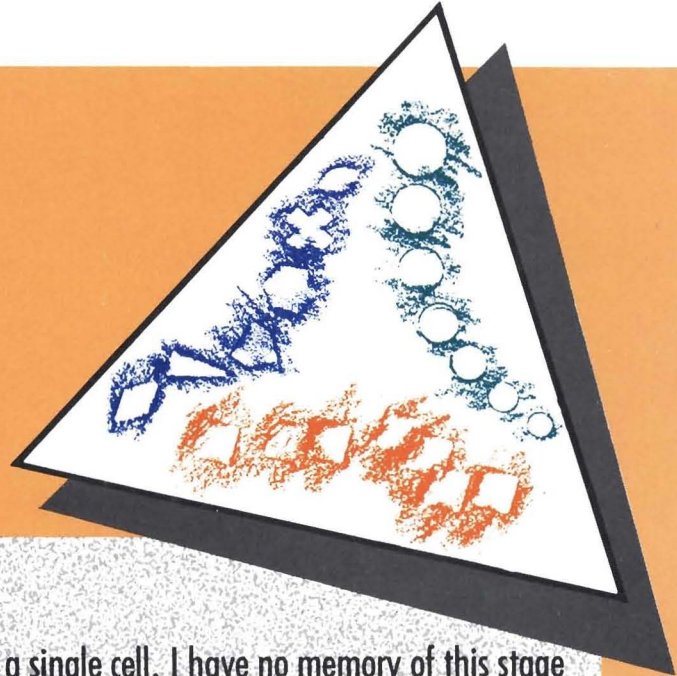


JOURNAL OF DENTISTRY FOR CHILDREN

/MJ 0022215
Dr. Milton I Houpt
251 Maple St
Englewood

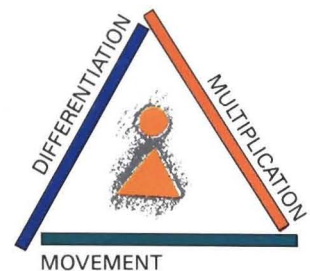


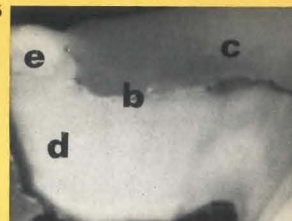
"... I was at one time, at my outset, a single cell. I have no memory of this stage of my life, but I know it to be true because everyone says so. . . . I know that I began dividing. I have probably never worked so hard, and never again with such skill and certainty. At a certain stage, very young, a matter of hours of youth, I sorted myself out and became a system of cells, each labeled for what it was to become, . . . all of them signaling to each other, calculating their territories, laying me out

By the time I was born, more of me had died than survived. It's no wonder I can't remember; during that time I went through brain after brain for nine months, finally contriving the one model that could be human, equipped for language."

Lewis Thomas
A Long Line of Cells, 1987

THE NEXT SEVERAL COVERS WILL SYMBOLIZE
RELATIONSHIPS BETWEEN DEVELOPMENTAL
STAGES OF COGNITIVE SKILLS AND SOCIAL
ELEMENTS OF CHILDREN'S ENVIRONMENTS.





JOURNAL OF DENTISTRY FOR CHILDREN

Volume 55 Number 1 January-February 1988

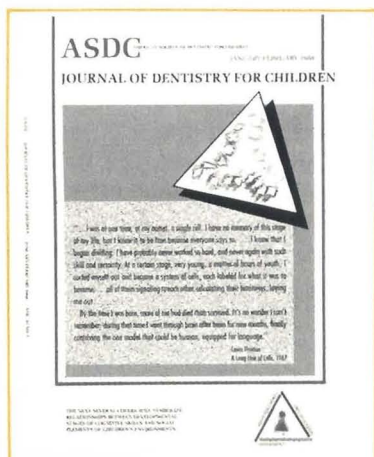
Copyright 1988 by the American Society of Dentistry for Children—ASDC JOURNAL OF DENTISTRY FOR CHILDREN, USPS # 279-480. Issued bimonthly—in January-February, March-April, May-June, July-August, September-October, and November-December—at 211 E. Chicago Avenue, Suite 1430, Chicago, IL, (312) 943-1244. Second class postage paid at Chicago, IL and additional mailing office. Subscription prices: within U.S.A., individuals \$60.00 per volume, institution \$80, single copies, \$15.00; Foreign (including Canada and Mexico) individuals \$70.00 per volume, institution \$90, single copies \$20.00. Thirty dollars and fifty cents (\$30.50) of the full membership dues are allocated to the Journal. Member—American Association of Dental Editors.

All copy and manuscripts for the journal should be sent directly to the Editorial Office, 730 Blaney Drive, Dyer, Indiana 46311, (219) 865-1184.

Prospective authors should consult "Information for Authors," which appears in the January and July issues. Reprints of this document may be obtained from the Editorial Office.

POSTMASTER

Change of address, subscriptions, advertising and other business correspondence should be sent to Executive Secretary, 211 E. Chicago Ave., Suite 1430, Chicago, Illinois 60611.



Lewis Thomas traces man's ancestry to the first bacterium to appear in the Earth's atmosphere and calls it Man's Ur-ancestor. To understand even a small part of a cell's function is to be fascinated with it.

Design and art by Sharlene Nowak-Stellmach.

- | | |
|-------------------|-------------------------|
| 14 Abstracts | 16 Editorial |
| 9 Annual meeting | 8 From the president |
| 4 Busy reader | 13 Index to advertisers |
| 12 Classified ads | 7 News |

SOCIAL FORCES

17 Dentistry and the children of poverty

Jimmy R. Pinkham, BS, DDS, MS; Paul S. Casamassimo, DDS, MS; Stephen M. Levy, AB, DDS, MPH

Despite huge expenditures for improvement, poor persons continue to suffer from problems of ill health, including dental disease.

COMPOSITE RESINS

25 Marginal adaptation of composite resins and dentinal bonding agents

Jorge M. Davila, DDS, MS; A. John Gwinnett, PhD, BDS, LDSRCS; Juan C. Robles, DDS

This investigation developed a method to determine whether gaps were present before desiccation procedures; the method was then applied to comparison of three commercially available dentinal bonding agents.

29 The effect of VLC Scotchbond and an incremental filling technique on leakage around class II composite restorations

Sergio Fisbein, CD; Gideon Holan, DMD; Rafael Grajower, PhD; Anna Fuks, CD

Incremental filling of class II cavities with a composite resin, employing two bonding agents, gave rise to less dye penetration than placement of the resin in bulk.

34 The invasive pit-and-fissure sealing technique in pediatric dentistry; an SEM study of a preventive restoration

G.P. DeCraene, DDS; C. Martens, DDS, PhD; R. Dermaut, DDS, PhD

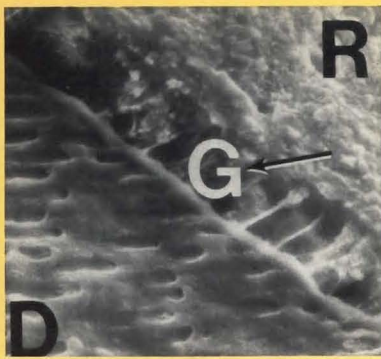
The application of sealants remains an important entity of clinical pediatric dentistry.

PATIENT EDUCATION

43 An alternative approach to prevention: computer-assisted patient education

Peter J. Fos, DDS, MPH

The patient of the 1980s is more concerned with what is necessary to ensure good health.



INFECTION CONTROL

47 Hygiene in dental practice - Part I: Potential pathogens and possibilities of contamination

William E. van Amerongen, DDS, PhD; Johannes de Graaff, PhD

Before an adequate prevention program can be designed for the dental practice, it is necessary to establish what microorganisms are in fact involved, and how these are transmitted.

56 Part II: Measures to reduce the risk of contamination

Johannes de Graaff, PhD; William E. van Amerongen, DDS, PhD; Grietje G. Mulder, DDS

Various possibilities of practicing contamination - reducing measures are discussed separately.

NUTRITION

64 Adolescent obesity

Laurel M. Mellin, MA, RD

Obesity in adolescence is distinct from that in other life stages in that the psychosocial and biological changes heralded by puberty profoundly influence obesity care.

TRAUMA

68 Emergency care in pediatric dentistry

MaryAnn Ready Battenhouse, DMD; M.M. Nazif, DDS, MDS; T. Zullo, PhD

Improved prognosis is often linked to availability and quality of treatment.

CASE REPORTS

72 Oral manifestations of Crohn disease: update of the literature and report of case

Robert A. Boraz, DDS

Because of the significant oral manifestations and the difficulty in diagnosis, a thorough understanding of Crohn disease by the dental practitioner is most important.

75 Oligodontia in the primary dentition with permanent successors: report of case

Takashi Ooshima, DDS, PhD; Keiko Sugiyama, DDS; Shizuo Sobue, DDS, PhD

Oligodontia is defined as the agenesis of numerous teeth, commonly associated with specific syndromes or severe systemic abnormalities.

OFFICERS

Prem S. Sharma President
Wieldon W. Crompton President-elect
Roland W. Hansen Vice President
Alfred C. Griffin Secretary-Treasurer

EDITORIAL STAFF

George W. Teuscher Editor-in-Chief
Donald W. Kohn Associate Editor
Jimmy R. Pinkham Associate Editor
Jane W. Teuscher Assistant Editor
730 Blaney Drive
Dyer, Indiana 46311

EDITORIAL AND PUBLICATIONS COMMISSION

Thomas K. Barber
Donald F. Bowers
Irving W. Eichenbaum
Donald W. Kohn
Ralph E. McDonald
John E. Nathan
Jimmy R. Pinkham
Prem S. Sharma
Robert Spedding
Paul P. Taylor

TRUSTEES

James L. Bugg, Jr.
Elliott J. Gordon
Donald W. Kohn
William H. Lieberman
Jimmy R. Pinkham
James F. Rundle
John M. Willis

EDITOR EMERITUS

Alfred E. Seyler

For the busy reader

Dentistry and the children of poverty—page 17

Poverty creates an environment that presents numerous obstacles to dental care, of which the lack of funds is only one. Others are: medical problems and health; communication; preventive practices; and social factors.

Requests for reprints should be directed to Dr. Jimmy R. Pinkham, Professor and Department Head, Department of Pediatric Dentistry, College of Dentistry, University of Iowa.

Marginal adaptation of composite resins and dental bonding agents—page 25

The method employed in this study showed that the gap is not an artifact resulting from preparation of the specimen for microscopy, but is in fact present *de novo*. Results show that the gap is probably accentuated by desiccation of the tissues, when they are readied for the SEM.

Requests for reprints should be directed to Dr. Jorge M. Davila, Senior Clinical and Research Associate, Department of Pediatric Dentistry, Eastman Dental Center, Rochester, NY.

The effect of VLC Scotchbond and an incremental filling technique on leakage around class II composite restorations—page 29

Visible light-cured dentin bonding agents are cured and adhere to dentin before the filling resin is applied. They are conceivably less prone, therefore, to displacement by contraction of the resin. They create stronger bonds between the filling resin and dentin than chemically cured bonding agents.

Requests for reprints should be directed to Dr. Anna Fuks, Senior Lecturer, Department of Pedodontics, the Hebrew University-Hadassah, Faculty of Dental Medicine, POB 1172, Jerusalem, Israel.

The invasive pit-and-fissure sealing technique in pediatric dentistry: a SEM study of a preventive restoration—page 34

The choice between the noninvasive and invasive techniques remains a matter of debate. The selection and application of other preventive restorations - avoiding amalgam - becomes important. Results here show clearly that by applying the invasive sealing technique, the choice of an adequate bur is important to obtain a caries-free fissure, in combination with minimal loss of tooth substance.

Requests for reprints should be directed to Dr. G.P. De Craene, Assistant Professor, Department of Pedodontics, State University of Ghent, De Pintelaan 185, B-9000 Gent, Belgium.

An alternative approach to prevention: computer-assisted patient education—page 43

Patients' attitudes about health care have changed significantly during the last several years. The opportunity to educate patients today about maintaining good dental and overall health has markedly improved. A computer-assisted instruction program to educate dental patients is described.

Requests for reprints should be directed to Dr. Peter J. Fos, Assistant Professor of Health Systems Management, Tulane University Medical Center, School of Public Health and Tropical Medicine, 1430 Tulane Avenue, New Orleans, LA 70112.

Hygiene in dental practice—Part I: Potential pathogens and possibilities of contamination—page 47

The patient's oral fluid, oral cavity, and mucous membranes are possible sources of potentially pathogenic microorganisms; furthermore, those of the dentist and assisting personnel are potential sources of contamination.

Requests for reprints should be directed to Dr. W. E. van Amerongen, Department of Paediatric Dentistry, ACTA, Vrije Universiteit, v.d. Boechorststraat 7, 1081 BT Amsterdam, The Netherlands.

Part II: Measures to reduce the risk of contamination—page 56

Reduction of the risk of contamination in the treatment of most infected patients – sometimes identified – can be achieved by the following measures: direct preventive behavior; protective measures; domestic cleaning; disinfection; sterilization; and indirect preventive behavior.

Requests for reprints should be directed to Dr. W. E. van Amerongen, Department of Paediatric Dentistry, ACTA, Vrije Universiteit, v.d. Boechorststraat 7, 1081 BT Amsterdam, The Netherlands.

Adolescent obesity—page 64

This topic arises from a number of origins. First, there has been a recent recognition that pediatric obesity has increased dramatically during the last 15 to 20 years. Second, as obesity in adults is refractory and intractable, prevention seems prudent.

Reprints are not available.

Emergency care in pediatric dentistry—page 68

This study investigated the incidence and classification of all pediatric dental emergencies in the ER at Children's Hospital of Pittsburgh during one calendar year.

Requests for reprints should be directed to Dr. Mary Ann Ready Battenhouse, Assistant Professor, Department of Pediatric Dentistry, Medical College of Georgia, 1459 Laney Walker Blvd., Augusta, GA 30912-0200

Oral manifestations of Crohn disease: update of the literature and report of case—page 72

The initial phase of Crohn disease begins with subtle clinical manifestations. Frequently, many months will pass between the first symptoms and the establishment of a correct diagnosis, which is based on clinical and laboratory findings. There are significant oral manifestations of the disease.

Requests for reprints should be addressed to: Dr. Robert A. Boraz, Associate Professor of Surgery and Pediatrics, University of Kansas Medical Center, 39th and Rainbow Boulevard, Kansas City, KS 66103.

Oligodontia in the primary dentition with permanent successors: report of case—page 75

This paper describes a two-year-old Japanese boy with oligodontia of the primary dentition who had the corresponding permanent teeth. An orthopantomogram was obtained when the patient was nearly five years old; all of the permanent tooth germs were recognized at that time.

Requests for reprints should be directed to Dr. Takashi Ooshima, Department of Pedodontics, Osaka University Faculty of Dentistry, 2-8, Yamada-Oka, Suita, Osaka 565, Japan.

Dentistry and the children of poverty

Social forces

Jimmy R. Pinkham, BS, DDS, MS
Paul S. Casamassimo, DDS, MS
Steven M. Levy, AB, DDS, MPH

In 1962, Michael Harrington's *The Other America* made Americans aware of the vastness of poverty in a seemingly affluent nation.¹ Soon thereafter, Lyndon Johnson declared war on poverty and created a federal bureaucracy to mount an offensive against poverty and the factors in society responsible for it. Since then, billions of private and federal dollars have been spent in charity, work programs, welfare, and health projects. More than twenty years later, poverty still exists and current economic trends and present governmental budget priorities suggest that it will remain a significant social problem in the years to come.² Despite the massive outlay of funds for improvement of the poor, the latter continue to suffer from problems of ill health, including dental disease.

The relevance of poverty to dentistry is tied to phenomena associated with societal and professional change. The American Dental Association and its components, as well as local communities, have increased access to dental care for the poor, targeting them directly or indirectly, by serving groups like the disabled, who tend to have a greater representation among the poor. In

Dr. Pinkham is Professor and Department Head, Department of Pediatric Dentistry, College of Dentistry, University of Iowa; Dr. Casamassimo is Professor and Chairman, Department of Growth and Development, School of Dentistry, University of Colorado; Dr. Levy is Assistant Professor, Department of Preventive and Community Dentistry, College of Dentistry, University of Iowa.

addition, dentists have participated in volunteer community efforts often directed at those who are poor or near poverty levels. Some dentists have added patients who are receiving public assistance to their practices, to solve personal economic problems. In many cases, the poor patient is a child with problems and needs created by an environment of poverty.

A purpose of this paper is to address the relevance of poverty and poor children to the practice of dentistry. The extent of the dental problems of poor children also is discussed and the difficulty encountered in obtaining treatment for these children will be explained. Lastly, the psychological aspects of poverty as they affect delivery of dental care to poor children are examined. The paper is presented in four sections:

- Poverty: a definition.
- Poverty's major obstacles to dental care and dental health.
- The dental health of children of poverty.
- Behavior management problems in the dental experiences of the children of poverty.

POVERTY: A DEFINITION

The definition of poverty is complex. Federal definitions are based on the cost of a subsistence diet and do not account for the other necessities of life or for those who earn even a few dollars above the poverty level. In 1979, the federal poverty level for a nonfarm family of four people was \$6,660. This amount was the cost of a subsistence diet for four people, assuming that the family shopper was efficient and bought foods supplying the nutrients needed for subsistence. In 1979, according to federal guidelines, the poor numbered in excess of forty million and accounted for about 25 percent of the population.³ Since these figures represent absolute poverty, they are misleading. For example, if those people within 125 percent of poverty were included, the number would have been swollen by almost ten million people.⁴

Today the income gap is greater than ever before. The poorest 40 percent of the nation is receiving only 15.7 percent of national income, while the top or wealthiest 40 percent is receiving 67.3 percent. Also, the number of families with incomes below \$5000, called the poorest of the poor, has grown faster than the poverty rate in general.²

Also excluded from the definition of poverty are many of the working poor: those people high enough above the poverty level to escape labeling, but poor enough not to afford more than the barest of essentials. No reliable measure of this population number is available, but if one expands the definition of poverty based on sugges-

tions by Townsend, "...inequities in distribution of income, capital assets, occupational fringe benefits, current public services and current private services," then the number of poor increases tremendously.⁵

Even when using the federal definitions, the poor present a significant challenge, both in number and in their gaining access to care. The poor are overrepresented among the aged, handicapped, and minorities, all of whom encounter obstacles to care, in addition to financial ones.^{2,6} Children also are prominent among the poor with approximately a fourth of them in this country living in or near poverty. A minority child has almost four times the likelihood of being reared in economic deprivation than does a white child.⁷

The poor can be found almost anywhere, but tend to inhabit the inner cities and outlying rural areas. The rural poor account for almost 40 percent of those in poverty and often do not benefit from the availability of services enjoyed by those in urban areas.⁷ Almost half of the poor inhabit cities.⁶ Although health and other services are more readily available, the concentration of the poor in urban areas tends to perpetuate factors that foster poverty with the close relationship between minority status and poverty, it is not surprising that the poor are most highly concentrated in the South and southeastern United States. Yet poverty isn't strictly a minority problem, since the total number of white poor is greater than the total number of nonwhite poor.

POVERTY'S MAJOR OBSTACLES TO DENTAL CARE AND DENTAL HEALTH

The impact of poverty on dentistry goes far beyond financial obstacles. Experience during Johnson's "War on Poverty" showed that even if financial obstacles were removed, many poor did not significantly increase their use of dental services. Poverty creates an environment that presents numerous obstacles to dental care, of which the lack of funds is only one.

Medical and Health Obstacles

Medical or health problems can present major obstacles to dental care. The poor suffer from disease at a disproportionately higher rate than the average American: rates of cardiovascular disease, sensory impairment and respiratory illness are four to eight times above the national average.⁸ These diseases tend to be serious and chronic.⁹ In fact, almost a third of all poor children come from a household where at least one parent has a chronic illness.⁸

Health problems can mean disability, unemploy-

ment, missed appointments, and compromised delivery of dental services. The health problems of the poor are compounded by a lack of medical care or by disjointed care by multiple practitioners in various clinics. The dentist treating the poor child may find that the patient is sick, malnourished, brain damaged or without adequate immunization or, at the very least, without a documented history of health status or previous care.¹⁰ The poor child is more likely to be sick and unaware of it. Parental ignorance about the signs of disease, failure to use available services, and other parental priorities often are the reasons.⁹ The poor child also may present with multiple diseases simultaneously, in what is called the clustering phenomenon.¹¹ In summary, the poor child seen by a dentist is more likely to be sick and to have an inadequate health history than the child from a family with an adequate income.

Obstacles to communication

Within the array of potential obstacles to dental care is inadequate communication, between dentist and child and between dentist and parent. The poor child is more likely to be learning-disabled or retarded and may present a difficulty in management because of a language problem.¹²⁻¹⁴ The poor have a higher incidence of mental illness, which also can compromise communication. Parental communication can also be affected by poverty. The attitude of the practitioner toward the parent can create friction and may be a reason why the poor tend to seek care at neighborhood clinics rather than private offices.^{8,15} Low educational levels and ignorance of health issues among the poor may further contribute to difficulties in communication.

Obstacles to preventive practices

Obstacles to the practice of preventive techniques are tied closely to communication. The inordinately high incidence of dental disease is in part the result of inadequate preventive health care, which can be the result of both a poor diet and attitudes incompatible with preventive behavior.^{16,17} Inadequate prevention practices among the poor seem to be a reasonable conclusion. Removal of plaque through regular oral hygiene requires knowledge and attitudes that many of the poor do not possess. Some authors have characterized the poor as being interested only in the present, needing immediate gratification, and having a low motivation to achieve.¹⁸ It would be unfair to thus label every poor person, but any one of these traits would tend to compromise a plaque control program. Diets high in car-

bohydrate and low in essential nutrients (diets typical of the poor) also contribute to the etiology of dental caries. Many rural poor also do not have access to optimal fluoride, either in communal water supplies or through professional fluoride therapy.

Secondary prevention, which aims at early detection and treatment, is lacking in the poverty population, where regular dental care is the exception. Contributing to the failure of secondary preventive efforts is a lack of knowledge about the signs of disease, differing attitudes about the significance of dental disease, and a differing set of priorities concerning the use of limited financial resources.⁸ For example, the parent whose child has many decayed primary teeth may assume that decay is normal, unimportant, or too costly to treat.

Social obstacles

A last set of obstacles to dental care and health are social factors that create a lifestyle inconsistent with seeking care from the private-practice sector. The private-practice system assumes a health-illness behavior model that begins with individual recognition of disease; knowledge of its seriousness; awareness of where to seek care; adequate resources, to pay for care; acceptance of professional recommendations; and availability of services.^{8,14} For the poor, each step of the model is complicated by problems. The poor person may not recognize or accept the signs of disease, because of ignorance or cultural norms. He may not have the resources to pay for care, or other related services needed to obtain care, such as daycare, transportation, or even a telephone call. Once in the dental care system, the patient may choose not to use scant resources for dental care, especially dental care of the primary dentition. Finally, services may not be readily available, because of a lack of transportation; isolated rural residence; or even fear of leaving one's home, because of the threat of crime and injury.

