percent are HIV infected.^{2,3}

Other reports indicate that single-parent families now account for 34 percent of the homeless population.

“Every night between 61,500 and 100,000 homeless children sleep in emergency shelters, abandoned buildings or cars. Some sleep exposed to the perils of the street.”⁴

During their critical formative years, homeless children lack the basic resources needed for normal development. Their experiences result in medical, emotional, behavioral, and educational problems that impact throughout their lives.⁴

No single review can provide a complete picture of the grim world of the homeless child. The following presentation (including an overview of the findings from a variety of local studies) is intended only as an introduction to the life of all too many children who exist in our communities in most instances beyond our consciousness.

“Families with children currently make up the fastest growing homeless population, and in several large cities families comprise more than half of the total homeless population.”⁵ e.g. “...50% in Portland, Philadelphia, Yonkers and Trenton and more than 75% in New York.”⁶

“...more than half of homeless children are less than 5 years of age and are preponderantly minority children.”⁶

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WHY HOMELESS

The idea that most, or even a sizeable number of the homeless are "homeless by choice" has been thoroughly discredited.⁷ (President Reagan had commented about the homeless, that "some of these people are there, you might say, by their own choice."⁴) The evident role of poverty in creating homelessness is associated with the increases in the number of poor women (two-thirds of the nation's poor adults are women) and is related to the increasing numbers of homeless children.⁸ "As a consequence, the poverty rate among American children is about twice that of adults."⁷ In the mid-1990s, four of ten poor people in this country are children.⁹

Poverty is a well known "risk factor" for poor health among children and contributes significantly to the poor health of homeless children both in terms of the consequences of their poverty as well as conditions resulting from their unsettled living conditions.

HEALTH CONDITIONS OF THE HOMELESS

While there are no nationwide studies of the health status of homeless children, numerous reviews of local conditions provide a general sense of the dire circumstances and resultant consequences faced by these youngsters.

- Boston: dental needs and conditions of the homeless (children and/or adults) included,
 - Ninety-seven percent needed some form of treatment.
 - Eighteen percent needed emergency treatment for pain and/or infection.
 - Twenty-eight percent had not visited a dentist for an average of 14.5 years.
 - Thirty-four percent did not own a toothbrush.
 - Children had six to seven times more untreated decayed tooth surfaces than regional and national norms.²
- Los Angeles: homeless children have higher rates of behavioral problems and school failure than housed poor children. Homeless children also have high rates of developmental delay and overweight problems—diets of homeless children frequently are imbalanced, depending on food from "fast-food" restaurants with frequent periods of deprivation. These diets, however, typically are high in cholesterol, fat, sugar, salt and starch, so that while they may offer adequate caloric intake, they are far short of optimal.⁷ Family problems include high rates of spousal abuse, as well as drug and alcohol

use.⁵ In addition, school-age, shelter children have a high level of unmet need for special education evaluation, the first step toward developing needed programs.¹⁰

- New York City: compared to children of similar socioeconomic status, the frequency of health problems among homeless children, (less than five years, who lived in "welfare hotels") was much greater—including delayed immunization, elevated blood lead levels, increased rate of admission to a hospital, and greater rates of child abuse and neglect reports.¹¹
 - Many "welfare hotels" are kept open because the city simply has no other place to shelter many of the 500 or more families that entered the homeless system each month. Upon entering the hotel, one is faced with human and social chaos. There are broken elevators and cracked walls, as well as evidence of drug dealing and prostitution. Children are everywhere, many were hungry. The parents are frustrated and frightened.¹²
- Philadelphia: children's health problems include high incidents of serious accidents and injuries, serious burns and lead toxicity. Associated with many of the difficulties faced by children is the reality that many of their parents suffer from depression, physical abuse, and substance abuse. School children tend to have low scores on tests of expressive vocabulary. Preschoolers have below age expectations in visual motor skills. Homeless children tend to score poorly on developmental and psychological skills.¹³
 - Homeless children experience more barriers to health care than low-income children living at home, resulting from frequent moves, loss of health insurance eligibility and extreme poverty. The shelter environment often poses a risk to health through crowded conditions, poor sanitation and nutritionally inadequate food.¹⁴
- Salt Lake City: the health conditions of predominantly white, two-parent families with few children (from 32 different states) who now live in the city, had similar problems to those reported nationally—delayed immunizations, dental decay, anemia and impaired vision.¹⁵
- San Diego: the most frequent needs that homeless families expressed for their children were: general non-emergency clinics (77 percent), emergency services (67 percent) and dental services (67 percent).¹⁶
- San Francisco: in comparing homeless youths (ages 10-18) with delinquent youths (in a detention cen-

ter) a greater percent of homeless youths had a history of high blood pressure, seizures, migraines, tuberculosis, abnormal bleeding, anemia and sexual abuse.¹⁷

And in larger areas:

- Massachusetts (several areas): approximately 50 percent of homeless children were found to have psychiatric problems, such as developmental delays, severe depression and anxiety, and learning difficulties. Nearly half required psychiatric evaluation.¹⁸

Even in family shelters, pediatric problems included a wide variety of acute and chronic medical conditions, unmet primary care needs and developmental and school difficulties.⁶

- Sixteen city study: homeless children are twice as likely as children from a national sample to be seen in a clinic for infestational ailments, anemia, neurologic disorders, seizures, eye disorders, heart and circulatory problems, gastrointestinal disorders and dentition problems.⁶
- Two Southern California Counties (Riverside and San Bernardino): studies show significantly lower levels of preventive health care for the high-risk-child homeless population than in the general population. Difficulties are associated with the priority of searches for food and shelter, as well as a lack of health insurance coverage, fear of being labeled and rejected by health personnel, and difficulties and complications in navigating the large bureaucracies of health institutions.¹⁹
- Washington State (Kings County): in emergency shelters, half of the children had a wide variety of acute and chronic health problems. Three out of five children had no regular health providers. Emergency room use was two-to-three times greater than U.S. general population.¹
- National study of homeless shelters: "...most Health Care for the Homeless Programs see dental health as an essential and integral part of overall health...(but there is) a lack of volunteer support from the local dental community."²⁰

LEGISLATION

The 1987 passage of the Stewart B. McKinney Act (the Homeless Assistant Act) offered new hope with the designation of \$490.2 million for aid to the homeless. An additional \$1.2 billion was appropriated during the next three years. The money was spent on supportive housing, residential programs, health and mental health care,

education for children and job training for all homeless persons.⁴ In 1992 and 1993, almost \$100 million in appropriation partially supported 120 grantees in forty-eight states, Washington, D.C. and Puerto Rico for more than 400,000 persons. Under these programs, almost 121,000 visits were for dental services (for an average of 302 users per program).²¹

In 1994 President Clinton proclaimed homelessness a high priority for his administration, when he signed an Executive Order directing seventeen federal agencies to develop a "single coordinated federal plan for breaking the cycle of existing homelessness and for preventing future homelessness."²²

COMMENTARY

A previous review in the *Journal of Dentistry for Children* introduced the dire economic, social and health conditions of an often neglected segment of our national population, "invisible children"—the children of migrant farm workers.²² It would not be difficult to argue and present the case that the children of migrant laborers (and the migrant laborers themselves) exist in a rural world which increasingly is alien to the experience of our general urban life style. But how do we rationalize our seeming indifference to another group of "invisible children" who reside IN our communities—homeless children? The reality is that,

"The large number of children now being seen on the streets and in the shelters and other facilities providing services to homeless people means that the homelessness of the 21st century is already being created today...the lives and futures of many tens of thousands of children around the nation are being destroyed..."²⁷

As practitioners intimately involved in the health and development of youngsters, surely our concern must extend beyond the confines of our individual practices to the world of this additional group of "invisible children." Do we really have any other choice?

REFERENCES

1. Miller, D.S. and Lin, E.H.B.: Children in sheltered homeless families: reported health status and use of health services. *Pediatrics*, 81:668-673, May 1988.
2. Allukian, Jr., M.: Oral health: an essential service for the homeless. *J Pub Health Dent*, 55:8-9, Winter 1995.
3. U.S. Conference of Mayors. A status report on hunger and homelessness in America's cities: 1994, a 30 city survey. Washington, D.C.: U.S. Conference of Mayors, 1994.
4. Bassuk, E.L.: Homeless families. *Scientific Am*, 265:66-74, December 1991.
5. Wood, D.L.; Valdez, R.B.; Hayashi, T. *et al*: Health of homeless children and housed, poor children. *Pediatrics*, 86:858-886, December 1990.

6. American Academy of Pediatrics, Committee on Community Health Services. Health needs of homeless children. *Pediatrics*, 82: 938-939, December 1988.
7. Wrights, J.D.: Children in and of the streets: health, social policy, and the homeless young. *Am J Diseases Child*, 145:516-519, May 1991.
8. Sidel, R.: *Women and children last: the plight of poor women in affluent America*. New York: Viking Penguin, 1986.
9. U.S. House of Representatives, Committee on Ways and Means. 1996 Green Book, background material and data on programs within the jurisdiction of the Committee on Ways and Means. Washington, D.C.: Government Printing Office, 1996.
10. Zima, B.T.; Bussing, R.; Forness, S.R. *et al*: Sheltered homeless children: their eligibility and unmet need for special education evaluations. *Am J Pub Health*, 87:236-240, February 1997.
11. Alperstein, G.; Rappaport, C.; Flanigan, J.M.: Health problems of homeless children in New York City. *Am J Pub Health*, 78:1232-1233, September 1988.
12. Redlener, I.: Health care for the homeless—lessons from the front line. Editorial. *New Eng J Med*, 331:327-328, August 4, 1994.
13. Parker, R.M.; Rescorla, L.A.; Finkelstein, J.A. *et al*: A survey of the health of homeless children in Philadelphia shelters. *Am J Diseases Child*, 145:520-526, May 1991.
14. Roth, L. and Fox, E.R.: Children of homeless families: health status and access to health care. *J Community Health*, 15:275-284, August 1990.
15. Page, A.J.; Ainsworth, A.D.; Pett, M.A.: Homeless families and their children's health problems, a Utah urban experience. *West J Med*, 158:30-35, January 1993.
16. Hu, D.J.; Covell, R.M.; Morgan, J. *et al*: Health care needs for children of the recently homeless. *J Community Health*, 14:1-8, Spring 1989.
17. Forst, M.L.; Harry, J.; Goddard, P.A.: A health profile comparison of delinquent and homeless youths. *J Health Care Poor and Underserved*, 4(4):386-400, 1993.
18. Bass, J.L.; Brennan, P.; Mehta, K.A. *et al*: Pediatric problems in a suburban shelter for homeless families. *Pediatrics*, 85:33-38, January 1990.
19. Riemer, J.G.; Van Cleve, L.; Galbraith, M.: Barriers to well child care for homeless children under age 13. *Pub Health Nursing*, 12: 61-66, February 1995.
20. Veal, E.T. and Stewart, A.V.: National study of homeless shelters and professional dental services provided. (Abstract) *J Dent Res*, 73:234, (Spec Iss) 1994.
21. Department of Health and Human Services, Health Resources and Services Administration, Division of Programs for Special Populations. 1994 user profiles for the health care for homeless program. Rockville, MD: Health Resources and Services Administration, 1994; in Allukian Jr., M. *op. cit*.
22. Waldman, H.B.: Invisible children: The children of migrant farm workers. *J Dent Child*, 61:218-221, May-June 1994.

ENAMEL SUBSURFACE DAMAGE IN TOOTH PREPARATION

To conclude, this study revealed the mechanisms of enamel subsurface damage induced by clinical tooth preparation. Median-type cracks and distributed microcracks were observed beneath the machined surface of enamel. The cracks extended preferentially along the boundaries between the enamel rods. Microcracks within individual enamel rods were also observed. The lengths of the median-type cracks depended on diamond particle size and enamel rod orientation, but not on removal rate. These cracks were significantly longer in the direction parallel to the enamel rods than perpendicular to the rods. The cracks produced by the use of coarse diamond burs were effectively removed by being finished with fine diamond burs. Such information supports the clinical practice of finishing by means of fine diamond burs for operative and fixed prosthodontics procedures.

Xu, H.H.K. *et al*: Enamel subsurface damage due to tooth preparation with diamonds. *J Dent Res*, 76:1698-1706, October 1997.

CLINIC

Orthodontic treatment of a patient with hypophosphatemic vitamin D-resistant rickets

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Hypophosphatemic vitamin D-resistant rickets (VDRR) is characterized by familial occurrence, a reduced serum phosphorous level, and a disturbed ossification that does not respond to the usual therapeutic dose of vitamin D. When this disease develops late in life, it is less severe and may not be characterized by rickets or other osseous deformities. The characteristic dental manifestations in VDRR are well known. Abnormal calcium and phosphate metabolism produces the usual retardation in skeletal and facial growth, in addition to the hypoplastic defects in enamel and poorly calcified and dystrophic dentin. The eruption of the teeth is retarded, and children with VDRR are often deformed and very susceptible to caries, with crowding of the dental arches a common occurrence. The facial growth in children with VDRR shows less retardation than the growth of the body stature; and mandibular growth is less retarded than maxillary growth. This paper presents the results of orthodontic therapy in a child with VDRR. Analysis included cephalometric measurements of craniofacial morphology and reaction of alveolar bone during tooth movement.

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CASE REPORT

Diagnosis

The patient was a nine-year-old girl with moderate Class II division 2 malocclusion. An overjet of 6.1mm and an overbite of 5.9mm accompanied the severe anterior crowding. The maxillary left incisor was in lingual crossbite and the palatal vault was very high (Figure 1). Panoramic X-ray films revealed that all primary and permanent teeth had large pulp chambers and were not normally calcified (Figure 2A). The patient's lips protruded and poor or insufficient mandibular growth was evident. The bone age was retarded by approximately three years.

History and clinical background

The patient was admitted to Nara Medical University Hospital at the age of one year and seven months for correction of her leg deformities. After a serum chemistry test, she was diagnosed with hypophosphatemic vitamin D-resistant rickets (VDRR) at Osaka University Hospital, Department of Pediatrics, for which she had received orally administered 4 μ g of 1 α -hydroxyvitamin D₃ (One-alfa@) and 1.0g of phosphorous daily. Throughout this period, serum phosphorous had never entered the normal range (3.4-6.3 mg/dl) (Figure 3).

Cephalometric analysis

Analysis of the skeletal relationships indicated an anteroposterior discrepancy of the maxilla to the mandible.

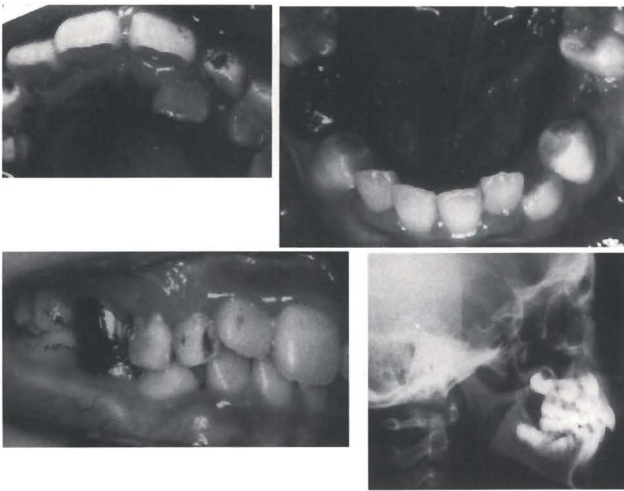


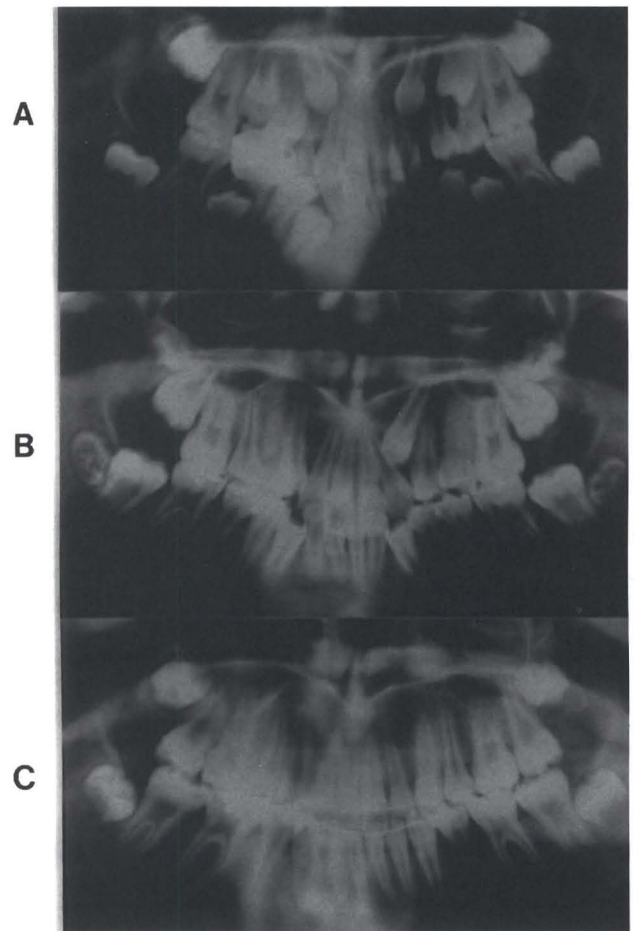
Figure 1. Patient before treatment.

This Class II division 2 malocclusion had an ANB angle of 9.3° ($SNA=83.9^\circ$, $SNB=74.7^\circ$). In terms of vertical dimensions, the mandibular plane angle was 31.2° . The maxillary incisors had essentially a lingual inclination, but IMPA (91.8°) was average (Figure 4).

Treatment progress

When the patient was nine years and ten months old, the maxillary left primary canine was extracted and the maxillary left permanent lateral incisor was moved labially with a lingual arch. Dental X-ray photographs obtained two months later indicated that there was no abnormality of the teeth and the periodontal tissue. In the first stage of treatment, the patient used an elastic open activator (EOA).¹

Two years and eight months later, the molar relationship achieved Class I. The maxillary canines were located labially, however, and crowding of the maxillary anterior teeth was noted. The size of all teeth was more than 1.S.D., and the mandibular arch had an arch-length tooth-mass discrepancy of 10.6 mm. Skeletally, the mandibular plane angle was high (FMA 38.1°), with an apical base discrepancy (ANB angle $+4.2^\circ$). The treatment plan was designed, therefore, to eliminate the anterior crowding and to place the anterior teeth over the basal bone. The maxillary left impacted canine was exposed surgically and retraction to the occlusal plane was achieved with elastics and an edgewise appliance ('018 slot). The three other premolars were then extracted. For approximately 16 months thereafter, various arch



Figures 2. Panoramic radiographs: A. Initial stage of orthodontic treatment. B. After two years and eight months of treatment. C. Final stage of treatment.

wires with open coil springs and chain elastics were used to align and coordinate the maxillary and mandibular dental arches as much as possible. Care was taken to prevent both alveolar bone loss and root resorption.

After debonding and debanding, a fixed lingual retainer bonded to several anterior teeth was used (Figure 5).

Treatment results

In the first EOA treatment stage, the molar relationship achieved Class I. The ANB angle was reduced from 9.3° to 6.6° . Superimposition revealed good downward and forward growth. The mandibular plane angle increased, from 31.2° to 38.1° .

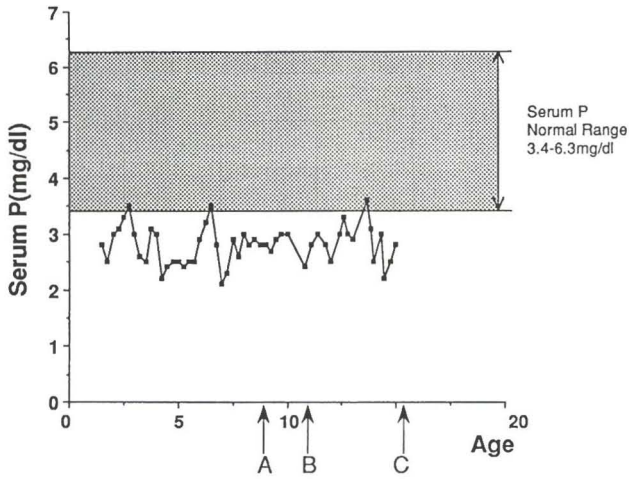
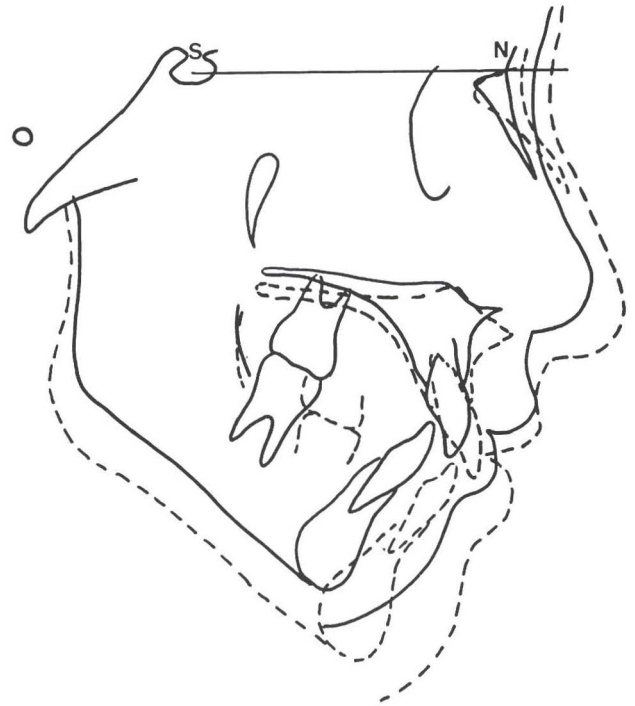


Figure 3. Serum phosphate. A. Initial stage of orthodontic treatment. B. After two years and eight months of treatment. C. Final stage of treatment.

In the second stage of treatment by use of the edge-wise appliance, mandibular growth and maturation continued, and the facial features became more pleasing. All crowding of the teeth was eliminated, and the maxillary incisors were positioned upright over the basal bone. Good root parallelism was obtained, and the extraction spaces were tightly closed. The SNA angle was decreased, from 81.7° to 80.3°, and the SNB angle was increased, from 75.1° to 76.2° (Figure 4). Panoramic X-ray films revealed a slight root distortion of the lateral incisors, no abnormalities of the alveolar bone, or evidence of root resorption (Figure 2C)

DISCUSSION

The term *rickets and osteomalacia* encompasses a group of diseases with similar clinical manifestations and histological alterations but with diverse etiologies.² VDRR is a type of rickets that is refractory to the usual therapeutic dose of vitamin D₃ (1, 25 (OH)₂ D₃).³ Clinically it cannot be distinguished from vitamin D deficient rickets. Some of the manifestations of VDRR are the need for continuous therapy and high doses of 1,25(OH)₂ D₃ and resistance of the bone metabolism defect to therapy. VDRR is inherited through an X-linked dominant gene.³ It is reported that 62.5 percent of VDRR patients in Japan, however, have no familial history.⁴ Early clinical signs are usually detected at about two years of age, and include lateral bowing of the legs and frontal bossing.³ The diagnosis is usually made when the child begins to walk.