The poor encounter more than just financial obstacles to dental care. Theirs is a world complicated by physical, psychological, and social factors often beyond their control. The dentist should look individually at the effects of poverty on the delivery of care and assess in its broadest terms the impact of poverty on the cycle of care, from initiation of treatment to home-care measures and follow-up.

THE DENTAL HEALTH OF CHILDREN OF POVERTY

Epidemiological studies at both the national and state levels have identified relationships between family in-

come and children's dental health. In these studies, oral health status was generally poorer for children from families with low incomes than for those from families with high incomes. Because information about family income is available less frequently, however, than is information about race in such studies, race (and sometimes family size, parent's educational level, and parent's occupation) is often used as a surrogate measure of family income. These socioeconomic indicators are correlated with family-income levels. Members of racial minorities, particularly those with little education or from large families, and those in certain occupations "...tend to have lower incomes than the general population."¹⁸

The status of children's dental health is frequently judged by the following criteria, when reporting epidemiological results and sociodemographic variables of interest:

- Utilization of dental health services.
- Dental caries experience.
- Status of periodontal health.

All three of these measures indicated relatively poor dental health among those with lower family incomes. Number of missing teeth is a fourth measure often used in studies of adults, which is less useful with children, however, and will not be discussed here.

Utilization of dental care service

Data from the National Health Interview Surveys concerning interval- since-last-dental-visit and mean-number-of-dental-visits show that those from families with low incomes continue to have dental visits less frequently than those with high incomes, although the extent of the differences has decreased from 1964 to 1976 to 1981.^{18,19} For example, in 1978 to 1979, those from families with high incomes were approximately 60 percent more likely than those with low incomes to have had a dental visit within the last year.^{18,20} This relationship to family income was evident both among whites and nonwhites, although nonwhites continue to receive dental services at lower levels than do whites.

A statewide survey in Iowa in 1980 showed similar patterns.²¹ In the Iowa Survey of Oral Health, the proportion of people with recent dental visits increased as family income increased and there was an apparent threshold at \$10,000 of income.

Decayed, missing, and filled teeth

The DMF and def (or dF) indices are composite measures of decayed (D), missing (M), or filled (F) permanent teeth and decayed (d), extracted (e), or filled (f)

primary teeth, respectively. They often are used to compare rates of dental caries experience among groups of children. Although data from the 1971-74 National Health and Nutrition Examination Survey showed black children to have lower total DMF and def scores than did white children, blacks consistently have had a greater number of decayed and missing teeth and far fewer filled teeth.^{18,22} For example, six- to eleven-year-old blacks had an average of 0.9 decayed permanent teeth vs 0.6 for whites; 0.3 vs 0.1 missing permanent teeth; and 0.5 vs 0.9 filled permanent teeth. The differences were more dramatic for twelve- to seventeen-year-old children.¹⁸

Data from the 1979-1980 National Dental Caries Prevalence Survey showed similar patterns.²³ Nonwhite children needed 69 percent more restorations in permanent teeth, 44 percent more restorations in primary teeth, and four times as many extractions as did white children.

Data from statewide surveys are consistent with the national data. North Carolina from 1976 found nonwhite children to have higher numbers of decayed and missing teeth than did white children, while total DMF and df scores were lower for nonwhites.²⁴

Data from the 1980 Iowa Survey of Oral Health showed that children from the lowest family income group had four times the mean number of actively decayed primary teeth as did the children from other income groups.²¹

Results from the 1982-83 South Carolina Dental Health and Pediatric Blood Pressure survey showed that nonwhite children had significantly higher mean numbers of decayed and filled (df) primary teeth (3.6 vs 2.8), decayed, missing, or filled (DMF) permanent teeth (4.3 vs 3.4), and higher proportions of untreated decayed permanent teeth (67 percent vs 39 percent).²⁵ When eligibility for free or reduced-price school lunch programs is used as the determinant of low vs high income group or of socioeconomic status (SES), then a comparison of low SES to high SES in South Carolina is essentially a comparison of nonwhites to whites. Eighty-two percent of the high SES group were white and 70 percent of the low SES group were nonwhite. For example, 55 percent of high income children were in need of restorative treatment, vs 76 percent of low income children, as compared with 54 percent of white vs 79 percent of nonwhite children.

Periodontal disease

National data from 1871-74 showed that twelve- to seventeen-year-old blacks had almost twice as high a mean

Periodontal Index (PI) score as did whites (0.53 vs 0.29).¹⁸⁻²⁶ There were only small differences among six- to eleven-year-old children. Average Simplified Oral Hygiene Index (OHI-S) scores were 22 percent higher for six- to eleven- year-old blacks than for whites and 63 percent higher for twelve- to seventeen-year-old blacks than whites.¹⁸

Statewide data from North Carolina, Iowa, and South Carolina were similar to the national data.^{21,24,25} In North Carolina, social class had a "...very strong influence on periodontal disease."²⁴ This was true both in terms of average PI and OHI-S scores. In South Carolina, moderate to severe gingivitis was seen more frequently in children from low-income families than in those from high-income families, 38 percent vs 30 percent.²⁵

Summary

The data clearly show that children from poor families have more dental disease in need of treatment and receive less dental care than do other children. Health care planners and dental practitioners must remember this when designing dental care delivery systems or developing individual treatment plans for poor children.

BEHAVIOR MANAGEMENT PROBLEMS FOR THE CHILDREN OF POVERTY

An important challenge for dentistry in addressing the dental needs of poor children is the increased likelihood of misbehavior or inappropriate behavior, during the dental experience of these children, compared to children from more privileged social environments.

A child's ability to satisfy the dentist's social and behavioral expectations, during a dental appointment, will be addressed by four questions; where pertinent, general considerations and recommendations are offered.

Are respect for authority and compliance with authority and the ability to work well with adults compromised in the children of poverty?

Conclusion: Yes

Evidence: One of the fundamental goals in rearing a child in a highly complex society is to foster appropriate responses to authority. Respect for authority is first introduced to the child in the home. Appropriate child rearing also grooms respect for the authority of older people, teachers, law enforcement officials, property owners, and employers. It appears almost axiomatic that proper function within society means proper function within the authority systems of society.

Hunt notes that children from the slums have little

opportunity to develop respect for laws, concern for the needs of others, basic honesty, and tenderness.²⁷ Instead, they tend to acquire the opposite values; thus it can be reliably predicted that certain aspects of their behavior will be aversive to organized society. This, of course, could mean that abnormal or inappropriate behavior can be the typical response to a certain situation, even though no emotional or mental health issue is involved.

The inability of a child to coordinate behavior with identified authority is one aspect of delinquent behavior. Juvenile delinquency, though not limited to the poor classes, is a significant problem for such classes. Conger and Miller studied delinquency as it relates to personality and social class.²⁸ Their work revealed a higher percentage of delinquents from moderately and severely deprived socioeconomic environments than from non-deprived environments. Similarly, these authors found a higher percentage of delinquents from culturally isolated and disadvantaged ethnic minority groups than would be expected on the bases of their representation in the population as a whole.

Conger and Miller's findings about delinquency in America emphasize the poor attitudes that delinquents have toward authority, including the failure to understand the need for rules and regulations in society and the need for abiding by these rules. These authors concluded that delinquents harbor both resentment toward and show rejection of authority in any form. Also, when asked to react to a roster of rules and behavioral expectations, delinquent children tend to give up more easily than nondelinquent children.²⁸

It is important to note that Conger and Miller do not consider intelligence to be strongly related to delinquency. Kvaraceus and Miller also concluded that delinquency among the poor is not necessarily predictive of emotional disturbance.²⁹ Their 1959 publication concluded that a middle-class youth who became involved in overt delinquency was much more likely to be emotionally disturbed than was a lower-class delinquent youth.

Discussion and recommendation

Since the dentist represents a powerful authority figure in the office and during the course of a dental appointment, there may be more rejection and resentment of the dentist among a group of socioeconomically and culturally disadvantaged children than there would be from a more socioeconomically and culturally advantaged group. The dentist should anticipate certain inabilities of the poor child to follow instructions, since

such an inability may be one of the ramifications of the child's poverty. Although it would be impractical for the dentist to abandon entirely a posture of authority, an emphasis on gentleness may help some children. Also, since there may be a tendency for poor children to deplete their limited ability to cope with the expectations of authority, their dental appointment should be conducted as efficiently as possible.

Are the abilities to learn and to communicate affected in the children of poverty?

Conclusion: Yes

Evidence: Hunt concluded that children from lower-class environments are substantially compromised in their preschool years, in the development of language and in being motivated to learn.²⁷ Hunt further concluded that, generally, these children begin their traditional schooling, incompetent and typically unfit to profit substantially from the schools' curricula. In support of this conclusion, Hunt cites research that shows that the children of poverty from many countries and cultures lack the opportunity to acquire language and number skills, motivational habits, and the values and standards that underlie confidence.

Many authors have drawn similar conclusions about the liabilities of language development in poor children. In some instances, the poor infants and younger children, because of the crowded circumstances in which they are reared, become habituated to a vocal din and show, therefore, inadequacies in auditory discrimination.³⁰ Also the verbal interaction between poor children and their elders is often limited to commands, and the children may have little or no experience in formulating answers to questions.^{31,32} Such experiences may predict that their development in prepositional relationships and syntactical rules of standard language may be completely unlearned.³³

It is important for individuals who need to communicate with poor children to realize that they may not be accustomed to receiving explanations. The processes or reasonings to explain certain phenomena may seldom or never be related to them.³⁴ In fact, it has been concluded that the children of poverty often are "shut up" as they ask questions or attempt conversations.³⁵ It is hardly surprising that words used in recognizing someone, use of general vocabulary, length of remarks, and complexity of sentence forms in these children fall substantially below the norms.^{31,32,36}

Discussion and recommendation

The ability to inform a child about the procedures that take place at the dental appointment is important in

overcoming his perceived or acquired fears. Because of their lack of language skills, the children from poverty may not understand the dental experience as quickly as will children from a more fortunate socioeconomic environment. This does not mean that they cannot learn, but rather implies that, as a general rule, they will be significantly slower than nondisadvantaged children in understanding information from oral communication alone.

The educational phase of a dental appointment that is structured in a certain manner and at a certain pace for middle-class children may be inappropriate in both content and time for poor children. A slower, more repetitive method may be preferred. Visual aids, which are helpful in educating all children, would be particularly advantageous for poor children.

In addition, because of the possibility of compromised language skills, the dentist should consider using non-verbal communication, particularly facial expression, when reinforcing, maintaining, or intercepting behaviors. The following excerpt from Coles's *Migrants, Sharecroppers, Mountaineers* discusses the communication style of a migrant worker mother with her children and may indirectly support the above statement.

"She can be very stern and very insistent with them. She doesn't really speak to them very much, explain this and that to them, go into details, offer reasons, appeal to all sorts of ideas and ideals and convictions. She doesn't coax them or persuade them or argue them down. She doesn't beat them up either, or threaten to do so. It is hard to see what she does, because words are shunned by her and anyway don't quite convey her sad, silent willfulness, a mixture of self-command and self-restraint; and it is hard to describe what she does, because whatever happens manages to happen swiftly and abruptly and without a lot of gestures and movements and steps and countersteps. There will be a word like 'here' or 'there' or 'OK' or 'now' or 'It's time,' and there will be an arm raised, a finger pointed, and most of all a look, a fierce look or a summoning look or a steady, knowing look—and the children stir and move and do."³⁷

Are emotional health and mental health major problems for the children of poverty and, if so, are the abilities of poor children to handle stress reduced?

Conclusion: Yes

Evidence: The lower social classes show the highest rates of mental and emotional disorders.³⁸ In 1969, Bruce and Barbara Dohrenwend published a review of the literature on studies that sought to link social status and psychological disorders.³⁹ In the report, they showed that approximately 80 percent of these studies had

shown a consistent trend in establishing that the highest rates of mental illness existed among those at the lower end of the social ladder. The lowest rates were among those at the top of the social ladder.

The Dohrenwends did not resolve whether individuals comprising the lowest levels of society are predisposed to psychological disability or whether the environmental conditions of the lowest social classes induce psychological disorders. Segal and Yahraes concluded, however, that both of these factors are true.⁴⁰ Rogler and Hollinghead also endorse this duality and suggest that the environmental stress of poverty, paired with genetic susceptibility, strongly predicts the onset of the signs and symptoms of emotional and mental illnesses.⁴¹ Unquestionably, the likelihood of stress is generally greater in the homes of the poor than in the homes of the more privileged.⁴⁰

Discussion and recommendation

Poor emotional and mental health can cause deleterious and undesirable behavior during rigorous and demanding dental appointments. It would seem, therefore, that the indications for the pharmacological control of patient behavior, because of poor emotional and mental health will be present more frequently for a population of poor children than for a matched population of more fortunate children.

Are perceptions of fear and of pain in the children of poverty different from those in children of higher social classes?

Conclusion: Information on this topic is not abundant. Increased fear and pain perception, apparently, are not problems.

Evidence: A more fearful psychological personality or a more pain-prone personality status in lower class children, when compared to higher class children, is not substantiated by the literature. Holmes found the children of lower-class environments in a daycare center to be less fearful than children of an upper-middle class environment in a nursery school.⁴² It should be noted that Holmes' study was conducted in 1935, during the Great Depression, when children of lower-class parents could be expected to have encountered more painful stimulation than children of the upper-middle class.

Other studies suggest that early encounters with painful stimulation may serve to raise what Helson labels the adaptation level for painful stimulation, thereby reducing the adversiveness of noxious stimuli.⁴³ Helson states that there probably are early experiences that predispose infants to being sensitive and anxious, but their nature cannot yet be clearly specified. He also

suggests that poor children probably are "tougher" regarding pain, if for no other reason than they may be accustomed to it.

Discussion and recommendation

Despite the scarcity of information addressing this question, the lack of language and communication skills in the children of poverty would seem to predict at least a more rigid persistence of those fears that are customarily addressed in the dental office by explanation, tell-show-do, and other techniques designed to gain cooperation and control behavior.

The fact that health care may be more threatening to the poor child's parents as well, may also influence the fear-status of such children.

It is recommended that the child of poverty not be regarded as either "tough" or "tender," but rather as an individual whose fears must be managed and who must be spared, as with all patients, any pain possibly associated with dental treatment.

SUMMARY

In this paper, the fact that poor people, and specifically poor children, are a problem for our society was discussed. The eradication of poverty is a goal that our nation has not been able to achieve and probably will not reach in the foreseeable future. Data that are supportive of links between poverty and increased needs for dental treatment and difficulty in the acquisition of professional dental care for children were reviewed. Finally, it was pointed out that social consequences surrounding the environments of poor children and, in some instances, the manner in which these children are reared, are predictive of misbehavior at dental appointments, particularly in younger age-groups. Such misbehavior paired with the finding that these children often do need restorative and surgical care may present challenges in patient management. The most important conclusion of this paper is that the dentist must be sensitive to the problems of poor children, responsive to their psychological needs, and prepared to give the extra energy and time that may be needed in management before and during the dental appointment.

REFERENCES

1. Harrington, M.: *The Other America*. New York: MacMillan Company, 1962.
2. Chicago Tribune Staff: *The American milestone*. Chicago: Contemporary Books, Inc., 1986.
3. American Dental Association: *Prevention and control of dental disease through improved access to comprehensive care*. Chicago: Am Dent Assoc, 1979, pp 11-18.

4. Couto, R.A.: *Poverty, politics, and health care: an appalachian experience*. New York: Praeger Publishing, 1975, pp 1-51.
5. Townsend, P.: *The concept of poverty*. New York: American Elsevier Publishing Company, 1970, pp 1-251.
6. Morrill, R.L. and Wohlenberg, E.H.: *The geography of poverty in the United States*. New York: McGraw-Hill Book Company, 1971, pp 1- 59.
7. *Social change and the mental health of children: report of Task Force VI and excerpts from the report of the committee on children of minority groups by the joint commission on mental health of children*. New York: Harper and Row, 1973, pp 17-88.
8. Kane, R.L.; Kasteler, J.M.; and Gray, R.M.: *The health gap: medical services and the poor*. New York: Springer Publishing Company, 1976, pp 40-58.
9. Bullough, B. and Bullough, V.L.: *Poverty, ethnic identity, and health care*. New York: Appleton-Century Crofts, 1972, pp 1-136.
10. Seham, M.: *Blacks and american medical care*. Minneapolis: University of Minnesota Press, 1973, pp 1-136.
11. Hurley, R.L.: *Poverty and mental retardation: a causal relationship*. New York: Random House, 1969, pp 128-164.
12. Birch, H.G. and Gussow, J.D.: *Disadvantaged children: health, nutrition, and school failure*. New York: Harcourt, Brace and World, 1970, pp 46-80.
13. Ingalls, R.P.: *Mental retardation: the changing outlook*. New York: John Wiley and Sons, 1978, pp 161.
14. Kosa, J.; Antonovsky, A.; and Zola, I.K.: *Poverty and health: a sociological analysis, Second Edition*. Cambridge, MA: Harvard University Press, 1975, pp 135-192.
15. Glazer, N.Y. and Creedon, C.F.: *Children and poverty: some sociological and psychological perspectives*. Chicago: Rand-McNally and Company, 1969, pp 118-120.
16. Rothman, D.J. and Rothman, S.M.: *On their own: the poor in modern America*. Reading, MA: Addison-Wesley Publishing Company, 1972, pp 211-213.
17. Luft, H.S.: *Poverty and health*. Cambridge, MA: Ballinger Publishing Company, 1978, pp 1 - 240.
18. *Health status of minorities and low income groups*. DHHS Publication No. (HRSA) HRS-P-DV 85-1. U.S. Department of Health and Human Services, Health Resources and Services Administration, Bureau of Health Professions, 1985.
19. *Health, United States, 1983*. DHHS Publication No. (PHS) 84-1232. U.S. Department of Health and Human Services, National Center for Health Statistics, Hyattsville, MD, December 1983.
20. Wilder, C.S.: *Dental visits, volume and interval since last visit, United States, 1978-1979*. Vital and Health Statistics, Series 10, No. 138. DHHS Pub. No. (PHS) 82-1566. U.S. Department of Health and Human Services, Public Health Service, Washington, D.C., April 1982.
21. *Iowa survey of oral health: 1980*. University of Iowa College of Dentistry and Iowa Dental Association, 1982.
22. Harvey, C.R.: *Decayed, missing and filled teeth among persons 1-74 years, United States, 1971-74*. Vital and Health Statistics, Series 11, No. 223. DHHS Publication No. (PHS) 81-1673. U.S. Department of Health and Human Services, Public Health Service, Hyattsville, MD., August, 1981.
23. *Dental treatment needs of United States children, 1979-1980*. NIH Publication No. 83-2246. U.S. Department of Health and Human Services, National Institute of Dental Research, December, 1982.
24. Hughes, J.T.; Rozier, R.G.; and Ramsey, D.L.: *Natural history of dental diseases in North Carolina, 1976-1977*. Durham, North Carolina: Carolina Academic Press, 1982.
25. *The South Carolina dental health and pediatric blood pressure survey 1982-83*. South Carolina Department of Health and Environmental Control.
26. Kelly, J.R.: *Basic data on dental examination findings of persons 1-74 years, United States, 1971-1974*. Vital and Health Statistics Series 11, No. 214. DHEW Publication No. (PHS) 79-1662. U.S. Department of Health, Education, and Welfare, Public Health Service, Hyattsville, MD, May, 1979.
27. Hunt, J.M.: *The challenge of incompetence and poverty*. Chicago: University of Illinois Press, 1969, pp 1-289.
28. Conger, J.J. and Miller, W.C.: *Personality, social class, and delinquency*. New York: John Wiley and Sons, Inc., 1966, pp 1-249.
29. Kvaraceus, W.C.; Miller, W.B. et al: *Delinquent behavior: culture in the individual*. Washington, D.C.: National Education Association, 1959.
30. Deutsch, C.P.: *Auditory discrimination and learning social factors*. Merrill-Palmer Quarterly, 10:277-296, 1964.
31. John, V.P.: *The intellectual development of slum children: some preliminary findings*. Am J Orthopsychiatry, 33:813-822, 1963.
32. John, V.P. and Goldstein, L.S.: *The social context of language acquisition*. Merrill-Palmer Quarterly, 10:265-275, 1964.
33. Bernstein, B.: *Language and social class*. Brit J Sociol, 11:271- 276, 1960.
34. Chilman, C.S.: *Child-rearing and family life patterns of the very poor*. Welfare in Review, 3:3-19, 1965.
35. Jones, K.L.: *The language development of Head-Start children* doctoral dissertation, University of Arkansas. Ann Arbor, MI: University Microfilms, No. 66-11, 1966, p 609.
36. Anastasi, A.: *Differential psychology*, 3rd ed. New York: Mac-Millan Company, 1958.
37. Coles, R.: *Children of crises: volume II - migrants, sharecroppers, mountaineers*. Boston: Little, Brown Publishers, 1976, pp 1-650.
38. Fried, M.: *Social differences in mental health*. In Kosa, A. and Kosa, Z. (eds), *Poverty and health*, Cambridge, MA: Commonwealth Fund Book, Harvard University Press, 1969, pp 113-168.
39. Dohrenwend, B.P. and Dohrenwend, B.S.: *Social status and psychological disorder: a causal inquiry*. New York: Wiley Interscience, 1969.
40. Segal, J. and Yahraes, H.: *A child's journey: forces that shape the lives of our young*. New York: McGraw-Hill, 1978.
41. Rogler, L.H. and Hollingshead, A.B.: *Trapped: families and schizophrenia*. New York: Wiley, 1965.
42. Holmes, F.B.: *An experimental study of children's fears*. In Jersild, A.T. and Holmes, F.B., (eds), *Children's fears*. New York: Child Development Monographs, No. 20, Columbia University Teachers College, 1935, pp 167-296.
43. Helson, H.: *Adaptation level theory*. In Koch, S., (ed), *Psychology, a study of science*, Vol. 1, *Sensory, perceptual, and physiological formulations*. New York: McGraw-Hill, 1959, pp 565-621.