	Pretreatment	Posttreatment	Ref. Norm.
1-APO	+1.5mm	+3.3mm	+3.0mm
FMA	31.2°	34.7°	31.2°
Wits	14.2mm	7.5mm	6.4mm
ANB	9.3°	4.1°	3.3°
1-SN	95.9°	108.5°	106.8°
SNA	83.9°	80.3°	80.8°
SNB	74.7°	76.2°	77.4°

Figure 4. Superimposed pretreatment and posttreatment cephalometric tracings. Note downward and forward growth pattern. —: pretreatment (9 years and 1 month). - - - - -: posttreatment (15 years and 1 month).

VDRR involves defective renal tubular transport of phosphate as well as a variable degree of impaired calcium and phosphorous absorption at the intestinal level.⁵ Biochemically it is characterized by hypophosphatemia, hyperphosphaturia, elevated level of serum alkaline phosphatase, and a normal or slightly elevated level of immunoreactive parathyroid hormone.⁶ It has been reported that, while the clinical symptoms in vitamin D-resistant rickets are very similar to those of vitamin D-deficient rickets, its treatment should be entirely different.⁷ In the treatment of VDRR, long-term trials with high-dose 1,25 (OH)₂ D₃ and phosphate supplementa-

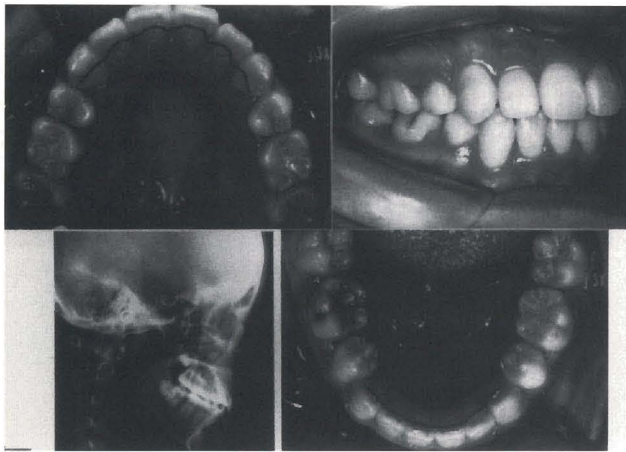


Figure 5. Patient after treatment.

tion are well documented in the medical literature.⁸⁻¹⁰ This patient had received continuously administered $1,\alpha(\text{OH})_2 \text{D}_3$ ($4,\mu\text{g}/\text{day}$) and phosphate ($1.0\text{g}/\text{day}$) since the age of one year and seven months. The serum phosphate level was consistently very low, however, and never entered the normal range.

The dental manifestations of VDRR can be the first indications of the disease. These pathoses usually occur when the patient has no other signs or symptoms of rickets. Abscesses with gingival fistulas, with or without gross evidence of pulpal exposure from caries, and fractures or attrition can all be seen in the very early stages of the disease.^{11,12} The dental findings in rickets have been reported, and many authors have considered that rickets patients have malocclusions.¹³⁻¹⁵ It is well known that orthodontic tooth movement is related to alveolar bone remodeling. Metabolic bone disease, such as VDRR, may affect periodontal tissue and the process of mineralization. There are, however, very few investigations of this phenomenon or case reports in orthodontics.

VDRR is a genetic disorder that is known to occur in humans and mice.^{16,17} Craniofacial abnormalities are a common physical characteristic; and retardation of maxillary and mandibular growth and bossing of the frontal and occipital regions have been reported.^{18,19} Tracy *et al* suggested that, in the chronic rachitic state, the cartilaginous growth in the nasal septum might be more readily affected than the growth of the mandibular condyle.³ Mostata's study revealed that the Hyp mouse, which is an animal model system for the investigation of VDRR, has a small anteroposterior skull and mandibular relationship and shorter upper face height than the normal mouse.²⁰ The facial pattern in children with VDRR

shows mandibular growth that is less retarded than maxillary growth. In the present patient as well, less retarded maxillary growth was not recognized, but the mandible was located posteriorly, indicating an anteroposterior discrepancy of the relation of the maxilla to the mandible. Narrow dental arch-form has been reported in the literature. This patient showed a crowded arch accompanied by very large tooth size. The large tooth size discrepancy in VDRR is reported to be related to the stress exerted on the maxilla by the muscles of the jaw during function, which influence its growth.²¹ The pathogenesis of the malocclusion in VDRR patients may be related to endocrine and other delayed developmental disorders.

Thus, we used the elastic open activator (EOA) in our patient in order to stimulate mandibular growth.¹ The elastic open activator, designed by G. Klammt, is one of the activators with reduced bulk that can be worn both day and night. The use of this appliance is advantageous in the improvement of a Class II relationship and for the correction of deep overbite. After the first stage of treatment, cephalometric examination revealed decrease of the ANB angle and effective anterior growth of the mandible, which we attributed to the activator.

In the second stage of treatment, to obtain desirable occlusion, we used a multi-bracket appliance. The patient's treatment outcome was fair to good. We noted that all teeth were repositioned without complication, as Isshiki *et al* noted.²¹ Panoramic radiographs obtained after the treatment revealed no unfavorable root resorption and bone defect. In a patient such as ours, however, when bone remodeling is abnormal, a longer period of retention and observation is necessary to confirm the stability of the resulting occlusion.

REFERENCES

1. Graber, T.M. and Neumann, B.: *Removable Orthodontic Appliances*, 2nd ed. Philadelphia: W.B. Saunders Co., 1984, pp 342-356.
2. Pitt, J.J. and Haussler, M.R.: Vitamin D, biochemistry and clinical applications. *Skeletal Radiol.* 1:191-208, 1977.
3. Tracy, W.E. and Campbell, R.A.: Dentofacial development in children with vitamin D-resistant rickets. *J Am Dent Assoc*, 76: 1026-1031, May 1968.
4. Seino, Y.; Shimotsuji, T.; Matsuura, N. *et al*: The survey of children calcium disorders in Japan, - Familial hypophosphatemia and vitamin D resistant rickets - *Acta Paediatrica Jpn*, 88:165-168, 1984.
5. Chesney, R.W.: *Nelson Textbook of Pediatrics*, 14th ed. Philadelphia: W.B. Saunders Co., 1992, pp 1748-1751.
6. Walton, J. : Familial hypophosphatemic rickets. A delineation of its subdivisions and pathogenesis. *Clin Ped*, 15:1007-1012, 1976.
7. Harrison, H.E. and Harrison, H.C.: Hereditary metabolic bone disease. *Clin Orthop*, 33:147-156, 1964.
8. Harrison, H.E.; Harrison, H.C.; Lifschitz, F. *et al*: Growth disturbance in hereditary hypophosphatemia. *Am J Dis Child*, 290-297, 1960.

9. West, C.O.; Blanton, J.C.; Silverman, F.N. *et al*: Use of phosphate salts as an adjunct to vitamin D in the treatment of hypophosphatemic vitamin D refractory rickets. *J Pediatr*, 64:469-477, 1969.
10. Seino, Y.; Shimotsuji, T.; Ishii, T. *et al*: Treatment of hypophosphatemic vitamin D-resistant rickets with massive doses of 1 α -hydroxy-vitamin D₃ during childhood. *Arch Dis Child*, 55: 49-53, 1980.
11. Cohn, S. and Becker, G.L.: Origin, diagnosis and treatment of the dental manifestations of vitamin D-resistant rickets; review of the literature and report of case. *J Am Dent Assoc*, 92:120-129, January 1976.
12. Harris, R. and Sullivan, H.R. : Dental sequelae in deciduous dentition in vitamin D resistant rickets, case report. *Aust Dent J*, 5: 200-203, 1960.
13. Seow, W.K.: X-linked hypophosphatemic vitamin D-resistant rickets. *Austra Dent J*, 29:371-377, 1984.
14. Archard, H.O. and Witkop, C.J.: Hereditary hypophosphatemia (vitamin D-resistant rickets) presenting primary dental manifestations. *Oral Surg Oral Med Oral Pathol*, 22:184-193, 1966.
15. Gigliotti, R.; Harrison, H.; Reveley, R.A. *et al*: Familial vitamin D-refractory rickets. *J Am Dent Assoc*, 82:383-387, February 1971.
16. William, T.F. and Winters, R.W.: *Metabolic Basis of Inherited Disease*. New York: McGraw-Hill, 1972, pp 1465-1485.
17. Eicher, F.H.; Suthard, J.L.; Scriver, C.R. *et al*: Hypophosphatemia: Mouse model for human familial hypophosphatemic (vitamin D-resistant) rickets. *Proc Natl Acad Sci USA*, 73:4667-4671, 1976.
18. Shafer, H.; Hine, M.; Levy, B.: *Oral pathology*. Philadelphia: W.B. Saunders Co., 1974, pp 593-595.
19. Marks, S.C.; Lindahl, R.L.; Bawden, J.W.: Dental and cephalometric findings in vitamin D-resistant rickets. *J Dent Child*, 32: 259-265, 1965.
20. Mostafa, Y.A.; El-Mangoury, N.H.; Meyer, Jr., R.A. *et al*: Deficient nasal bone growth in the X-linked hypophosphatemic (Hyp) mouse and its implication in craniofacial growth. *Arch Oral Biol*, 27:311-317, 1982.
21. Isshiki, Y.; Takahashi, I.; Maruyama, H.: On three clinical cases of vitamin D resistant rickets. *J Jpn Orthod Soc*, 26:70-82, 1967.

TREATING TOBACCO ADDICTION

The safety of both nicotine-replacement therapy and bupropion seems to be excellent. Many smokers as well as physicians believe that nicotine is dangerous and that medications containing nicotine should be avoided or at least used for as short a time as possible. Although there was concern in 1992 that nicotine patches might cause heart attacks, recent clinical trials and experimental studies suggest that nicotine does not have clinically significant adverse cardiovascular effects, even in people with heart disease who take nicotine while continuing to smoke. There is no evidence that nicotine causes cancer. Nicotine could theoretically contribute to adverse outcomes of pregnancy, including fetal neurotoxicity, although no safety data on nicotine-replacement therapy are available to determine whether this is a significant risk. People can also become dependent on nicotine-replacement therapy, an effect that seems to be related to the rate of nicotine absorption by the body. Thus, the risk of dependence is expected to be greatest with nicotine nasal spray, less with nicotine gum, and minimal with nicotine patches.

Like nicotine, bupropion has some sympathomimetic actions. No data are available on the safety of bupropion in smokers with coronary heart disease or in pregnant smokers. Bupropion in excessive doses, but occasionally even at therapeutic doses, can cause severe anxiety, hypertension, and seizures. The risk of seizures seems to be higher in people with eating disorders (bulimia or anorexia nervosa), many of whom are cigarette smokers. Nortriptyline, like all tricyclic antidepressants, can have serious anticholinergic side effects, and overdose is potentially lethal. Bupropion and nortriptyline probably pose little or no risk of dependence.

The movement of nicotine-replacement therapies from prescription drugs to over-the-counter medications has the potential disadvantage of taking smoking-cessation therapy out of mainstream medical practice. However, since over-the-counter therapy is unsuccessful in many smokers, the availability of new medications offers physicians the opportunity and the obligation to become involved in smoking-cessation therapy. We can expect future research to yield a wide variety of novel therapies, such as drugs that are agonists or antagonists of specific nicotinic receptor subtypes, which would have the potential to prevent or reverse specific withdrawal symptoms without affecting other psychological or physiologic systems.

Benovitz, N.L.: Treating tobacco addiction—nicotine or no nicotine (Editorial).
N Engl J Med, 337:1230-1231, October 23, 1997

The differences in the chronology and calcification of second molars between Angle class III and class II occlusions in Japanese children

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It has been suggested that tooth eruption is intimately associated with normal dentofacial growth and occlusal development, and that the time of eruption is clinically important in clinical experiments. Excessive or deficient posterior eruption, increased or decreased lower facial height, and the time of eruption are frequently important factors in controlling molar relationships. From clinical observation, we have learned that the eruption of the mandibular second molar in class III patients was earlier than that in class II patients. There are many studies concerning the developmental and calcification stages of the permanent dentition and the sequence of eruption of the permanent teeth.¹⁻¹¹ Despite years of investigation, the relationship between the development of second molars and skeletal malocclusion is not well-understood. For class III or class II patients, correcting the molar relationship is one of the most important and the most challenging problems in orthodontic treatment. For patients with these molar relationships, we often use orthopedic forces, such as face bow or face mask. It is

well known that these orthopedic forces are most effective when applied before the eruption of the second molar. This article presents new data regarding the differences in eruption and calcification times of second molars between class II patients and class III patients; the relationship between the calcification of the second molar and the type of malocclusion; the relationship between the time of eruption of the second molar and the type of malocclusion in Japanese non-orthodontically treated children; and discusses the possible significance of the new findings and their clinical implications.

SUBJECTS AND MATERIALS

Included in the records of 2200 cases are cephalometric radiographs, panoramic radiographs, profile photographs, and dental casts from the Kanomi Orthodontic and Pedodontic Clinic at Himeji in Japan; fifty-three cases, ages seven to ten years, who had no orthodontic treatment and no serious dental caries were selected. Of these, twenty-six children (twelve boys and fourteen girls) were Angle class III with minus ANB and of a concave facial type according to profile photographs. Their mean age was 8.67 years (SD 0.87 years). We named them the "Class III group". Twenty-seven children (eleven boys and sixteen girls) were Angle class II with 5 degrees or more ANB and of a convex facial type according to profile photographs. Their mean age was 8.75 years (SD 1.02 years) and were called the "Class II

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Table 1 □ Sample characteristics.

	Class III		Class II	
Number				
Male	12		11	
Female	14		16	
Pool	26		27	
Age (year)	8.67	SD 0.87	8.75	SD 1.02
Cephalometric characteristics				
SNA	77.61	SD 3.12	82.14	SD 3.01
SNB	79.08	SD 3.29	74.94	SD 2.88
ANB	-1.57	SD 1.46	7.84	SD 1.49
FMA	27.31	SD 3.29	30.93	SD 4.29
IMPA	88.81	SD 7.22	97.59	SD 5.21
Saddle angle	126.08	SD 5.28	126.40	SD 3.66
Articular angle	140.78	SD 5.49	145.32	SD 5.07
Conial angle	128.95	SD 5.84	127.41	SD 6.36
ANS-PNS (mm)	47.21	SD 3.50	51.25	SD 3.10
Xi-PM (mm)	64.05	SD 4.88	60.27	SD 2.71

group" (Table 1). There was no statistically significant difference in age between the groups.

METHOD

Nolla's stage of development of the teeth

The equipment used for the experiment was an AUTO-2, a high-voltage (200 volt) panoramic and cephalometric radiographic instrument produced by Asahi

Roentgen Ltd. Co., Japan. The first study examined the calcification of maxillary and mandibular second molars. The calcification criteria incorporated Nolla's developmental stages for mandibular and maxillary teeth.^{12,13} The reproductivity of these criteria on the panoramic radiographs was assessed by calculating Kappa between judgments made on two separate occasions on twenty panoramic radiographs selected at random. The result was 0.82 and was clinically significant. Pairs of judgments never exceeded one stage.

Chronology by panoramic radiograph

The reliability of measurement of panoramic images was investigated by Suzuki.¹⁴ Vertical distortion was within 1 percent, ranging from +110 mm to -110 mm in the vertical position at the mid-horizontal plane. Suzuki concluded that the method was sufficiently reliable for use in clinical research. We also planned the preliminary experiment to test the reliability of the distance between panoramic images in our previous study. The results were consistent with those reported by Suzuki. The second study examined chronology as shown by panoramic radiographs, which we made using a new method (Figure 1). First, we measured the length of the completely erupted first molars between the tops of the crowns and the apices of the roots. This length was regarded as 100

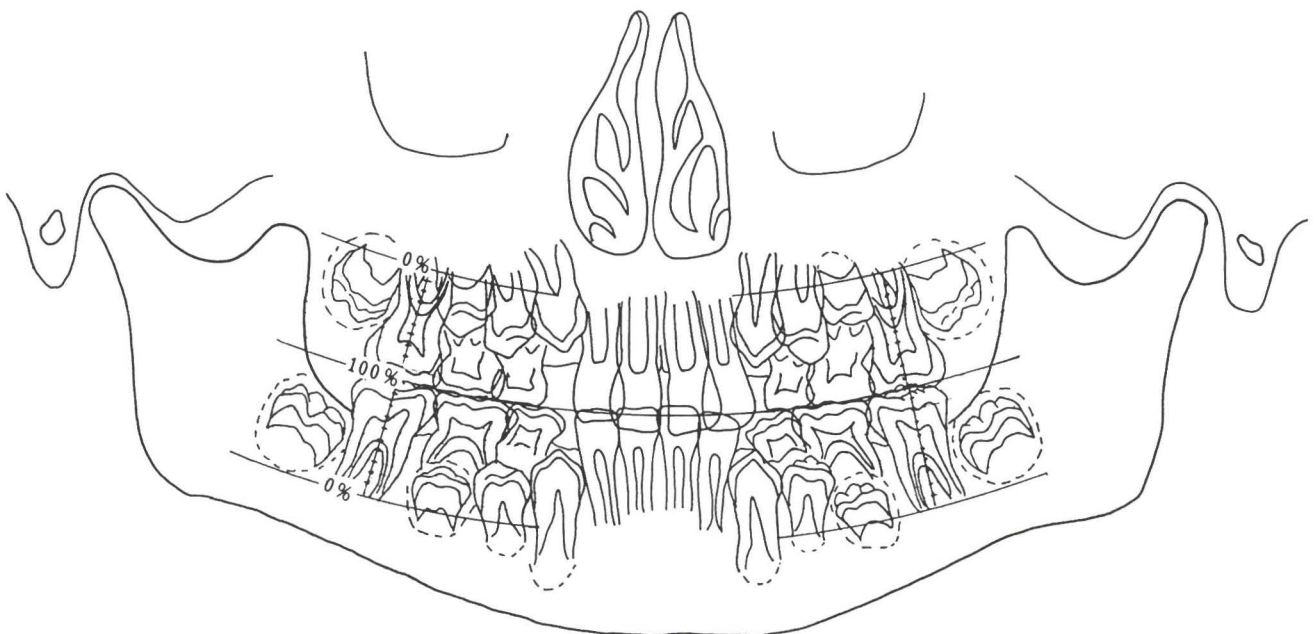


Figure 1. Stage of eruption of permanent dentition by panoramic radiography.

percent of the eruption. Next, we measured the distance between the top of second molar and the occlusal line. We defined the occlusal line as follows: The occlusal line in panoramic radiographs is that part of the curved line that extends from the mid-point between the tips of the maxillary and mandibular left incisors to the anterior contact between maxillary and mandibular first molars. Finally, we calculated how much of each second molar was erupted in comparison with the first molar of the same segment.

Chronology by cephalometric radiograph

The third study examined chronology by cephalometric radiograph. We measured the length between the occlusal line (the line from the mid-point between the tip of the maxillary and mandibular incisors to the anterior contact between the maxillary and mandibular first molars in occlusion) to the mid-point between the top of both crowns in the same jaw (Figure 2). Using this method, however, the smaller the distance is the greater the eruption, we used the minus sign to correct this phenomenon. In order to reduce examiner bias, the panoramic and cephalometric radiographs were numbered and subsequently measured "blind" in random order. Digital calipers (Caliper Ultra-Call Mark III, Fowler

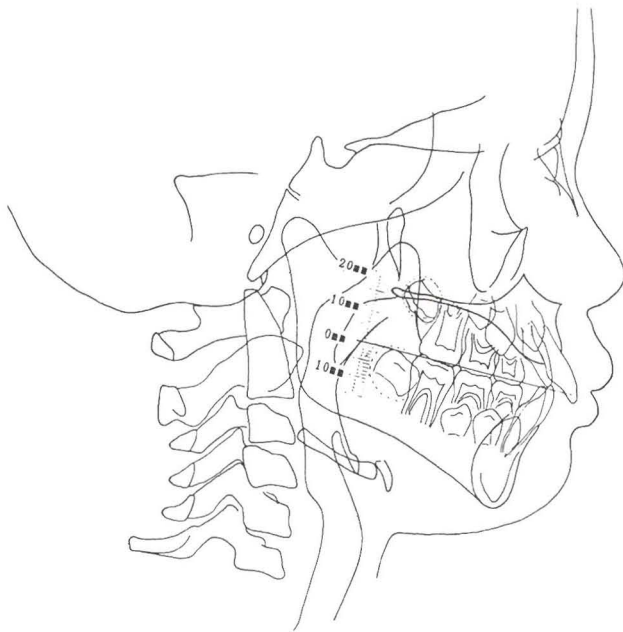


Figure 2. Stage of eruption of permanent dentition by cephalometric radiography.

Co., Inc., Newton, Mass.) were used to measure (at 0.01 mm) panoramic radiographs. Cephalometric landmarks were digitized with the digitizer and NEC Versa-STAT program (Yasunaga computer laboratory Inc. Japan) was used. The reproductivity of the measurements on the panoramic and cephalometric radiographs was assessed by calculating Pearson's product-moment correlation coefficients between two measurements made on two separate occasions on fifteen panoramic radiographs and fifteen cephalometric radiographs selected at random. The correlation coefficients for both panoramic and cephalometric measurements ranged from 0.97 to 0.99. Each error in assessing panoramic and cephalometric radiographs ranged within 0.30 mm.

Estimation of correlation

The results of calcification were discussed in relation to skeletal malocclusion and time of eruption. Significant differences between results were noted. In this study, twenty measured values that were deemed to be important were selected for study. The cephalometric criteria studied include the sella nasion A-point angle (SNA), the sella nasion B-point angle (SNB), the relationship between SNA and SNB (ANB), the Frankfort mandibular angle (FMA) and the incisor mandibular plane angle (IMPA). We also used the Jaraback analysis for this study, one of the most adequate analyses for understanding skeletal patterns of mixed dentitions. We measured the distance between ANS and PNS for maxillary length. We also measured the distance between Xi and PM, as well as the distal surfaces of mandibular first molars and mesial surfaces of the mandibular bone on the occlusal plane for mandibular bone length. These measurements show both horizontal and vertical relationships that are critical to a better understanding of skeletal malocclusion. Groups were compared by using the MANOVA test for independent groups.¹⁶ Association between variables was evaluated by the Pearson product-moment correlation coefficient.

RESULTS

A significant difference in mean values of calcification, using Nolla's classification stages between the class III group and class II group was noted in the maxillary second molars. In the mandibular second molars, however, there was no significant difference of mean values of Nolla's calcification stages between the groups (Table 1). Calcification of the maxillary second molar in the class I group was significantly earlier than that of the class III group, whereas no significant difference for the mandib-

ular second molar was found. There was no statistically significant difference of mean values of calcification stages, using Nolla's classification, between boys and girls in both the class III and class II groups.

Significant differences of mean values for the eruption of the maxillary second molar between the class III and class II groups was noted, using our new panoramic-radiograph method (Table 1). Also, significant differences in mean values for the eruption of the mandibular second molar, between the class II and III groups using our new panoramic-radiograph method, was noted for the second molar. For the maxillary molar, the erupting stage of class II subjects was earlier than that of class III subjects. In contrast, for the mandibular molar the erupting stage for class III subjects was earlier than that of class II subjects.

Significant differences of mean values for eruption of maxillary second molars, using our new cephalometric-radiograph method was noted for the second molar (Table II). There was a significant difference in mean values for the erupting stages of the mandibular second molar. For the maxillary second molar, the erupting stage for class II subjects was earlier than that of class III subjects. In contrast, for mandibular teeth the erupting stage for class III subjects was earlier than that of class II subjects—as panoramic methods were used.