Marginal adaptation of composite resins and dentinal bonding agents

Composite resins

Jorge M. Davila, DDS, MS
A. John Gwinnett, PhD, BDS, LDSRCS
Juan C. Robles, DDS

While bonding to dental enamel is well established in clinical practice, the lack of adhesion of commercially available restorative resin materials to dentin and cementum is an acute problem. Current research has been directed toward the investigation of agents which form a bond between the dentin and resin. The establishment of an adhesive bond through a "chemical bridge" was first investigated by Buonocore *et al* (1956).¹ Bonding was achieved with bifunctional molecule containing a dimethacrylate group and a reactive phosphate group. The latter is postulated to form an ionic bond with the calcium in the dentin, while the former would copolymerize with the restorative resin. Commercially available systems currently use this same principle with variations in the type of reactive component from product to product. The subject has been eloquently discussed by Asmussen and Munksgaard (1985).²

One exception to the above system is the use of agents containing isocyanate groups. These groups react with collagen to form urethane and urea derivatives. Reactions may also occur with the inorganic phase of the tissue. Numerous laboratory studies have been pub-

Dr. Davila is Senior Clinical and Research Associate, Department of Pediatric Dentistry, Eastman Dental Center, Rochester, NY; Dr. Gwinnett is Professor, Oral Biology and Pathology, State University of New York at Stony Brook, Stony Brook, NY; Dr. Robles is Pediatric Dentist, Guatemala City, Guatemala, C.A.

lished on the bond-strengths of dentin bonding agents (e.g. Nakamichi *et al*, 1983; Asmussen *et al*, 1984) with the values being significantly lower than those reported for enamel bonding.^{3,4} Several reports (e.g. Davidson *et al*, 1984) have shown that stress created by shrinkage during polymerization probably exceeds the dentinal bond-strength in the three dimensional configuration of a cavity preparation.⁵ These workers and others have noted contraction gaps during the examination of the interface between the dentin and resin, by scanning electron microscopy (SEM). Since severe desiccation of the specimens is necessary for examination by SEM, artifactual gaps may have been created. This may be especially true for dentin due to its high water content, where significant volumetric change will occur upon dehydration.

The purpose of this investigation was to develop a method to determine whether gaps were present before the desiccation procedures. The method was then applied to a comparison of three commercially available dentinal bonding agents.

MATERIALS AND METHODS

The study was divided in two parts. The first was concerned with an examination of the interface between dentin and restorative resin, and the second involved a microleakage study.

Class I cavity preparations were made in forty-eight extracted, caries-free, human permanent molar teeth. After cavity preparation, the specimens were assigned to one of the following groups:

Group 1. Thirty specimens for scanning electron microscopy.

Group 2. Eighteen specimens to study dye penetration of the interface of enamel and dentin with the restorative resin.

The preparations were filled with Heliomolar in combination with Dentin Adhesit, Visar Fil with Creation Bond, and P-30 with Scotchbond. Equal numbers of specimens were assigned to the three products. Half of the specimens in each group of products were thermocycled for four hours at temperature extremes of 4°C and 80°C. The dwell time was a minute in each case. The specimens were evaluated for leakage, using a 0.5 percent solution of basic fuchsin. Before immersion in the dye, the teeth were covered with wax, leaving only the restoration and 2 mm beyond its margin exposed. The remaining samples were not subjected to thermocycling, but stored in a moist environment at room temperature. Those samples in the group designated for

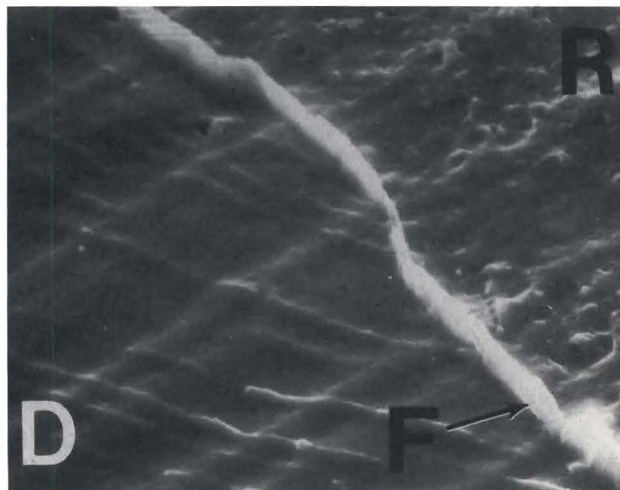


Figure 1. Scanning electron micrograph showing an impression of the resin (R)/dentin (D) interface. A flash of impression material (F) registers the gap at the interface. Original magnification x 750.

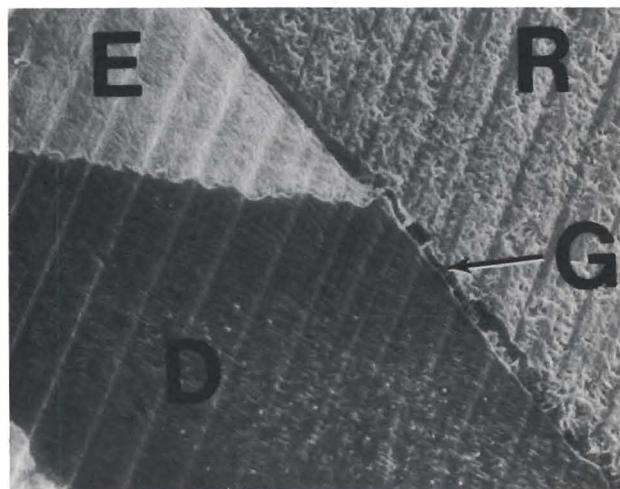


Figure 2. Scanning electron micrograph showing the relationship between the restoration (R), enamel (E) and dentin (D). A gap (G) exists at the interface between resin and dentin. Original magnification x 150.

microscopy were not immersed in dye, although half were thermocycled.

Evaluation of dye penetration was made by examining the sections cut longitudinally through the restoration. The sections were cut on a water-cooled, rotating diamond blade. Examination was made using a light optical stereomicroscope at 20x magnification. The penetration of dye was scored according to categories reported by

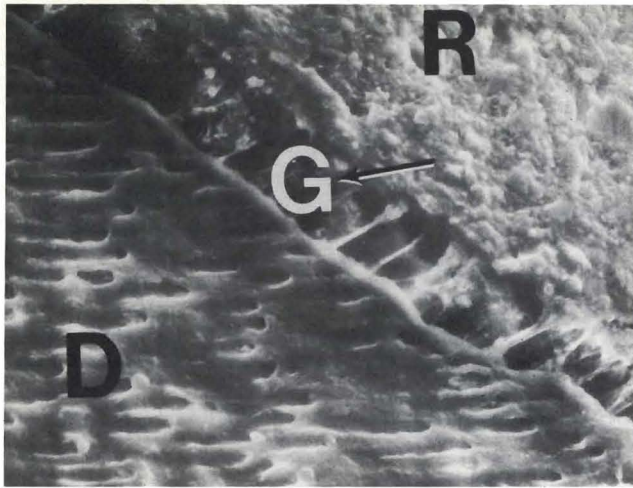


Figure 3. Scanning electron micrograph showing the gap (G) between resin (R) and dentin (D). Small, sparse connections cross the gap. The gap is significantly wider than recorded in the impression probably because of enhancement through desiccation of the dentin. Original magnification x 750.

Eriksen and Buonocore (1975).⁶ The categories were as follows:

- O - no marginal penetration.
- I - penetration of dye limited to the dentin-enamel junction.
- II - penetration of dye limited to the walls of the cavity.
- III - penetration of dye to include walls and floor of the cavity.
- IV - penetration of dye partly or completely through dentin to the pulp.

Preparation of the specimens for scanning electron microscopy was as follows: Each specimen was pared longitudinally through the restoration with a water-cooled, rotating diamond blade. Care was taken to ensure that the specimens remained wet at all times. After lightly drying, the cut surface of a half of each specimen was cleaned with 37 percent phosphoric acid for ten seconds. After washing and lightly drying, a polyvinyl

siloxane impression was made of the surface. Since this impression material is known to reproduce detail in the order of a few microns (Pameijer, 1979), it was used to replicate the interface between the resin and tissue before its dehydration in preparation for microscopy.⁷ Both the impression and specimen halves were mounted and conductively coated with gold for examination in a scanning electron microscope. Observations were recorded on Polaroid film.

FINDINGS

Group 1

An examination of the impressions in the scanning electron microscope showed clear evidence of a gap at the interface between dentin and all the restorative resins tested. The gap was registered as a thin flash of impression material (Figure 1). Both thermocycled and non-thermocycled teeth showed gaps before desiccation. Direct observation of the specimens showed the gaps to be wider than defined by the impression (Figures 2, 3). More detail was resolved than was possible with the impression material. Close examination of the gap between resin and tissue (Figure 3) showed sparse connections across the region. These may represent tearing of the resin or extrusion of resin that might have penetrated into dentinal tubules.

Group 2

The results obtained from experiments of marginal leakage are illustrated in the Table.

It is evident from the group of thermocycled specimens, that Visar Fil showed the least dye penetration, followed closely by P-30. Heliomolar exhibited the greatest amount of leakage. When no thermocycling was employed, the leakage was less severe in all cases and P-30 showed no leakage at all.

DISCUSSION

It is apparent from the results of this study that no bond exists between the dentin and the restorative material systems tested. Davidson *et al* (1984) demonstrated contraction gaps in association with dentin bonding and additionally the role that shrinkage of the resin during polymerization plays in causing the gaps: in essence,

Table 1. Eighteen specimens, assigned equally to two categories (thermocycled and nonthermocycled); each group of six specimens was filled with one of the three materials to be tested.

Marginal leakage code	Thermocycled		
	Group A (Heliomolar)	Group B (Visar-Fil)	Group C (P-30)
0		1	2
1		2	1
2			
3	1		
4	2		
Total	3	3	3

Marginal leakage code	Nonthermocycled		
	Group A (Heliomolar)	Group B (Visar-Fil)	Group C (P-30)
0	1	2	3
1	1	1	
2	1		
3			
4			
Total	3	3	3

that the stress created in the restoration exceeds the strength of the bond to dentin.⁵ The method employed in our study shows that the gap is not an artifact resulting from preparation of the specimen for microscopy, but is in fact, present *de novo*. The results show that the gap is probably accentuated by desiccation of the tissues, when they are readied for the scanning microscope. This finding also supports the work of Brannstrom *et al* (1984), who was able to penetrate the contraction gap with a resin tagged with a fluorescent dye.⁸ They reported contraction gaps with P-30 as well as with other restorative materials not tested by us at this time. The results also show no merit to either dentin system: namely, the one utilizing reactive phosphate groupings or isocyanate.

We conclude, as have others, that most dentin bonding agents as currently formulated, do not provide in clinical use, continuity between dentinal tissue and restorative resin, and that our clinical expectations must be tempered accordingly. Further research is needed and many useful new directions were compiled under the editorship of Vanherle and Smith (1985).⁹

REFERENCES

1. Buonocore, M.S.; Wileman, W.; and Brudevold, F.: A report on resin composition capable of bonding to human dentin surfaces. *J Dent Res*, 35:846-851, 1956.
2. Asmussen, E. and Munksgaard, E.C.: Adhesion of restorative resins to dentinal tissues. In *Posterior composite resin dental restorative materials*. Ed. Vanherle, G. and Smith, D.C. The Netherlands: Peter Szulc Publishing Co., 1985, pp 217-229.
3. Nakamichi, I.; Iwaku, M.; and Fusayama, T.: Bovine teeth as possible substitutes in the adhesion test. *J Dent Res*, 62:1076-1081, 1983.
4. Asmussen, E. and Munksgaard, E.C.: Formaldehyde as bonding agent between dentin and restorative resins. *Scand J Dent Res*, 92:480-483, 1984.
5. Davidson, C.L.; deGee, A.J.; and Feilzer, A.: The competition between the composite dentin bond strength and the polymerization contraction stress. *J Dent Res*, 63:1396-1399, 1984.
6. Eriksen, H. and Buonocore, M.G.: Marginal leakage with different composite restorative materials "in vitro". *J Oral Rehabilitation*, 3:315-322, 1976.
7. Pameijer, C.H.: Replication techniques with new dental impression materials in combination with different negative impression materials. *Scan Electron Micros*, II: 571-574, 1979.
8. Brannstrom, M.: Composite resin restorations: biological considerations with special reference to dentin and pulp. In *Posterior composite resin dental restorative materials*. Ed. Vanherle, G. and Smith, D.C. The Netherlands: Peter Szulc Publishing Co., 1985, pp 71-81.
9. Vanherle, G. and Smith, D.C.: *Posterior composite resin dental restorative materials*. The Netherlands: Peter Szulc Publishing Co., 1985.

DENTINAL BONDING

Dentin bonding is of tremendous significance in restorative dentistry. Long-term clinical observations (Jordan *et al*, 1977) have documented clearly that enamel bonding procedures are ultraconservative, highly reliable, and biologically innocuous. In the event that dentin bonding is eventually rendered equally reliable, the entire conceptual basis of restorative dentistry will undergo significant change (Bowen *et al*, 1982). The reason for this is that, until recently, the fundamental approach to retention of restorative materials has been almost completely dependent on the resistance retention form of box-type undercut cavity preparation procedures, which invariably result in extensive sacrifice of sound tooth structure. The necessity for this has been dictated by the fact that few if any restorative materials bond reliably to tooth structure. Should materials be developed that in fact bond to both enamel and dentin mechanically and/or chemically, the textbooks relating to restorative dentistry will have to be rewritten since teeth may be restored in the future using infinitely more conservative techniques than have ever been used in the past.

Jordan, R.E. *et al*: *Esthetic Composite Bonding*.
St. Louis: The C.V. Mosby Company, 1986, p 184.

The effect of VLC Scotchbond and an incremental filling technique on leakage around class II composite restorations

Sergio Fisbein, CD
Gideon Holan, DMD
Rafael Grajower, PhD
Anna Fuks, CD

Composite resins have gained great popularity as anterior filling materials. In recent years a large number of "posterior composites" for occlusal restorations have appeared on the market. Their use for class II restorations, however, has not yet been fully accepted. Insufficient sealing of margins is a major disadvantage of these materials.¹⁻³ This occurs especially at the gingival margins, where little or no enamel is present, and where it is technically difficult to obtain dry margins for etching.⁴ Gaps at the margin between resin and tooth may result from contraction due to polymerization of the setting resin.⁵ Their size and shape may be affected, thereafter, by masticatory forces, thermal changes and water sorption.⁶ Among the methods that have been suggested to decrease marginal leakage, one may mention: increasing the resin-enamel contact area by the preparation of bevels at the margins, employing dentin bonding agents, or applying the filling material in increments.^{1,2,7,8}

In an earlier investigation, it was found that the ap-

plication of chemically cured Scotchbond does not reduce leakage at the margins of class II composite restorations.⁴ Setting of this bonding agent occurs only when the overlying resin is cured. It is possible, therefore, that the bonding agent is pulled away from dentin by the contracting resin during setting. VLC (visible light cured) dentin bonding agents are cured and adhere to dentin before the filling resin is applied. They are conceivably less prone, therefore, to displacement by contraction of the resin. They create stronger bonds between the filling resin and dentin than chemically cured bonding agents.⁹ VLC dentin bonding agents may be more effective, therefore, in preventing leakage than agents that set in contact with filling resins.

Curing an increment of a filling gives rise to a smaller contraction than curing of an entire filling, placed in bulk. Part of the space resulting from contraction of the first increment will be filled by the second increment, etc. In addition, if the first increment is placed on the dentin bonding agent at the cervical floor, without being anchored on other surfaces, it may be expected to contract toward dentin and not away from it.

The object of this study was to determine the effect of a VLC dentin bonding agent and of an incremental filling technique on microleakage around class II composite restorations *in vitro*.

Dr. Fisbein is a postgraduate student, Department of Pedodontics, presently in private practice, in Curitiba, Brazil; Dr. Holan is instructor, Department of Pedodontics; Dr. Grajower is head, Laboratory for Dental Materials; Dr. Fuks is senior lecturer, Department of Pedodontics, the Hebrew University-Hadassah, Faculty of Dental Medicine, POB 1172, Jerusalem, Israel.

MATERIALS AND METHODS

Conventional mesial or distal class II cavities were prepared in sixty-two extracted or exfoliated primary molars. All gingival margins were prepared in enamel. Small carious lesions or amalgam fillings, which had been present in some of the teeth, were removed during cavity preparation. A bevel was prepared in the enamel at all cavity margins. The pulpal and axial walls were covered with a lining of Dycal.*

The teeth were randomly assigned to one of four experimental groups, according to the type of bonding material or filling technique, as shown in Table I. VLC Scotchbond ** was applied to enamel and dentin of the teeth of groups A and B. Enamel Bond ** was applied to enamel margins only of the teeth in groups C, D. Matrix bands were adapted to the teeth, using a Toffelmire matrix holder. All teeth were restored with the VLC posterior composite P-30. In groups A and C, the resin was placed into the cavities in three increments, starting with the proximal box. Each increment was condensed with an amalgam condenser and cured with light *** for twenty seconds, before placement of the next increment. In groups B and D, increments of the filling material were placed and condensed as before; but irradiation for forty seconds was carried out only after the entire filling was condensed. The proximal surfaces of the fillings were irradiated for an additional twenty seconds, after removal of the matrix bands.

The occlusal surfaces and margins were finished with Alpine stones followed by Soflex discs.** Those proximal surfaces with excess resin at the margins were polished with Soflex discs to remove the resin flashes. Other proximal surfaces were not finished, since they were smooth and showed no excess resin as the result of close adaptation of the matrix bands.

The restored teeth were subjected to thermocycling between 4 ± 2 and $60 \pm 2^\circ\text{C}$ for 100 cycles. The dwell times in each bath and the intervals at room temperature between baths, were one minute. The teeth were coated, immersed in a 2 percent solution of basic fuchsin, washed, embedded, sectioned mesiodistally, ground off and polished, as already described.⁴ The procedure of grinding and polishing was repeated to allow evaluation of four to six sections on each tooth.

The depth of dye penetration was considered an indicator for marginal leakage. Six degrees of marginal

leakage were distinguished, as suggested in the Fuks and Shey modification of the standard developed by Going *et al.*^{10,11} Degrees of leakage at the occlusal and cervical margins are represented by Arabic and Roman numerals, respectively.

Degree 0: No penetration of dye.

Degree 1 or I: Penetration of dye along the occlusal or gingival wall of the filling, adjacent to enamel only.

Degree 2 or II: Penetration of dye along the entire length of the occlusal or gingival wall of the filling, but not along the pulpal wall.

Degree 3 or III: Penetration of dye along the entire length of the occlusal or gingival wall of the filling, including the pulpal wall.

Degree 4 or IV: Penetration of dye along the filling, and diffusion of dye into dentin from the pulpal wall.

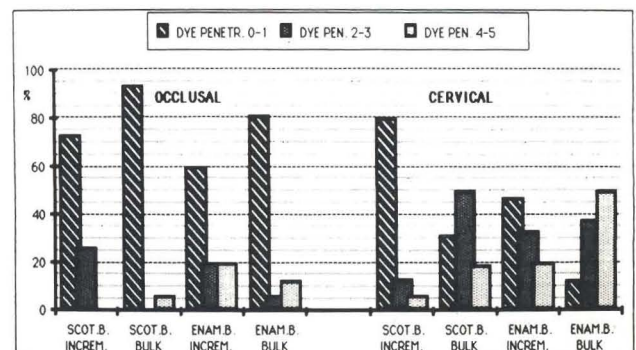
Degree 5 or V: Penetration of dye along the filling, and diffusion of dye through dentin to the pulp chamber.

RESULTS

The results are given in Table 2, for occlusal and cervical margins. In Figure 1 the results are represented as three levels of penetration, each corresponding to the combination of two degrees from the Tables. Figure 2 is a photomicrograph of a sample showing no leakage.

Leakage was generally found to be lower at the occlusal margin than at the cervical margin (Figures 3,4). Only for the experimental group A, involving the application of Scotchbond and placement of the resin in increments, a similar, low degree of leakage was found at both margins. For the occlusal margins, the differences between the experimental groups were not statistically significant. Table 2 shows that no leakage was found at 60 percent to 88 percent of these margins. The statistical significance of differences between the results of various

Figure 1. Bar diagram, representing the results at three levels of dye penetration, each corresponding to the combination of two degrees from the tables.



*L.D. Caulk Co. Milford, DE

**3M, Dental Products, St. Paul, MN

***Helipar II, ESPE, Seefeld, W.Germany

Table 1 □ Experimental groups of restored teeth.

Group	Number of teeth	Bonding agent	Filling technique
A	15	VLC Scotchbond	Incremental
B	16	VLC Scotchbond	Bulk
C	15	VLC Enamel Bond	Incremental
D	16	VLC Enamel Bond	Bulk

Table 2 □ Assessment of marginal leakage by depth of dye penetration.

Degree of dye penetration	VLC Scotchbond				Enamel Bond			
	Incremental		Bulk		Incremental		Bulk	
	N	%	N	%	N	%	N	%
Occlusal margins								
0	9	60	14	88	9	60	12	75
1	2	13	1	6	0	0	1	6
2	1	7	0	0	2	13	1	6
3	3	20	0	0	1	7	0	0
4	0	0	0	0	2	13	2	13
5	0	0	1	6	1	7	0	0
Cervical margins								
0	10	67	5	31	6	40	1	6
I	2	13	0	0	1	7	1	6
II	0	0	4	25	3	20	3	19
III	2	13	4	25	2	13	3	19
IV	0	0	0	0	0	0	3	19
V	1	7	3	19	3	20	5	31

Table 3 □ Levels of significance (P) of differences in leakage at the cervical margin, between groups of teeth restored by various methods.

Student t Test	Group	Scotchbond		Enamel Bond		Mann Whitney U test
		Incremental	Bulk	Incremental	Bulk	
		A	B	C	D	
	A	—	<0.1	N.S.	<0.02	
	B	<0.05	—	N.S.	N.S.	
	C	N.S.	N.S.	—	<0.05	
	D	<0.01	<0.1	<0.05	—	

Levels of significance to the left of the dashed lines were calculated according to the Student t test, and those to the right of the dashed lines according to the Mann Whitney U test.

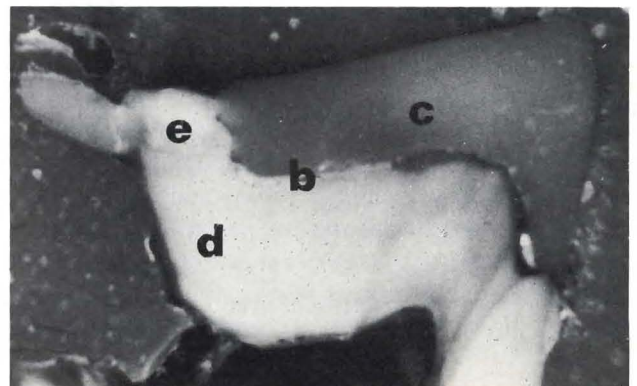
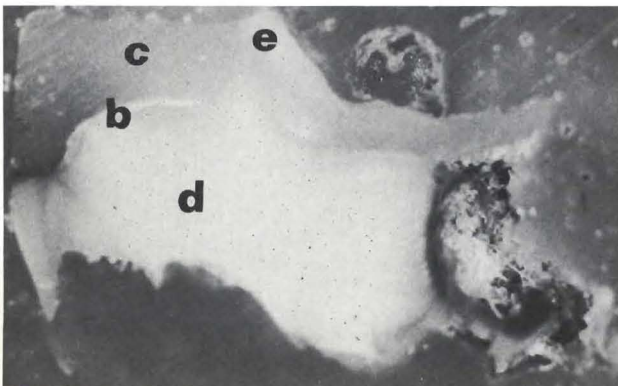
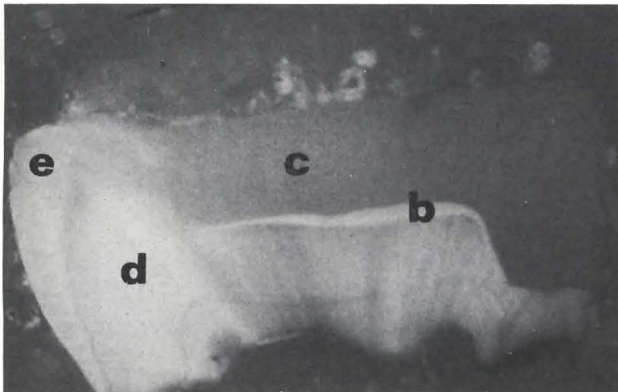
treatments, at the cervical margins, are presented in Table 3. The levels of significance were evaluated by means of the Student t test and Mann Whitney U test. These tests showed that the degree of dye penetration at the cervical margin decreased by application of the resin in increments. The differences resulting from using the two bonding agents were not significant, if the same packing techniques were applied. The greatest differences were observed, however, between restorations prepared in increments on Scotchbond and those prepared in bulk on Enamel Bond.

Pictured here (counterclockwise, from left) are:

Figure 2. Longitudinal section of a class II composite restoration, prepared with VLC Scotchbond and an incremental technique. This sample did not exhibit leakage at the occlusal or the cervical margins. e enamel, d dentin, c composite resin and b base.

Figure 3. Section of a restoration prepared with VLC Scotchbond and a bulk filling technique, showing no leakage at the occlusal margin and degree III leakage at the cervical margin.

Figure 4. Section of a restoration prepared with VLC Enamel Bond and a bulk filling technique, showing degree 3 leakage at the occlusal margin and degree V leakage at the cervical margin.



DISCUSSION

Donly and Jensen attached strain gauges to the buccal surfaces of teeth, in order to study the stresses created by different filling techniques, during polymerization of posterior resin restorations.⁷ They found that a faciolingual incremental technique resulted in significantly lower strain values than bulk polymerization. The differences obtained with respect to gingivoclusal increments were not statistically significant. The correlation between strain at the buccal surface and gap formation at the margin, however, remains to be established. The results of the present investigation show, that the technique involving placement of the filling in gingivoclusal increments, decreases leakage at the cervical margin. The results of this study are in agreement with the observations of LeClaire *et al*, the theoretical considerations of Davidson, and the suggestions of Vanherle *et al*.^{8,12,13}

In a previous study, class II restorations of the chemically cured resin P10 were placed in bulk on chemically cured Scotchbond or Enamel Bond.⁴ The results for Enamel Bond were better than obtained in the present investigation, whereas the results for Scotchbond were worse. The penetration of resin tags into etched enamel, and hence the retention, may be expected to be enhanced by a low viscosity of the uncured resin; a low surface energy of the enamel-resin interface; and a prolonged time-interval, during which the resin is sufficiently fluid to flow into enamel surface irregularities.¹⁴ The difference in the results for the two types of Enamel Bond can possibly be attributed to the fact that VLC Enamel Bond is cured in less than thirty seconds after its application, whereas setting of the chemically cured material takes much longer.

Chemically cured Scotchbond does not set at all, before a layer of filling resin is cured over it. Hence it can be pulled away from the tooth surface by contraction of the filling resin during polymerization. VLC Scotchbond, on the other hand, sets before application of the overlying resin, which could possibly aid in withstanding the forces exerted during contraction. The observed degree of leakage on the VLC Scotchbond samples was slightly lower than on the VLC Enamel Bond samples, but this difference was not statistically significant. The use of VLC Scotchbond and the incremental filling technique were synergetic in decreasing leakage, as the greatest difference was found between the type A samples, prepared in this manner, and the type D samples, prepared with Enamel Bond and the bulk filling technique.

Since the contraction during polymerization increases with the size of restorations, its effect on the leakage of small cervical restorations could be of less importance than of large class II restorations. Chem C Scotchbond has been found to decrease the leakage of restorations for cervical erosion lesions that have cervical dentin walls, as well as of class V restorations, surrounded by enamel.¹⁵⁻¹⁸ A similar degree of leakage was found around cervical restorations prepared by bulk filling as by an incremental filling technique.¹⁵ In another study, however, involving small restorations surrounded by enamel on facial tooth surfaces, smaller microgaps were found after incremental filling than after application of the resin in bulk.¹⁹

An additional factor which must be considered when comparing the results of this study with those of the previous one, is operator variability. The restorations in the two studies were placed by different clinicians. Hence, the occurrence of minor differences in cavity preparation, filling and finishing, could have been possible.

CONCLUSIONS

Incremental filling of class II cavities with a composite resin, employing two bonding agents, gave rise to less die penetration than placement of the resin in bulk. The lowest degree of leakage was found after application of Scotchbond and filling the cavity in increments. None of the investigated methods provided a perfect seal of the margins.

REFERENCES

1. Moore, D.H. and Vann, W.F.: A method to study marginal leakage of posterior composite restorations. *J Dent Res*, 65: Abstract 778, March, 1986.
2. Moore, D.H. and Vann, W.F.: Effect of the cavosurface bevel on posterior composite marginal leakage. *J Dent Res*, 65: Abstract 898, June, 1986.
3. Browne, R.M. and Tobias, R.S.: Microbial leakage and pulpal inflammation. A review. *Endod Dent Traumatol*, 2:177-183, October, 1986.
4. Holan, G.; Fuks, A.B.; Grajower, R. *et al*: *In vitro* assessment of the effect of Scotchbond on the marginal leakage of class II composite restorations in primary molars. *J Dent Child*, 53:188-192, May-June, 1986.
5. Jensen, M.E. and Chan, D.C.N.: Polymerization shrinkage and microleakage. In *Posterior composite resin dental restorative materials*. Vanherle, G. and Smith, D.C. editors. St. Paul: Minnesota Mining and Mfg. Co., 1985, pp 243-262.
6. Erickson, J. and Jensen, M.E.: Effect of pressure cycling on microleakage of composite restoration margins. *J Dent Res*, 65: Abstract 895, June, 1986.
7. Donly, K.J. and Jensen, M.E.: Posterior composite polymerization shrinkage in primary teeth: an *in vitro* comparison of three techniques. *Pediatric Dent*, 8:209-212, September, 1986.

8. LeClaire, C.C.; Blank, L.W.; Hargrave, L.W. *et al*: A 2 stage composite resin fill technique and microleakage below the CEJ. *J Dent Res*, 65: Abstract 799, March, 1986.
9. Bassiouny, M.A.: Adhesive tensile bond strength of light activated dentin bonding agents. *J Dent Res*, 65: Abstract 1306, March, 1986.
10. Fuks, A.B. and Shey, Z.: *In vitro* assessment of marginal leakage of combined amalgam-sealant restorations on occlusal surfaces of permanent posterior teeth. *J Dent Child*, 50:425-429, November-December, 1983.
11. Going, R.E.; Massler, M.; Dute, H.L.: Marginal penetration of dental restorations. *J Dent Res*, 39:273-284, March-April, 1960.
12. Davidson, C.L.: Resisting the curing contraction with adhesive composites. *J Prosthet Dent*, 55:446-447, April, 1986.
13. Vanherle, G.; Verschueren, M.; Lambrechts, P. *et al*: Clinical investigation of dental adhesive systems; Part I: An *in vivo* study. *J Prosthet Dent*, 55:157, February, 1986.
14. Fan, P.L.; Seluk, L.W.; O'Brien, W.J.: Penetrativity of sealants. *J Dent Res*, 54:262-264, March-April, 1975.
15. Crim, J.A. and Chapman, K.W.: Prevention of marginal leakage by four dental adhesives. *Gen Dent*, 235-236, May-June, 1986.
16. Hembree, J.H., Jr.: *In vitro* microleakage of a new dental adhesive. *J Prosthet Dent*, 55:442-445, April, 1986.
17. Fuks, A.B.; Hirschfeld, Z.; Grajower, R.: Marginal leakage of cervical resin restorations with a bonding agent. *J Prosthet Dent*, 54: 654-657, May, 1985.
18. Ben Amar, A.; Liberman, R.; Gordon, M. *et al*: Comparison of the effect of a new bonding agent (Scotchbond) and the conventional bonding agent on marginal sealing in composite resin restorations. *Dent Med J Isr Dent Assoc*, 2:14-17, January, 1984.
19. Herrin, H.K. and Berry, E.A.: Variables affecting the microgap of the enamel composite interface. *J Dent Res*, 65: Abstract 777, March, 1986.

CURRENT STATUS OF POSTERIOR COMPOSITE MATERIALS

Although patients universally demonstrate tremendous enthusiasm relative to the new "tooth-colored" posterior restorative materials, their routine use at the present time must be regarded as experimental. Although 3-year recall results (Wilder et al, 1983) relative to wear resistance appear promising, particularly regarding light-cured composite materials, an acceptable substitute for silver amalgam in the posterior region has not yet been found (ADA Status Report, 1983). Until longer-term clinical observations relative to the durability of composite materials in the posterior region are available, posterior composite materials should only be planned under the following circumstances:

1. Where esthetic requirements are primarily essential, i.e., in maxillary and mandibular canines and premolars.
2. In situations in which the buccolingual width of the cavity preparation can be restricted.
3. When the patient is fully informed as to the experimental nature of the posterior materials.
4. When the dentist is prepared to minimize the technique-sensitivity of the materials by means of a meticulously controlled clinical procedure involving (a) conservative cavity design, (b) "prewedging" and customized matricing techniques, (c) controlled pulp protection, and (d) a controlled insertion technique which minimizes the need for extensive finishing procedures.

Jordan, R.E. et al: *Esthetic Composite Bonding*. St. Louis: The C.V. Mosby Company, 1986, p. 230.

The invasive pit-and-fissure sealing technique in pediatric dentistry: an SEM study of a preventive restoration

G.P. De Craene, DDS
C. Martens, DDS, PhD
R. Dermaut, DDS, PhD

Along with good hygiene, optimal fluoridation, dietary counseling and regular six-month recalls, pit-and-fissure sealants have been recommended for longer than fifteen years. Their ease of application and their clinical success have been demonstrated by several authors.¹⁻¹⁰ Although the prevalence of dental caries has decreased, Nikoforuk reminds us that the pits and fissures found on many occlusal surfaces make those surfaces vulnerable sites for caries attacks.¹¹⁻¹⁴ Consequently, the application of sealants remains an important entity of clinical pediatric dentistry. The choice between the non-invasive and the invasive techniques remains, however, a matter of debate.¹⁵ Keeping in mind that the clinician desires to conserve the maximum amount of sound tooth structure, questionably carious fissures cause problems. The selection and application, therefore, of other preventive restorations (avoiding amalgam) become important.

Also, in the present SEM study, various types of burs are compared, to learn which of them will permit optimal preparation of fissures. In addition, several choices of preventive restorations are shown in the Table.

Dr. G.P. De Craene and Dr. C. Martens are Assistant Professors, Department of Pedodontics and Dr. R. Dermaut is Chairman, Department of Orthodontics and Pedodontics at the State University of Ghent, De Pintelaan 185, B-9000 Gent, Belgium.

MATERIALS AND METHODS

In this study, the study sample consisted of permanent human maxillary premolars extracted for orthodontic, prosthetic, or periodontal reasons. They were obtained from the university clinic or from private practitioners. A group of specimens with open fissures, apparently noncarious; and another group with deep, narrow, discolored fissures, apparently carious, were stored in a 10 percent formalin solution. They were then embedded in a synthetic resin cube of self-curing acrylic resin.

Longitudinal sections across the fissures were cut, using a water-cooled diamond saw, rotated at 3000 rpm. Preceding the sample preparation for the scanning electron microscope (SEM), a transverse section was made, separating the crown from the root.

For the fissure preparations six types of burs were used.* Four were diamond burs, used at high speed with water-cooling (type 1-4); and two of them were small round steel burs, at low speed (type 5-6). A first series of SEM photographs was made of the six different bur types (see Figures 1A-6A). With each type of bur, the fissures of five teeth were prepared. All these were examined in the SEM. The specimens were first spray-

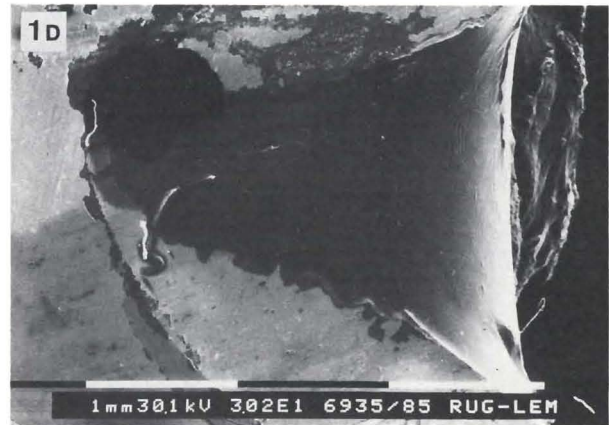
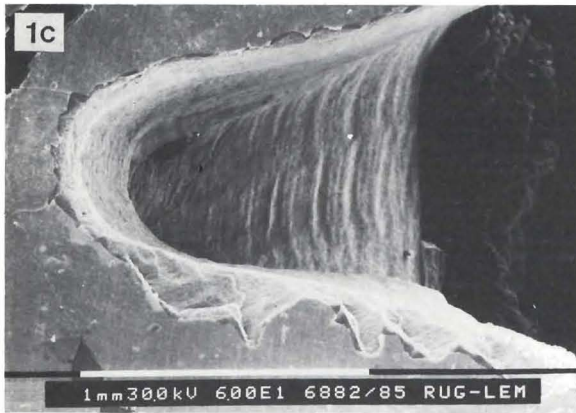
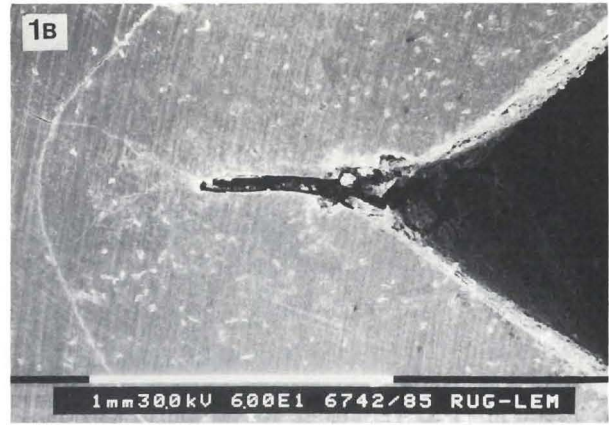
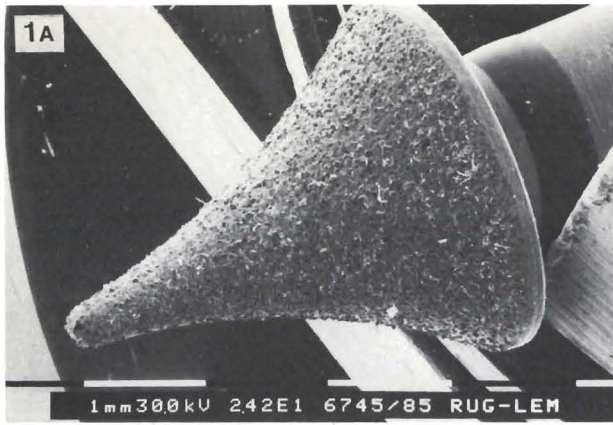


Figure 1A. Bur type nr. 806-314-466514-031 (Komet, SEM x 24,2) 1B. The fissure before preparation (SEM x 60). 1C. The fissure after preparation (SEM x 60). 1D. Sealed fissure (Helioseal, Vivadent, SEM x 30,2).

Table □ Procedures used in the treatment of occlusal fissures.

No caries, "open" fissures not suspected of caries	Deep, narrow, discolored fissures, suspected of caries	Beginning, shallow local caries in the fissures		Fissures with obvious caries	
Prophylactic treatment (limited to enamel)	Prophylactic treatment (limited to enamel)	Minimal restoration (in dentin)		Restorative treatment (in dentin)	
PFS	PFSI	PPRR	PGIR	PRR	AA
Isolation Etching Rinsing Reisolation Drying Applying sealant Polymerization Control	Isolation Preparation Reisolation Etching Rinsing Reisolation Drying Applying sealant Polymerization Control	(Anesthesia) Isolation Preparation Reisolation Dentin treatment Ca(OH) ₂ Etching Rinsing Reisolation Drying Applying bonding agent + posterior composite Polymerization (Etching) Applying sealant Control	(Anesthesia) Isolation Preparation Reisolation Dentin treatment Ca(OH) ₂ GIC Etching Rinsing Reisolation Drying Applying sealant Control	Anesthesia Isolation Preparation Reisolation Dentin treatment Ca(OH) ₂ +/- or cement Etching Rinsing Reisolation Drying Applying bonding agent + posterior composite Polymerization Control	Anesthesia Isolation Preparation Reisolation Dentin treatment Ca(OH) ₂ +/- or cement AA Control

Abbreviations:
PFS = Pit and fissure sealing, noninvasive technique.
PFSI = Pit and fissure sealing, invasive technique.
PPRR = Preventive posterior resin restoration
PGIR = Preventive glass ionomer restoration.
GIC = Glass ionomer cement.
PRR = Posterior resin restoration.
AA = Amalgam restoration.

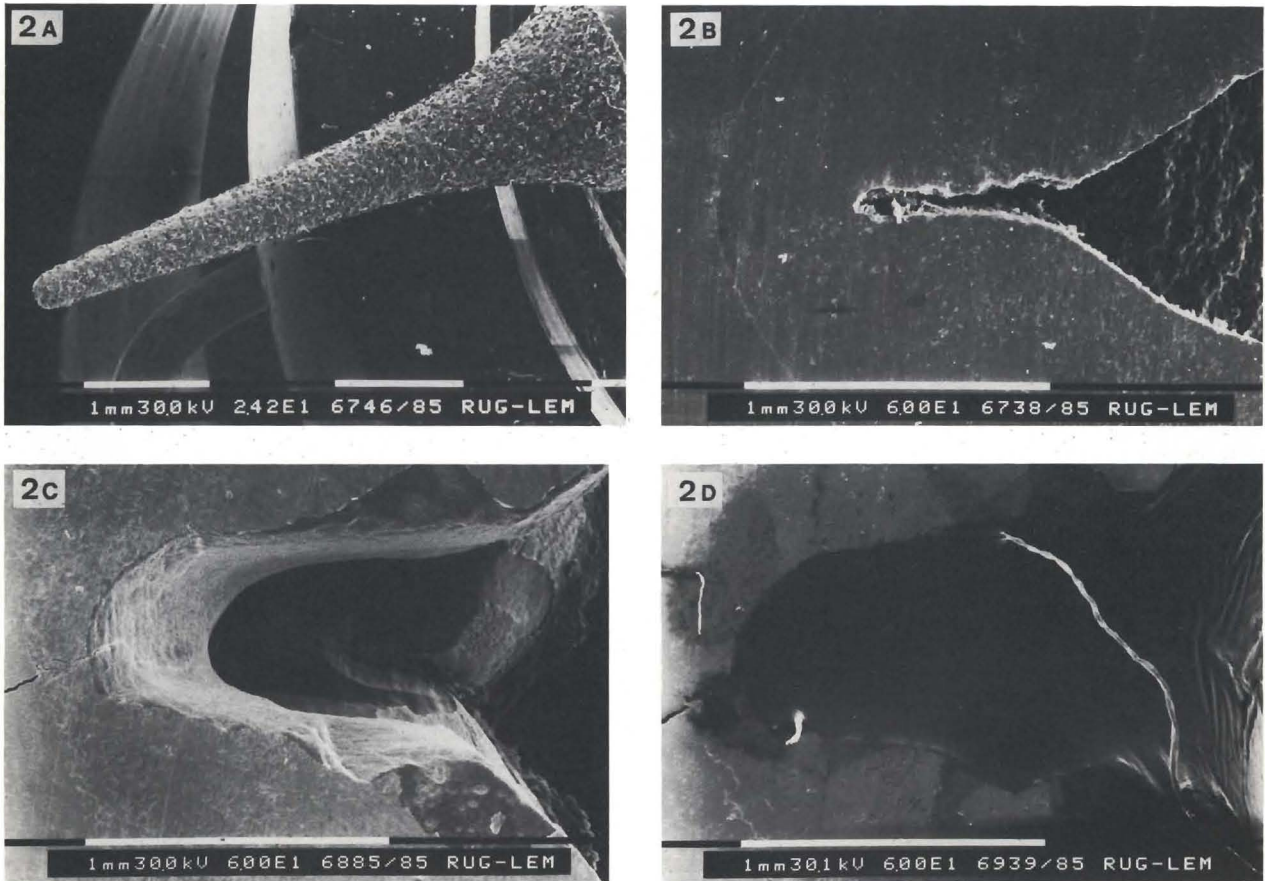


Figure 2A. Bur type nr. 806-314-465514-016 (Komet, SEM x 24, 2). 2B. The fissure before preparation (SEM x 60). 2C. The fissure after preparation (SEM x 60). 2D. Sealed fissure (Helioseal, Vivadent, SEM x 60).

washed, air-dried and gold-coated by an ionspatter. The SEM photographs were made with a SEM, Philips 505, 30 KV, magnification factor 60, for all the samples. The most representative specimen was selected and photomicrographs were made. Figures 1B-6B illustrate the nontreated fissures before the preparation with the burs.

Another series of photomicrographs was made after preparation of all fissures with the respective types of burs. After etching the specimens for 60 seconds with 37 percent phosphoric acid, rinsing, and air-drying for a similar period, the fissures were sealed with a white colored, visible-light-cured sealant.* A final series of photographs was made of the sealed fissures.

RESULTS

Figures 1C and 2C show that during fissure preparation, using bur types 1 and 2, a minimal loss of tooth structure occurred. In both cases, the preparation was limited to the enamel. It seems easy to manipulate these burs clinically, which permits a good and accurate preparation. It should be emphasized, however, that bur type n⁰ 2 is recommended for deeper fissures.

The use of bur type n⁰3 (Figure 3C) also results in a limited loss of tooth structure. This type of bur, however, cuts extremely fast, and its use should be limited to fully and adequately trained clinicians. The clinical application of type n⁰4 seems very satisfying, but Figure 4C shows the greatest loss of enamel, causing an obvious weakening of the tooth.