Table III contains the intercorrelation matrix for chronology and calcification of the maxillary second molar as compared to cephalometric analysis. There was a significant correlation between calcification and the length of ANS-PNS. There was also a significant correlation between the time of eruption in both panoramic and cephalometric radiographs and the length of ANS-PNS. For the maxillary second molar, the longer the ANS-PNS the earlier the calcification and eruption. There

were significant correlations between both calcification and chronology of maxillary second molar and ANB angles. The maxillary second molar with the largest ANB had the earliest calcification and chronology. There was a significant correlation between both calcification and chronology of the maxillary second molar to SNA and SNB angles. The maxillary second molar with greater SNA angles showed earlier calcification and eruption. In contrast, the maxillary second molar with greater SNB angles exhibited later calcification and chronology.

Table IV shows the intercorrelation matrix of the chronology and calcification of the mandibular second molar to cephalometric analysis. There was a significant correlation between the chronology of the mandibular second molar in panoramic radiographs and SNB, ANB and IMPA angles. The mandibular second molar with the greater SNB angle had the earlier eruption. In contrast, the greater ANB and IMPA angles were found in the later eruption of the mandibular second molar. There was no significant relation between the length of mandibular bone and its calcification or chronology.

DISCUSSION

It is suggested that tooth eruption is intimately associated with normal dentofacial growth and occlusal development, and the timing of eruption is clinically important. Now we can say that second molar eruption is intimately associated with normal dentofacial growth and occlusal development, and the timing of eruption is clinically important in our study. Our clinical observation that the eruption of the mandibular second molar in the class III patient was earlier than that in the class II patient was confirmed in this study. The panoramic radiographs showed that the calcification of the maxillary second molar in class II patients occurred

Table 2 □ The development and erupting stage in Class III and Class II groups.

	Class III		Class II		MANOVA (p-level)	t-test (unpaired)
	A.V.	S.D.	A.V.	S.D.		
The development stage using Nolla's classification						
Maxillary left second molar	4.92	1.11	6.92	0.83	0.000	**
Maxillary right second molar	5.00	0.95	6.89	0.75	0.000	**
Mandibular left second molar	5.64	0.86	5.89	0.80	0.285	
Mandibular right second molar	5.64	0.95	5.89	0.80	0.311	
The eruption stage of teeth using panoramic radiographs (%)						
Maxillary left second molar	37.72	10.17	53.85	7.28	0.000	**
Maxillary right second molar	36.76	8.86	51.78	5.79	0.000	**
Mandibular left second molar	54.76	8.80	45.89	6.82	0.001	
Mandibular right second molar	55.60	8.68	49.33	5.69	0.003	
The erupting stage using cephalometric radiographs (mm)						
Maxillary second molar	17.26	1.84	14.22	1.74	0.000	**
Mandibular second molar	7.62	1.24	8.20	1.21	0.093	*

**p<0.01 *p<0.05

Table 3 □ Correlation between cephalometric analysis and developing stage (maxillary).

	Nolla's classification		Panorama		Cephalo	
SNA	0.43	**	0.49	**	0.47	**
SNB	-0.45	**	-0.43	**	-0.38	**
ANB	0.68	**	0.71	**	0.69	**
FMA	0.33	*	0.29	*	0.36	*
IMPA	0.36	*	0.35	*	-0.15	
ANS-PNS	0.60	**	0.61	**	0.58	**
Xi-PM	-0.35	*	-0.25		-0.22	
Sa.a.	0.15		0.06		-0.02	
Ar.a.	0.17		0.31	*	0.30	*
Co.a.	-0.06		-0.17		-0.05	
S-N	0.03		0.17		0.13	
Go-Me	-0.03		0.05		-0.05	
Go-Me/S-N	0.09		-0.04		-0.04	
S-Go	0.26		0.27		0.10	
N-Me	0.26		0.34	*	0.25	
PFH/AFH	-0.05		-0.09		-0.18	
S-Ar	0.35	*	0.32	*	0.27	
Ar-Co	0.03		0.03		-0.20	
Ar-Co/S-Ar	-0.26		-0.25		-0.36	*
L6-Mand.b.	-0.22		-0.30	*	-0.04	

**p<0.01 *p<0.05

Table 4 □ Correlation between cephalometric analysis and developing stage (mandibular).

	Nolla's classification		Panorama		Cephalo	
SNA	0.11		-0.03		0.00	
SNB	-0.18		0.42	**	0.16	
ANB	0.16		-0.41	**	-0.16	
FMA	0.18		0.00		-0.02	
IMPA	0.09		-0.41	**	-0.15	
ANS-PNS	0.52	**	0.01		-0.16	
Xi-PM	0.02		0.33	*	0.13	
Sa.a.	0.28		-0.21		-0.26	
Ar.a.	-0.05		-0.04		0.08	
Co.a.	0.00		0.13		0.00	
S-N	-0.15		-0.11		0.12	
Go-Me	0.26		0.33	*	-0.13	
Go-Me/S-N	0.22		0.15		-0.18	
S-Go	0.23		-0.06		-0.02	
N-Me	0.28		-0.05		-0.07	
PFH/AFH	-0.09		-0.01		0.07	
S-Ar	0.27		-0.16		-0.02	
Ar-Co	0.13		0.09		0.03	
Ar-Co/S-Ar	-0.11		0.18		0.03	
L6-Mand.b.	0.21		0.24		-0.22	

**p<0.01 *p<0.05

earlier than in class III patients, as measured by Nolla's stages of development for mandibular and maxillary teeth. The time of eruption of maxillary second molars in class II patients was earlier than in class III patients using both our new panoramic and cephalometric methods. It was surprising to find a significant difference in development of the second molars between the class III patient and the class II patient. These findings suggested to us that we should be very careful in determining the time of application of the face bow to the first molars in class II patients, if we expect it to be effective. The time of eruption and calcification of the maxillary second molar was related significantly to the length of ANS-PNS. The longer ANS-PNS, the faster the time of eruption and calcification. In contrast, there was no significant relationship between the time of eruption and the length of the mandibular bone. We considered that the mandibular second molar was erupting mesiolingually as the compact mandibular bone was very thin on the lingual side. And also there was space for eruption of the second molar mesiolingually. There was a significant relationship between the ANB angle and the time of eruption, as well as the ANB angle and calcification. The smaller the ANB, the earlier the time of eruption and calcification of the mandibular teeth. Timing of treatment is very important, because our growing patients' oral environment changes day by day. We should consider the timing of treatment as well as the kinds of appliances, if the patient has a skeletal malocclusion, because the development of the second molar will differ between class II and class III patients.

REFERENCES

- Hotz, R.: Relation of dental calcification to chronological and skeletal age. *Europ Orthodont Soc Trans*, 35:40, 1959.
- Lebret, L.: Reproducibility of rating stages of tooth formation. *Amer J Orthodont*, 60:90, 1971.
- Logan, W.H.G. and Kronfeld, R.: Development of human jaw and surrounding structure from birth to age of 15 years. *J Amer Dent Assoc*, 20:379-427, 1933.
- Hellman, M.: Development of face and dentition. *Amer J Orthodont*, 26:431, 1940.
- Hurme, V.O.: Ranges of normalcy in the eruption of permanent teeth. *J Dent Child*, 16:11, 1949.
- Lo, R.T. and Moyers, R.E.: Studies in the etiology and prevention of malocclusion of the permanent dentition. *Amer J Ortho*, 39: 460-467, 1953.
- Moorrees, C.; Fanning, E.; Hunt, E.: Age variation of formation stages ten permanent teeth. *J Dent Res*, 42:1490, 1963.
- Garn, S.M. and Rohmann, C.: International of nutrition and genetics in the timing of growth. *Pediat Clin North America*, 13:353, 1966.
- Steedle, J.R. and Proffit, W.R.: The pattern and control of eruptive tooth movements. *Amer J Orthodont*, 87:56-66, 1985.
- Burn-Murdoch, R.A.: The role of the vasculature in tooth eruption. *Eur J Orthod*, 12:101-108, 1990.
- Lee, C.F. and Proffit, W.R.: The daily rhythm of tooth eruption. *Amer J Orthodont*, 38:107, 1995.
- Nolla, C.: Development of the permanent teeth. *J Dent Child*, 27: 254-272, 1960.
- Moyers, R.E.: *Handbook of Orthodontics*, third edition. Chicago: Yearbook medical publishers, Inc., 1973, pp 166-241.
- Suzuki, Y.: Measurements of image on the panoramic tomogram. (in Japanese) *Jpn J Pedo*, 18:591-605, 1980.
- Haruki, T.; Kanomi, R.; Shimono, T.: Application of panoramic radiography for estimation of the crown width of the mandibular permanent lateral teeth in III A dental age. *Pediatric Dent J*, 4(1): 1-10, 1994.
- StatSoft Technical Support: *Statistica for the Macintosh Vol. 1*, pp 375-400. Oklahoma: StatSoft, Inc., 1994.

Inherited dental anomalies and abnormalities

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Familial and twin studies have established the importance of hereditary factors in determining craniofacial form. Similarly, dental size, morphology and development have been shown to be genetically determined in many instances, although it is generally agreed that most genetic influences are polygenic with the expression of traits being influenced by other modifying genes and environmental factors. While the genetic background of the tooth in specific localized anomalies has been documented, recent evidence suggests that the etiology of less specific anomalies, such as impacted teeth, may also have a significant genetic background, although the mechanism of expression is not understood. Furthermore, there seems to be a pattern of association between multiple dental anomalies. Whether the association is a linked genetic relationship or the environmental consequence of one genetically controlled trait is yet to be unraveled. Twin studies will play an important role in this investigation process. Other anomalies that may have a genetic etiology include canine-premolar transposition and primary-tooth submergence. This review reexamines the genetic etiologic aspects of various localized dental anomalies and underlines the importance of an accurate family dental history in diagnosis and treatment planning in both pediatric dentistry and orthodontics.

PATTERNS OF HYPODONTIA AND OTHER ANOMALIES

The reported prevalence rate for missing teeth, excluding third molars, varies depending on the population with mandibular incisors being the most frequently missing teeth in Asians, while in Caucasians it is the maxillary lateral incisor and mandibular and maxillary second premolars.^{1,2} Females are affected more often than males.³ Of increasing interest is the pattern of association between a range of other dental anomalies and hypodontia.

Garn and Lewis studied 100 individuals with one or more missing third molars and noted fifty-three other teeth were also missing.⁴ In 398 controls with all third molars present, however, only seventeen additional teeth were missing. The same authors were able to demonstrate an anteroposterior gradient of relative size reduction of the remaining teeth in 101 subjects with one or more missing teeth (including third molars), compared to 557 control patients without missing teeth.⁵ Keene noted that when one or more third molars were missing, the remaining teeth in both arches were more spaced and less crowded.⁶ The above relationships were more pronounced when all four third molars were missing. This pattern of associated or correlated anomalies has been confirmed by other studies. The association between submergence of primary molars and congenitally absent successors has been well documented. These associations highlight the value of careful clinical recording and radiographic examination in the expectation that where one

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anomaly is present, there is a relatively high probability that a complementary anomaly will also occur.

HEREDITARY NATURE OF HYPODONTIA

An hereditary tendency may be proposed by demonstrating that missing teeth occur more frequently among relatives than in the general population. A study of children with missing teeth found that up to half of their siblings or parents also had missing teeth.⁷ This is higher than the rate of 6 percent found in the general population. Hume reported an interesting example of three siblings, two of whom had missing maxillary canines and maxillary lateral incisors, while the third had all maxillary premolars missing.⁸

Markovic investigated missing teeth in ninety-nine monozygous and sixty-six dizygous twin pairs.⁹ Eight of the nine monozygous twin pairs with missing teeth were completely concordant, while all five dizygous twin pairs were discordant. Other studies have confirmed a relatively high rate of monozygous concordance, although concordance cannot always be expected. The statistical probabilities point to the need, however, for an awareness of a strong genetic link for congenital absence in monozygous twins who may exhibit concordance with similar expression or concordance with variable expression. The family and twin studies outlined reveal the hereditary nature of missing teeth. The exact mode of transmission remains unclear. A single autosomal dominant gene with incomplete penetrance has been suggested.

ANOMALOUS MAXILLARY LATERAL INCISORS

Alvesalo and Portin have provided substantial evidence supporting the view that missing and malformed maxillary lateral incisors are the result of a common gene.¹¹ The prevalence of anomalous maxillary lateral incisors varies for missing, peg-shaped, and microdont teeth. A female preponderance, familial occurrence, and an association with other dental anomalies, including ectopic maxillary canines, maxillary canine-first premolar transposition, and other missing teeth, suggest a polygenic etiology.^{12,13}

ECTOPIC MAXILLARY CANINES

Ectopic maxillary canines occur in about two percent of the population. A female predilection exists and canines are the most frequent ectopically impacted teeth in Caucasians.¹⁴ An investigation of orthodontic patients dem-

onstrating at least one palatal ectopic canine, showed the lateral incisors adjacent to these canines were missing in a high percentage of cases.¹⁵ Bjerklin, Kurol and Valentin investigated 273 children, ninety-three with infraclusion of primary molars, ninety-one with ectopic eruption of maxillary canines, ninety-seven with missing premolars, and ninety-two with ectopic eruption of maxillary first permanent molars. Ectopic eruption of maxillary canines occurred with a higher frequency than normal in children with any of the other three dental anomalies.

HEREDITARY NATURE OF ECTOPIC MAXILLARY CANINES

Repeated patterns of tooth malposition, malformation, and hypodontia (excluding third molars), occur simultaneously in family members in proportions that clearly exceed the proportion of these anomalies occurring in the general population.¹⁷ Zilberman, Cohen, and Becker studied twenty-five patients who had been treated for ectopic maxillary canines and subsequently investigated 105 of their first degree relatives.¹⁵ In the treated propositus group, 64 percent of the ectopic canines were palatal and they were associated with anomalous maxillary lateral incisors in 46 percent of cases. Among the thirty-nine parents, 5.1 percent demonstrated palatally ectopic canines and 30.8 percent had anomalous maxillary lateral incisors. Of the sixty-six siblings, 11 percent had palatally ectopic canines and 28 percent had anomalous maxillary lateral incisors. Peck *et al* concluded that palatally ectopic canines were an inherited trait, being one of the anomalies in a complex of genetically related dental disturbances, often occurring in combination with missing teeth, tooth size reduction, supernumerary teeth, and other ectopically positioned teeth.¹⁴

The normal buccal eruption path of the maxillary canine is exaggerated by an arch-length deficiency in the case of most labially ectopic canines. The absence of local anatomical guiding factors due to anomalous maxillary lateral incisors has been implicated in the etiology of palatally ectopic canines; racial variation and sex linked phenotypic differences, as well as association with other dental anomalies, however, is more suggestive of a polygenic etiology.^{16,17}

MAXILLARY CANINE-PREMOLAR TRANSPOSITION

Maxillary canine-first premolar transposition is a positional interchange of these two teeth. It is a relatively

rare phenomenon with a reported prevalence of about 0.25 percent in the general population; but a study of forty-three orthodontic patients with maxillary canine-first premolar transpositions, revealed one or more missing teeth (excluding third molars) in 37 percent of cases. The most frequently missing teeth were the maxillary lateral incisors and mandibular second premolars. Pegshaped maxillary lateral incisors occurred in 16 percent of cases. In total, 49 percent of individuals had other dental anomalies.¹³

Schachter reported an example of a nine-year-old boy with a unilateral maxillary canine-first premolar transposition and an associated supernumerary tooth.¹⁸ He claimed that "transposition of a tooth, either partial or complete, is a comparatively rare anomaly. It may be considered a symptom, a result of some disturbance early in the developmental period of the teeth. It is often accompanied by other anomalies in the same patient, such as absence or malformation of other teeth, or supernumerary or supplemental teeth."

SUBMERGED PRIMARY MOLARS

The reported prevalence of primary molar submergence varies widely, although comparison of previous studies suggests that about 9 percent of primary dentitions might be expected to show the trait, with mandibular first and second molars most often affected.^{19,20} The siblings of affected children are likely to be similarly affected in about 18 percent of cases. In monozygous twins, nearly identical patterns and degrees of submergence are likely to be found. The association between primary submergence and absence of the succedaneous tooth is well known. These findings along with the fact that a variety of dental anomalies are associated with other missing teeth and maxillary canine-first premolar transposition, implies a significant genetic component in the etiology.

OTHER ANOMALIES

Jarvinen and Vaataja reported a pair of monozygous twins with ectopic eruption of the permanent maxillary first molar occurring bilaterally in one twin and unilaterally in the other.²¹ Oikarinen, Guven and Silaste presented radiographs of a pair of monozygous twins with similarly impacted maxillary and mandibular second and third molars.²² In addition, one of the twins demonstrated a supernumerary tooth germ distal to the maxillary left third molar.

Nik-Hussein and Salcedo illustrated a pair of monozygous twins with multiple dental anomalies.²³ One had a joined maxillary right primary central incisor and supernumerary tooth, missing primary and permanent left lateral incisors and a microdont permanent maxillary left lateral incisor. The co-twin had a joined maxillary left primary central and lateral incisor, joined mandibular left primary lateral incisor and left primary canine and was missing a right primary mandibular lateral incisor, permanent mandibular lateral incisors and a permanent right maxillary lateral incisor.

MIRRORING OF DENTAL ANOMALIES

Mirror imaging is a phenomenon observed in monozygous twins and is characterized by asymmetric occurrence of the same phenomenon. There is no evidence that mirroring arises from the twinning process.²⁴ Characteristics and traits such as handedness, direction of hair whorl and fingerprints are mirrored in monozygous twin pairs. Mirror imaging of missing teeth, eruption disturbances, supernumerary and supplemental teeth, gemination of primary incisors and missing and peg-shaped permanent maxillary lateral incisors, have been reported. The etiology of mirror imaging remains controversial and there is no conclusive evidence to rule out chance occurrence.

SUMMARY

A considerable body of evidence exists to suggest that genes play a dominant role in the etiology of the dental anomalies reviewed. It has been postulated that some kind of genetically controlled interrelationship may exist for some of these coincidental dental anomalies, as evidenced by their frequency of association. It has also been speculated that a "common genetic defect" may give rise to different phenotypic manifestations, including missing, malformed, and even ectopic and malpositioned teeth. The maxillary teeth that develop in the critical marginal areas of the dental lamina, namely the lateral incisors, canines and second premolars, seem most susceptible. Such dental anomalies may be symptoms of an inheritable developmental disturbance of tooth structures.

Knowing that these dental anomalies can be inherited, a familial history and early clinical or radiographic detection of one anomaly, could alert parents and clinicians to the high likelihood of detection of other defects in the same individual and similar defects in other family

members. Early diagnosis is important so that interceptive pediatric and orthodontic opportunities in relation to missing, malformed and ectopically erupting teeth are not overlooked. Further family studies are necessary to reveal the mode of inheritance of some of these dental anomalies and twin studies comparing monozygous and dizygous twins would enable an estimation of the extent of their inheritance.

REFERENCES

1. Davis, P.J.: Hypodontia and hyperdontia of permanent teeth in Hong Kong schoolchildren. *Community Dent Oral Epidemiol*, 15:218-220, August 1987.
2. Graber, L.W.: Congenital absence of teeth: a review of the literature with emphasis on inheritance patterns. *J Am Dent Assoc*, 96:266-275, February 1978.
3. Stamatiou, J. and Symons, A.L.: Agenesis of the permanent lateral incisor: distribution, number and sites. *J Clin Pediatr Dent*, 15: 244-246, Summer 1991.
4. Garn, S.M. and Lewis, A.B.: The relationship between third molar agenesis and reduction in tooth number. *Angle Orthod*, 32:14-18, January 1962.
5. Garn S.M. and Lewis, A.B.: The gradient and the pattern of crown size reduction in simple hypodontia. *Angle Orthod*, 40:51-58, January 1970.
6. Keene, H.J.: Third molar agenesis, spacing and crowding of teeth, and tooth size in caries-resistant naval recruits. *Am J Orthod*, 50: 445-451, June 1964.
7. Grahnen, H.: Hypodontia in the permanent dentition. *Odontol Revy*, 7:1100, Supplement 1956.
8. Hume, W.J.: Oligodontia. A case report. *Br Dent J*, 71:71-72, January 1972.
9. Markovic, M.: Hypodontia in twins. *Swed Dent J Suppl*, 15:153-162, Supplement 1982.
10. Boruchov, M.J. and Green, L.J.: Hypodontia in human twins and families. *Am J Orthod*, 60:165-174, August 1971.
11. Alvesalo, L. and Portin, P.: The inheritance patterns of missing, peg-shaped, and strongly mesio-distally reduced upper lateral incisors. *Acta Odontol Scand*, 27:563-575, December 1969.
12. Brin, I.; Becker, A.; Shalhav, M.: Position of the maxillary permanent canine in relation to anomalous or missing lateral incisors: a population study. *Eur J Orthod*, 8: 12-16, February 1986.
13. Peck, L.; Peck, S.; Attia, Y.: Maxillary canine-first premolar transposition, associated dental anomalies and genetic basis. *Angle Orthod*, 63:99-109, Summer 1993.
14. Peck, S.; Peck, L.; Kataja, M.: The palatally displaced canine as a dental anomaly of genetic origin. *Angle Orthod*, 64:249-256, 1994.
15. Zilberman, Y.; Cohen, B.; Becker, A.: Familial trends in palatal canines, anomalous lateral incisors, and related phenomena. *Eur J Orthod*, 12:135-139, May 1990.
16. Bjerklind, K.; Kuroi, J.; Valentin, J.: Ectopic eruption of maxillary first permanent molars and association with other tooth and developmental disturbances. *Eur J Orthod*, 14:369-375, October 1992.
17. Svinhufvud, E.; Myllarniemi, S.; Norio, R.: Dominant inheritance of tooth malpositions and their association to hypodontia. *Clin Genet*, 34:373-381, December 1988.
18. Schachter, H.: A treated case of transposed upper canine. *Brit Soc for Study of Orthodont Tr*, 76-94, 1951.
19. Kuroi, J.: Infraocclusion of primary molars: an epidemiologic and family study. *Community Dent Oral Epidemiol*, 9:94-102, April 1981.
20. Kuroi, J.: Infraocclusion of primary molars with aplasia of the permanent successor. A longitudinal study. *Angle Orthod*, 54:283-294, October 1984.
21. Jarvinen, S. and Vaataja, P.: Ectopic eruption of the maxillary first permanent molar in twins. *Proc Finn Dent Soc*, 76:175-178, 1980.
22. Oikarinen, V.; Guven, O.; Silaste, H.: Similarly impacted second and third maxillary and mandibular molars in a pair of monozygotic twins. *Dentomaxillofac Radiol*, 19:133-134, August 1990.
23. Nik-Hussein, N.N. and Salcedo, A.H.: Double teeth with hypodontia in identical twins. *J Dent Child*, 54:179-182, May-June 1987.
24. Brown, T.; Townsend, G.C.; Richards, L.C. *et al*: A study of dentofacial morphology in South Australian twins. *Aust Dent J*, 32:81-90, April 1987.