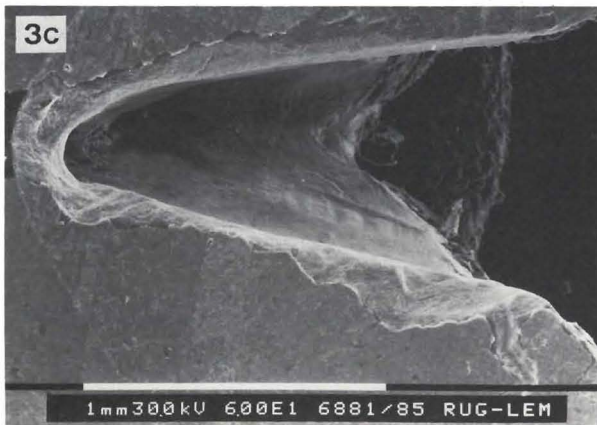
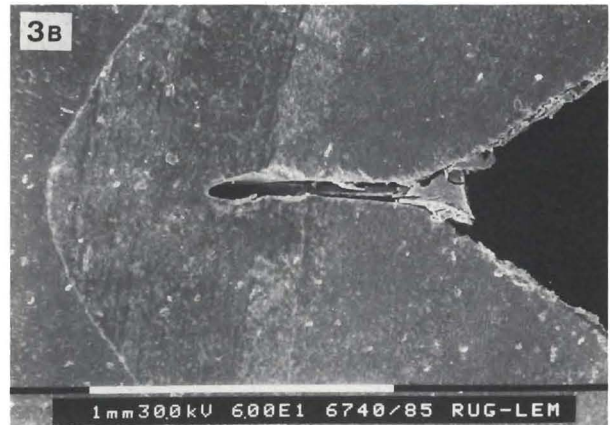


Figure 3A. Bur type nr. 806-314-540514-009 (Komet, SEM x 27, 6). 3B. The fissure before preparation (SEM x 60). 3C. The fissure after preparation (SEM x 60). 3D. Sealed fissure (Helioseal, Vivadent, SEM x 60).

Finally, an evaluation of the fissures prepared with the two round steel burs also reveals a marked loss of tooth substance (Figure 5C and 6C). Moreover, from a practical point of view, it seems more difficult to penetrate the enamel with these burs.

DISCUSSION

Several authors have reported that when decay is sealed, a significant reduction of viable microorganisms occurs.¹⁶⁻¹⁹ Others proved that the acid-etching procedure itself causes an immediate 75 percent reduction in the bacterial count.²⁰⁻²¹ Nevertheless, clinical and radiographical findings suggest that sealants placed over carious enamel and dentin decrease the penetration of caries, compared with untreated controls.²²⁻²⁴ The retention rate of a sealant applied in carious occlusal fis-

tures was reported to be higher than in clinically sound fissures.²⁵

In spite of these results, sealing of carious fissures cannot be considered acceptable clinical practice.

The most important advantage of the invasive technique is the ability to diagnose the extent of the carious lesion. Furthermore, it was suggested in some studies that higher retention rates for sealants were obtained following mechanical preparation of the fissure area.²⁶⁻³¹ The risk of microleakage was also reduced when the fissure was enlarged.

According to Tadokoro, the sealant easily penetrates the enlarged artificial fissure and adheres to the walls resulting in a better retention. Consequently, there is no need to cover a wide area outside the fissures for adequate retention. Another possible explanation for better retention following mechanical preparation was

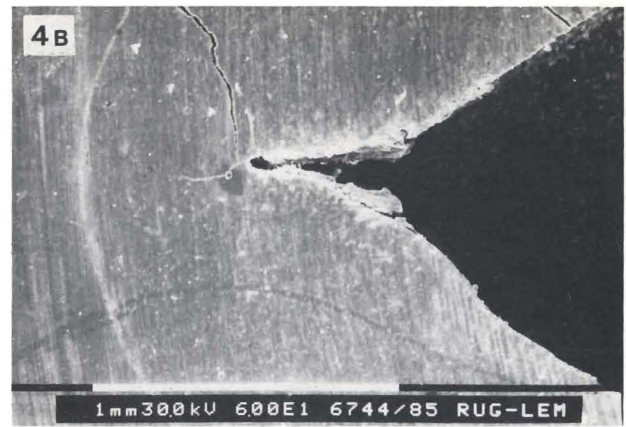


Figure 4A. Bur type nr. 806-314-257514-016 (Komet, SEM x 27,6). 4B. The fissure before preparation (SEM x 60). 4C. The fissure after preparation (SEM x 60). 4D. Sealed fissure (Helioseal, Vivadent, SEM x 60).

given by Shapiro.²⁷ He mentioned that this procedure widens and deepens the fissure by eliminating organic material and plaque and a very thin layer of enamel, resulting in a thicker layer of sealant with better retention.

According to Le Bell, good results may be explained by the fact that the fissures were opened up before sealing, which allowed a plug of resin to be formed, instead of a thin layer of varying thickness.³⁰ This plug would adhere better to the etched surface. In addition, Conniff and Hamey, in their study on primary teeth, suggested that the retentive strength of the acid-etch bonding system was increased after partial or complete removal of the outermost prismless surface layer.³³ This hypothesis was described earlier by Gwinnett, who found that the presence of this prismless layer on the enamel surface would create a reduction in mechanical retention.³⁴ According to this author, this might be due to a difference in topography between the prismless and prismatic enamel. Consequently, the removal of this layer by applying the invasive technique could be a solution to the questionable retention of sealants in primary teeth.^{35,36} Moreover, Shiota and Ripa reported

that the bases of occlusal grooves or fissures in molars usually have a prismless enamel layer that should be removed.^{37,38} In contrast to these findings, Horsted stated that this layer has a minimal effect on the retention of the sealant.³⁹

The clinical application of the burs offering the best results in this study, is presented in Figure 7A-D. Sometimes, type nr.5 and 6 are used to finish the preparation, after the superficial discolored enamel is taken away. These two burs were used in some clinical studies to enlarge the fissures.²⁷⁻²⁹

Finally, it should be noticed that some authors recommended a flame type, pointed diamond (0.8 mm), using ultrahigh speed to open up the fissure and to remove surface enamel or enamel that appears carious; but they did not specify a type of bur.^{29,30}

In an *in vitro* study, Tadokoro investigated a vibratory etching technique in which occlusal pits and fissures are simultaneously cleaned and acid-etched, using a fissure needle mounted on an electromagnetic vibrator.³² According to this author, a clinical study on children is now in progress, to examine the efficacy of this vibratory etching technique.

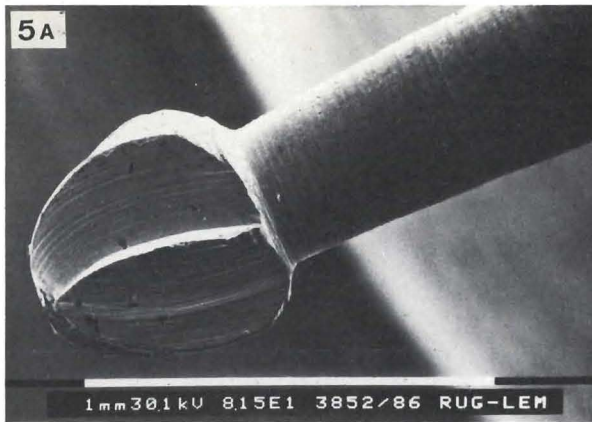


Figure 5A. Bur type nr. 500-204-001001-006 (Komet, SEM x 81,5). 5B. The fissure before preparation (SEM x 60). 5C. The fissure after preparation (SEM x 60). 5D. Sealed fissure (Helioseal, Vivadent, SEM x 60).

So far, the invasive pit-and-fissure sealing technique was discussed. In fact this is only one of the preventive restorations in pedodontics.

In our department, the procedures illustrated in the Table are used. Before examination of the occlusal surface with a sharp, short, straight probe, the tooth is cleaned with a dry brush and rinsed with water. Depending upon the diagnosis, one of the following methods of treatment is used.

- In a tooth with open fissures, not suspected of being carious, a prophylactic treatment will probably be chosen, usually the non-invasive pit-and-fissure sealing technique (PFS), without mechanical preparation of the fissures.
- In cases of deep and narrow fissures that are discolored and suspected of being carious, the invasive pit-and-fissure sealing technique (PFSI) should be chosen, which again can be considered as a prophylactic treatment. The purpose of the treatment is to clean the fissure entrance, to allow inspection of the lesion and to determine the degree of its extension toward the dentinoenamel junction (DEJ). It should be emphasized that it is not always necessary to clean deep fissures to the deepest

point, to assure that they are caries-free (Figure 8, A-C): fissure caries does not start at the base, but along the side walls of deep fissures.^{32,40}

- The third type of clinical situation is a tooth with beginning, shallow, caries localized in the fissures. In this case, a preventive posterior resin restoration (PPRR) is the restoration of choice. It is a minimal restoration (in dentin), as described by Simonsen and Houpt or a preventive glass ionomer restoration as described by Franklin Garcia-Godoy.⁴¹⁻⁴³ Using this technique, only the carious portion of the fissure is removed and the clinically sound fissure is protected against future caries by sealing.
- Finally, the traditional extension for prevention (EFP) technique is only indicated for carious fissures. This technique should be considered the point of no return.

An advantage of these procedures is the possibility of switching from one method to another. Furthermore, a combination of techniques is possible. The latter is opposed to the classical clinical procedure of "extension for prevention", which, however, always results in weakening the tooth.⁴⁴

Although a six-month recall is required, watchful

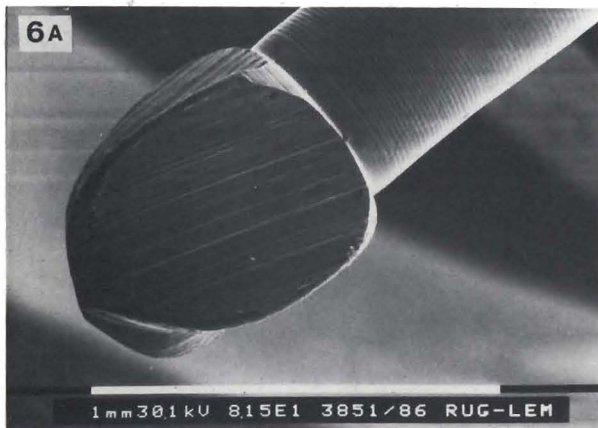


Figure 6A. Bur type nr. 500-204-001001-008 (Komet, SEM x 81, 5). 6B. The fissure before preparation (SEM x 60). 6C. The fissure after preparation (SEM x 60). 6D. Sealed fissure (Helioseal, Vivadent, SEM x 60).

waiting is an acceptable and often recommended approach.

CONCLUSION

According to the literature, it is suggested that higher retention rates for sealants are obtained following mechanical preparation of the fissure area and that the risk of microleakage is also reduced, when the fissure is enlarged.²⁶⁻³¹

The results of this study clearly show that by applying the invasive sealing technique (PFSI), the choice of an adequate bur is important to obtain a caries-free fissure in combination with a minimal loss of tooth substance.

Type nr. 1 and 2 were found to perform the best, in terms of the amount of tooth substance removed: only a small amount of enamel was removed, thus no weaken-

ing of the tooth occurred. Moreover, from a clinical point of view, they were also found to be the most efficient ones, because an ideal preparation for sealing could be accomplished (Figures 1C, 2C). These burs also facilitate an adequate preparation without penetration into the dentin. At this point in the procedure, the distinction between a superficial carious lesion and one extending to the DEJ can be readily made.

Traditional "extension for prevention techniques" as described above have predominated in the past and were an acceptable treatment of the invasion of dentin by bacteria. An accurate diagnosis and an accurate rendition of the described procedures, using newly developed materials, however, would provide clinicians with a truly conservative occlusal-fissure-management technique.

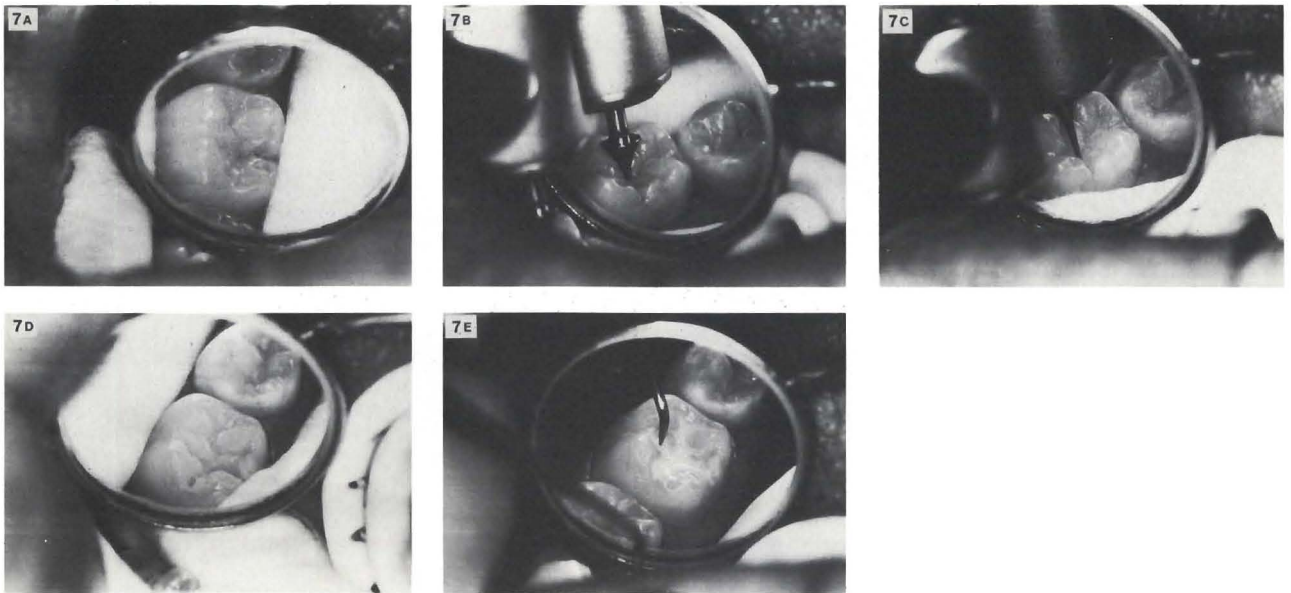


Figure 7A. The fissure before preparation. 7B. Preparation with Bur type nr. 1. 7C. Preparation with Bur type nr. 2. 7D. The fissure after preparation with Bur type nr. 1, 2. 7E. Sealed fissure (Helioseal, Vivadent).

REFERENCES

1. Simonsen, R.J.: Preventive aspects of clinical resin technology. *Dent Clin N Am*, 25:291-305, April, 1981.
2. Mertz-Fairhurst, E.J.; Fairhurst, C.W.; Williams, J.E.: A comparative clinical study of two pit and fissure sealants: 7 years results in Augusta, GA. *J Am Dent Assoc*, 109:252-255, August, 1984.
3. Ripa, L.W.: Occlusal sealants: rationale and review of clinical trials. *Clin Prevent Dent*, 45:3-9, September-October, 1982.
4. Houpt, M. and Shey, Z.: The effectiveness of a fissure sealant after six years. *Pediatr Dent*, 5:127-130, March-April, 1983.
5. Fuks, A.B.; Eidelman, E.; Biton, N.: A comparison of the retentive properties of two filled resins used as fissure sealants. *J Dent Child*, 49:127-130, March-April, 1982.
6. Rock, W.P.: Potential use of fissure sealants in the NHS. *Brit Dent J*, 157:445-448, December, 1984.
7. Johnsen, J.: Pit and fissure sealant use: an issue explored. *J Am Dent Assoc*, 108:310-322, March, 1984.
8. Leverett, D.H. and Handelman, S.L.: Use of sealants in the prevention and early treatment of carious lesions: cost analysis. *J Am Dent Assoc*, 106:310-322, March, 1983.
9. Bernstein, : Consensus development conference statement on dental sealants in the prevention of tooth decay. *J Am Dent Assoc*, 108:233-236, February, 1984.
10. DeCraene, L.G.; Dermaut, L.R.; Martens, L.C.: Pit- en fissuursealing: fictie of werkelijkheid? *Belg Tijdschr Tandheelk*, 41:38-46, Mei, 1986.
11. Glass, R.L.: Secular changes in caries prevalence in two Massachusetts towns. *J Dent Res*, 61(Sp. Iss.):1352-1355, 1982.
12. Brunelle, J.A. and Carlos, J.P.: Changes in the prevalence of dental caries in V.S. schoolchildren, 1961-1980. *J Dent Res*, 61(Sp. Iss.): 1346-1351, 1982.
13. Kalsbeek, H.: Evidence of decrease in prevalence of dental caries in the Netherlands: an evaluation of epidemiological caries surveys on 4-6 and 11-15 year old children, performed between 1965 and 1980. *J Dent Res*, 61(Sp. Iss.):1321-1326, 1982.
14. Nikoforuk, G.: *Understanding dental caries*. I. Etiology and mechanisms. Basel: S. Karger, 1985.
15. Meiers, J.C. and Jensen, M.E.: management of the questionable carious fissure: invasive vs non-invasive techniques. *J Am Dent Assoc*, 108:64-68, January, 1984.
16. Handelman, S.L.; Washburn, F.; Wopperer, P.: Two-year report of sealant effect on bacteria in dental caries. *J Am Dent Assoc*, 93:967- 970, November, 1976.
17. Jeronimus, D.J.; Till, M.J.; Sveen, O.B.: Reduced viability of micrororganisms under dental sealants. *J Dent Child*, 42:275-279, July- August, 1975.
18. Going, R.E.; Loesche, W.; Syed, S.: The viability of microorganisms in carious lesions five years after covering with a fissure sealant. *J Am Dent Assoc*, 97:455-462, September, 1978.
19. Meiers, J.C.; Wirthlin, M.R.; Shklair, I.L.: A microbiological analysis of human early carious and non-carious fissures. *J Dent Res*, 61:460-464, March, 1982.
20. Theilade, E.: Effect of fissure sealing on the microflora in occlusal fissures of human teeth. *Arch Oral Biol*, 22:251-259, 1977.
21. Jensen, O.E. and Handelman, S.L.: Effect of an autopolymerizing sealant on viability of microflora in occlusal dental caries. *Scand J Dent Res*, 88:382-388, October, 1980.
22. Handelman, S.L.; Leverett, D.H.; Solomon, E.S.: Radiographic evaluation on the sealing of occlusal caries. *Community Dent Oral Epidemiol*, 9:256-259, December, 1981.
23. Mertz-Fairhurst, E.J.: Clinical progress of sealed and unsealed caries. Part I: Depth changes and bacterial counts. *J Prosthet Dent*, 42:521-526, November, 1979.
24. Mertz-Fairhurst, E.J.: Arresting caries by sealants: results of a clinical study. *J Am Dent Assoc*, 112:194-197, February, 1986.
25. Handelman, S.L.: *Questions about sealants in viewpoint on preventive dentistry*. The role of pit and fissure sealants. New Jersey: Medical Education Dynamics, 1978, p 19.
26. Shapira, J. and Eidelman, E.: The influence of mechanical preparation of enamel prior to etching on the retention of sealants. *J Pedod*, 6:283-287, Summer, 1982.



Figure 8A. Discolored fissure before preparation (SEM x 60).
8B. The fissure after preparation (SEM x 60). Note that the preparation is not extending to the deepest point of the fissure.
8C. Sealed fissure (Helioseal, Vivadent, SEM x 60).

27. Shapira, J. and Eidelman, E.: The influence of mechanical preparation of enamel prior to etching on the retention of sealants: 3-year follow-up. *J Pedod*, 8:272-277, Spring, 1984.
28. Shapira, J. and Eidelman, E.: Fissure topography after combined 20- and 60-seconds etching and mechanical preparation viewed by SEM. *Clin Prev Dent*, 7:27-30, July-August, 1985.
29. Shapira, J. and Eidelman, E.: Six-year clinical evaluation of fissure sealants placed after mechanical preparation: a matched pair study. *Pediatr Dent*, 8:204-205, September, 1986.
30. LeBell, Y. and Forsten, L.: Sealing of preventively enlarged fissures. *Acta Odontol Scand*, 38:101-104, July, 1980.
31. Raadal, M.: Microleakage around preventive composite fillings in loaded teeth. *Scand J Dent Res*, 87:390-394, May, 1979.
32. Tadokoro, Y. and Fusayama, T.: A laboratory report on vibration etching for fissure sealants. *J Dent Res*, 61:780-784, June, 1982.
33. Conniff, J.N. and Ray Hamby, G.: Preparation of primary tooth enamel for acid conditioning. *J Dent Child*, 177-179, June, 1976.
34. Gwinnett, A.J.: Human prismless enamel and its influence on sealant penetration. *Arch Oral Biol*, 18:441-444, March, 1973.
35. Ripa, L.W.: Sealant retention on primary teeth: a critique of clinical and laboratory studies. *J Redodont*, 275-290, Summer, 1979.

36. Simonsen, R.J.: Fissure sealants in primary molars: retention of colored sealants with variable etch times, at twelve months., *J Dent Child*, 46:382-384, September-October, 1979.
37. Shiota, K.; Yaoi, H.; Yamauchi, T.: Submicroscopic structures and histogenesis of "Rodless Enamel". *Jap J Oral Biol*, 11:41-48, 1963.
38. Ripa, L.W. and Gwinnett, A.J. and Buonocore, M.S.: The "prismless" outer layer of deciduous and permanent enamel. *Arch Oral Biol*, 11:41-48, 1956.
39. Horsted, M.; Fejerskov, O.; Larsen, M.J.: The structure of surface enamel with special reference to occlusal surfaces of primary and permanent teeth. *Caries Res*, 10:287-296, 1979.
40. Silverstone, L.M.: *Dental caries: Aetiology, pathology and prevention*. London: The McMillan Press Ltd., 1981, p 135.
41. Houpt, M.; Eidelman, E.; Shey, Z.: Occlusal restoration using fissure sealants instead of "extension for prevention". *J Dent Child*, 270-273, July-August, 1984.
42. Simonsen, R.J.: Preventive resin restorations: innovative use of sealants in restorative dentistry. *Clin Prev Dent*, 4:27-29, July-August, 1982.
43. Garcia-Godoy, F.: The preventive glass ionomer restoration. *Quintess Intern* 17/10:617-619, October, 1986.
44. Larson, T.D.; Douglas, W.H.; Geistfeld, R.E.: Effect of prepared cavities on the strength of teeth. *Oper Dent*, 6:2-5, 1981.

This study was supported by KOMET, Gebr. Brasseler (Lemgo, Germany) and by VIVADENT (Schaan, Liechtenstein).

The authors wish to thank Mrs. B. Vandevoorde-Jouret for typing the manuscript, Mr. G. Dermout for the photography and Mr. W. Bohijn from the Laboratory for Electron Microscopy - State University of Ghent, for his help in using the scanning electron microscope.