PULSE OXIMETRY AND CAPNOGRAPHY

Prompted by the Harvard Medical School report on standards of monitoring during anesthesia, the American Society of Anesthesiologists has become a leader in the adoption of standards of care and guidelines for practice. As a result, pulse oximetry and capnography (the analysis of carbon dioxide in exhaled air) are now used routinely to monitor general anesthesia in virtually all surgical patients in the United States. Despite optimistic predictions that the use of pulse oximetry would reduce the incidence of hypoxic injuries during anesthesia, in a prospective, randomized study of 20,000 patients at five Danish hospitals, there were no significant differences in cardiovascular, respiratory, neurologic, or infectious complications between patients who were monitored with pulse oximetry and those who were not. However, even a sample of 20,000 patients may be too small to identify a benefit of pulse oximetry, since major complications are fortunately rare. Clearly, noninvasive pulse oximetry provides the physician with an early warning of impending hypoxia. Infrared capnography is useful for diagnosis and the management of esophageal intubation, endobronchial intubation, airway obstruction, bronchospasm, hypermetabolic states, pulmonary embolism, venous air embolism, and cardiogenic shock. In addition, it is useful for identifying mechanical problems in anesthetic circuits.

Wiklund, R.A. and Rosenbaum, S.H.: *Anesthesiology*. *N Engl J Med*, 337:1215-1219, October 23, 1997

Mandibular lateral incisor - canine transposition in monozygotic twins

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Tooth transposition is a rare form of ectopic eruption of permanent teeth. It is characterized by the positional interchange of two adjacent teeth or the development/eruption of a tooth into a position occupied normally by a nonadjacent tooth.

Seven types of tooth transposition have been identified in the human dentition, five in the maxilla and two in the mandible.¹⁻³ They have been classified according to the involved teeth:^{2,3}

- Maxillary canine - first premolar (abbreviated as Mx.C.P1)
- Maxillary canine - lateral incisor (Mx.C.I2)
- Maxillary canine to first molar site (Mx.C to M1)
- Maxillary lateral incisor - central incisor (Mx.I2.I1)
- Maxillary canine to central incisor site (Mx.C to I1)
- Mandibular lateral incisor - canine (Mn.I2.C)

Mandibular canine transmigrated/erupted (Mn.C trans-erupted)

All tooth-transposition types have very low prevalence rates. The most frequently observed transposition is the Mx.C.P1 type, which is found in less than 0.25 percent of orthodontic patients.⁴ Until now, Mx.C.P1 has been the only transposition type with published reports of occurrence among families, including first-degree and sec-

ond-degree relatives.⁵⁻⁸ Mx.C.P1 transposition is an occlusal anomaly which has been shown to be of genetic origin.¹

Mandibular lateral incisor - canine transposition (Mn.I2.C) is one of the rarer types of tooth transposition, having an estimated prevalence rate of 0.03 percent (or 3 cases per 10,000 individuals).^{9,10} This article reports the occurrence of Mn.I2.C transposition in genetically identical-twin sisters. It appears to be the first documentation of familial occurrence of this abnormality.

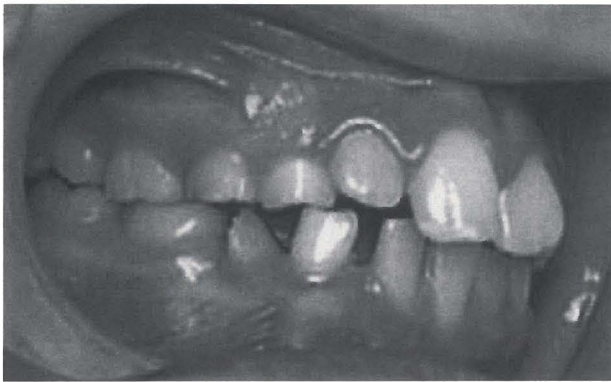
PATIENT HISTORY AND CLINICAL FINDINGS

Two sisters, monozygotic (MZ) twins, presented for orthodontic examinations at 9.1 years of age. Medical history was unremarkable for each of the girls.

Both girls exhibited normal facial form for their age, including strong facial resemblance and the presence of a 3mm interlabial gap at rest position. Both girls had Angle Class I malocclusions in the mixed-dentition stage of development (Figures 1,2). Dental developmental timing and sequencing appeared normal. The maxillary incisors for both girls were erupted and showed tooth rotations and interdental spacing. In the mandibular arch, ectopic distal eruption of the mandibular right lateral incisor was evident for both patients. In one of the twins (A.L.), the ectopic lateral incisor was erupting rotated mesiolingually between the right primary second molar and the right primary canine, having provoked

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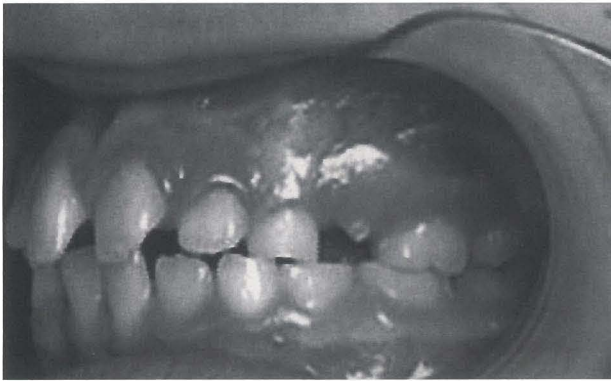
Dr. Hirsh is in San Diego, California.



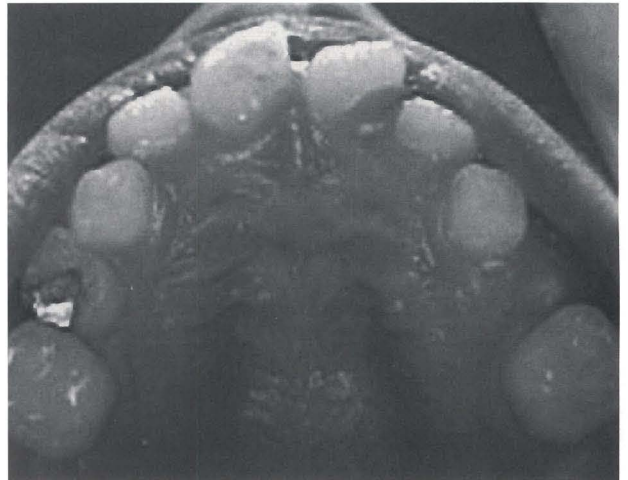
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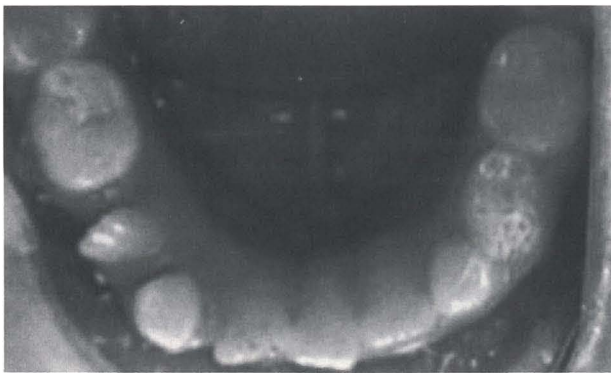
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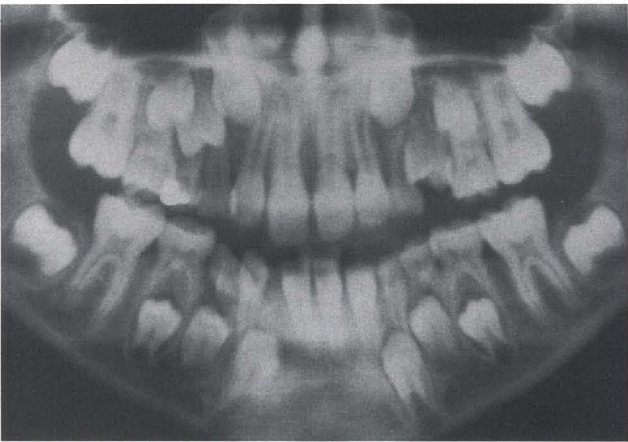
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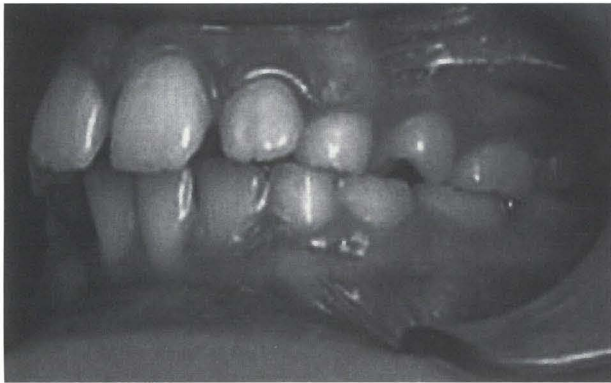
Figure 1 (A-F). Monozygotic twin, A.L., 9.1-year-old girl. Intraoral photographs and panoramic radiograph showing developing mandibular lateral incisor - canine (Mn.I2.C) transposition.



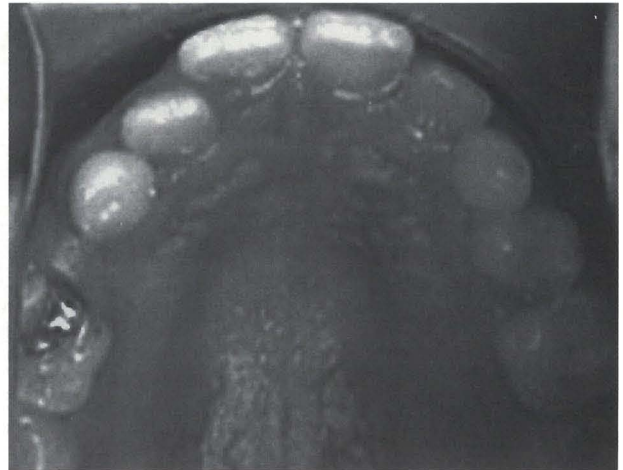
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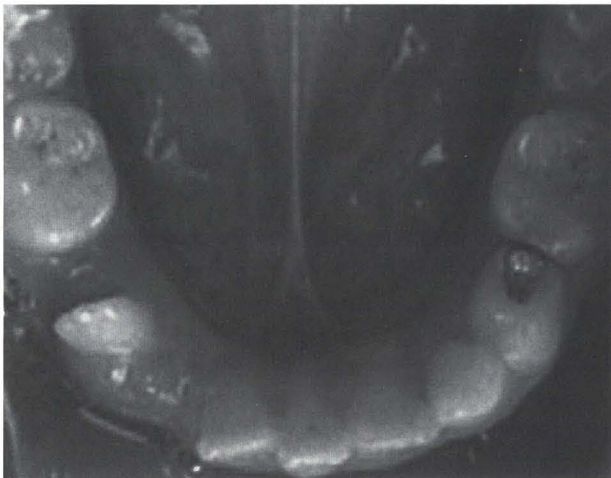
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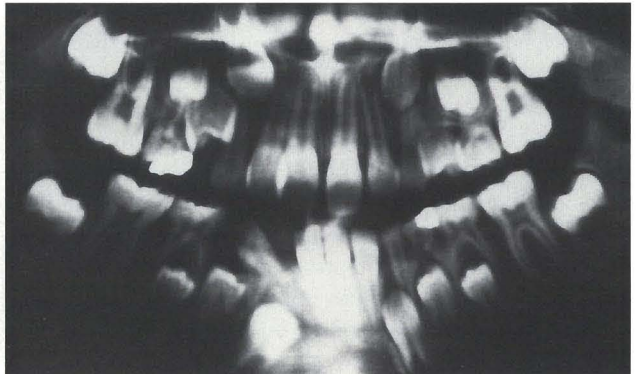
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Figure 2 (A-F). Monozygotic twin, N.L., 9.1-year-old girl. Intraoral photographs and panoramic radiograph showing developing mandibular lateral incisor - canine (Mn.I2.C) transposition.

early exfoliation of the primary first molar. In her twin sister (N.L.), the ectopically erupting lateral incisor had caused early exfoliation of the right primary canine and right primary first molar. Panoramic radiographs (Figures 1,2) for both girls showed that the mandibular right canine was developing mesially to its normal position and that the root apex of the lateral incisor was still in its normal site, contiguous to the central incisor.

The diagnosis for this occlusal abnormality observed in both children is a developing right-side mandibular lateral incisor - canine (Mn.I2.C) transposition. It is characterized by early distal tipping, displacement and mesiolingual rotation of the mandibular right lateral incisor, prompting the adjacent canine to develop transposed mesially.

DISCUSSION

Early-stage Mn.I2.C transposition, such as that present in the MZ twins reported here, is usually diagnosed between ages seven and ten years, at which time the anomaly typically exhibits transposition of the mandibular lateral incisor and canine crowns only, with the roots of these teeth not yet in interchanged positions. Over time these teeth tend to upright. When Mn.I2.C is detected later than age ten years, the crowns of the canine and lateral incisor clearly appear transposed and radiographically the root positions are either fully transposed or superimposed. Usually by age fourteen years, all untreated Mn.I2.C transpositions appear complete.³

Early treatment of the Mn.I2.C transposition between ages seven and ten years yields the best prognosis for interception of the malocclusion and correction of the tooth order. For unilateral occurrence of this transposition, such as seen with these 9.1- year-old twin sisters, extraction of the same-side retained mandibular primary canine and primary lateral incisor, if present, is indicated. Then, a stage of fixed-appliance orthodontic treatment on the mandibular teeth is performed to upright the ectopic lateral incisor into normal position. This simple interceptive treatment strategy assures that the affected mandibular canine will erupt eventually into proper position. In the years after age ten, interception of a Mn.I2.C transposition becomes far more difficult, because during these years the transposition tends to develop naturally into a total positional interchange of the affected teeth.

Unilateral right-side location and female occurrence seem to be predominant patterns of Mn.I2.C expression, based on previous case reports of this occlusal variation

(Peck *et al*).³ In addition, bilateral occurrence of Mn.I2.C transposition has been often noted, as well as the concomitant occurrence of other dental anomalies, such as tooth agenesis.¹⁰⁻¹⁴

The MZ-twin sisters reported here, exhibiting same-side occurrence of Mn.I2.C transposition, show concordance with similar expression for the anomaly. Concordance with either similar or variable expression has been the dominant finding in previous studies of MZ twins exhibiting dental abnormalities, such as tooth agenesis, palatally displaced canines or Mx.C.P1 transposition.^{1,15-17} Given the low estimated prevalence (0.03 percent) of Mn.I2.C transposition in the population, the probability is remote (less than one chance in 3000) that a second occurrence of this rare dental phenotype would be found in the family of a proband simply as a chance happening. More likely, significant genetic influences are operative in the expression of the Mn.I2.C abnormality in these two siblings with identical genotypes.

CONCLUSIONS

Human twin studies are particularly useful in identifying physical variations that may have a genetic etiology. The monozygotic twin girls with mandibular lateral incisor - canine transposition (Mn.I2.C), unilateral same-side expression, featured in this report, provide evidence suggesting genetic transmission of this rare dental anomaly. Early orthodontic intervention, before age ten years, is essential for simple, predictable correction of mandibular anterior tooth order in cases of Mn.I2.C transposition.

REFERENCES

1. Peck, L.; Peck, S.; Attia, Y.: Maxillary canine - first premolar transposition, associated dental anomalies and genetic basis. *Angle Orthod*, 63:99-109, April 1993.
2. Peck, S. and Peck, L.: Classification of maxillary tooth transpositions. *Am J Orthod Dentofac Orthop*, 107:505-517, May 1995.
3. Peck, S.; Peck, L.; Kataja, M.: Mandibular lateral incisor - canine transposition, concomitant dental anomalies and genetic control. *Angle Orthod* (in press).
4. Sandham, A. and Harvie, H.: Ectopic eruption of the maxillary canine resulting in transposition with adjacent teeth. *Tandlaegebldet*, 89:9-11, January 1985.
5. Allen, W.A.: Bilateral transposition of teeth in two brothers. *Br Dent J*, 123: 439-440, November 1967.
6. Payne, G.S.: Bilateral transposition of maxillary canines and premolars: report of two cases. *Am J Orthod*, 56:45-52, July 1969.
7. Newman, G.V.: Transposition: orthodontic treatment. *J Am Dent Assoc*, 94:554-557, March 1977.
8. Feichtinger, C.H.; Rossiwall, B.; Wunderer, H.: Canine transposition as autosomal recessive trait in an inbred kindred. *J Dent Res*, 56:1449-1452, December 1977.

9. Niczky, E.; Müller, K.; Slávik, J.: Transpozície. *Cesk Stomatol*, 67: 227-233, April 1967.
10. Järvinen, S.: Mandibular incisor-cuspid transposition: a survey. *J Pedodont*, 6:159-163, Winter 1982.
11. Pifer, R.G.: Bilateral transposed mandibular teeth. *Oral Surg*, 36: 145, July 1973.
12. Shapira, Y.: Bilateral transposition of mandibular canines and lateral incisors: orthodontic management of a case. *Br J Orthod*, 5(4): 207-209, 1978.
13. Kryshchalskyj, B.: A rare case of bilateral mandibular canine-lateral incisor transposition. *Ontario Dent*, 59:31-35, July 1982.
14. Werner, A.: Transposicion bilateral en la mandibula. *Odontol Chilena*, 37:274-276, December 1989.
15. Gravely, J.F. and Johnson, D.B.: Variation in the expression of hypodontia in monozygotic twins. *Dent Practit*, 21:212-220, February 1971.
16. Markovic, M.: Hypodontia in twins. *Swed Dent J*, 15 Suppl:153-162, 1982.
17. Kotsomitis, N.; Dunne, M.P.; Freer, T.J.: A genetic aetiology for some common dental anomalies: a pilot twin study. *Austral Orthod J*, 14:172-178, October 1996.

MUSCULOSKELETAL SYMPTOMS IN NEW SOUTH WALES DENTISTS

The findings from the present study clearly indicate that current work practices by dentists, whether reporting the use of four-handed dentistry or otherwise, do not prevent musculoskeletal or neurological symptoms. Only 18 percent of those studied were symptom-free. In fact, the significant increase in the frequency of pain reported by the group claiming to practice four-handed dentistry may be due to the reduction of movement associated with greater utilization of a dental assistant or with longer periods of working without taking a break. The latter finding may not have been apparent in the sample as a whole because of the binomial distribution of working periods reported. Alternatively, those who reported that they practiced four-handed dentistry may not have received formal training in this method of practice.

The high prevalence of musculoskeletal symptoms in dentists is a common feature of visually dependent occupations in which the visual demands require the adoption of fixed postures for extended periods of time. Other occupations with high visual demands in which the prevalence of musculoskeletal symptoms is also high include musicians and draftsmen. It has been hypothesized that the type and/or severity of symptoms might be related to the duration of working without a break. In both the present study and that of Rundcrantz *et al.*, no relationship has been identified.

The present study found that distal neurological symptoms in the upper limb (finger weakness and paraesthesia) were more common in the dominant than non-dominant arm, that shoulder pain did not vary substantially according to dominance and that bilateral presentation of symptoms was common. Similar findings were reported by Milerad and Ekenvall, who also found that the relative risk of developing neurological symptoms in the dominant hand in dentists was high when this group was compared with pharmacists. These investigators likened the symptoms to those of Raynaud's disease and postulated that the symptoms may be associated with the use of high speed dental equipment.

Marshall, E.D. *et al.*: Musculoskeletal symptoms in New South Wales dentists. *Australian Dent J*, 42:240-246, August 1997.

Temporary and permanent restorations for fractured permanent teeth with immature apices: A clinical study

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A frequent reason for seeking dental treatment is fracture of the anterior teeth, especially among the school-age population. Statistics show that annually 1.5 percent to 3.5 percent of the children in sports suffer dental trauma. These injuries are more frequent in patients with anterior overjets.¹⁻³

Many authors have pointed out that fracturing a permanent tooth is a tragic experience for both child and parents, who are more concerned with the esthetic rather than the symptomatic aspects of the problem. This causes an apprehensive behavior demanding that the dentist restore "normalcy" as soon as possible.⁴⁻⁶

When it is a matter of fractures involving the enamel and dentin, the primary objective should be to assure pulp vitality. The tooth should be protected from thermal or chemical irritation of the exposed dentin, as well as from bacteria that could enter the dental tubules. It is also important to stabilize the position and restore the anatomic form of the tooth when the points of contact are lost as a consequence of an extensive crown fracture, in order to prevent malposition or over-eruption.^{4,7-9} When it is a matter of trauma involving incisors with

incomplete apical growth, therapy should include protection of the pulp and evaluation of pulpal response, to ensure continuation of apical growth.

Conventionally, therapies used to treat these cases have used the temporary restorative techniques that emphasize preservation of pulp vitality and thus assure physiological completion of the apices. In one of these techniques, a metal band is cemented with zinc-oxide-eugenol cement and, if deemed advisable, the pulp is protected with calcium hydroxide. This option requires the patient to undergo provisional treatment for a period of time in which emphasis is placed on maintaining pulp vitality, rather than on esthetics. Presumably, restorative materials could affect the traumatized dental tissues unfavorably.^{1,4,7,9,10} The use of this technique is based on the premise that definitive restoration is only advisable when the repair of the damaged dentin and completion of the apical foramen have occurred.

Today, however, the biological and physical properties of dental materials currently available offer the use of a technique that will protect the pulp and provide an esthetic restoration without affecting the health of the pulp.

Reported here is a comparative evaluation of two techniques for treating Ellis Class I, II, or III fractures

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Table 1 Evaluation of pulp vitality at 3, 6 and 12 months in fractured permanent anterior teeth reconstructed with metal bands and crown techniques. Maracaibo, State of Zulia, 1.995.

Time	Band technique						Crown technique					
	3 months		6 months		12 months		3 months		6 months		12 months	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Pulp vitality												
Yes	40	91.0	40	91.0	40	91.0	40	97.5	40	97.5	40	97.5
No	4	9.0	4	9.0	4	9.0	1	2.4	1	2.4	1	2.4
Totals	44	100.0	44	100.0	44	100.0	41	100.0	41	100.0	41	100.0

of permanent teeth with physiologically incomplete apices. The criteria for evaluation were pulp vitality and apical growth. The first technique called for protection of the pulp as the primary concern and esthetics as of secondary concern; the second technique was equally concerned with pulp vitality and with restoration of the esthetic qualities of the human tooth.

MATERIALS AND METHODS

The techniques were used on equal numbers of the study sample of fifty-six patients of both genders, ages seven to eleven years, all of whom had at least one permanent incisor with an Ellis Class I, II or III fracture with a physiologically incomplete apex. The following clinical steps were followed with all subjects:

- Isolating the tooth, using a lip retractor and cotton rolls.
- Cleaning the affected area with a physiological salt solution.
- Cleaning the tooth with a rubber cup and a mixture of pumice stone and water.
- Washing, irrigating and protecting the dentin-pulp with calcium hydroxide (Life, Ken Dental Products).

Fifty percent of the sample were restored using the conventional technique: metal bands (if needed) cemented with zinc-oxide-eugenol cement, to which zinc acetate was added as accelerator. The bands were constructed from a metallic matrix material between 0.005 and 0.180 mm thick, that can be adapted to the tooth with no pressure and electrically soldered to another piece of matrix material extending from the labial surface to the lingual surface in an attempt to restore the original height of the incisor. For the patients in the remaining 50 percent of the sample, the fractured teeth were restored with photopolymerizable glass ionomer (Vitrebond™, 3M Dental Products) and a photopolymerizable resin (Silux™ 3M Dental Products). All patients were clinically examined after three, six and twelve

months. The pulp vitality was determined, using mechanical and electrical tests. A radiographic evaluation was also made to determine the state of apical growth, using apical convergence as the criterion.

RESULTS

The sample comprised 53.6 percent males and 46.3 percent females: 92.7 percent of the fractures were located in the maxilla; 77.4 percent were Ellis Class II, (fractures involving both dentin and enamel); 51.7 percent of the fractures were a week or less old; and existing overjets increased their degrees of overjet in 73.2 percent of the cases.

When the cases were evaluated at three, six and twelve months, it was found that, of a total of forty-four teeth reconstructed using the band technique, 91.0 percent maintained their vitality in the first and successive evaluations. Of the teeth reconstructed with crowns, 97.5 percent maintained their vitality in the first and successive evaluations (Table 1). Application of Fisher's exact test verified that there is no significant relationship between the type of restorative technique used and the preservation of pulp vitality.

Radiographs showed that physiological completion of the apices of the roots occurred after three months in 17 percent of the cases restored with the crown technique, as opposed to only 9 percent of those treated with the band technique. A similar difference was observed at six months: 48.7 percent of the cases treated with the crown technique had completed apices, whereas only 43.1 percent showed apical completions using the band technique. At twelve months, 73.1 percent of the cases reconstructed with the crown technique achieved completion of the apices, as opposed to only 54.5 percent of those treated with the band technique (Table 2 and Figure).

The statistical analysis proved that there was no significant relationship between the type of restoration and the time at which the physiological completion of the apices occurred ($p > 0.05$).

Table 2 □ Time at which radicular formation ends, in accordance with the type of technique used. Maracaibo, State of Zulia, 1.995.

Technique	Crown			Band		
	No.	%	Accumulated frequency	No.	%	Accumulated frequency
3 months	7	17.0	17.0	4	9.0	9.0
6 months	13	31.7	48.7	15	34.1	43.1
12 months	10	24.3	73.1	5	11.3	54.5
+12 months	11	26.8	100.0	20	45.4	100.0
Totals	41	100.0	100.0	44	100.0	100.0

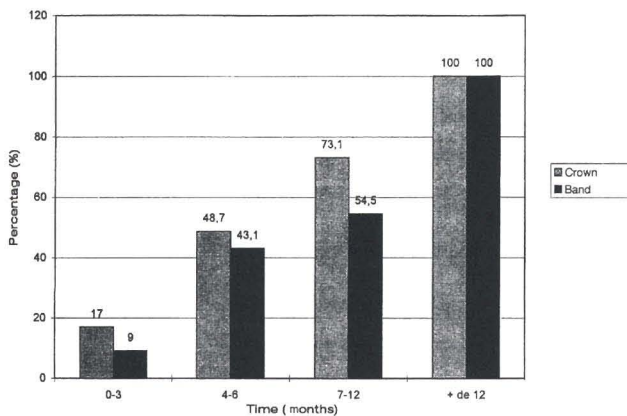


Figure. Time at which radicular formation ends, in accordance with the technique used. Maracaibo, State of Zulia, 1.995.

DISCUSSION

The results of this study corroborate the hypothesis that immediate restoration of a fractured incisor (Ellis Class I, II, or III) does not affect the vitality of its pulp and the growth of its apex. In fact the full restoration appears to enhance the developmental process, based on radiographic observations. The findings suggest an alternative to Andreasen, who recommends definitive restoration only when radicular formation is complete.¹

Furthermore, this study corroborates data reported in the literature with respect to the production and distribution of traumatic dental injuries: a predominance of injuries among males, a greater frequency among school-aged children, a greater incidence in maxillary teeth.^{1,2,8,10-12} Falls precede most fractures, and the

greatest percentage of injuries is found in patients with pronounced overjets.^{1,7}

CONCLUSION

From the results of this study, it may be concluded that both the band and crown restoration techniques allow preservation of pulp vitality and the physiological completion of apical growth in fractured permanent teeth with immature apices. The use of the crown technique allows, however, a quick restoration of the esthetics, anatomy, and function of the fractured tooth.

REFERENCES

- Andreasen, F.M. and Daugaard, Jensen J.: Treatment of traumatic dental injuries in children. *Tandlaegermess-Tidsskr*, March (3): 76-89, 1992.
- Forsberg, C.M. and Tedestam, G.: Traumatic injuries to teeth in Swedish children living in an urban area. *Swed Dent J*, 14(3):115-122, 1990.
- McLean, John W.: Clinical applications of glass-ionomer cements. *Operative Dentistry*, Supplement 5, 1992,184-190.
- Maiwald, H.J.: Therapy for fractured crown of juvenile permanent teeth. *Dtsch Stomatol*, October, 40 (10):426-427, 1990.
- Garcia-Godoy, F.: Reason for seeking treatment after traumatic dental injuries. *Endodont Dent Traumatol*, 5 (4):180-181, 1989.
- Garcia-Godoy, F.: Reasons for traumatic injuries to teeth in Swedish children living in an urban area. *Swed Dent J*, 14(3):115-122, 1990.
- Andreasen, J.O.: Lesiones traumáticas de los dientes. Segunda Edición. Barcelona, España: Labor, S.A., 1980, pp 8-60.
- Grossman, L.: *La Práctica Endodóntica*. Buenos Aires: Editorial Mundi, S.A.I.C. y F. 1993 Cap.18, pp 346-349.
- Kaba, A.D. and Maréchaux, S.C.: A fourteen-year follow-up study of traumatic injuries to the permanent dentition. *J Dent Child*, 56: 417-425, November-December 1989.
- Andlaw, R.J. and Rock, W.P.: *Manual de Odontopediatría*. Segunda Edición Editorial Interamericana, 1989, pp 187-191.
- Rusmach, M.: Traumatized anterior teeth in children. A 24-month follow up study. *Australian Dent J*, 35 (5):540-543, 1990.
- Medina, M.: Fractura Coronaria en dientes permanentes antero-superiores. Evolución. Trabajo de Ascenso. Facultad de Odontología, LUZ, 1993.

Intentional extraction and reimplantation of an immature invaginated central incisor

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Dens invaginatus, also known as dens in dente is a condition in which there is invagination of ameloblasts into the dental papilla that results in a malformed tooth that appears to have a separate odontogenic entity developing within it.¹⁻³ The stimuli for the invagination of ameloblasts remains obscure at this time, but some have speculated that it may be a result of

- Apical proliferation of ameloblasts.
- Local growth retardation.
- Increased external pressure.
- Inadequate nutrition of the epithelium.^{1,4}

The diagnosis of dens invaginatus is often made only after clinical signs and symptoms of pulpal necrosis and acute periapical periodontitis are present.^{1,2,5} Early pulpal necrosis that occurs soon after the eruption of the tooth suggests that there is communication between the external surface of the invagination and the dental pulp.^{1,5-8} The complications of this condition can range from mild discomfort to an acute odontogenic infection with associated cellulitis.^{1,5,8}

Dens invaginatus has been categorized by three types:

- Type I - an enamel lined cavity confined within the crown of the tooth.
- Type II - an enamel lined cavity that invades the root but remains within its confines (may or may not be in communication with the pulp).
- Type III - the invagination extends beyond the CEJ and perforates apically or laterally at a foramen (there is no communication with the pulp).⁸

The incidence associated with dens invaginatus has been reported to range from .25 percent to 10 percent.^{2,6,7,9-11} This condition occurs only in the permanent dentition and is most commonly seen in maxillary permanent lateral incisors, followed by central incisors, premolars, canines and molars.^{3,6,8,9}

Treatment of a tooth that exhibits dens invaginatus is a complicated process. Treatment modalities range from prevention, conventional endodontic therapy, surgical endodontic therapy, intentional reimplantation, and extraction.^{2,8,10,11} No absolute method of treatment can be proposed for dens invaginatus, because of the variables of the malformation; treatment is based, therefore, on the patient's signs and symptoms of the anomaly. If the diagnosis is made before the patient experiences any associated morbidity, the treatment is based on prevention of pulpal contamination by sealing the invagination with composite resin or amalgam and maintaining good oral hygiene.⁷

If associated morbidity exists, the best course of treatment will be determined by clinical considerations.⁷ Access to the pulpal chamber is often difficult because

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signs of morbidity may be present in a young child with a tooth that may not be fully erupted.^{3,7,9} In a young child, multiple visits that require important patient cooperation may be necessary.^{3,7} A complex morphology can make proper instrumentation impossible; a combined endodontic and surgical endodontic approach, therefore, may be necessary.^{3,7} Root maturity may indicate an apexification procedure.^{3,7,9}

The following presentation reflects the fact that treatment associated with dens invaginatus is complex, and no one treatment modality can fit every case. After careful evaluation of the patient and the invaginated tooth, the decision was made that the best chance for success would be attained by attempting an intentional reimplantation of the tooth with the dens formation.

CASE REPORT

E.D. (9 yr 7 mo) presented to the primary author's office for evaluation of spontaneous pain associated with the maxillary right central incisor. Clinical examination reveals a mixed dentition with Class II skeletal and Class II dental occlusion; bilateral posterior crossbite; anterior open-bite, secondary to an active digit habit; premature loss of the maxillary right primary canine and associated midline shift; asymmetry in the mesiodistal width of the central incisors; a mild amount of dental caries; and good oral hygiene. Radiographic examination revealed an invaginated left central incisor (Figure 1). The patient's symptoms indicated an irreversible pulpitis. A diagnosis of dens invaginatus was made and a sealant was placed on the lingual surface of the tooth. Antibiotic treatment was begun and the patient was referred for endodontic treatment.

Consultation presented a number of treatment options, all with limited prognosis, due to the complex nature of the tooth's root canal system. The treatment options included:

- Conventional endodontics (would probably allow proper instrumentation of the distal canal system, but not the invaginated canal system, because it is so deep within the tooth [Figure 1]).
- Endodontic surgery to fill the apices; this option was rejected, however, due to the angulation of the tooth resulting from an active digit habit, and the close proximity of the developing permanent canine.
- Extraction and intentional reimplantation. Risks and benefits of this option were explained, including loss of the tooth and possible ankylosis.

A conservative approach was taken initially to allow



Figure 1. Radiographic presentation of the maxillary right central incisor.

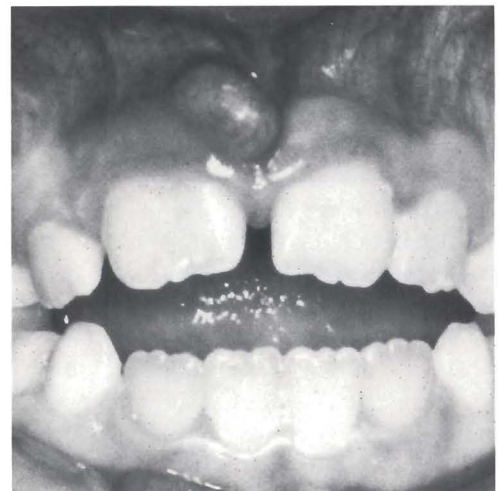


Figure 2. Clinical presentation before treatment. Note evidence of periapical granuloma.

the operator GKP to consult with colleagues about appropriate treatment and other possible options. The patient's parents were primarily concerned with the child's discomfort. They were informed early that regardless of the treatment, the long-term prognosis was poor.

During this time a draining fistula developed (Figure 2), and as a result, conventional endodontics was begun with the hope that this might resolve the patient's symptoms. Access into the distal or main (normal) root canal



Figure 3. Radiographic evaluation after treatment.

system revealed a vital pulp. Instruments were used 8-10 mm into the canal. The canal was washed, a small amount of formocresol was placed and a caviti temporary restoration was inserted. No access to the invaginated canal system was achieved; an intentional reimplantation, therefore, was scheduled.

A local anesthetic was administered and the soft tissues were gently released. The incisor was extracted, including a large mass of granulation tissue measuring 6 × 7 × 8 mm. The apical tissues were removed from the tooth and the latter was transferred to a wet sterile gauze. The crown was held with the wet gauze during instrumentation. Care was taken to prevent damage to the radicular soft tissues throughout the procedure. The canals were debrided, washed, dried and filled with a thick mix of PCA sealer. The tooth was replaced in the socket, the soft tissues were adapted, and the tooth was stabilized with an orthodontic wire (Figure 3).

Following treatment the patient was asymptomatic. The orthodontic splint was left in place for four weeks. After removal of the splint, mobility of the tooth was within normal limits. At the six-month follow-up visit, the tooth was slightly discolored and eruption asymmetry was apparent (Figure 4). Thirteen months posttreatment the eruptive asymmetry and discoloration are more apparent, but the patient remains symptom free (Figure 5).



Figure 4. Six-month postoperative photograph.



Figure 5. Thirteen-month postoperative photograph. Note failure of maxillary right central incisor to continue to erupt.

DISCUSSION

Success of any form of treatment is subjective. In this case, before treatment the professional consensus was that the tooth would be lost. To date the prognosis is guarded.

The lack of eruptive symmetry has been a cause of concern for the authors (Figure 5). Initially this was thought to be a result of ankylosis, but the tooth does not exhibit any of the classic signs of ankylosis. The invaginated incisor has maintained a physiologic mobility that is comparable to the adjacent tooth. This movement appears to be between the tooth and the periodontal ligament, rather than a result of flexure of the alveolar process associated with ankylosis. Auscultation of the teeth during percussion testing has yielded results that

were determined to be within normal limits and similar to results from the adjacent teeth. During treatment the eruptive potential of the invaginated incisor was severely disrupted. Conservative treatment options have been recommended, due to the guarded prognosis. The eruptive asymmetry and the discoloration can be treated with composite veneering until the prognosis is more firmly established.

This case illustrates some of the difficulties of managing an invaginated incisor with a complex radicular morphology. Intentional reimplantation may be a viable treatment option, but may be better suited for those cases where root maturity has occurred and eruption is complete.

REFERENCES

1. Benyon, A.D.: Developing Dens Invaginatus (Dens in Dente). *Br Dent J*, 153(7):255-260, October 1982.
2. Schindler, W.G. and Walker, W.A.: Continued root development after apexification of an immature tooth with dens invaginatus. *J Endod*, 9:430-433, October 1983.
3. Hicks, M.J. and Flaitz, C.M.: Dens invaginatus with partial coronal agenesis: Report of a case. *J Dent Child*, 52:217-219, May-June 1985.
4. Wells, D.W. and Meyer, R.D.: Vital root canal treatment of a dens in dente. *J Endod*, 19:616-617, December 1993.
5. Hata, G. and Tuda, T: Treatment of dens invaginatus by endodontic therapy, apicocurretage, and retrofilling. *J Endod*, 13:469-472, September 1987.
6. Kaufman, A.Y.; Kaffe, I.; Littner, M.M.: Vitality preservation of an anomalous maxillary central incisor after endodontic therapy. *Oral Surg-Oral Med-Oral Path*, 57: 668-672, June 1984.
7. Rothstein, I.; Stabholz, A.; Heling, I. *et al*: Clinical considerations in the treatment of dens invaginatus. *Endod-Dent-Traumatol*, 3: 249-254, October 1987.
8. Rakes, G.M.; Aiello, A.S.; Kuster, C.G. *et al*: Complications occurring resultant to dens invaginatus: case report. *Ped Dent*, 10: 53-56, March 1988.
9. Ferguson, F.S.; Friedman, S.; Frazzetto, V.: Successful apexification technique in an immature tooth with dens in dente. *Oral Surg-Oral Med-Oral Path*, 49:356-359, April 1980.
10. ElDeeb, M.E.: Nonsurgical endodontic therapy of a dens invaginatus. *J Endod*, 10:107-109, March 1984.
11. Szajkis, S. and Kaufman, A.Y.: Root invagination treatment: A conservative approach in endodontics. *J Endod*, 19:576-578, November 1993.
12. Feinglass, J.C.: Reimplantation treatment for infected dens in dente. *Dental Survey*, 52:48-49, July 1976.

THE ENIGMA OF LOW BIRTH WEIGHT AND RACE

The racial identification of African Americans is at best speculative. The authors estimate that African Americans derive one quarter of their genetic makeup from Europeans, and in their study they apply this figure uniformly to all African Americans. Individual blacks' racial backgrounds cover the entire spectrum, from virtually no European bloodline to nearly all European. Furthermore, a substantial percentage of African Americans have Native-American lineage, a factor the authors did not consider.

To look for a genetic cause of the difference in birth weight between the races is premature until the sociocultural questions have been answered. This has not been done. Race very often serves as a proxy for poverty. The importance of "nonmedical" barriers to a good health care outcome has not been fully appreciated by the medical profession. Medical sociologists recognize attitudinal and organizational barriers to health care. Attitudinal barriers are those that prevent individual persons from becoming motivated to seek needed health services. Organizational barriers, in contrast, prevent motivated persons from obtaining the services they know they need. Examples of attitudinal barriers are cultural isolation, fatalism, customs, superstition, and fear. Examples of organizational barriers are lack of health insurance, lack of transportation, poor access to health care providers, a paucity of health care facilities, and the like. It is known that these barriers are greatest in minority communities.

Foster, Jr., H.W.: The enigma of low birth weight and race, (Editorial).
N Engl J Med, 337:1232-1233, October 23, 1997.

EPIDEMIOLOGY

Caries experience in inner-city preschoolers at the time of their initial dental visit

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A recent review indicated that between 1980 and 1986, the number of preschool children in the U.S. increased by almost 11 percent.¹ Most preschoolers live in metropolitan areas.² Government data indicate that of the 10.8 million two- to four-year-olds residing in the U.S. in 1986, 3.2 million (29.6 percent) lived in central (inner-city) metropolitan statistical areas, 5.1 million (47.2 percent) lived in noncentral metropolitan statistical areas and 2.5 million (23.2 percent) lived in nonmetropolitan statistical areas.² Reports also indicate that those residing in inner-city metropolitan statistical areas were more likely to live in households whose income was below the poverty level, compared to children who lived in non-inner-city areas.²

Data regarding caries experience in preschool-age children, especially in the younger segment of preschoolers, are scant. The few international reports on caries prevalence in young preschoolers have shown steep increase in caries experience with age. Internationally, caries has been reported to affect 0.5-2.0 percent of children at around one year of age, 7.7 percent to 26.3 percent at around two years of age and 28.0 percent to 36.6 percent at around three years of age.³⁻⁵ In the United States, reports regarding caries in pre-

schoolers generally relate to children three years of age and older. Johnsen *et al* reported that for Head Start children in Ohio (three and a half to five years of age), caries experience ranged from 50 percent of those residing in optimally fluoridated nonurban areas to 64 percent in nonfluoridated urban areas.⁶ Data from broadly based assessments of caries experience in children under three years of age in the United States have not been fielded. The dearth of information on children less than three years old is noteworthy, because Louie *et al* have suggested that most dental caries in primary teeth occurs shortly after tooth eruption.⁷ Although baby bottle tooth decay—an oftentimes severe form of caries that typically affects young preschoolers—continues to receive considerable attention, the broader issue of caries experience in preschoolers has, by and large, been neglected.⁸

The lack of caries experience data on preschoolers, coupled with the observation that low socioeconomic status has been shown to be associated with increased risk for caries development, underscores the importance of fielding data on caries prevalence in inner-city preschoolers.⁹ Thus the objective of this retrospective study was to determine caries experience in a sample of inner-city children aged five years and under at the time of their initial dental visit (i.e., for preschoolers with no history of previous dental treatment).

MATERIALS AND METHODS

Clinical and demographic data for all children aged five

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years and under who presented for initial treatment during a continuous six-month period in 1991 were obtained from the records of an inner-city community health center clinic located in a fluoridated area (n=103). Radiographs had been taken where indicated. Children who had received prior dental treatment were excluded. Data for children who could not be treated in an outpatient setting (typically children less than three years of age with severe caries experience requiring hospitalization) were not included.

Caries prevalence, caries distribution by surface and caries severity were determined. Caries severity was determined by noting the number of decayed surfaces (ds) and the percent of all teeth present that were found to be carious (number of carious teeth/total number of teeth). The percent carious teeth factor was used because some children in the sample did not have all twenty primary teeth erupted at the time of their initial visit. For purposes of analysis, the sample was divided into two groups based on the age of the children at their first visit: under three years of age and three to five years of age. Caries pattern assignment was done for the caries-positive children using the "caries analysis system" as described by Douglass *et al.*¹⁰ Children could be included in more than one category during caries pattern assignment.

RESULTS

Sociodemographic characteristics

The sample consisted of fifty-eight males and forty-five females (Table 1). The mean age of the subjects was 44.1 months (S.D. \pm 10.5 months). The sample was comprised of predominantly African-American children (73.8 percent), with Hispanic (20.4 percent), non-Hispanic Caucasian (2.9 percent) and other children (2.9 percent) constituting the rest. The majority (80.6 percent) were three-to-five-years old at the time of their initial dental visit. Ninety-seven subjects (94.2 percent) were covered by Medicaid, while six children (5.8 percent) were self-pay/commercially insured patients.

Caries experience

Overall 67 percent of preschool subjects who presented to the inner-city clinic for their first dental visit exhibited caries (Table 2). Those under three years of age averaged 4.30 decayed surfaces, while those aged three-to-five-years averaged 7.08 decayed surfaces. Mean caries prevalence increased with age for all surfaces. Buccal/

Table 1 □ Demographic characteristics of sample subjects.

Number (and percent) of children	Under 3 years	3-5 years	Totals
Males	13 65.0%	45 54.2%	58 56.3%
Females	7 35.0%	38 45.8%	45 43.7%
Totals	20 19.4%	83 80.6%	103 100.0%
Mean Age (months)	27.7	48.1	44.1
\pm S.D. of Children	\pm 5.7	\pm 6.9	\pm 10.5

Table 2 □ Mean number of decayed surfaces (ds) and standard deviations by surface and age.

Mean ds for:	Under 3 years	3-5 years	Totals
All surfaces	4.30 \pm 6.00	7.08 \pm 7.34	6.54 \pm 7.16
Occlusal surfaces	1.45 \pm 2.48	3.24 \pm 3.20	2.89 \pm 3.15
Proximal surfaces	1.10 \pm 2.27	1.90 \pm 3.04	1.75 \pm 2.91
Buccal/lingual surfaces	1.75 \pm 2.51	1.94 \pm 2.79	1.90 \pm 2.73
Percent experiencing caries:	45.0%	72.3%	67.0%

Table 3 □ Mean caries levels and standard deviations, and levels of caries severity (ds and percent) for caries-positive children.

Mean ds:	Under 3 years	3-5 years	Totals
All surfaces	9.56 \pm 5.39	9.80 \pm 6.92	9.77 \pm 6.70
Occlusal surfaces	3.22 \pm 2.86	4.48 \pm 2.93	4.32 \pm 2.93
Proximal surfaces	2.44 \pm 2.92	2.63 \pm 3.30	2.61 \pm 3.23
Buccal/lingual surfaces	3.89 \pm 2.37	2.68 \pm 2.97	2.84 \pm 2.91
Caries severity (ds):	Number (and percent) of children		
ds: 1-5	3 (33.3%)	17 (28.3%)	20 (29.0%)
ds: 6-10	2 (22.2%)	23 (38.3%)	25 (36.2%)
ds: 11-15	3 (33.3%)	7 (11.7%)	10 (14.5%)
ds: > 15	1 (11.2%)	13 (21.7%)	14 (20.3%)
Caries severity (percent):	Number (and percent) of children		
Carious teeth: 1-25%	2 (22.2%)	26 (43.3%)	28 (40.6%)
Carious teeth: 26-50%	5 (55.6%)	25 (41.7%)	30 (43.5%)
Carious teeth: 51-75%	2 (22.2%)	8 (13.3%)	10 (14.5%)
Carious teeth: 76-100%	0	1 (1.7%)	1 (1.4%)
Total children	9 (13.0%)	60 (87.0%)	69 (100.0%)

Table 4 □ Caries patterns analysis for caries-positive children.