An alternative approach to prevention: computer-assisted patient education

Patient education

Peter J. Fos, DDS, MPH

Patient attitudes concerning health care have changed significantly during the last several years. The patient of the 1980s is more concerned with what is necessary to ensure good health. As a result, the opportunity to educate today's patient about maintaining good dental and overall health has improved noticeably. The key to decreasing the incidence of dental caries, for example, is prevention; which can succeed, however, only if the dentist shares his knowledge about prevention with his patients and the public.¹

The purpose of this paper is to describe a computer-assisted instruction (CAI) program to educate patients about their dental health. The program was designed as an interactive, realistic learning experience. Over the past several years, computers have gained popularity in use as conversation devices. With increasing availability and decreasing cost of the equipment, the microcomputer is becoming a useful and powerful educational tool. Today, computers are used extensively in primary, secondary, and college education, but their use lags in health education.²

Despite the concern that computer-assisted instruction may prove ineffective for lack of patient acceptance, recent findings show that the majority of those surveyed

Dr. Fos is Assistant Professor of Health Systems Management at Tulane University Medical Center, School of Public Health and Tropical Medicine. Dr. Fos also serves as the director of Executive Education Programs in Health Systems Management at Tulane University Medical Center, 1430 Tulane Avenue, New Orleans, LA 70112.

indicated that computer interaction was a positive experience.³ According to one study, 88 percent of those tested felt that a computer interview was as easy as an interview with a clinician.⁴ Another study showed that 82 percent of the patients polled were favorably inclined to computer interviews. The study found that patients' interactions with a computer were easy and provided positive reinforcements, to the extent that they preferred the computer to human interviewers.⁵

CURRENT EDUCATIONAL METHODOLOGY

Several methods of educating patients are in use today: pamphlets, brochures, and letters have proven to be very successful; one-on-one educational sessions; and lectures at schools and community organizations are a few of the most commonly used methods. Audiovisual techniques, especially videocassettes, have gained recent popularity. All of these methods have a common, but often difficult-to-reach goal: to get the information to the greatest number of people with the least expenditure of time by the dental staff. Unfortunately, the average practicing dentist does not have the necessary time to educate patients properly.

Since a large majority of people do not seek dental services regularly, the computer looms as a viable educational tool. Approximately 12 percent of the U.S. population already have home computers (25 million), a trend strongly supported by the continually decreasing cost of equipment. In the near future a personal computer will become a household necessity.

COMPUTER-ASSISTED INSTRUCTION (CAI)

Computers in the dental office are gaining similar popularity. Most computerized dental practices, however, do not fully utilize the capabilities of their computers, particularly as valuable instructional tools.⁶

As a component of the patient education center, the computer becomes an adjunct to videocassettes and other traditional methods. As the technology advances, the computer can become a feature of the reception area and a source of information.

CAI APPLICATION: A PROGRAM FOR DENTAL EDUCATION

An interactive CAI program has been developed to present instruction in dental health. The program is written in the popular programming language known as *PILOT* (Programmed Inquiry, Learning Or Teaching), a "dialect" of *PASCAL*, a modification for use with micro-

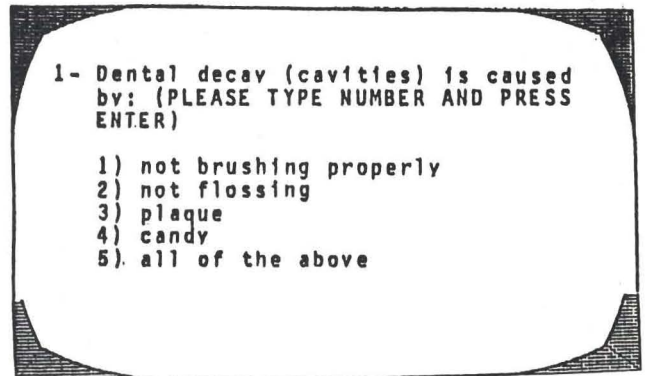


Figure 1. Monitor screen: question.

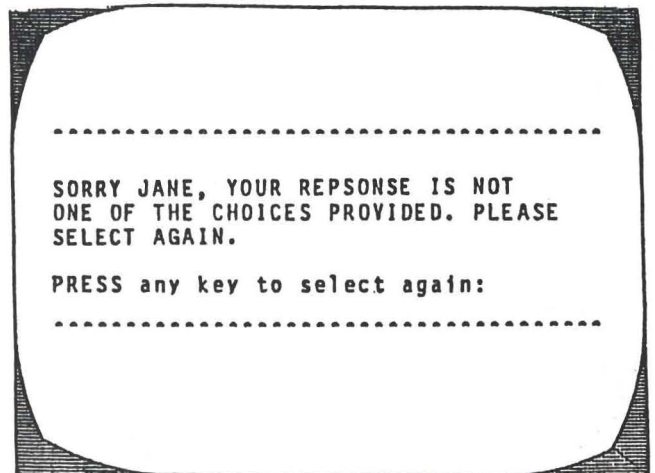


Figure 2. Monitor screen: unrecognized response.

computers and CAI.

To develop a CAI program that is an effective educational tool, several criteria must be met. The program must be easily accessible to the user, who must be able to interact easily with the computer, and not become frustrated because of the complexity of the program. "User-friendliness", with ample instructions throughout the program, is essential. And lastly, the user should have increased knowledge, when the program is completed.

What is meant by the concept "user-friendly"? This slogan is much abused today in reference to computer usage. Which features of a program that make it "friendly" are not very clear. The following examples are features that should be included in a CAI program to ensure its effectiveness:

- Menu-driven*: beginning users of computers can be greatly helped by step-by-step instructions which can be afforded by using menus.

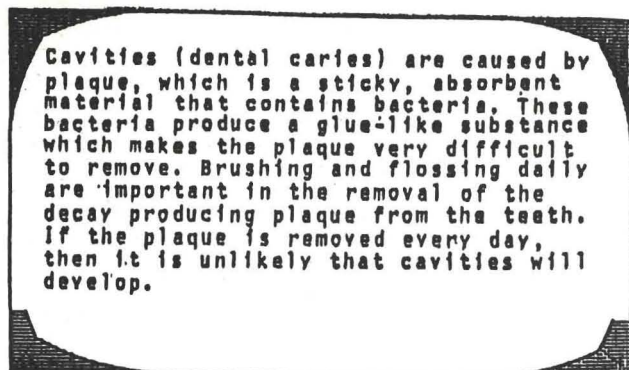


Figure 3. Monitor screen: feedback for correct response.

- User-modified syntax*: a program must not be written in a vocabulary contrary to what is expected.
- Control over the sequence of questions*: the user must feel a sense of control, and have the ability to skip around through the questions and be able to decide which questions are to be asked next.
- Anticipate errors*: an effective CAI program must have the ability to anticipate user errors, with error messages that demonstrate how to avoid problems incorporated into the program.
- Compatibility with users' cognitive style*: the cognitive style of the user must be taken into account by designing screen presentations appropriate to the information processing skills of proposed users.
- English*: an effective program must be written in proper English, following the rules of grammar.

The CAI program was designed to meet several instructional objectives:

- To evaluate the user's current knowledge of proper dental care.
- To educate the user as to what he should know about proper dental health.
- To provide a better understanding of proper dental health and care.

The program's content consists of six sections: preventive dentistry, endodontics, restorative dentistry, periodontics, pediatric dentistry, and nutrition. The user has the option to proceed through each section in order or skip around to specific sections. The computer asks the users questions concerning current knowledge of dental health (Figure 1). The users are prompted to type the number of their selection. If the incorrect response is entered, the computer displays a very friendly response indicating a mistake (Figure 2). After displaying this error on the screen, the program returns to the same question. If the user now types the number of the correct selection, the computer displays positive, encouraging feedback (Figure 3).

It is important to note that the computer not only acknowledges a correct response, but displays screens of positive educational feedback. Also note that in response to incorrect selections the computer displays a personalized, friendly correction.

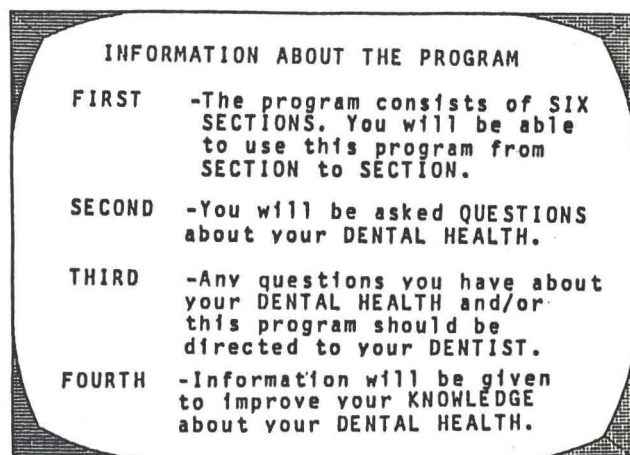


Figure 4. Monitor screen describing keyboard.

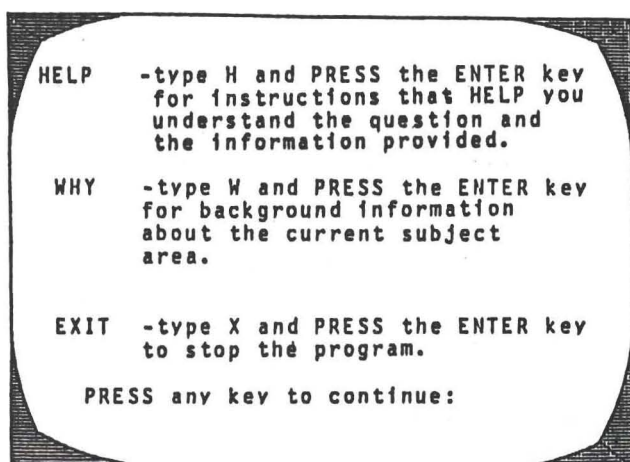


Figure 5. Monitor screen describing exit, help, why.

To ensure ease in use of the program, specific instructions are given to the user in the introduction. The user's previous computer experience (or lack of previous knowledge) is a concern, so keyboard information is displayed (Figure 4). The intent of this information is to facilitate smooth execution of the program. As previously mentioned, the program may be used in order (section one to section six) or the user may skip around (these options are given at the beginning and at the end of each section). The user may proceed at his own pace, and any section may be repeated as often as the user desires. At any time during the execution of the program, the user may stop (exit the program), ask "why", or seek "help" (Figure 5). These options are useful to allow the user to understand the purpose and objectives of the program.

If an unrecognized response is entered, the computer displays a screen with a very cordial statement (Figure 2). The goal is for the user to be at ease, to be comfortable, and not intimidated during the program session.

DISCUSSION

As an educational tool, CAI has several potentially useful applications. To fit in with traditional dental health education, the computer can become a component of an in-office, patient-education center. In addition to written information and videotapes, the computer may be very helpful during the patient's educational visit to the dental office. The program may be utilized as an initial exposure to the proper care and treatment, and it has unlimited usefulness as a means of reinforcing information. The program has the flexibility of allowing the patients to increase their knowledge of a specific topic of dental care.

With the rapid and continual decrease in cost, the numbers of computers in homes have greatly increased in the last five to ten years. If the trend continues at the present rate, computers will be as commonplace in the home as videocassette players. This creates the potential of the development of a patient loan system. Patients with home computers can borrow educational programs from the dental office for short periods of time, similar to a library loan-program. After review of the program the patients may direct any questions or comments to the doctor or the dental support staff.

Unquestionably, the most useful application for CAI programs is as a teaching aid in schools. The majority of secondary schools (and increasingly in elementary schools) provide computer instructions to their students. The program's effectiveness as an educational tool is two-fold: a) an exercise in the use of the computer and computer software; and b) a health-care educational exercise. School-based health fairs, health-education courses, and library-centered, self-instruction courses are just a few of the many potential educational uses of CAI.

The program discussed in this paper is an example of how the dental health-care profession and the health-care profession as a whole can begin to utilize computer technology to improve an individual's overall health.

With the increasing pressures of time and the need for increased patient-contact-hours, the use of CAI is a useful tool to provide health-care education. The cost of the necessary computer hardware and software for CAI presentation is more than off-set by the savings in doctor and staff time. Moreover, the profession by definition has the responsibility to educate the public about proper dental care.

CAI programs may be generic (as with the program described here) or the programs may focus on specialized topics. CAI can be a useful vehicle for all dental care providers and serve a dual purpose: patient education, and as a replacement for dental staff members who are needed for clinical duties.

SUMMARY

Computer-assisted instruction is a reliable, viable, and increasingly popular method for patient education. CAI programs are easy to use, nonintimidating to the patient, and enjoyable. CAI is a relatively new, but effective method of "spreading the word" to those who most desperately need the information.

As technology advances, more computer-based patient education programs will become available to the dental health-care profession. The computer based education center will become a useful and integral component of the dental office of the future.

REFERENCES

1. Technical Report: The National Preventive Dentistry Demonstration Program (NPDDP). *Am J Public Health*, 76:245-251, 1986.
2. Erdman, H.P.; Klein, M.H.; Griest, J.H.: Direct patient computer interviewing. *J Consult Clin Psychol*, 53:760-773, 1985.
3. Angle, H.V.; Ellingwood, E.H.; Hay, W.H. *et al*: Computer-aided interviewing in comprehensive behavioral assessment. *Behav Ther*, 8: 747-754, 1977.
4. Carr, A.C.; Ghosh, A.; Ancill, R.J.: Can a computer take a psychiatric history? *Psychol Med*, 13:151-158, 1983.
5. Lucas, R.W.: A study of patients' attitudes to computer interrogation. *Internat J Man-Machine Studies*, 9:69-86, 1977.
6. Richards, J.G.: Computer-assisted instruction and the use of PILOT. *J Fam Med*, 19:255-257, 1984.

Hygiene in dental practice— Part I: Potential pathogens and possibilities of contamination

Infection control

William E. van Amerongen, DDS, PhD
Johannes de Graaff, PhD

The purpose of general hygiene in dental practice is to prevent dissemination of microorganisms. Preventive efforts are focused on potentially pathogenic microorganisms.

The patient's oral fluid, oral cavity and mucous membranes are possible sources of such microorganisms; furthermore, the oral fluids, oral cavities and mucous membranes of the dentist and assisting personnel are potential sources of contamination.

The aerosols formed by the use of rotating instruments and multipurpose syringes disperse vast numbers of microorganisms. In principle, then, the entire operating area, including the personnel in it, should be regarded as contaminated. To prevent further contamination, various preventive measures of general hygiene should be taken; i.e. measures aimed at optimal limitation of the risk of contamination.

Before an adequate prevention program for dental practice can be designed, it is necessary to establish what microorganisms are in fact involved, and how these are transmitted. It is not the intention of this publication to discuss all conceivable pathogens of infectious diseases, because the human organism has an adequate defence system against many of them; furthermore, all of them do not pose a threat to public health. Moreover, hygienic measures effective against infections that pose a

Dr. van Amerongen, Department of Paediatric Dentistry and Dr. de Graaff, Professor in Oral Microbiology, are with ACTA, Vrije Universiteit, v.d. Boechorststraat 7, 1081 BT Amsterdam, The Netherlands.

threat to public health, will as a rule also be effective in preventing the infectious diseases not mentioned here.

In view of this, only the most relevant pathogens will be discussed, with reference to the way in which contamination can take place in dental practice. Measures to minimize the risk of contamination will be discussed in Part II.

POTENTIAL PATHOGENS

Tuberculosis

The lungs are the principal site of primary infections caused by *Mycobacterium tuberculosis*. These primary infections can be encapsulated and persist for years as dormant foci. These may be reactivated (e.g. by diabetes or corticosteroid therapy), whereupon the bacteria multiply and may spread to other parts of the body via expectorates. Tuberculosis thus is disseminated extrapulmonary in 15 percent of cases and involves the oral cavity in 3.5 percent of cases. Almost all oral tissues (except the teeth) may be infected with tuberculosis.¹

Tubercle bacilli can long survive outside the body. Dissemination of tuberculosis by aerosol formation (coughing) is widely known, and this is why a dentist treating a patient with open tuberculosis is running a risk of contamination. On the other hand, patients treated by a tuberculosis-positive dentist are also at risk.² The number of new cases of tuberculosis reported annually in The Netherlands is approximately 1600. In this country (The Netherlands) the disease has unmistakable epidemic characteristics.

Gonorrhoea

Infections with *Neisseria gonorrhoeae* are not confined to the genital mucosa, but may also encompass the mucous membranes of the oral cavity, the tonsils and the pharynx.³⁻¹¹ Conjunctival infections are found especially in neonates, via contamination by an infected birth canal; this leads to ophthalmia neonatorum. If left untreated, such infections often lead to blindness of the infant.^{12,13}

Approximately 15,000 cases of infection with *Neisseria gonorrhoeae* are now reported annually in The Netherlands. The bacterium is highly sensitive to hypothermia and dehydration. Yet, dental personnel may develop infections of the eyes, and nasal and oral mucosa via airborne transmission from patients with oral infections. The transmission takes place via exudate from infected mucosal lesions.

Syphilis

This four-stage infectious disease is caused by *Treponema pallidum*. The hard chancre of primary syphilis and the multiform skin eruptions of secondary syphilis are the major sources of contamination. Contamination is also possible via infected blood. Contamination via objects is less likely to occur, because the bacteria are highly sensitive to cooling and oxygenation, and cannot survive long in the atmosphere. The disease is worldwide; and, the highest morbidity is found in the fifteen- to thirty-year age-group (sexually most active). Approximately 1000 instances of infection are reported annually in The Netherlands (primary and secondary cases). Dentists and oral hygienists run an increased risk of contamination through contact with skin lesions and mucosal lesions in and around the oral cavity.^{14,15} It is often difficult to diagnose syphilis, the oral manifestations of which are frequently overlooked.¹⁶⁻¹⁸ Microscopic demonstrability of spirochaetes in smears from lesions does not clinch the diagnosis, because the oral cavity contains numerous spirochaetes, indistinguishable from *Treponema pallidum*. This is why serological studies are indispensable in these cases. The formation of aerosol may lead to infection of the dentist's own mucous membranes and eyes, but transmission of *Treponema pallidum* may also occur via microlesions on the hands of dentists and oral hygienists.¹⁴

Herpes simplex virus infections

Of the various herpes virus infections, infections with herpes simplex virus (HSV) is most common in man. In this group, two human types are distinguished: type 1 (HSV1) and type 2 (HSV2). HSV1 is generally (in 75 percent of cases) the pathogen causing orofacial infections, visceral infections (in the case of immune disorders), and encephalitis.

HSV2 causes genital infections in some 75 percent of cases and is often the pathogen of neonatal infections.¹⁹ HSV has been related to tumors of the cranial and cervical region.^{20,21} It is the HSV infections in the orofacial region that expose providers of dental care to an increased risk of infection.²²⁻²⁵ These orofacial HSV infections will, therefore, be discussed here.

Herpes virus infections are characterized by the fact that a primary focus persists in latent form in the trigeminal ganglion of the infected dermatomic area and constitutes a potential pathogen.²⁶ Although the primary infection may be accompanied by a whole range of symptoms of varying severity, e.g. gingivostomatitis,

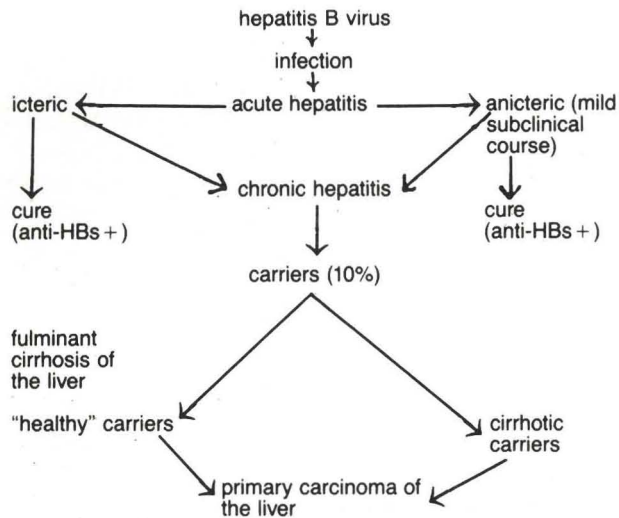


Figure. Schematic representation of possible courses of hepatitis B.

keratoconjunctivitis, acute herpes meningoencephalitis and primary cutaneous herpes (especially in medical and paramedical personnel), the infection usually takes a subclinical or asymptomatic course. Virtually everyone is sooner or later contaminated with the virus. Recurrent herpes develops after reactivation of latent HSV in the ganglion; migration of the virus along the axon to the dermatomic area in question; and multiplication of the virus in the skin, giving rise to vesicular lesions. Such reactivation of HSV may occur in response to endogenous or exogenous triggers. Endogenous triggers are usually stress-related and exogenous triggers may be fever, sunburn or traumatic injuries. Dental treatment, too, may reactivate latent HSV, e.g. through epithelial irritation or axonal damage entailed in an extraction.^{27,28}

Approximately 50 percent of persons in the USA are suffering from reactivated herpes simplex.¹ Most lesions develop on the lips, nose, chin or cheeks; but in principle, any skin area may be infected, in dentists very often the fingers.²²⁻²⁴ Immediately before eruption, the skin starts to burn and itch, whereupon erythema and vesicles develop. Contamination takes place through contact with the fluid from these vesicles, and also through contact with sputum and blood. HSV is relatively stable, especially in the presence of proteins (blood, sputum, etc.) and may survive hours or even days, at room temperature. The virus is heat-sensitive and acid-sensitive (pH < 6.8).

Due to its ubiquitousness and the many asymptomatic carriers, it is difficult to avoid contact with the virus. Dentists, oral hygienists, and auxiliary personnel, however, should take precautions and use protective aids.

Viral hepatitis

Four different types of virus have so far been identified: hepatitis A, hepatitis B, non-A non-B hepatitis, and hepatitis D (delta). Because hepatitis A has no special implications for dentistry, and because nothing is known about possible dental implications of non-A non-B hepatitis, only hepatitis B and hepatitis D will be discussed here. Infections with hepatitis B virus (HBV) may take any of several courses, as schematically represented in the Figure.

Man is the only HBV reservoir. In principle, anything that contains a HBV particle is infectious. This applies to blood and blood products, as well as to all bodily discharges: sputum, saliva, breast milk, tears, wound fluid, sperm, sweat, urine, feces, vaginal discharges, crevicular fluid, etc.²⁹⁻³⁴ Minute quantities may be sufficient for parenteral transmission of the infection (e.g. 0.1 µl blood).³⁵ There are several possible modes of transmission:

By percutaneous inoculation

- Via poorly sterilized needles or other instruments (used in acupuncture, tattooing, piercing of ears to accommodate earrings, mainlining by drug addicts).^{36,37}
- Via blood-drinking insects (especially in the tropics).³⁸
- Via microscopic skin lesions in dental practice (cuticle lesions, eczema, microtraumata caused by fast-moving airborne particles).³⁹⁻⁴¹

Nonpercutaneous infection via intact barriers

- Via the mucosa, contaminated material and saliva coming into contact with the mucous membranes of oral and nasal cavities, conjunctiva or genitals.³⁶
- Via sexual contact through infected sperm, mucosal contact, vaginal discharge, and blood (menstruation).⁴²

Indirect transmission is also possible. Since the virus is very stable outside the organism, the environment must be regarded as contaminated after treatment of a HBV-positive patient.⁴³ Due to the formation of aerosols, virus particles are dispersed in the room; and persons not operating at the chair are thus exposed to the virus (maintenance personnel, dental technicians, persons in adjacent treatment units, as in group-practices and main clinics).⁴⁴⁻⁴⁶ Transmission from a HBV-positive

dentist and oral hygienist to the patient is likewise possible, as several case reports have testified.^{47,48} In the USA and in the Federal Republic of Germany dentists have been sued and convicted in court, because they had not taken adequate precautions to protect their patients from contamination (see below).⁴⁹

Approximately 0.5 percent of the adult population in The Netherlands are HBsAg-positive (carriers), while 5-10 percent are anti-HBs-positive. The percentages in other countries differ widely, but very high carrier prevalences are reported from parts of Africa, the Far East, and South East Asia.^{38,50} High prevalences of HBV are also reported from some parts of Southern Europe. Kalyvar reported a 25-32 percent prevalence of HBV markers (HBsAg or anti-HBV antibodies) in the population of Athens.⁵¹ Dentists and oral hygienists are a group at risk in view of the fact that the prevalence of hepatitis B infections in dental personnel is three to ten times that in the general population.^{47,51} This is why routine basic prevention is urgently needed in the treatment of patients; it should aim at preventing transmission of this virus from patient to dentist and oral hygienist and vice versa. Patient-to-patient transmission should also be prevented so far as possible. Standardized application of preventive measures is required, in view of the presence of asymptomatic carriers. In this respect, it should be borne in mind that HBV is a heat-resistant virus, which is not killed until it has been heated to 95° C for five minutes.