Number (and percent) of children	Under 3 years	3-5 years	Totals
Fissure caries	6 66.7%	51 85.0%	57 82.6%
Maxillary anterior smooth surface caries	8 88.9%	29 48.3%	37 53.6%
Posterior proximal caries	0	25 41.7%	25 36.2%
Posterior buccal/lingual smooth surface caries	1 11.1%	9 15.0%	10 14.5%
Total children	9 (13.0%)	60 (87.0%)	69 (100.0%)

lingual caries was the predominant type in subjects under three years of age, with a mean of 1.75 decayed surfaces. Occlusal caries was predominant in three-to-five-year-olds, with a mean of 3.24 decayed surfaces.

Mean caries prevalence for the caries-positive group was approximately one and a half times the mean for the entire sample (Table 3). Caries severity data demonstrated very high levels of disease ($ds > 10$) in about one-fourth of the sample, irrespective of age. Caries severity data also showed that two-fifths of the sample had more than 25 percent of their teeth decayed at the time of their initial visit, irrespective of age.

Caries patterns

Maxillary anterior smooth surface caries was predominant (88.9 percent) under three years of age, while fissure caries was the most observed pattern (85.0 percent) at three to five years of age (Table 4). Prevalence of maxillary anterior smooth surface caries declined with age, while fissure caries and posterior buccal/lingual smooth surface caries increased with age. Posterior proximal caries was not seen under three years of age but increased significantly at three to five years of age affecting approximately two-fifths of the children (41.7 percent).

DISCUSSION

The predominance of three-to-five-year-olds in this sample probably reflects the usual utilization trend whereby children are more likely to access dental care with increasing age. This situation parallels data from a 1989 National Health Interview Survey that demonstrated that only about one-third of two-to-four-year-olds had visited a dentist in the preceding year, a rate that was less than half of that observed for five-to-seventeen-year-olds.¹¹

Data from this sample of preschool-aged children are consistent with the findings of the National Preventive Dentistry Demonstration Program which noted that children from low socioeconomic segments of the population exhibit high levels of caries prevalence and untreated disease.⁹ The mean number of decayed surfaces in our sample was 6.54, almost twice the level of 3.40 mean dfs reported by the National Institute of Dental Research for a random sample of U.S. children at five years of age in 1986-1987, but considerably less than the mean dfs of 9.85 reported for three-and-a-half-to-five-and-a-half-year-old children in USDHHS Region IX Head Start programs in 1986-1987.^{7,12} Our caries severity data also demonstrate very high levels of disease in one-fourth to two-fifths of the sample, depending on the severity criteria used, irrespective of age. These results are consistent with the findings of Margolis *et al* who

reported that a segment of the child population experiences severe dental disease.¹³

The percentage of caries-free children in this sample decreased with age, while caries levels increased for all surface types. The observation that 72.3 percent of the three-to-five-year-old children in our sample had experienced caries, a figure that is considerably higher than the 42.0 percent prevalence reported for three-year-old Head Start children in Connecticut, undoubtedly is due in part to the nature of our sample (i.e., individuals seeking care versus all Head Start enrollees) and the higher age-range of our subjects.¹⁴ These factors also probably explain the greater mean decayed surface of 6.54 for our sample versus the mean dfs of 2.20 reported for three-year-old Head Start children in Connecticut.¹⁴

Maxillary anterior smooth surface caries was the predominant caries pattern under three years of age reflecting nursing caries experience. Fissure caries was the most observed pattern at three to five years of age, in agreement with previous reports, for that age-group.^{10,14} These findings contradict the observation of Louie *et al*, however, who reported that proximal caries was predominant in Head Start children (three-and-a-half to five-and-a-half-years old) in USDHHS Region IX.⁷

Majority of the children in our sample had caries levels that place them at high risk for caries in the permanent dentition.¹⁵ Combined with the findings of Steiner *et al*, which indicate that children who experience high levels of caries in the primary dentition are at increased risk for caries in the permanent dentition, the results of this study are significant from both epidemiological and public health perspectives, and support arguments for targeted early intervention programs.¹⁶

The findings are also relevant to those who are responsible for financing and delivering dental services for preschoolers, especially for indigent populations. With respect to financing, the concept of managed care increasingly is being advocated as a mechanism for redistributing financial risk and controlling health care costs in diverse groups (including those presently covered by publicly financed programs such as Medicaid). The observation that an extremely high proportion of preschoolers from households of low socioeconomic status exhibit considerable untreated dental disease is a factor that bears consideration in the formulation of managed-care arrangements. An additional aspect that has both financial and delivery system implications concerns the need to use sedation or general anesthesia to provide dental services for many preschoolers with advanced dental treatment needs. This requirement not only increases treatment cost, but also requires a higher

level of training and expertise on the part of clinicians providing services for these children.

CONCLUSIONS

Consistent with the findings of recent large-scale epidemiologic surveys in older children, data from this study suggest that dental caries remains a significant problem for many inner-city preschoolers. Data from this study also indicate that caries prevalence and severity increase with increasing age within the preschool population. The results have important epidemiological, public health and public policy implications.

REFERENCES

1. Waldman, H.B.: A litany for change. *J Dent Child*, 57:194-197, May-June 1990.
2. Waldman, H.B.: Reaching more children with needed dental services. *J Dent Child*, 57:417-420, November-December 1990.
3. Wendt, L-K.; Hallonsten, A-L.; Koch, G.: Dental caries in one- and two-year old children living in Sweden. *Swed Dent J*, 15:1-6, 1991.
4. Fujiwara, T.; Sasada, E.; Mima, N. *et al*: Caries prevalence and salivary mutans streptococci in 0-2 year-old children of Japan. *Community Dent Oral Epidemiol*, 19:151-154, 1991.
5. Holt, R.D.; Joels, D.; Bulman, J. *et al*: A third study of caries in preschool aged children in Camden. *Br Dent J*, 165:87-91, August 6, 1988.
6. Johnsen, D.C.; Bhat, M.; Kim, M.T. *et al*: Caries levels and patterns in Head Start children in fluoridated and non-fluoridated, urban and non-urban sites in Ohio, USA. *Community Dent Oral Epidemiol*, 14:206-210, August 1986.
7. Louie, R.; Brunelle, J.A.; Maggiore, E.D. *et al*: Caries prevalence in Head Start children, 1986-87. *J Public Health Dent*, 50:299-305, Fall 1990.
8. Crall, J.J.: Delivery systems for preschool children. *Dent Clin N Amer*, 39:897-907, October 1995.
9. Graves, R.C.; Bohannon, H.M.; Disney, J.A. *et al*: Recent dental caries and treatment patterns in U.S. children. *J Public Health Dent*, 46:23-29, Winter 1986.
10. Douglass, J.M.; Wei, Y.; Zhang, B.X. *et al*: Dental caries in preschool Beijing and Connecticut children as described by a new caries analysis system. *Community Dent Oral Epidemiol*, 22:94-99, 1994.
11. Gift, H.C. and Newman, J.F.: Oral health activities of U.S. children: Results of a National Health Interview Survey. *J Am Dent Assoc*, 123:96-106, October 1992.
12. National Institute of Dental Research: Oral health of United States Children: The National Survey of Dental Caries in U.S. School Children, 1986-1987. NIH Publication No. 89-2247, 1989.
13. Margolis, M.Q.; Hunt, R.J.; Vann, W.F. Jr. *et al*: Distribution of primary tooth caries in first-grade children from two nonfluoridated U.S. communities. *Pediatr Dent* 16:200-205, May-June 1994.
14. Tinanoff, N.; Crall, J.; Thibodeau, E. *et al*: Dental caries patterns and treatment in Connecticut Head Start children: preliminary results. *Conn State Dent Assoc J*, 67:21-23, Winter 1991.
15. ter Pelkewijk, A.; van Palenstein Helderma, W.H.; van Dijk, J.W.E.: Caries experience in the deciduous dentition as predictor for caries in the permanent dentition. *Caries Res*, 24: 65-71, 1990.
16. Steiner, M.; Helfenstein, U.; Marthaler, T.M.: Dental predictors of high caries increment in children. *J Dent Res*, 71:1926-1933, 1992.

IMPACT VELOCITIES OF THE TEETH

In theory, high bite forces cause high impact velocities of the lower teeth onto the upper teeth when an individual bites through hard and brittle food. However, in reality, high bite forces decrease quickly as soon as the mandible starts to move, as has been shown in unloading experiments in which the resistance to a forceful static bite is suddenly withdrawn (Hannam *et al.*, 1968; Miles and Wilkinson, 1982; Van Willigen *et al.*, 1997). Consequently, an almost-constant small velocity of the mandible is reached (Slager *et al.*, 1995), since jaw acceleration returns to zero when net muscle force has vanished and with it the measured bite force. The quick reduction in bite force and the limitation in jaw velocity cannot be explained by a sudden inactivity of the jaw-closing muscles (delay of 5 to 20 ms) or an increased activity of the jaw-opening muscles (delay of 20 to 40 ms) after the mouth starts closing (Hannam *et al.*, 1968; Lamarre and Lund, 1975; Miles and Wilkinson, 1982; Yoshida and Inoue, 1995; Van Willigen *et al.*, 1997), since reflexes were recorded well after the bite force had vanished.

There are several hypotheses explaining the sudden decrease in bite force and the resulting limitation in jaw velocity. From unloading experiments, it has been shown that the jaw-opening (digastric) and jaw-closing (masseter) muscles co-contract during the static phase of the initial bite (Miles and Wilkinson, 1982). These authors suggest that the resistance to elongation of the activated digastric muscles is responsible for the limitation in jaw closing. This resistance is perhaps due to distortion of cross-bridges between myofilaments (Rack and Westbury, 1974).

Nagashima, T. *et al*: Impact velocities of the teeth after a sudden unloading at various initial bite forces, degrees of mouth opening, and distances of travel. *J Dent Res*, 76:1751-1759, November 1997.

First-molar caries experience among Taiwanese first-grade children

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Dental caries is one of the most common diseases in the world, particularly among children. Its prevalence among populations and its extent in individuals varies, however, among nations and over time. In general, there have been relatively few data reported in the literature concerning the prevalence of dental caries among Taiwanese school children, and accurate and consistent methods of assessing dental caries have not been used. A few studies from the 1960s suggested that dental caries among the children of Taiwan was less prevalent and less extensive than among children in other industrialized nations, such as the United States. Current evidence suggests, however, that while the dental caries problem has diminished in many parts of the world, it may have risen dramatically among Taiwanese children during the past twenty years.

A 1960 study comparing the caries experience of Taiwanese and Australian children found that DMFT values for eight, ten, and eleven-year-old children in Taiwan were 1.7, 1.8 and 3.8, respectively, while rates for Australian children of the same ages were 3.2, 5.3 and 7.0.¹ A study in 1972, which compared DMF values for two cities in Taiwan to findings of DMF values in Japan, the

United States, and Canada, found that the DMF values were substantially lower for Taiwanese children at all age levels.² A comparison in the early 1970s revealed that eleven-year-olds in Taiwan had lower DMF values than in most other countries in the Asian area of the Pacific Basin.³

During the past twenty years, the rate of dental caries has apparently risen dramatically among Taiwanese children. A twelve-year cross-sectional study begun in 1971 and conducted in the fluoridated city of Chung-Hsing New Village and the nonfluoridated city of Tsao-Tun demonstrated the dramatic rise in dental caries rates among Taiwanese school children, regardless of fluoridation status. The mean DMF scores for children in the fluoridated community roughly doubled from 1971 to 1984, while in the nonfluoridated community, mean DMF scores roughly quadrupled over the same time period.⁴

Studies conducted during the past decade have demonstrated that this trend has reached the point where Taiwanese children now experience dental caries at rates much higher than many other countries. A study in 1983 comparing caries rates among children in Taiwan to children in Flint, Michigan, USA, showed that Taiwanese children had much higher mean dft and DMFT scores than their counterparts in the United States.⁵ Another study reported in 1987 found that elementary students in Taipei, Taiwan had the highest DMFS scores and greatest number of DMF teeth per child when com-

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pared to children of the same age in Korea and Japan.⁶ A 1988 study indicated that the dental caries rate among Taiwan children continued to be high.⁷

Although increases in caries prevalence among Taiwanese children have been documented as described above, there is very little further information available, such as distribution of surfaces affected, proportions of decayed, missing, or filled, and the relationship between caries and socioeconomic status.

PURPOSE

As part of a long-term project to develop a model caries prevention program for children in Taiwan, the purpose of the present study was to collect detailed caries data on a limited sample of Taiwanese children.

METHODS

Through personal and professional contacts, an affiliation between the University of Iowa College of Dentistry and the National Defense Medical Center (NDMC) Dental School in Taipei was established in 1990. Subsequently, NDMC, the Taiwan Department of Public Health and Iowa personnel worked to plan a preventive dentistry demonstration project in selected schools. It quickly became apparent, however, that before an effective preventive strategy could be developed, more detailed information was needed regarding the prevalence, severity and location of caries in Taiwanese children. We collected, therefore, baseline caries data for a selected sample of Taiwanese school children.

Before conducting the examinations for the project, two examiners were trained according to criteria established by NIDR by an experienced dental examiner from the University of Iowa. The examiners were provided with background information relative to the project, including specific criteria and examples for caries examinations that were adapted from NIDR and a detailed examination protocol. In addition, examiners completed both simulated examinations and practice examinations on a small number of children.

Examinations were subsequently conducted by the two examiners at four selected elementary schools in Taiwan in late 1993 and early 1994. These schools were selected to provide a balance of urban/non-urban populations and socioeconomic statuses. Due in part to limited time and resources, it was decided that the data collection would be limited to first grade children. Part of this rationale was that any preventive efforts would most likely be directed toward younger children, so that

information on first grade children would be the most useful. Data were collected, therefore, for the 401 first grade students in the four schools. Collection of data was also restricted to the permanent first molar teeth, due to limited time and resources, and because most of the permanent teeth in children in this age-group are first molars, and most decay could be expected, therefore, to be concentrated in these teeth.

In addition to dental caries information, data were gathered on children's socioeconomic status, by asking parents about their occupations and levels of education on the consent forms sent home with the children. Socioeconomic statuses were categorized in five groups based on an index rating parents' occupations and levels of education. To compare children from low socioeconomic status families to children in higher categories, the five socioeconomic status categories were dichotomized into two categories:

- Low and moderately low socioeconomic status.
- Moderate, moderately high, and high socioeconomic status.

In this way, 181 children were categorized as "low socioeconomic status" and 193 as "high socioeconomic status," with parents of twenty-seven children not responding.

All data were collected on paper forms which were sent to The University of Iowa for computer entry, verification, and analysis.

RESULTS

The sample included 481 first grade children from four elementary schools, of whom 401 consented to participate and were present for the examination (83.4 percent response rate). The mean age was 6.5 years and only 16 percent of the children were in the moderate-high or high socioeconomic categories. The number of children per school and the socioeconomic status varied among the four schools as presented in Table 1. The number of permanent first molars present per child also varied, and this distribution is presented in Table 2.

DMFS scores, restricted to the first molar teeth, were computed for the sample. The mean DMFS for the entire sample was 1.21. Because a large number of children had no permanent first molars present, the DMFS scores may be artificially low and difficult to interpret. The balance of the analyses, therefore, included only those children with one or more permanent first molars present. The exclusion of the sixty-eight children with no permanent first molars present resulted in a mean DMFS of 1.46 and a mean DMFT of 1.19. Males and

Table 1 □ School characteristics: number of children and socioeconomic status (SES).

School	n	% with high or moderately-high SES
Urban schools		
1. Kuo-Tung	94	33
2. Kuang-Fun	100	23
Rural schools		
3. Chung-Hsiao	149	2
4. Chung-Ho	58	6
Totals	401	16

Table 2 □ Distribution of the number of first permanent molar teeth present per child.

Number of 1st molars present	Number of males (%)	Number of females (%)	Totals (%)
0	41 (20%)	26 (13%)	67 (17%)
1	9 (4%)	7 (4%)	16 (4%)
2	15 (7%)	12 (6%)	27 (7%)
3	14 (7%)	14 (7%)	28 (7%)
4	127 (62%)	135 (70%)	262 (65%)

Table 3 □ Proportion of decayed filled surfaces by type of surface.

Surface	%
Buccal	15.4
Lingual	2.6
Mesial	0.3
Distal	1.2
Occlusal	80.6

Table 4 □ DMFS by school.

School	DMFS
1. Kuo-Tung	1.57
2. Kuang-Fun	0.62
3. Chung-Hsiao	1.71
4. Chung-Ho	1.87

females did not differ significantly in DMFS or DMFT. The 164 males had a mean DMFS of 1.35 and mean DMFT of 1.14, while the 168 females had mean DMFS and DMFT scores of 1.57 and 1.24, respectively. There were no teeth missing due to caries in this sample of first grade children.

Based on the number of permanent first molars present (Table 2) and DMFS values, an attack rate can be computed. For this sample, the overall caries attack rate was 8.1 DMF surfaces per 100 surfaces present, with females having a higher attack rate (8.6) than males (7.5).

The distribution of DMF by surface is presented in Table 3. It should be noted that if occlusal, buccal, and lingual surfaces are combined and all are assumed to be

pit and fissure decay, then over 98 percent of the decay occurred in pits and fissures. In addition, 88.9 percent of the DMF surfaces were "decayed" and 11.1 percent were "filled."

Overall, 48 percent of the children in the sample had caries-free permanent first molars, 14 percent had one tooth affected, 20 percent had two teeth affected, 5 percent had three teeth affected, and 13 percent had all four permanent first molar teeth affected with caries.

There were significant differences in DMFS between children in high and low socioeconomic groups, and significant differences in mean DMFS between children from different schools. The 152 children included in the low socioeconomic group had a mean DMFS of 1.71, while the 158 children included in the moderate/high socioeconomic group had a mean DMFS of 1.12. Student's t test indicated that these two groups were significantly different in mean DMFS at $p < 0.05$. As demonstrated in Table 4, the two schools with larger proportions of high socioeconomic children (Table 1) had lower mean DMFS scores.

DISCUSSION

The results of this study suggest that dental caries has become a much more serious problem in Taiwanese first-graders than it is in the United States, where surveys conducted in the 1970s and 1980s have demonstrated a steady decline in DMFS and DMFT among children in all age-groups, although no national caries surveys have been conducted in the U.S. since 1987.⁹⁻¹¹ To provide some perspective for the caries levels in our Taiwan sample, it is illustrative to compare findings to those the authors recently reported for the state of Iowa, which were collected at approximately the same time in early 1994, used identical criteria, and shared some key personnel as the data presented here for Taiwan.¹² Comparing first molar caries data among first grade children with at least one permanent first molar present between the two sites shows that mean DMFS was 1.46 for Taiwan, compared to 0.24 for Iowa, while the caries attack rates per 100 surfaces present was 8.1 DMF surfaces for Taiwan, and 1.3 DMF surfaces for Iowa.¹³ In addition, the caries prevalence scores in Taiwan are slightly higher than those found in studies conducted there during the 1980s, suggesting that the upward trend in caries has continued among Taiwanese children.

The results of this study must be interpreted with the following in mind: study limitations included the use of non-random, selected schools, a relatively small sample, the inclusion of only first grade children, and the inclu-

sion of only first permanent molar teeth. Clearly, larger, more comprehensive studies are needed before firm recommendations regarding caries prevention strategies can be made. It appears that the level of dental caries among Taiwanese children, however, is quite high, and the need for caries preventive programs appears, therefore, to be great. Fortunately, the success of many different preventive strategies in the United States, Europe, and many other areas suggests that similar successes can be achieved in Taiwan. The cultural, economic, and political characteristics of Taiwan are unique, however, so that programs that have proved effective in other areas may need to be modified to be effective in Taiwan.

Further studies should be initiated to document caries experience among children of different ages, and if results are generally consistent with our findings, a comprehensive caries prevention program for Taiwan should be undertaken. Consideration should be given to a broad range of caries preventive strategies, including school-based or community-based pit and fissure sealant programs, particularly for children who are at highest risk, improved public awareness and access to dental preventive services, and increased exposure to sources of fluoride.

The results of this study showing that nearly all of the decay among Taiwanese children occurred in pits and fissures suggests that pit and fissure sealant programs may be particularly effective. The high prevalence of decay in the first molars of these young children also suggests, however, that, to be effective, sealants must be placed as soon as possible after eruption. Although the low prevalence of interproximal decay further suggests that pit and fissure sealants would be appropriate, it is possible that interproximal decay is more prevalent, but had not progressed to the point of clinical detection in these newly-erupted teeth. Data concerning interproximal decay in older children should be gathered, therefore, as part of any comprehensive preventive program.

In regard to sealants, it appears that dentists in Taiwan, like those in the United States, may need to be better informed about the rationale for using sealants. In planning this project, many Taiwanese dentists (including several pediatric dentists) expressed strong reservations about placing sealants in children as young as first-graders, and suggested that children should be at least in the 3rd grade or older before they could be expected to cooperate sufficiently for sealant placement. In addition, many of these Taiwanese dentists considered rubber dams to be absolutely necessary for sealant placement, and were skeptical of our plans to place sealants with cotton roll isolation in a school setting.

In addition to sealants and other preventive programs, the high level of untreated decay among Taiwanese first-graders suggests that a large number of children already have substantial restorative needs. Efforts need to be undertaken to increase the amount of restorative dental

care available to Taiwanese children. In particular, Taiwanese children in lower socioeconomic groups appear to be especially in need of dental care as evidenced by higher DFS values which are mostly "decayed" surfaces. Whether through efforts to raise public awareness or increase access via improved financing, increased supply of dentists, or more efficient delivery of services, the high decay rates among the current generation of children should not be ignored.

CONCLUSION

In conclusion, the results of this project suggest that although widespread use of fluorides and other preventive methods may have reduced the level of dental caries among children in many parts of the world, there are challenges that remain. Children in some parts of the world, including Taiwan, appear to experience dental caries at a high rate. Thus, the methods of preventive dentistry must not be confined by national boundaries, and must continue to be promoted throughout the world.

REFERENCES

1. Pu, M.Y. and Lilienthal, B.: Dental caries and mottled enamel among Formosan children. *Arch Oral Biol*, 5:125-136, March-April 1961.
2. Hsieh, C.C.: Dental diseases among the children in Chung-Shin village and Tsaotung. *J Formosan Med Assoc*, 71(8):537-548, August 28, 1972.
3. Moreira, B.J.: The pattern of dental disease in the Asian area of the Pacific Basin. *Int Dent J*, 23(4):559-572, April 1973.
4. Hsieh, C.C.; Guo, M.K.; Hong, Y.C. *et al*: An evaluation of caries prevalence in Chung-Hsing New Village after 12 years of water fluoridation. *J Formosan Med Assoc*, 85:822-831, August 1986.
5. Chen, B.C.S. and Wang, T.M.: Sex differences in dental caries prevalence of primary schoolchildren in Flint (Michigan, USA) and those reported in Taiwan. *Chinese Dent J*, 5(1):12-21, June 1986.
6. Ishii, K.: Study on the relation between dental caries prevalence of permanent teeth in school children and their living environment by quantification. *J Fukuoka Dent Coll*, 14(3):226-248, November 1987.
7. Yao, J.H.; Hsu, B.S.; Chao, C.F.: Survey of factors associated with non-treated caries cases among junior high school first-year students in Taipei. *Chinese Dent J*, 53-64, June 1988.
8. Oral Health Surveys of the National Institute of Dental Research—Diagnostic Criteria and Procedures. U.S. Department of Health and Human Services. NIH Pub No. 91-2870. Bethesda, MD: National Institute of Dental Research, January 1991.
9. National Institute of Dental Research, U.S. Department of Health and Human Services. Oral Health of United States Children—The National Survey of Dental Caries in U.S. School Children: 1986-87, National and Regional Findings. NIH Pub. No. 89-2247. Bethesda, MD: National Institute of Dental Research, September 1989.
10. Hicks, M.J. and Flaitz, C.M.: Epidemiology of dental caries in the pediatric and adolescent population: A review of past and current trends. *J Clin Pediatric Dent*, 18(1):43-49, Fall 1993.
11. National Caries Program, NIDR. The Prevalence of Dental Caries in United States Children, 1979-80. NIH Pub. No. 82-224S, December 1981.
12. Warren, J.J.; Levy, S.M.; Hand, J.S. *et al*: Results of the 1994 Iowa oral health survey. *Iowa Dent J*, 82:55-60, January 1996.
13. Hand, J.S.; Warren, J.J.; Yao, J.H. *et al*: First molar caries prevalence in Iowa and Taiwan first graders. (abstract #266) *J Dent Res*, 74 (AADR abstracts): 45, March 1995.

REPORTS

Dentinal dysplasia type I: Review of the literature and report of a family

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Dentinal dysplasia (DD) so named by Rushton (1939) is a very rare autosomal dominant condition affecting dentin formation.¹ The prevalence has been recorded as 1 in 100,000 population.² This disorder was first described by Ballschmiede (1920) under the term "rootless teeth".³ Shields (1973) classified dentinal dysplasia in two different types: Type I also called radicular DD involved only radicular dentin; Type II or coronal DD where the radicular dentin is normal and the coronal dentin is dysplastic, and associated with frequent pulp stones.^{1,4-7} The clinical findings of DD type I show normal enamel formation and color, unlike dentinogenesis imperfecta where the enamel is attrited or lost. Mobility of the teeth has been mentioned in the literature, and would usually be followed by early exfoliation of teeth. Radiographic views of DD type I show short and blunted roots with obliterated pulp canals, as well as periapical radiolucencies, which are a frequent finding, although not always presented.^{4,8} Clinically normal attrition is found in both dentitions.⁹

In DD type II the primary teeth are brown or blue opalescent in color similar to dentinogenesis imperfecta. Radiographs of the primary teeth show pulp obliteration with a small number of pulp stones, but in permanent teeth these findings are not present. Enlargement of the pulp cavity is seen, however, in radiographic views particularly of the coronal area of the pulp chamber and

also the coronal part of the root. This specific appearance is called "thistle-tube" shape. Pulp stones are also present in these cases, but there is no evidence of radiolucencies in the periapical areas.⁴

In 1978 a third type of the condition was described in which radiographic findings of both type I and II DD are found.¹⁰ In type II DD the primary dentition undergoes rapid attrition, but attrition is normal in the permanent dentition.

There are a few reports of DD associated with some tumoral calcinosis.⁷ Witcher *et al* reported six siblings who showed evidence of DD on radiographs and tumoral calcinosis in different parts of the body.¹¹ Hoggins and Marsland had similar findings in their report in 1952.¹² In 1985 Wannfoss and associates reported a case of fibrous dysplasia of bone with DD and suggested that the parallel occurrence of dysplastic changes in bone and teeth may be a sign of a generalized defect in the mesenchymal hard tissue of forming cells, with lesions in the left mandible, in the maxilla, in frontal and occipital bones, in the ilium, in the proximal ulnae, and in the ribs.^{13,14} DD has also been reported in association with fibrous dysplasia.^{7,11,12}

Witkop in 1989 suggested that dentinal dysplasia type II according to the clinical, radiographic, histologic, and structural protein alterations is exactly the same as dentinogenesis imperfecta type II, and fibrous dysplasia of dentin should be considered as a type of dentinogenesis imperfecta.¹⁴ Scola and Watts in 1986 suggested a sub-classification of type I dentinal dysplasia:

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- Total DD type I is characterized by considerably reduced or obliterated pulp spaces and permanent teeth with diminutive roots.
- Subtotal DD type I is differentiated by the permanent teeth with roots of intermediate length.¹⁵

CASE REPORTS

A family was referred by their general dental practitioner to the consultant clinic in the Child Dental Health Department in Glasgow Dental Hospital because of irregular mobility of several teeth and a family history of early loss of teeth. The medical history of all three patients was unremarkable.

Case I

The seven-year-old male patient had the following teeth present: 16, 55, 54, 53, 52, 62, 63, 64, 65, 26, 46, 85, 84, 83, 82, 81, 71, 73, 74, 75, 36.

The primary maxillary central incisors were exfoliated a year previously and the mandibular left primary lateral incisor was exfoliated recently. The patient's caries rate was high and uncontrolled, but the oral hygiene was adequate (Figure 1). The patient reported occasional mobility of his teeth, specifically after meal times. The orthopantomogram (Figure 2) showed both primary and permanent teeth present with the pulp canals and pulp chambers obliterated. The short roots were characteristic of DD type I. Periapical radiolucencies were obvious around the apices of the primary molars.

The furcation areas in the first permanent molars were positioned farther apically than in normal teeth and similar to those found in taurodont teeth. The clinical appearance of the teeth was slightly yellowish gray in color, but without any sign of attrition.



Figure 1. Closed bite, anterior view of first case, note dark color of incisors.

The patient was cooperative but nervous and would not accept the dental handpiece for cavity preparation. In these circumstances a chemomechanical caries removal system and hand excavation without injection were performed, using rubber dam.¹⁶ Glass ionomer filling material was placed in the treated teeth. At the final visit unsavable teeth: 74, 75, 84 were extracted using local anaesthesia without any problem.

CASE II

The nine-year-old sister of the first case had clinical findings similar to those of her brother. Her caries rate was controlled, but oral hygiene was inadequate, particularly in the anterior mandibular region (Figure 3). Two factors contributing to the inadequate oral hygiene were

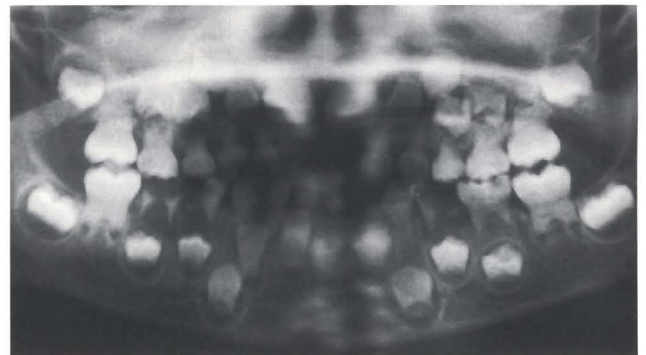


Figure 2. Dental orthopantomograph of the first case, note obliterated pulp chambers and linear pulp appearance on molar teeth.



Figure 3. Closed bite, anterior view of the second case.



Figure 4. Dental orthopantomograph of the second case shows the characteristics of dentinal dysplasia.

crowding and mobility of the teeth. The patient was visibly nervous at examination.

Teeth present were as follows: 16, 55, 54, 53, 12, 11, 21, 62, 63, 64, 65, 26, 46, 85, 84, 83, 42, 41, 31, 32, 72, 73, 74, 75, 36. The color of the teeth was yellowish gray and there was no abnormal loss of tooth structure. There was an anterior open bite. The orthopantomogram showed obliterated pulp canals and pulp chambers associated with typically short roots. The furcation areas of the primary and permanent molars were located quite far from the normal location. Radiolucent appearance of the pulp is seen in Figure 4. Apical radiolucencies were related to the maxillary and mandibular left first permanent molars. A treatment plan was formulated and involved prevention and routine conservation to achieve a caries free mouth and retain the teeth for as long as possible.

CASE III

The twenty-six-year-old father of the two reported children, was a previously reported case of type I DD.¹⁷ He had lost his anterior permanent teeth due to periodontal involvement of the affected teeth and short roots. Teeth present were as follows: 14, 15, 17, 18, 23, 24, 25, 26, 27, 28, 31, 32, 33, 34, 35, 36, 37, 38, 41, 42, 43, 44, 45, 47, 48.

The maxillary permanent incisors were extracted eight years ago due to discomfort and mobility, and were replaced with a removable partial denture. Radiographs showed the anterior mandibular teeth to have short roots and the permanent molars's furcation areas were far from the usual cervical region. The radiolucent linear pulp chambers in the cervical portion of molars



Figure 5. Dental orthopantomograph of the father of the family shows the whole dentition involved with the same condition.

and also premolars are visible on the orthopantomogram (Figure 5).

Histopathology report of the exfoliated mandibular primary lateral incisor and mandibular first primary molar in the second case indicated similar features to those reported earlier.^{12,16,19} In the ground sections of both teeth, the enamel appeared structurally normal. The early stages of dentin formation appeared normal, but there was a sharp transition to very irregular abnormal dentin. These features are entirely consistent with the clinical diagnosis of dentinal dysplasia type I.

DISCUSSION

To date a total of fifty-two cases have been reported in the literature involving DD, including one case of each DD type II and III, since 1920. These reports included clinical, radiographic and in some cases histological investigations (Table 1).

This investigation was conducted in order to assess the similarity of the characteristics presented by our cases in comparison to those reported in the current literature.

Our clinical, radiographic, and histological findings confirm the characteristics described by the literature. Both children of the affected father had the disorder, indicating an autosomal dominant inheritance trait. This developmental anomaly appears in both dentitions as short roots, amber color teeth, normal clinical crown shape and hardness, and occasional mobility of the teeth. Radiographs showed calcified pulp chambers and pulp canals, periapical radiolucent areas, blunted root appearance as a generalized pattern and are the characteristic findings of dentinal dysplasia type I. The affected

Table □ Presentation of the number of cases reported so far in the literature including the investigations conducted.

Year	Author	Number of reported cases	Reported documents (investigations)	Treatment	Journal of publication
1920	Ballschmiede	1	clinical description	—	Dissertation
1952	Hoggins	1	photo of ext teeth/histology	—	Br Dent J
1939	Rushton	1	radiographs/histology	—	Guy's Hosp. Report
1962	Logan ²⁰	5	radiographs/histology	—	Oral Surg Oral Med Oral Pathol
1967	Elzay ²¹	1	radiographs/histology	—	Oral Surg Oral Med Oral Pathol
1968	Brookreson ²²	1	radiographs/histology	—	J Am Dent Assoc
1972	Sauk	1	SEM(electron optic)	—	J Oral Surg
1972	Peterson ²³	1	radiographs	—	Oral Surg Oral Med Oral Pathol
1973	Shields	1(type II)	histology/classification/genetics	—	Arch Oral Biol
1974	McFarlane	3	radiographs	—	J Oral Surg
1976	Wesley	1	SEM/radiograph/histology/genetic	—	J Oral Surg
1977	Morris	2	Radiograph/Histology/Assoc. Synd.	—	Oral Surg Oral Med Oral Pathol
1977	Eastman ²⁴	1(type III)	clinical/radiographs/classification	—	Oral Surg Oral Med Oral Pathol
1977	Perl ²⁵	1	histology/clinical report	—	Oral Surg Oral Med Oral Pathol
1979	Tidwell	1	clinical report	RCT	J Endod
1979	Coke	1	clinical report/pulp histology	RCT	J Oral Surg
1980	Melnick	1	SEM	—	J Oral Surg
1981	Petrone ²⁶	1	clinical report/radiographs	—	J Am Dent Assoc
1982	Dym ²⁷	1	clinical report	—	J Dent Child
1984	Steidler ²⁸	8	clinical report/radiographs/histology	—	Br J Oral Maxillofac Surg
1985	Wannfors	1	clinical report/radiographs/histology	—	Oral Surg Oral Med Oral Pathol
1986	Bakaeen ²⁹	1	clinical report/radiographs	—	J Dent Child
1986	Luffingham	1	clinical report/radiographs	—	Br Dent J
1987	Scola	1	clinical report/radiographs	—	Br J Orthod
1988	Brenneise ³⁰	1	clinical report/radiographs/histology	—	J Am Dent Assoc
1989	Witcher	6	clinical report/radiographs	—	Oral Surg Oral Med Oral Pathol
1989	Van Dis ³¹	4	clinical reports/radiographs	—	Dento Maxillofac Radiol
1990	Miers ³²	1	clinical report/radiographs	—	J Dent Child
1991	O'Carroll ³³	1	Classification/radiographs	—	Oral Surg Oral Med Oral Pathol
1993	Flynn ³⁴	1	radiographs	—	Dento Maxillofac Radiol

teeth are predisposed to fracture even during careful extraction procedure.^{17,27,31} All features have been reported to be present before tooth eruption. Mobility, malposition, and early exfoliation of the teeth are explained by root developmental defects and lack of periodontal support.

Shortened roots of the maxillary and mandibular incisors may contribute to early loss of these teeth. Discomfort due to the mobility of these teeth particularly after a meal is a common complaint.^{12,17,21,27} Histological findings have shown that the enamel and dentin of the crown appear to be normal, but below the normal dentin a slit like space is the only evidence of a pulp chamber. Apically from this space are large calcified masses of tubular dentin and osteodentin fused with the normal-in-appearance dentin of the root.

As a possible cause of this disorder Rushton explained: "multiple foci of degeneration in the dental papilla are the principal factors leading to retarded growth and final obliteration of the papilla and to the sporadic formation of the dentin about the calcified foci".^{1,5,12,27} DD type I has been described as a defect in the epithelial component in which the invagination of the root sheath, first, occurs too soon and, in a sequence of futile attempts to correct itself, results in a stunted root form with an unusual whorl-like pattern of dentin obliterating the pulp

chamber.^{18,22,29} In pathologic findings the first layer of the coronal dentin is normal mantle dentin.^{1,22} The dentinal tubules are blocked and shunted from their usual paths by numerous and sometimes massive true denticles.²⁰ In most instances the extremely thin cementum and the short, pointed root suggest the failure of Hertwig's sheath to develop to a normal length.²⁰ Logan also suggested that the histogenetic pattern of the dentin formation is by no means always the same.²⁰ There is no sex dependency in this disorder. Eruption dates of the teeth are reported to be normal, although a few cases of slightly delayed eruption are reported in the literature.²⁰ Blood tests have been performed in some of the reported cases with results well within normal limits.^{2,12,17,22}

Sauk and associates conducted a study on the early stages of development of the tooth in these cases, believing that the invagination of the epithelial root sheath occurred too soon in the tooth's developmental stages and caused a lack of a normal developmental pattern of radicular dentin with an unusual whorl-like pattern of dentin obliterating the pulp chambers and canals.¹⁵ The abnormal root morphology of DD type I is secondary to the abnormal differentiation and function of odontoblastic derivatives. Crown morphology in those teeth is normal, because the initial differentiation of onto-

blasts and their functions are normal.¹⁹ The primary factor is the formation of multiple areas of abnormally calcified tissue within the dental papilla, which appear to have acted as centers around which true denticle was formed.¹² These multiple degenerations are formed and subsequently bound together by scattered areas of tubular dentin.¹² Report of the result of scanning electron microscopic analysis of the primary dentition suffering from DD type I, revealed the following: normal enamel, a thin layer of normal dentin adjacent to the dentino-enamel junction, a crescent-shaped pulpal remnant below the normal dentin and finally masses of dysplastic dentin both atubular and tubular.^{18,19}

The differential diagnosis includes a number of syndromes such as Ehlers-Danlo syndrome, Ellis-Van Creveld syndrome (chondro-ectodermal dysplasia); osteogenesis imperfecta associated with dentinogenesis imperfecta; different types of disorders of the dentinal structure of the teeth; and also "dwarfed teeth" caused by radiation of the jaws. Peterson suggested that dentinal dysplasia and calcification of the dentinal papilla might be two different entities.²³

Tumoral calcinosis lesions by their definition appear in the first two decades of life. They are firm, nodular, non-tender lesions that seem to be fixed to the underlying structures. The overlying skin is freely movable over the tumors. These lesions expand for two years and then cease growth. Over time they tend to perforate the skin, draining a chalky fluid that is initially sterile. These abnormal calcifications of cystic masses usually form in the fibrous tissues adjacent to the joint spaces without any joint involvement.^{7,11,12}

There are a few reports of malocclusions in association with this disorder, but malocclusion is not a specific feature of this condition. Anterior open-bite in association with DD type I, Angle Class III malocclusion tendency, and also Angle Class II malocclusion associated with DD type I have been reported.^{7,17,28} Linear semilunar radiolucency parallel to the amelodentinal junction of the teeth even in unerupted third molars at the early stages of development is another specific characteristic of these cases. Care should be taken during extraction of these teeth, due to the tendency of root separation from the crown.

SUMMARY

A family is reported with dentinal dysplasia type I affecting both dentitions. Presenting features included unusual mobility of the teeth, followed by early exfoliation; normal clinical shape of the crowns of the teeth, but with

an amber color without any sign of attrition or abnormal loss of enamel. Radiographic findings showed pulp-chamber and root-canal obliteration, poor root formation, radiolucent linear appearance of the pulp chamber parallel to the cemento-enamel junction and frequent periapical radiolucencies. Histological studies have reported large masses of calcified tubular dentin, atypical osteodentin, and also true denticle.

REFERENCES

1. Rushton, M.A.: A case of dentinal dysplasia. *Guys Hospital Report*, 89:369-373, 1939.
2. Stewart, R.E. and Prescott, G.H.: *Text book of Oral Facial Genetics*. St. Louis: Mosby Co., 1976, pp 237-239.
3. Ballschmiede: Dissertation, Berlin, 1920 in Herbst, E. and Apffelstaedt, M: *Malformations of the jaws and teeth*. New York: Oxford University Press, 1930, p 286.
4. Sheilds, E.D.; Bixler, D.; ElKafrawy, A.M.: A proposed classification for heritable human dentin defects with a description of a new entity. *Arch Oral Biol*, 18:543-553, April 1973.
5. Mcfarlane, M.W. and Cina, M.T.: Dentinal dysplasia, report of a family. *J Oral Surg*, 32:867-869, November 1974.
6. Wesley, R.K.; Wysoki, G.P.; Mintz, S.M. *et al*: Dentin dysplasia type I: clinical morphologic, and genetic studies of a case. *Oral Surg Oral Med Oral Pathol*, 41:516-524, April 1976.
7. Morris, M.E. and Augsburg, R.H.: Dentinal dysplasia with sclerotic bone and skeletal anomalies inherited as an autosomal dominant trait. *Oral Surg Oral Med Oral Pathol*, 43:267-283, February 1977.
8. Coke, J.M.; Rosso, G.D.; Remeikis, N. *et al*: Dentinal dysplasia type I, report of a case with endodontic therapy. *Oral Surg Oral Med Oral Pathol*, 48:262-268, September 1979.
9. Tidwell, E. and Cuninghame, C.J.: Dentinal dysplasia: Endodontic treatment, with case report. *J Endod*, 5:372-376, December 1979.
10. Ciola, B.; Bahm, S.L.; Govva G.L.: Radiographic manifestations of an unusual combination type I and type II dentin dysplasia. *Oral Surg Oral Med Oral Pathol*, 45:317-322, February 1978.
11. Witcher, S.L.; Drinkard, D.W.; Shapiro, R.D. *et al*: Tumoral calcinosis with unusual dental radiographic findings. *Oral Surg Oral Med Oral Pathol*, 68:104-107, July 1989.
12. Hoggins, G.S. and Marsland, E.A.: Developmental abnormalities of the dentin and pulp associated with calcinosis. *Br Dent J*, 92: 305-311, June 1952.
13. Wannfors, K.; Lindskog, S.; Olander, K.J. *et al*: Fibrous dysplasia of bone and concomitant dysplastic changes in the dentin. *Oral Surg Oral Med Oral Pathol*, 59:394-398, April 1985.
14. Witkop, J.R.: Amelogenesis imperfecta, dentinogenesis imperfecta and dentin dysplasia revisited: Problems in classification. *J Oral Pathol*, 17:547-553, December 1988.
15. Scola, S.M. and Watts, P.G.: Dentinal dysplasia type I: A sub-classification. *Br J Orthod*, 14:175-179, July 1987.
16. Punwani, I.C.; Anderson, A.W.; Soh, J.M.: Efficacy of caridex in children and adults. *J Pedod*, 12:351-361, Summer 1988.
17. Luffingham, J.K. and Noble, H.W.: Dentinal dysplasia. *Br Dent J*, 160:281-283, April 1986.
18. Sauk, J.J.; Lyon, H.W.; Trowbridge, H.O. *et al*: An electron optic analysis and explanation for the etiology of dentinal dysplasia. *Oral Surg Oral Med Oral Pathol*, 33:763-771, May 1972.
19. Melnick, M.; Levin, L.S.; Brady, J.: Dentin dysplasia type I: A scanning electron microscopic analysis of the primary dentition. *Oral Surg Oral Med Oral Pathol*, 50:335-340, October 1980.
20. Logan, G.; Becks, H.; Silverman, S. *et al*: Dentinal dysplasia. *Oral Surg Oral Med Oral Pathol*, 15:317-333, March 1962.
21. Elzay, R.P. and Robinson, C.T.: Dentinal dysplasia: Report of a case. *Oral Surg Oral Med Oral Pathol*, 23:338-342, March 1967.

22. Brookreson, K.P. and Miller, A.S.: Dentinal dysplasia: Report of a case. *J Am Dent Assoc*, 77:608-611, September 1968.
23. Peterson, A.: A case of dentinal dysplasia and/or calcification of the dentinal pupilla. *Oral Surg Oral Med Oral Pathol*, 33:1014-1017, June 1972.
24. Eastman, J.R; Mehlick, M.; Goldblatt, L.I.: Focal odontoblastic dysplasia: Dentin dysplasia type III? *Oral Surg Oral Med Oral Pathol*, 44:909-914, December 1977.
25. Perl, T. and Farman, A.G.: Radicular (type I) dentin dysplasia. *Oral Surg Oral Med Oral Pathol*, 43:746-753, May 1977.
26. Petron, J.A. and Noble, E.R.: Dentinal dysplasia type I: A clinical report. *J Am Dent Assoc*, 103:891-893, December 1981.
27. Dym, H.; Levy, J.; Sherman, P.M.: Dentinal dysplasia type I: Review of the literature and report of a family. *J Dent Child*, 49:437-440, November-December 1982.
28. Steidler, N.E.; Radden, B.G.; Reade, P.C.: Dentinal dysplasia: A clinicopathological study of eight cases and review of the literature. *Br J Oral Maxillofac Surg*, 22:274-286, August 1984.
29. Bakaeen, G.; Synder, C.W.; Bakaeen, G.: Dentinal dysplasia type I: Report of a case. *J Dent Child*, 52:128-129, March-April 1985.
30. Brenneise, C.V.; Dwornik, R.M.; Brenneise, E.E.: Clinical, radiographic, and histopathological manifestations of dentin dysplasia type I: Report of a case. *J Am Dent Assoc*, 119:721-723, December 1988.
31. Van Dis, M.L. and Allen, C.M.: Dentinal dysplasia type I: Report of four cases. *Dentomaxillofac Radiol*, 18:128-131, August 1989.
32. Miers, D.R. and Herbert, F.L.: Dentinal dysplasia type I: Report of case. *J Dent Child*, 57:299-302, July-August 1990.
33. O'Carroll, M.K.; Duncan, W.K.; Perkins, T.M.: Dentinal dysplasia review of the literature and a proposed subclassification based on radiographic findings. *Oral Surg Oral Med Oral Pathol*, 72:119-125, July 1991.
34. Flynn, C.M. and High, A.S.: The diagnosis of dentinal dysplasia type I. *Dentomaxillofac Radiol*, 22:43, February 1993.