Hepatitis D (delta)

The hepatitis D virus (HDV) is a RNA virus which replicates only in the presence of HBsAg. It is a so-called defect virus, in that it needs a helper virus (HBV) for the outside (HBsAg), because it lacks a protein cover.⁵²⁻⁵⁵ This is why infection takes place exclusively in the presence of HBV: either in combination with an acute HBV infection or as superinfection of chronic HBV patients. In both cases fulminant hepatitis develops with a very high mortality.^{56,57} HDV infections are mainly problems of i.v. drug users and hemophiliacs.^{58,59} The transmission of HDV is the same as that of HBV. HDV infections are endemic in southern Italy and have been demonstrated worldwide, since their discovery in 1977.^{52,60} In view of the dependence of HDV on HBV, the prevalence of HDV infections depends on that of HBV infections. Since dental care providers run an increased risk of developing HBV infections, it seems plausible that they are also at risk of HDV infections. HDV infections can be avoided, however, by vaccination against HBV.⁶¹

Acquired immunodeficiency syndrome

The acquired immunodeficiency syndrome (AIDS) is caused by the human retrovirus LAV/HTLV-III (lymphadenopathy-associated virus/human T-cell lymphotropic virus type III). Today the virus is called HIV (human immunodeficiency virus). Infection with this virus can give rise to a wide variety of symptoms, ranging from hardly specific manifestations to generalized lymph node swelling (LAS lymphadenopathy syndrome). After an incubation period of a few months to a few years (the maximum incubation period is still unknown), the patient may develop AIDS or ARC (AIDS-related complex), which is characterized by manifestations indicating attenuated or absent cellular defence.^{62,63} A wide variety of opportunistic infections may develop. Moreover, the lymphadenopathy may be accompanied by Kaposi's sarcoma.⁶⁴ According to current estimates some 23 percent to 26 percent of the seropositives are likely to develop AIDS or ARC. The assumption at this time is that seropositive persons are a source of infection for a period as yet unknown. Transmission of the infection may take place through sexual contact, blood transfusion, pregnancy, and via artificial insemination with infected semen; but the virus has also been found in saliva, tears, breast milk and cervical and vaginal discharge.^{63,65-68} In the Western world, homosexuals, i.v. drug users, and prostitutes are a group at risk.⁶⁹ In African countries, the disease is seen in men as well as in women, and in these countries there are indications of vertical infections in families.^{63,64}

The expectation is that in the Western world, too, AIDS will not remain confined to these groups at risk, but as is already the case in Africa, will develop more and more as a general sexually transmitted disease.⁶³ For prevention it is advised to avoid sharing toothbrushes, razors, etc. with asymptomatic infected persons.⁷⁰ Airborne transmission of the virus is improbable according to current opinion; nor has transmission been demonstrated by normal household contacts between nonsexual partners.⁷¹ Apart from transmission via parenteral routes (accidents with blood-contaminated syringes and instruments, contaminated blood in blood transfusions), transmission results from intimate, direct personal (mucosa/mucosa) contact. Only a very limited number of instances of infection with LAV/HTLV-III has so far been demonstrated in medical, nursing and laboratory personnel engaged in caring for AIDS patients.^{72,73} Dentists and oral hygienists are a group at risk due to intensive contacts with saliva and blood during the treatment of patients. This is why basic pre-

ventive measures should be routinely taken in patient treatment (see Part II).

The virus is thermolabile, and is killed by heating to 56° C for ten minutes; moreover, it is sensitive to a large number of disinfectants.⁷⁴

Legionnaires' disease

A form of pneumonia discovered fairly recently is caused by *Legionella pneumophila*. This is a new (rediscovered) bacterium which owes its name to the first description of an epidemic of this severe pneumonia during a reunion of Korea veterans in Philadelphia in 1976.

It is difficult to culture *Legionella pneumophila* in the laboratory (diagnosis) but the bacterium survives quite well in water. We now know that this water-loving bacterium can often be found in the pipelines of heating systems (hotels, hospitals, apartment buildings, etc.) and in humidifiers of air-conditioning systems.⁷⁵⁻⁷⁷ Transmission of *Legionella* infections takes place via aerosols formed from contaminated water.⁷⁸

Although *Legionella* have not to date been described in dental practices, the possibility of such an infection is to be taken into account. After all, there have been previous reports on infections caused by water-loving bacteria such as *Pseudomonas*, *Acinetobacter* and *Alcaligenes* contaminating water in multiple-function syringes and drill-cooling systems.^{79,80} There seems to be no conceivable reason why *Legionella* might not have to be added to these microorganisms in the future.

Research into the presence of *Legionella-specific* antibodies in the personnel of a dental department revealed that these persons had significantly higher antibody titres, and that a larger percentage of persons had antibodies than in a matched control group. These findings warrant the conclusion that *Legionella* must be present in this dental department and may constitute an additional risk to personnel and patients.⁸¹

Since these bacteria can multiply very quickly, water left stagnant at room temperature for a few hours may prove to contain vast numbers of microorganisms. The pipeline system of the unit should therefore be regularly washed out and the contents of water reservoirs should be refreshed.

POSSIBILITIES OF CONTAMINATION

In dental practice, contamination may take place as follows:

- Transmission from dentist and assistant to patient;
- Transmission from patient to dentist and assistant;
- Transmission from patient to patient.

In some cases there may be direct transmission of microorganisms, while in other cases contamination is caused by indirect transmission via aids or instruments used in dental treatment. Although direct transmission entails a graver risk of contamination than indirect transmission, this will not be emphasized here, because we proceed from the postulate that, in principle, any risk of contamination is to be avoided. The following presentation does not claim to indicate any order of importance, because contamination may occur by any one or any combination of the following avenues.

Hand instruments

Contamination may result from pricking or cutting with contaminated instruments, with blood as the principal vehicle. Transmission of hepatitis B virus, for instance, can take place in minute quantities of contaminated blood (0.1 µl).³⁵ Since many dental instruments are very sharp, the risk of contamination in this way in dental practice is very grave, at least theoretically.

Not only blood, however, but also oral fluids can serve as a vehicle. Both hepatitis B antigen and LAV/HTLV-III have been demonstrated in saliva.^{30,31,68} That the concentration of microorganisms is bound to increase in blood-tinged saliva is evident. The risk of contamination through hand instruments exposed to such saliva is, therefore, correspondingly higher.

Direct contamination from patient to dentist and assistant or indirect contamination from patient to patient, as described here, can also take place without lesions caused by instruments. There may be preexistent lesions (inflammations, traumata) in the mouth. Contamination is even possible through intact mucosal membranes.

Rotating instruments, multiple-function syringes and ultrasonic scalers

Transmission can take place via the aerosol reflected from the mouth via the multiple-function syringe, drill-cooling system, and ultrasonic scaler; this aerosol can contain traces of blood and saliva as well as water. Moreover, the aerosol may disperse sharp particles of dental tissue, calculus, and filling material with considerable force. This dispersion may cause microlesions of the hands, eyes and face that can become a portal of entry for infectious agents. Research has shown that particles > 0.1 mm can be dispersed distances up to six meters, at speeds of 50-60 kmh. In this way, dentists may sustain microtraumata of the hands, eyes and face.^{39,82,83} Contamination may also take place, however, in the

absence of microlesions. Aerosols may be inhaled and the microorganisms contained in them can cause an infection via the mucous membranes of the oronasopharyngeal cavity. Both possibilities of contamination concern the transmission of microorganisms from the patient to dental personnel. When the instruments used are inadequately cleaned, patient-to-patient transmission is also possible. Transmission of pathogenic microorganisms may for instance take place via the suction mechanism of modern dental turbines; they prevent dripping, but at the same time suck microorganisms from the patient's mouth, which may then enter the system.⁸⁴ In this context, drills, scaling cups and brushes, discs, etc. should also be mentioned, if perhaps superfluously.

Finally the possibility of airborne transmission from one treatment unit to the next should be borne in mind, when several units are set up in the same room without adequate isolation.

Instruments used in endodontics

Root canal treatments are invasive interventions in which microorganisms often play a role. The purpose of these treatments is to eliminate the microorganisms causing local inflammation. That these microorganisms may also entail a risk of contamination is often disregarded. Disinfectants are widely used in endodontics, but it should be borne in mind that the mere application of a disinfectant does not guarantee that all conceivable infectious microorganisms are rendered harmless. Cross-infection and contamination through inadvertent cuts or perforations during treatment remain possible. In this context, it may also be pointed out that the handling of reamers, files, etc. may cause microlesions on the hands through which transmission is also possible.

Hands and face

Hands in particular are likely to be smeared with blood during treatment. In this context, Allen and Organ studied the presence of blood remnants on the fingers of dentists.⁴¹ After treatment, blood was demonstrable on the hands of 80 percent of the dentists examined, particularly under the nails of thumb and index finger; in 40 percent, blood was still found after the weekend.

This study also revealed microlesions of the skin in 30 percent to 40 percent of the dentists studied. The eyes, too, are vulnerable in this respect.^{39,85} The findings warrant the conclusion that the dentist's contact with

patient blood and saliva is very intensive and that consequently the risk of transmission of pathogenic microorganisms is serious.

Clothing

Not only the exposed parts of the body, but also those covered by clothing are contaminated with (pathogenic) microorganisms, in particular via aerosols. Dissemination of microorganisms to rooms outside the treatment unit via the clothing of dental personnel is a very real risk.

Treatment units

The treatment unit* as a whole is also a potential source of transmission of pathogenic microorganisms. On the one hand, all surfaces touched by hand or exposed to aerosols are contaminated; on the other hand, the entire suction mechanism is constantly in contact with oral fluids or solid particles from the patient's mouth. The same applies to cuspidors and, to a lesser extent, to washbasins. It is theoretically conceivable, moreover, that the water used in dental treatment is contaminated with pathogenic microorganisms (e.g. *Legionella*). And cross-infection via roentgen apparatus is likewise possible.⁸⁶ Finally, nailbrushes may be a source of microorganisms.

DISCUSSION

Although the pathogenic microorganisms mentioned in this article constitute an unmistakable risk of contamination for the dental profession, the question is whether this risk is really so grave that efforts should be made to ensure reduction of this risk through adequate precautionary measures. For most of the microorganisms mentioned here, the real risk of contamination of dental personnel has never been measured, let alone the risk of cross-infection. Only on hepatitis B does the literature give some relevant data. It is questionable, however, whether detailed investigations in this context would be meaningful, for a risk of contamination depends on many different factors:

- *The type of microorganism.* Type, virulence and number are of importance with regard to the risk of contamination, when treating an infected patient.

*Treatment unit is defined not only as the unit in which actual dental treatment is performed, but also as the unit where radiographs are made.

- *National factors.* In the discussion of the various pathogens, an attempt has always been made to give an impression of the number of contaminated patients, usually related to the situation in The Netherlands. These data are highly dependent, however, on numerous factors, such as general hygiene, nutritional habits, and sexual inclinations of the population. In other countries, therefore, quite different figures may apply and consequently there are different risks of contamination in dental practice.
- *Local factors.* Even within the same country there may be marked differences in the prevalence of infected patients. Intravenous drug users, prostitutes and homosexuals, for example, are high-risk groups often concentrated in big cities. Thus the prevalence of hepatitis B antigen in the average Dutch population is between 0.1 percent and 0.5 percent, but for Amsterdam it is estimated to be between 0.5 percent and 1 percent. Similar differences in concentration are observed for AIDS patients: eighty-four were registered in The Netherlands in October 1985, and fifty of these were living in Amsterdam (the number of seropositives is significantly higher, and is estimated to be 100 times as high).
- *Changes in the numbers of infected patients.* As apparent from this article, the numbers of infected patients mentioned are related to a particular year. There may be significant annual changes in these figures. Shifts in favor of other sexually transmitted diseases may be expected with the increasing number of AIDS patients. There are distinct indications that these diseases are decreasing as a result of an increased use of contraceptives. Although it may be concluded that risks may differ in different dental practices, there remains the fact that, in principle, any risk is to be avoided.

Measures which can be taken to reduce these risks will be discussed in part II.

SUMMARY

In order to ensure adequate reduction of risks of contamination in dental practice, potential pathogens should be known and insight should be gained into possibilities of contamination. The first part of this article presents brief descriptions of the most relevant pathogenic microorganisms such as the causative agents of tuberculosis, gonorrhoea, syphilis, herpes simplex, hepatitis, AIDS and legionnaires' disease. Possibilities

of contamination in dental practice are considered next, with special reference to various ways in which microorganisms can be transmitted:

- Hand instruments.
- Rotating instruments, multiple-function syringe and ultrasonic scaler.
- Instruments used in endodontics.
- Hands and face.
- Clothing.
- Treatment unit. Measures that can be taken in order to minimize the risk of contamination will be discussed in Part II.

REFERENCES

1. Schuster, G.S.: Oral microbiology and infectious diseases. Baltimore/London: Williams and Wilkins, 1983.
2. Roderick Smith, W.H.; Davies, D.; Mason, K.D. *et al*: Intraoral and pulmonary tuberculosis following dental treatment. *The Lancet* II, 842-844, 1982.
3. Schmidt, H.; Hjorting-Hansen; Philipsen, H.P.: Gonococcal stomatitis. *Acta Derm Vener*, 41:324-327, 1961.
4. Merchant, H.W. and Schuster, G.S.: Oral gonococcal infections. *J Am Med Assoc*, 95:807-809, 1977.
5. Jamsky, R.J. and Christen, A.G.: Oral gonococcal infections. *Oral Surg*, 53:358-362, 1982.
6. Escobar, V.; Farman, A.G.; Arm, R.N.: Oral gonococcal infection. *Int J Oral Surg*, 13:549-554, 1984.
7. Jamsky, R.J.: Gonococcal tonsillitis. *Oral Surg*, 44:197-200, 1977.
8. Stolz, E. and Schuller, J.: Gonococcal oro- and nasopharyngeal infection. *Br J Vener Dis*, 50:104-108, 1974.
9. Bro-Jorgensen, A. and Jensen, T.: Gonococcal tonsillar infections. *Br Med J*, 4:660-661, 1971.
10. Bro-Jorgensen, A. and Jensen, T.: Gonococcal pharyngeal infections. *Br J Vener Dis*, 49:491-499, 1973.
11. Wiesner, P.J.; Tronca, E.; Bonin, P. *et al*: Clinical spectrum of pharyngeal gonococcal infection. *N Engl J Med*, 288:181-185, 1973.
12. Thompson, T.R.; Swanson, R.E.; Wiesner, P.J.: Gonococcal ophthalmia neonatorum. Relationship of time infection to relevant control measures. *J Am Med Assoc*, 228:186-188, 1974.
13. Handsfield, H.H.; Lipman, T.O.; Harnish, J.P. *et al*: Asymptomatic gonorrhoea in men: Diagnosis, natural course, prevalence and significance. *N Engl J Med*, 290:117-123, 1974.
14. Wijngaarde, J.B. and Smith, L.H. (eds): *Cecil textbook of medicine*. 16th edn. Philadelphia: W.B. Saunders, 1982, pp 1573-1584.
15. Manton, S.L.; Egglestone, S.I.; Alexander, I. *et al*: Oral presentation of secondary syphilis. *Br Dent J*, 160:237-238, 1986.
16. Meyer, I. and Shklar, G.: The oral manifestations of acquired syphilis. *Oral Surg*, 23:45-57, 1967.
17. Wong, P.N.C.: Secondary syphilis with extensive oral manifestations. *Austr Dent J*, 30:22-24, 1985.
18. Ginnta, L.J. and Fiumara, N.: Ampicillin allergy presenting as secondary syphilis. *Oral Surg*, 57:152-157, 1984.
19. Rawls, W.E. and Camione-Piccardo, J.: Epidemiology of herpes simplex virus type 1 and 2 infections. In: Nahmias, A.J.; Dowdle, W.R.; Schinazi, R.E. (eds). *The human herpes virus: an interdisciplinary perspective*. New York: Elsevier, 1981.
20. Scully, C.: Viruses and cancer: Herpes viruses and tumors in the head and neck. *Oral Surg*, 56:285-292, 1983.
21. Shillitoe, E.J. and Silverman, S.: Oral cancer and herpes simplex virus. *Oral Surg*, 48:216-225, 1979.
22. Manzella, J.P.; McConville, J.H.; Valenti, W. *et al*: An outbreak of

- Herpes simplex virus type I gingivostomatitis in a dental hygiene practice. *J Am Med Assoc*, 252:2019-2022, 1984.
23. Miller, J.B.: Herpes simplex virus infection of the finger of a dentist. *J Dent Child*, 43:99-102, 1976.
 24. Rowe, N.H.; Heine, C.S.; Kowalski, C.J.: Herpetic whitlow: an occupational disease of practicing dentists. *J Am Dent Assoc*, 105:471-473, 1982.
 25. Brooks, S.L.; Rowe, N.H.; Drach, J.C.: Prevalence of herpes simplex virus disease in a professional population. *J Am Dent Assoc*, 102:31-34, 1981.
 26. Baringer, J.R.: Herpes simplex virus infection of nervous tissue in animals and man. *Prog Med Vir*, 20:1-26, 1975.
 27. Openshaw, H. and Bennet, H.E.: Recurrence of herpes simplex virus after dental extraction. *J Infect Dis*, 146:707, 1982.
 28. Openshaw, H.; Schizawa, T.; Wohlenberg, C. *et al*: The role of immunity in latency and reactivation of herpes simplex virus. In: Nahmias, A.J.; Dowdle, W.R. and Schinazi, R.F. (eds): *The human herpes virus: an interdisciplinary perspective*. New York: Elsevier, 1981, pp 289-296.
 29. Boxall, E.H.; Flewett, T.H.; Dane, O.S.: Hepatitis B surface antigen in breast milk. *The Lancet*, II: 1007-1008, 1974.
 30. Villarejos, V.M.; Kirsten, P.H.; Visoma, A. *et al*: A role of saliva, urine, and faeces in the transmission of type B hepatitis. *N Engl J Med*, 291:1375-1380, 1974.
 31. Karayiannis, P.; Novick, D.M.; Lok, A.S. *et al*: Hepatitis B virus DNA in saliva, urine and seminal fluid of carriers of hepatitis B antigen. *Brit Med J*, 290:1853-1855, 1985.
 32. de Flora, S. and Forci, F.: Hepatitis B surface antigen in pleural fluid. *Lancet*, I:1269, 1977.
 33. Hourani, M.R.; Mayer, G.H.; Greenbourn, D.S. *et al*: Hepatitis B surface antigen in urine of hemodialysis patients. *Kidney*, 13:324, 1978.
 34. Darrell, R.W. and Jacob, G.B.: Hepatitis surface antigen in human tears. *Arch Ophthalmol*, 96:674-676, 1978.
 35. Barker, L.J.: Transmission of serum hepatitis. *J Am Med Assoc*, 211:1509-1512, 1970.
 36. Mosley, J.W.: Present knowledge of viral hepatitis. *Int Dent J*, 4:110-114, 1984.
 37. Dienstag, J.L. and Ryan, D.M.: Occupational exposure to hepatitis B virus in hospital personnel: infection or immunization? *Am J Epidemiol*, 115:26-39, 1982.
 38. Japp, P.G.; Prozesky, C.W.; McElligott, S.E. *et al*: Infection of the common bedbug *Cimex lectularis* L with hepatitis B virus in South Africa. *S Afr Med J*, 53:598-601, 1978.
 39. Hartley, J.L.: Eye and face injuries resulting from dental procedures. *Dent Clin North Am*, 22:505-516, 1978.
 40. Kramer, R. von: The dentist's health: high-speed rotary equipment as a risk factor. *Quint Internat*, 16:367-371, 1985.
 41. Allen, A.L. and Organ, R.J.: Occult blood accumulation under finger nails: a mechanism for the spread of blood-borne infection. *J Am Dent Assoc*, 105:455-459, 1982.
 42. Szmuness, W.; Much, M.E.; Prince, A.M. *et al*: On the role of sexual behaviour in the spread of hepatitis B infection. *Ann Int Med*, 83:489-495, 1975.
 43. Favero, M.S.: Detection methods by study of the stability of hepatitis B antigen on surfaces. *J Inf Dis*, 129:210-212, 1974.
 44. Holbrook, W.P.; Muir, K.F.; MacPhee, J.T. *et al*: Bacterial investigation of the aerosol from ultrasonic scalers. *Brit Dent J*, 144:245-247, 1978.
 45. Rosen, S.; Scheid, R.C.; Kin, C.K. *et al*: Potential pathogens in dental aerosols. *Clin Prev Dent*, 5:17-20, 1983.
 46. Bass, B.D.; Andors, L.; Pierri, L.K. *et al*: Quantification of hepatitis B viral markers in a dental school population. *J Am Dent Assoc*, 104:629-633, 1982.
 47. Cottone, J.A. and Goebel, W.M.: Hepatitis B: The clinical detection of the chronic carrier dental patient and the effects of immunization via vaccine. *Oral Surg*, 56:449-454, 1983.
 48. Kane, M.A. and Lettau, L.A.: Transmission of HBV from dental personnel to patients. *J Am Dent Assoc*, 110:634-636, 1985.
 49. Baker, C.H. and Hawkins, V.L.: Law in the dental workplace: legal implications of hepatitis B for the dental profession. *J Am Dent Assoc*, 110:637-642, 1985.
 50. Editorial: Prevention of perinatally transmitted hepatitis B infection. *The Lancet*, 1:939-941, 1984.
 51. Kalyvas, O.G.: Prevalence and risk factors of hepatitis B virus infection in a sample of the dental population of Athens. Thesis, University of Athens, 1985.
 52. Rizzetto, M.; Canese, M.G.; Arico, S.: Immunofluorescence detection of new antigen-antibody system (delta/anti-delta) associated with hepatitis B virus in liver and in serum of HBsAg carriers. *Gut*, 18:997-1003, 1977.
 53. Rizzetto, M.; Hoyer, B.; Canese, M.G. *et al*: Delta agent: association of delta antigen with hepatitis B surface antigen and RNA in serum of delta-infected chimpanzees. *Proc Natl Acad Sci*, 77:6124-6128, 1980.
 54. Bonino, F.; Hoyer, B.; Ford, E. *et al*: The delta agent: HBsAg particles with delta antigen and RNA in the serum of an HBV carrier. *Hepatology*, 1:127-131, 1981.
 55. Rizzetto, M.; Canese, M.G.; Gerin, J.L. *et al*: Transmission of the hepatitis B virus-associated delta antigen to chimpanzees. *J Inf Dis*, 141:590-602, 1980.
 56. Govindarajan, S.; Chin, K.P.; Redeker, A.G. *et al*: Fulminant B viral hepatitis: role of delta agent. *Gastroenterology*, 86:1417-1420, 1984.
 57. Hadler, S.; Monzon, M.; Ponzetto, A.: Delta virus infections and severe hepatitis: An epidemic in the Yucca Indians in Venezuela. *Ann Int Med*, 100:339-344, 1984.
 58. MMWR: Delta hepatitis - Massachusetts. *J Am Med Assoc*, 252:1666-1667, 1984.
 59. Centers for Disease Control: Fulminant hepatitis B among parenteral drug abusers - Kentucky, California. *MMWR*, 33:70-77, 1984.
 60. Purcell, R.H. and Gerin, J.L.: Epidemiology of the delta agent: an introduction. In: Rizzetto, M.; Verne, G.; and Bonino, F. (eds). *Viral hepatitis and delta infection*. New York: Alan R. Liss, 1983, pp 113-119.
 61. Centers of Disease Control: ACIP recommendations for protection against viral hepatitis. *MMWR*, 34:313-335, 1985.
 62. Leads from the MMWR 35 Update: Acquired immunodeficiency syndrome U.S. *J Am Med Assoc*, 255:580-593, 1986.
 63. Acheson, E.D.: AIDS: a challenge for the public health. *The Lancet*, 1:662-666, 1986.
 64. Biggar, R.J.: The AIDS problem in Africa. *The Lancet*, 1:79-83, 1986.
 65. Vogt, M.W.; Witt, D.J.; Craven, D.E. *et al*: Isolation of HTLV/LAV-III from cervical secretions of women at risk for AIDS. *The Lancet*, 1:525-527, 1986.
 66. Wofsky, C.B.; Cohen, J.B.; Hauer, L.B. *et al*: Isolation of AIDS-associated retrovirus from genital secretions of women with antibodies to the virus. *The Lancet*, 1:527-529, 1986.
 67. Stewart, G.J.; Tyler, J.J.P.; Cunningham, A.L. *et al*: Transmission of human T-cell lymphotropic virus type III (HTLV-III) by artificial insemination by donor. *The Lancet*, II:581-584, 1985.
 68. Ziegler, J.B.; Johnson, R.O.; Cooper, D.A. *et al*: Postnatal transmission of AIDS: Associated retrovirus from mother to infant. *The Lancet*, 1:896-897, 1985.
 69. Levy, J.A. and Shimabukurno, J.: Recovery of AIDS-associated retrovirus from patients with AIDS of AIDS-related conditions and from clinically healthy individuals. *J Infect Dis*, 152:734-738, 1985.
 70. CDC: Additional recommendations to reduce sexual and drug abuse related transmission of human T-lymphotropic virus type III/lymphadenopathy-associated virus. *MMWR*, 35:10, 1986.
 71. Friedland, G.H.; Salzman, R.B.; Rogers, M.F. *et al*: Lack of transmission of HTLV III/LAV infection to household contacts of patients with AIDS of AIDS-related complex with oral candidiasis. *N Engl J Med*, 314:344-349, 1986.
 72. Weiss, S.H.; Saxinger, W.C.; Rechtman, D. *et al*: HTLV-III infec-