DOUBLE LIP

A double lip results from hyperplasia of labial salivary glands or connective tissue (Barnett et al.; Dingman and Billman). It is not the result of chronic inflammation, in contrast to the enlarged tongue of granulomatous cheilitis. When present at birth, a double lip is believed to represent the failure of the pars glabrosa and the pars villosa to fuse along the horizontal sulcus during early lip formation. The acquired form may follow trauma, be idiopathic, or associated with the Ascher syndrome. Typically, the double lip is present at birth but first becomes prominent as the permanent teeth erupt.

Most often only the upper lip is affected; the extra tissue consists of labial mucosa that is everted and folded parallel to the original lip. Histologic examination usually reveals excessive salivary gland tissue. Less often the lower lip or both lips may be involved. Typically the double lip is only seen when the mouth is open and becomes especially marked during speaking and laughing.

Plastic surgical repair can be accomplished when the extra tissue is cosmetically disturbing or interferes with function. Recurrences occur with the acquired form, but are uncommon with the congenital form.

Bork, K. *et al*: *Diseases of the oral mucosa and the lips*. Philadelphia: W.B. Saunders Company, 1996, p 244.

BEHAVIOR

Effects of training in management of child behavior for dental hygiene students

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Child behavior management is very important to the dentist and the dental staff in pediatric dentistry. Successful appointments for child patients are primarily a function of the behavior of the dentist and the dental staff. This concept focuses on their behavior, knowledge, and orientation, not on the behavior of the child. Expectations play a significant role in child management. There have been indications that confidence may be an important factor in the success of a startling variety of management approaches. Similar to the use of the placebo, when staff expect the approach to work, this is communicated to the child, who alters his or her behavior. Wurster *et al* substantiated the common-sense notion that dental student confidence affects the interaction with child patients.¹ They found that dental students who expressed greater confidence performed more effectively and experienced less uncooperative behavior. Thus, it would be advantageous if dental student confidence in managing child patients could be increased by some means less trying to both student and patient than actually treating children in the clinical setting.

Weinstein *et al* established the reliability and validity of a scale measuring the confidence of dental practitioners in child management.² After examining the reliability and validity of the Confidence of Dental Hygienist in Child Management Scale, we used the scale to evaluate

the effect of behavioral science training for dental hygiene students. U.S. and Canadian dental schools offered an average of 41 hours of interpersonal skills instruction in 1990.³ But, little interpersonal skill training is conducted for dental students and dental hygiene students in Japan. The purposes of this study are to develop a useful Confidence of Dental Hygienist in Child Management Scale and evaluate the effect of the training for dental hygiene students.

MATERIALS AND METHODS

The reliability and validity of a twenty-item, ten-point behaviorally-anchored confidence of dental practitioners in child management scale was established by Weinstein *et al*.² We examined the reliability and validity of a Confidence of Dental Hygienist in Child Management Scale (Table 1). The Confidence Scale was composed of ten items, some of which were from the original scale established by Weinstein *et al*.² Forty-eight dental hygiene students, nine dental staff and thirty-one dentists were subjects for the internal consistency and test-retest reliability and validity. Cronbach's coefficient α was calculated for the internal consistency.⁴ After the subjects were requested to fill out the additional Confidence Scale one week later, the correlation between the first and second confidence scores was calculated, and the Pearson correlation analysis was performed to examine stability. The validity was evaluated by the contrast be-

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Table 1 □ Confidence of dental hygienist in child management scale.

1	3	5	7	10
I feel my skills are disturbingly inadequate. [I do] not think I am able to control this behavior more than 1 time in 10.	I feel my skills are adequate. I think I am able to control this behavior about every other time (5 out of 10) it occurs.	I feel my skills are completely adequate. I am able to control this behavior whenever it occurs (10 out of 10).		
1. After introducing yourself to three-year-old Yumeko and asking her to come with you, she refuses tearfully and clings to her mother with both hands. 2. Four-year-old Takashi walks into your operatory with you, but he rejects your invitation to climb into the chair by shaking his head from side to side. 3. As soon as four-year-old Michiyo climbs into the chair, she clings to her mother and cries. 4. Five-year-old Tsutomu can climb into the chair by himself. After he meets the dentist, he begins to whimper constantly, tears running down his cheeks. 5. You repeatedly ask three-year-old Michiko to open her mouth during prophylaxis, but she refuses to open her mouth. 6. When you apply the fluoride gel to four-year-old Kumiko with a cotton applicator, she puts out her tongue and refuses it. 7. Five-year-old Kenji begins to gag and choke when you are placing the rubber dam. 8. Six-year-old Susumu is very withdrawn and timid. He does not respond to you at all during the application of the fissure sealant. He just sits rigidly with his eyes closed and his mouth open. 9. Masashi, aged seven, gags repeatedly when you are taking study model impressions. 10. After the dental treatment of four-year-old Satsuki is completed, she sits on the sofa in the waiting room and cries loudly that she won't come again.				

Table 2 □ Contrast between groups of students and clinicians.

Group	Mean Score	(S.D.)
Students	43.4	(16.4)
Clinicians	62.5	(21.1)

***p < 0.001

Table 3 □ Effect of training on total confidence scores.

Group	1st test	2nd test
Control grp.	32.5 (14.4)	32.0 (8.6)
Training grp.	29.0 (17.5)	52.3 (14.4)

(): S.D.
 N.S.: not significant
 * p < 0.05

tween the group of dental hygiene students and the group of dentists and dental staff. The data were analyzed using a t-test.

Next, we evaluated the effect of behavioral science training on twelve dental hygiene students. Six students took the training. For the first step, the training group heard a lecture concerning child management skills produced by Levy *et al.* They were provided brief descriptions of ten problem situations. For the second step, each trainee received individual videotape feedback. An actual child management situation was recorded on videotape. Every member of the training group watched the replayed situations, and discussed them with the investigators. A ground rule stipulated that all feedback was to be positive. Each individual's problems in child management were to be discussed personally. The third step was role playing related to the problems. The aim of the training was spontaneous behavior modification based on positive reinforcement. The training was given over a two-week period. We measured the confidence scores before and after the training. The confidence

scores were compared with those of a control group consisting of six other students who had no training. These data were analyzed using a t-test. The confidence scores of the control group were measured at Time 1 and Time 2, two weeks later.

RESULTS

Cronbach's coefficient α was 0.95. The stability of the Scale was estimated by correlating total scores obtained by the same subjects at Time 1 and Time 2, one week later. The correlation was 0.91. The confidence scores in the group of the clinicians were significantly higher than those of the students (Table 2).

There was no significant difference between the confidence scores of the training group and the control group before the training, but the mean score of the training group was significantly higher than the mean score of the control group after the training (Table 3). Table 4A and Table 4B show the first and second test scores on all items.

Table 4A □ First test scores for control and training groups on all items.

Group	Item No.									
	No. 1	No. 2	No. 3	No. 4	No. 5	No. 6	No. 7	No. 8	No. 9	No. 10
Control grp.	2.8 (1.2)	3.8 (2.0)	3.7 (2.2)	3.8 (2.0)	3.2 (1.3)	3.3 (1.7)	3.0 (2.2)	2.8 (2.0)	3.2 (1.7)	2.8 (1.6)
Training grp.	3.2 (2.5)	3.8 (2.6)	3.0 (2.3)	3.3 (2.3)	3.2 (2.3)	2.3 (0.9)	1.8 (1.1)	3.3 (2.0)	1.8 (0.9)	3.2 (2.1)
Significance										
(): S.D.										

Table 4B □ Second test scores for control and training groups on all items.

Group	Item No.									
	No. 1	No. 2	No. 3	No. 4	No. 5	No. 6	No. 7	No. 8	No. 9	No. 10
Control grp.	3.3 (0.5)	3.8 (1.1)	3.7 (1.5)	4.0 (0.8)	3.5 (1.0)	3.2 (1.3)	2.2 (1.3)	3.0 (1.5)	2.5 (1.5)	2.8 (0.9)
Training grp.	4.5 (1.7)	5.5 (1.7)	5.5 (1.7)	6.2 (1.7)	5.7 (2.4)	4.7 (1.7)	3.8 (1.6)	6.2 (2.0)	4.3 (1.6)	6.0 (1.3)
Significance										
(): S.D., *p < 0.05, **p < 0.01										

DISCUSSION

Weinstein *et al* produced the Confidence in Child Management Scale.² It is not for dental hygienists and dental hygiene students, but for dentists and dental students. In this study, we examined the reliability and validity of our Confidence of Dental Hygienist in Child Management Scale. Cronbach's coefficient α must be more than 0.80 for the acceptable level of internal consistency. The stability of the scale appeared to be adequate, because the result showed the significant level of test-retest reliability. The confidence scores in the group of clinicians were significantly higher than those of the students. The construct validity of the scale, therefore, was provided. In all, the Scale appeared to be a viable research instrument to assess the confidence in child management of dental hygienists and dental hygiene students.

A fair number of studies have dealt with the related issue of interpersonal skills training of health-care students. Medical educators have been particularly concerned with interviewing skills and have demonstrated successful teaching with a variety of approaches, using both simulated and real patients, often employing videotape feedback.⁶⁻¹² One of the earliest reports on the effectiveness of interpersonal skills training of dental students was by Jackson in which he reported suggestive evidence for the success of such training.¹³ A few dental school studies have documented gains in interpersonal skills.¹⁴⁻¹⁶ Kress *et al* reported that role playing was effective for the development of the confidence of dental students in child behavior management.¹⁷

We evaluated the effectiveness of the training in increasing the confidence of dental hygiene students. The confidence scores of the training group were significantly higher than those of the control group. There were several factors in the development of confidence. The lecture was effective in the cognitive domain, because the training group received beneficial knowledge concerning child behavior management. The members of the training group could look at themselves objectively through the videotape feedback. The simulated experience produced reliable improvement in their perceived ability to manage children through role playing. We speculate that spontaneous behavior modification based on positive reinforcement was one of the main factors for the improvement in confidence. Wurster *et al* reported that greater dental student confidence resulted in superior child management.¹ There is every reason to be assured that greater confidence in dental hygiene students who receive training will result in superior child management.

CONCLUSION

There was an acceptable level of reliability and validity of our Confidence of Dental Hygienist in Child Management Scale. The confidence scores of the training group were significantly higher than those of the control group. Training, using videotape feedback and role playing in child behavior management, was effective for dental hygiene students.

REFERENCES

1. Wurster, C.A.; Weinstein, P.; Cohen, A. J.: Communication patterns in pedodontics. *Perc Mot Skills*, 48:159-166, February 1979.
2. Weinstein, P.; Domoto, P.; Getz, T. *et al*: Reliability and validity of a measure of confidence in child management. *Pediatr Dent*, 2: 7-9, March 1980.
3. Lange, B.; Dunning, D.; Lewis, M.: A baseline study of behavioral science instruction in dental schools. *J Dent Educ*, 57:244-247, March 1993.
4. Cronbach, L.: Coefficient alpha and the internal structure of tests. *Psychometrika*, 16:297-334, September 1951.
5. Levy, R.; Domoto, P.; Olson, D. *et al*: Evaluation of one to one behavioral training. *J Dent Educ*, 44:221-222, April 1980.
6. Fine, V.K. and Therrien, M.E.: Empathy in the doctor-patient relationship: Skill training for medical students. *J Med Educ*, 52: 752-754, September 1977.
7. Neumann, M. and Elizur, A.: Group experience as a means of training medical students. *J Med Educ*, 54:714-718, February 1980.
8. Kauss, D.R.; Robbins, A.S.; Abrass, I. *et al*: Long-term effectiveness of interpersonal skills training in medical schools. *J Med Educ*, 55:595-596, July 1980.
9. Bird, J. and Lindley, P.: Interviewing skill: the effects of ultra-brief training for general practitioners—a preliminary report. *J Med Educ*, 53:349-355, September 1979.
10. Hannary, D.: Teaching interviewing with simulated patients. *J Med Educ*, 54:246-248, July 1980.
11. Hollifield, G.; Rousell, C.; Bachrach, A.: A method of evaluating student-patient interviews. *J Med Educ*, 32:853-858, December 1957.
12. Cassata, D.; Conroe, R.; Clements, P.: A program for enhancing medical interviewing using video-tape feedback in the family practice residency. *J Fam Pract*, 4:673-677, April 1977.
13. Jackson, E.: Convergent evidence for the effectiveness of interpersonal skill training for dental students. *J Dent Educ*, 42:517-520, September 1978.
14. Dunning, D.G. and Lange, B.M.: The effect of feedback on student use of interpersonal communication skills. *J Dent Educ* 51: 594-596, October 1987.
15. Meskin, L.H.; Loupe, M.; Martins, L.: Postgraduation assessments of a dental school TEAM program experience. *J Dent Educ*, 41: 571-573, September 1977.
16. Davis, E.L.; Tedesco, L.A.; Nicosia, N.E. *et al*: Use of videotape feedback in a communication skills course. *J Dent Educ*, 52:164-166, March 1988.
17. Kress, G.C. and Ehrlich, M.A.: Development of confidence in child behavior management through role playing. *J Dent Educ*, 54:619-622, October 1990.

WHY WE WISH TO KILL

Most people who are successful in their professions, particularly in politics, sports, and the entertainment industry, activate jealousy and resentment in the less successful. The more success we enjoy, especially if it brings great acclaim, the more we will be the recipient of derogatory remarks, even from family and supposedly dear friends.

The secure politician, who is aware of a sense of purpose and self-identity, does not feel endangered. He is able to debate issues, disagree clearly and firmly with his angered opponents. That is, if he does not feel like a child and expect to be loved by everybody, for then he will feel in a constant rage. He may become physically assaultive, even murderous, or sink into deep depression.

In any discussion of how our murderous wishes evolve, it is important to keep in mind that most children and most adults try their best to deny and repress their violent thoughts because rage, hatred, and the idea of murder never feel bearable for long.

Furthermore, as we have indicated, the murderous impulse is most commonly felt toward those we need—need desperately at times—such as parents and siblings, and later, spouses, bosses, and colleagues. Many serious emotional illnesses are wishful expressions of murder turned against the self rather than against another person.

Strean, H. and Freeman, L.: *Our wish to kill*. New York: St. Martin's Press, 1991, pp 28-29.

ABSTRACTS

Waldman, H.B. and Perlman, S.P.: Children with disabilities are aging out of dental care. J Dent Child, 64:385-390, November-December 1997.

Each year thousands of poor children with disabilities "age out" of dental programs that were established to meet their needs. But now they are older. Most states provide minimal if any dental services (with limited reimbursement levels) for adults within the Medicaid system. In an effort to stimulate an awareness of these difficulties, a review is provided of 1) the number of the "former" children with disabilities who are faced with the unavailability of dental services and 2) the limitations of the adult Medicaid dental program.

Children with disabilities: Medicaid limitations

Waldman, H.B.: Homeless children. J Dent Child, 64:391-394, November-December 1997.

Homeless children are an "invisible" population within our community. A review is provided of the economic, social, medical and dental conditions of these children.

Children; Homeless

Kawakami, M. and Takano-Yamamoto, T.: Orthodontic treatment of a patient with hypophosphatemic vitamin D-resistant rickets. J Dent Child, 64:395-399, November-December 1997.

Hypophosphatemic vitamin D-resistant rickets, when developed later in life, is less severe and may not be characterized by rickets or other osseous deformities. A Japanese girl, age nine years and one month, was first seen in the Dental Hospital of Osaka University, complaining of the crowding of the maxillary teeth. At one year of age, the patient was admitted to Osaka University Hospital for her leg deformities. Although the patient has been administered 4 μ g 1 α -hydroxyvitamin D₃ and 1.0g phosphorous daily, the serum phosphate has been low

and never reached normal level. This case was a Class II division 2 malocclusion with severe anterior crowding and retarded mandibular growth. We treated her with a functional appliance (elastic open activator), followed by the extraction of four premolars and the use of an edgewise appliance. No unfavorable root resorption or bone defect occurred. Good occlusion was achieved and the facial features were pleasing.

Rickets; Hypophosphatemic vitamin D-resistant rickets; Orthodontic treatment

Haruki, T.; Kanomi, R.; Shimono, T.: The differences in the chronology and calcification of second molars between angle class III and class II occlusions in Japanese children. J Dent Child, 64:400-404, November-December 1997.

The purpose of this study was to examine the differences in the times of eruption and calcification of the permanent dentition between skeletal class III and class II groups. (And also to examine the relationship between the time of eruption and the type of malocclusion) Fifty-three children, ages seven to ten years, were selected. Of these, twenty-six children (twelve boys and fourteen girls) were Angle class III with minus ANB and twenty-seven children (eleven boys and sixteen girls) were Angle class II with five or more ANB. Panoramic radiographs and cephalometric radiographs were used. The panoramic radiographs showed that the calcification of the maxillary second molars in class II were earlier than in class III using Nolla's classification. There was no statistically significant difference, however, for mean values of calcification stages, using Nolla's classification, between boys and girls. The cephalometric and panoramic radiographs showed that the times of eruption and calcification were earlier in the maxillary second molars than in the mandibular second molars for class II. In contrast, the times of eruption and calcification were earlier in

the mandibular second molars than in the maxillary second molars for class III. The times of eruption and calcification of the maxillary molars were significantly related to the length of the ANS-PNS. The longer the ANS-PNS, the earlier were the times of eruption and calcification. There was a significant relationship between the ANB angle and the time of eruption, as well as the ANB angle and calcification. The larger ANB had earlier calcification and chronology of maxillary second molars.

Eruption times; Dental calcification; Skeletal malocclusion

Kotsomitis, N. and Freer, T.J.: Inherited dental anomalies and abnormalities. J Dent Child, 64:405-408, November-December 1997.

Various dental anomalies and abnormalities are known to have a genetic etiology, probably due to a polygenic influence in many cases, although at least one anomaly has been identified as resulting from a single gene. Of particular interest and clinical significance in pediatric dentistry and orthodontics is the pattern of associations between a wide variety of conditions, including the impaction or ectopic eruption of teeth. The etiology of the latter problem is not understood, but the available evidence points strongly to a genetic etiology in many cases and a high likelihood of association with other anomalies of development. These findings highlight the importance of obtaining a complete family dental history.

Dental anomalies; Etiology; Genetics; Family history

Peck, Sheldon; Peck, Leena; Hirsh, Gary: Mandibular lateral incisor - canine transposition in monozygotic twins. J Dent Child, 64:409-413, November-December 1997.

Mandibular tooth transposition, involving the right lateral incisor and canine, is described in monozygotic twin girls. This report provides evidence suggest-

ing that genetic factors influence the development of mandibular lateral incisor - canine (Mn.I2.C) transposition. Interceptive orthodontic treatment for children with this rare occlusal abnormality should be considered.

Tooth transposition; tooth eruption, ectopic; Malocclusion; Twins, monozygotic; Clinical genetics

Viada, S.; Rivera, N.; Nava, S. et al: Temporary and permanent restorations for fractured permanent teeth with immature apices: A clinical study. J Dent Child, 64:414-416, November-December 1997.

Clinical and radiological studies of the fractured teeth of patients between seven and eleven years old were conducted to assess the use of permanent and immediate (emergency) techniques for reconstructing anterior permanent teeth with immature apices. The general hypothesis states that, given the physical and biocompatible properties of the materials used in the immediate (emergency) technique, there are no significant differences between the two techniques as far as pulp vitality and apical growth are concerned. A sample of fifty-six patients was selected. They had Class I, II or III fractures, for which permanent and immediate (emergency) techniques were used in equal numbers. Thermal, mechanical, and electrical tests were used to evaluate pulp vitality compared with the homologous tooth. Apical convergence was the radiological criterion used for determining the end of the radicular process. After one year, both techniques allow preservation of pulp vitality. Results show that, unlike the conventional technique (permanent), radicular formation was completed first in teeth reconstructed using the immediate (emergency) technique. Statistical analysis shows no definitive relationship between the technique used and completion of apical growth time ($P > 0.05$). In conclusion, comparison between the mediate (permanent) and immediate (emergency) techniques shows that the immediate (emergency) technique is an adequate alternative for reconstructing fractured teeth, because of the greater

esthetic and functional advantages at the patient's disposal.

Fractured permanent teeth; Emergency treatment; Permanent treatment

Nainar, S.M. Hashim and Crall, James J.: Caries experience in inner-city preschoolers at the time of their initial dental visit. J Dent Child, 64: 421-424, November-December 1997.

This retrospective study examined caries experience in a population of inner-city preschoolers at their initial dental visit. Clinical and sociodemographic data for 103 patients aged five years and under at the time of their first dental visit were obtained from the dental records of an inner-city community health center clinic located in a fluoridated area. The sample consisted of fifty-eight males and forty-five females who were seen during a continuous six-month period in 1991. The mean age of the subjects was 44.1 months (S.D. ± 10.5 months). Sixty-seven percent of the children in the sample were found to have caries. The mean number of decayed surfaces (ds) was 6.54 (± 7.16), with the following distribution by surface type: occlusal—2.89 (± 3.15); proximal—1.75 (± 2.91); buccal/lingual—1.90 (± 2.73). Caries prevalence and severity increased with age. Findings from this sample demonstrate that dental caries remains a significant problem in inner-city preschoolers, thereby underscoring the importance of early intervention programs.

Dental caries; Inner-city children, five years and under; First dental visit

Warren, J.J.; Hand, J.S.; Yao, J-H: First-molar caries experience among Taiwanese first-grade children. J Dent Child, 64:425-428, November-December 1997.

Evidence suggests that dental caries levels may have risen dramatically among Taiwanese children during the past twenty years. Accurate and consistent methods of assessing the dental caries in Taiwanese children, however, have not been widely employed. This project assessed, in detail, the caries status of 333

first grade children attending four schools in Taiwan. Mean DMFS and DMFT computed solely from first-molar data for these children were 1.46 and 1.19, respectively. Mean DMFS for females was 1.57, while for males it was 1.35. Overall, 48 percent of the children were caries-free in their permanent first molars. Nearly 90 percent of DMF was "decayed," while the remainder was "filled." Children from families in lower socioeconomic status (SES) categories had significantly higher mean DMFS than did higher SES children. Results suggest that caries prevention and dental treatment programs should be developed or enhanced to reduce the caries problem among Taiwanese children, particularly those at high risk.

Taiwanese children; Caries status

Ishikawa, Takayoshi; Kuwahara, Satsuki; Nagasaka, Nobuo: Effects of child behavior management training for dental hygiene students. J Dent Child, 64:435-438, November-December 1997.

The reliability and validity of a Confidence of Dental Hygienist in Child Management Scale were examined. Forty-eight dental hygiene students, nine dental staff, and thirty-one dentists were subjects for the reliability and validity scale. The acceptable level of the reliability on the Scale was shown by internal consistency and stability. The confidence scores of the clinician's group were significantly higher than the confidence scores of the students. The reliability and validity of the Confidence Scale were established.

Next, we evaluated the effect of behavioral science training, using videotape feedback and role playing on twelve dental hygiene students. Six students received the training, the other six students, the controls, had no training. The confidence scores of the training group were significantly higher than those of the control group, demonstrating that the training was effective for dental hygiene students.

Confidence scale; Child management; Dental hygiene student; Behavioral science training