- tion among health care workers. *J Am Med Assoc*, 254:2089-2093, 1985.
73. CDC Update: Evaluation of human T-lymphotropic virus type III/lymphadenopathy-associated virus infection in health-care personnel - United States. *MMWR*, 34:5/5-578, 1985.
 74. Martini, L. S.; McDougal, J.S.; Loskoski, S.J.: Disinfection and inactivation of the human T lymphotropic virus type III/lymphadenopathy-associated virus. *J Infect Dis*, 152:400-403, 1985.
 75. Boldur, I. and Ergaz, M.: A prevalence study of Legionella species in geriatric institutions. *J Hyg Camb*, 92:37-43, 1984.
 76. Stout, I.; Yu, L.V.; Vickers, R.M. *et al*: Ubiquitousness of Legionella pneumophila in the water supply of a hospital with endemic Legionnaire's disease. *N Engl J Med*, 306:466-468, 1982.
 77. Zuravleff, J.J.; Yu, V.L.; Shonnard, J.W. *et al*: Legionella pneumophila contamination of a hospital humidifier. *Am Rev Respir Dis*, 128:657-661, 1983.
 78. Baskerville, A.; Fitzgeorge, R.B.; Hambleton, P. *et al*: Experimental transmission of Legionnaire's disease by exposure to aerosols of Legionella pneumophila. *The Lancet*, 1:1389-1390, 1981.
 79. Exner, M.; Hann, F.; Kocikowski, R.: Zahnärztliche Einheiten als Kontaminationsquelle für *Pseudomonas aeruginosa*. *Dtsch Zahnärztl Z*, 36:819-824, 1981.
 80. Just, H.M. and Michel, R.: Infektionsgefährdung durch Bakterien, Pilze und Amöben in Kühl- und Spülwasser Zahnärztlicher Einheiten. *Dtsch Zahnärztl Z*, 39:60-64, 1984.
 81. Fotos, P.G.; Westfall, H.N.; Snyder, I.S. *et al*: Prevalence of Legionella-specific IgG and IgM antibody in a dental clinic population. *J Dent Res*, 64:1382-1385, 1986.
 82. Grandy, J.R.: Enamel aerosols created during use of the air turbine handpiece. *J Dent Res*, 46:409-416, 1967.
 83. Kramer, R. von: The dentist's health: high-speed rotary equipment as a risk factor. *Quint Internat*, 16:367-371, 1985.
 84. Graf, W. and Vollmuth, G.: Design-dependent transmission of bacteria by internal infection of dental turbines. *Zbl Bakt Hyg I Abt Orig B*, 165:444-457, 1978.
 85. Cooley, R.L. *et al*: Ocular injuries sustained in the dental office: methods of detection, treatment and prevention. *J Am Dent Assoc*, 97:985-988, 1978.
 86. White, S.C. and Glaze, S.: Interpatient microbiological cross-contamination after dental radiographic examination. *J Am Dent Assoc*, 96:801, 1978.

PERIODONTAL DISEASE IN CHILDREN

Periodontal disease is one of the most common bacterial conditions affecting mankind, though it is sometimes rather naively considered to concern only the adult population. It is clear . . . that this is far from the truth. The sentiment does, however, reflect the paucity of published data concerning periodontal conditions in children and adolescents. The dentist should interact at an early stage in order to give these young people the maximum chance of entering adulthood with a healthy status throughout adult life.

Periodontal disease is thought to be the result of a reaction of the host tissues to bacterial plaque, though in a few periodontal conditions it may be due to specific microorganisms within the plaque. Gingivitis, the earliest clinical sign of periodontal disease, can be seen, to a greater or lesser extent, in almost every individual with permanent teeth. However, the preschool child appears to have a reduced host immune response to plaque deposits, making gingivitis less common than in children at the mixed dentition stage, even with similar amounts of plaque deposit. This may be due to an immature system or to a different oral flora in the very young.

If allowed to persist, gingivitis usually progresses to irreversible loss of periodontal attachment, bone loss and eventually exfoliation of the teeth, a process which may take a lifetime or just a few years. There is considerable individual variability in the rate of progression of periodontal disease, some individuals being far more susceptible than others. These latter constitute an 'at-risk' group. The progression of periodontal disease is not continuous, there being periods of active progression and quiescence. In some instances, regression has been reported.

Elderton, R.J.: *Positive Dental Prevention*. London: William Heinemann Medical Books, 1987, pp 7, 8.

Hygiene in dental practice Part II: Measures to reduce the risk of contamination

Johannes de Graaff, PhD
William E. van Amerongen, DDS, PhD
Grietje R. Mulder, DDS

Taking adequate hygienic measures in general dental practice requires some insight into possibilities of contamination during or as a result of treatment of patients. This is why Part I discussed risks of contamination in dental practice in relation to tuberculosis, gonorrhoea, syphilis, herpes, hepatitis, legionnaires' disease and AIDS.¹ The descriptions of these diseases warrant the following conclusions:

- Contamination may occur via the mucosal membranes, lungs, eyes and (micro) lesions of the skin, especially the skin of the hands and face.
- Hygienic measures should focus especially on protection of these organs and tissues.
- When hygienic measures to prevent transmission of hepatitis B and AIDS are routinely taken in the treatment of patients, the risk of transmission of other infectious diseases is minimized as well.

Part I also reviewed conceivable possibilities of transmission of pathogenic microorganisms in dental practice. Generally speaking, transmission may take place via instruments, aerosols, hands and face, clothing, and treatment unit. Although possibilities of reducing trans-

mission of pathogenic microorganisms are complex, each measure can often contribute to improvement of general hygiene.

For the sake of reader convenience, therefore, various possibilities of practicing contamination-reducing measures will be discussed separately, so far as possible. Measures to reduce the risk of contamination will be discussed first. Measures with regard to patients specifically identified as infected come next, and measures to protect personnel immediately involved in dental treatment follow. Finally there will be a discussion of the consequences of a stringent introduction of a range of measures taken to improve hygiene in dental practice.

GENERAL MEASURES TO REDUCE THE RISK OF CONTAMINATION

Reduction of the risk of contamination in the treatment of most infected patients, sometimes unidentified, can be achieved by the following measures: direct preventive behavior, protective measures, domestic cleaning, disinfection, sterilization, indirect preventive behavior.

Direct preventive behavior

Direct preventive behavior can be defined as behavior of dental personnel based on constant awareness of the risk of contamination or transmission. This involves:

- Frequent washing of the hands with water and soap.

Dr. de Graaff is Professor of Oral Microbiology. Dr. van Amerongen and Dr. Mulder are with the Department of Paediatric Dentistry, ACTA, Vrije Universiteit. Dr. de Graaff should be addressed at that university, v.d. Boechorststraat 7, 1081 BT Amsterdam, The Netherlands; and Dr. van Amerongen and Dr. Mulder at the same university, de Boelelaan 1115, P.O. Box 7161, 1007 MC Amsterdam, The Netherlands.

- Avoidance of contact with the entire treatment unit, during the treatment of patients, so far as possible.
- Close attention to personal general hygiene.
- Working cleanly and in an orderly fashion.

Protective measures

WEARING UNIFORM

Wearing a uniform* is necessary in view of the immediate contact with the patient and the aerosol problem.²⁻⁷ Wearing a uniform is of little actual use, however, unless it is changed after each patient. This would mean that an enormous stock of uniforms must be maintained, to assure a ready supply. Disposable clothing might be an alternative. Since both methods are very expensive, however, the wearing of a plastic apron over the uniform seems an acceptable solution. The apron may be submitted to simple domestic cleaning after each treatment, and the uniform can be worn a little longer. Moreover, this combination can also largely prevent transmission of microorganisms through the building: an apron can easily be removed, when one leaves the treatment unit.

LAUNDRY

The laundering of uniforms is obviously the next subject to be given attention. Despite the apron, uniforms may be contaminated with pathogenic microorganisms. An adequate laundering program is required, therefore, to ensure effective cleaning. Laundering at 80° C for ten minutes as a rule suffices to kill most microorganisms. To make sure that even highly resistant microorganisms such as hepatitis B virus are killed, it is advisable to add an amount of chlorine (0.75 l per 100 kg) to the laundry. Some laundries specialize in laundering hospital clothing.

USE OF DISPOSABLES

The use of disposable items may contribute in many ways to improve hygiene in practice. The following items may be considered in this context.

- Rubber gloves.* Inadequate facilities for proper cleaning of the hands and the risk of direct contamination via microlesions may be compensated for or avoided by wearing rubber gloves.⁸⁻¹⁰ Although of course it would be preferable to use a new pair of sterile gloves for each patient, this may

not be feasible for financial reasons. Even non-sterile gloves used, however, for several patients can considerably improve hygiene. Unlike the hands, rubber gloves are smooth and offer few sites for retention of microorganisms. When the gloved hands are regularly washed with water and soap, the risk of transmission of microorganisms via the hand is small.¹¹

- Masks.* Inhalation of possibly infected aerosols and infliction of microlesions on the face can be reduced by wearing masks to cover the nose and mouth.^{10,12} The mask should be discarded and replaced by a new one after each patient, because moistened masks do not ward off microorganisms.
- Caps.* In particular to prevent cross-contamination as a result of hair "polluted" by aerosols, it is advisable to wear a cap. Their use is subject to the same rules as the use of masks.
- Protective glasses.* Damage to the eyes, accompanied by contamination with pathogenic microorganisms, can readily occur during dental work with water cooling, excavation, removal of calculus, and removal of chemical and irritant preparations.¹⁰⁻¹⁶ It is highly advisable, therefore, that dental personnel wear protective glasses, which should be cleaned with water and soap after each patient.

DOMESTIC CLEANING

Domestic cleaning can be defined as removal of blood, oral fluids, dental tissue and dental materials from the instruments used on patients (both rotatory and manual instruments). This procedure is required for biophysical and microbial reasons. The sterilization that follows this procedure is frequently unsatisfactory unless preceded by a thorough cleaning as described above: the high temperature of sterilization may burn remnants of dirt into the instruments or cause marked oxidation of blood stains; thus, microorganisms in the debris may survive the sterilization process.

Consistent thorough cleaning of rotatory and manual instruments by brushing with water and soap might be regarded as an adequate preparation for sterilization. In actual practice, however, the contrary has proved true. Moreover, the handling of instruments for cleaning poses a substantial risk of suffering lesions caused by sharp instruments. This is why the use of a professional dishwasher is to be preferred. In these machines, the instruments are as a rule prerinsed at a low temperature to prevent coagulation of proteins. The actual washing then takes place at about 90° C, for ten minutes. The

*In view of the aerosol problem the uniform should consist of jacket and trousers.

reasonably high temperature contributes to the killing of a fair number of microorganisms and is certainly sufficient to kill the hepatitis B virus (HBV) and the AIDS virus (HIV). It should be borne in mind, however, that the instruments are not yet sterile.

DISINFECTION

Disinfection can be defined as reduction of the level of contamination by treatment with certain chemicals. Unfortunately, the disinfectants currently in use do not assure that all pathogenic microorganism and their spores will be destroyed. This procedure, therefore, cannot replace sterilization, assuming sterilization is possible.

This applies to surfaces of tables, cabinets, units, plastic aprons, vulnerable instruments and skin surfaces. The basis of disinfection should be thorough cleaning with water and soap. For skin surfaces this may generally have to suffice, because effective disinfectants are too irritating. So far as the material permits, the use of effective chemicals is advisable. Examples of reasonably effective disinfectants are: formaldehyde, 1.4 percent to 2 percent; hypochlorite (NaOCl), 0.5 percent; glutaraldehyde.

The efficacy of these disinfectants against HBV particularly has been demonstrated in several studies. It should be borne in mind, however, that the published data pertain to experimental conditions, which cannot be achieved when these agents are used to disinfect surfaces.

Other disinfectants previously recommended by The Netherlands Health Council are still under investigation.¹⁷ Research on 70 percent ethylalcohol has been resumed. The reports on acetic acid are not unequivocal. Contrary to previous recommendations (Health Council 1976), ethylalcohol and acetic acid can no longer be recommended for use in practice as long as research is in progress.¹⁸ In view of the toxic effects of formaldehyde preparations, the current preference is for glutaraldehyde or sodium hypochlorite.^{16,19-21}

STERILIZATION

Sterilization is defined as the destruction of all viable microorganisms. Although there are several conceivable methods, only one is generally accepted in dental practice: moist heat sterilization (sterilization by steam under pressure).

The suitable instrument for this is the autoclave. The instruments should be heat-stable, and in this respect considerable progress has been made in recent years.

Practically all manual instruments can now be sterilized. Parts with which this is difficult or impossible are widely available as disposables.²²

Apart from a few brands, the technically vulnerable handpieces and angle-pieces can likewise be sterilized by steam under pressure. It is advisable, therefore, to replace any nonsterilizable angle-pieces as quickly as possible.

Problems related to endodontic treatment are to be considered somewhat separately in this context. The limited efficacy of disinfectants and the risk of accidental perforations necessitate the use of sterile instruments. The best way to achieve this, even during treatment, is to use a glass bead sterilizer.²³⁻²⁵

Indirect preventive behavior

Indirect preventive behavior consists of taking measures that may indirectly influence the reduction of the risk of contamination. These measures concern:

THE COMPONENTS OF THE TREATMENT AREA

Particularly with regard to the components of the treatment area some aspects of hygiene should be further discussed, namely:

□ *The suction system*

The use of a high-velocity suction system is recommended for all interventions involving aerosols (e.g. multiple-function syringe and drill cooling). This type of system sucks off most of the aerosol and reduces the germ count by about 99 percent. In addition it is advisable to use cofferdam, because this forms a mechanical screen from the other parts of the oral cavity. Most suction systems show a high degree of internal contamination, and this is why the preference is for models with automatic drainage and a special rinsing system obviating the need for periodical cleaning of the separation drum. Apart from this, it is necessary to flush the entire system at least twice daily with ample amounts of water containing one of the cleaning agents especially developed for this purpose.

□ *The multifunction syringe*

Since the use of a multi-function syringe also involves aerosol formation, thus contaminating the instrument, sterilization of the entire cartridge is required. In procuring multi-function syringes, it should be ensured that the entire cartridge is detachable and sterilizable.

□ *The cuspidor*

Since the possibilities of efficient cleaning of cuspidors are limited, these should not be used; if possible, they should be removed altogether. If necessary, they may be replaced with a sterilizable rinsing funnel.

□ *Unit handles*

Component parts that are not used during treatment should not be touched with the hands during treatment. Inevitably, however, parts like the operating lamp and the arm holding the angle-pieces, multifunction syringe, and suction tips must regularly be moved to different positions. These parts should be fitted, therefore, with detachable, sterilizable handles, or covered by disposable foil, which should be replaced after each patient.

□ *The treatment chair*

To avoid touching this with the hands, any manual positioning controls should be inactivated and replaced with foot controls.

□ *Water supply for multifunction syringe and spray cooling*

Water to be used in the mouth is slightly warmed in the unit, in a small boiler or by a heater element. In units equipped with a boiler, the water remains stagnant for a considerable time during holidays. Although this remains to be properly investigated, it is theoretically possible that pathogenic microorganisms such as *Legionella pneumophila* develop in this water during such periods. It is advisable, therefore to change the water in these boilers (about 1.5 l) by draining for five minutes with the multifunction syringe and replenishing with fresh water before resumption of work after holidays.

□ *Disposables*

Disposables should be used, whenever possible. Indispensable disposables are plastic suction tips and rinsing cups, and paper covers on treatment trays and instrument cabinets. Instead of plastic tips and cups, however, sterilizable tips and cups might be used in deference to environmental concern.

□ *Dividing walls between treatment units in group practices and large clinics in dental schools*

As already pointed out, aerosols may cover large distances at high velocity, and particles containing pathogenic microorganisms may thus enter an adjacent treatment unit. Divisions between units, therefore, should be ceiling-high.

THE ANESTHETIC SYRINGES

The syringes used in anesthesia may be threaded to accommodate disposable needles. To remove such a needle after treatment, the protective cover must first be replaced and thus the risk of accidental perforation as the needle is detached from the syringe is quite considerable. It is advisable, therefore, to use a syringe with a clip-on needle. This needle can be detached simply by inserting it in the aperture of a specially designed needle container and twisting; the risk of accidental perforation is thus avoided.

THE WASHBASIN

All washbasins should carry a nailbrush, because this is certainly needed after removal of rubber gloves. The heat retention caused by the gloves creates a climate that favors the growth of microflora. Thorough cleaning of the hands after treatment is, therefore, necessary. The nailbrush, which should be sterilizable, should be replaced after use. The use of liquid soap from an arm- or foot-controlled dispenser and an arm-controlled mixing tap are useful contributions to a hygienic handwashing.

ULTRASONIC CLEANING

For instruments not easily cleaned by washing (drills, endodontic instruments, etc.) the ultrasonic cleaner is an acceptable solution, particularly if the fluid used also contains a disinfectant. A disadvantage of ultrasonic cleaning is the aerosol it creates, resulting in a cloud of pathogenic microorganisms that spreads through the room. To avoid this occurring, the ultrasonic cleaner should have a close fitting lid. With regard to sterilization of drills, the problem of oxidation is to be considered. This complication can be avoided by replacing steel drills with tempered metal drills.

VACCINATION AGAINST HEPATITIS B

In Part I it was stated that infection with the hepatitis B virus is a serious threat. The erratic course, the lack of an effective therapy, and the highly contagious nature of minute quantities of blood, as well as the continuous presence of carriers in the general population, are all factors contributing to the menace of hepatitis B, more especially for high-risk groups, such as those involved in the dental care of patients.²⁶⁻⁴² In view of this risk, The Netherlands Health Council advises all workers in dental health care — dentists, dental assistants, oral hygienists, dental laboratory technicians and mainte-

nance personnel — to be vaccinated against hepatitis B.

There is as yet no consensus about periodical serum antibody determinations after vaccination and reactivation of the vaccination status with subsequent booster doses. According to the literature, a lower limit of 10 MIU/ml seems desirable.

The newly developed vaccine against HBV has produced very good results. Some 95 percent of the persons vaccinated produce anti-HBsAG antibodies, and effective protection is proved to correspond with this finding.^{43,44} It may be expected that vaccination against hepatitis B will play an important role in the protection of individuals.⁴⁵⁻⁴⁷ It would not be meaningful, however, to vaccinate all patients against hepatitis B.

Although some reports in the literature mention contamination of patients by HBsAG-positive dentists (probably via minute lesions on the dentists' hands), other reports indicate that such contaminations can be prevented by wearing gloves.⁴⁸⁻⁵⁰

SUPPLEMENTARY HYGIENIC MEASURES IN TREATING PATIENTS WITH IDENTIFIABLE INFECTIONS

Unfortunately, it is not always possible to prevent all contamination by using a number of general hygienic measures. With regard to patients with a specific disease entity, such as hepatitis B and AIDS, basic hygienic measures should be expanded without being unrealistic. Only occasionally is an infected patient identified, and the majority of patients are treated, therefore, as though normal. It seems ill-advised to provide a special treatment room for these patients. This opinion is supported by the following arguments.

- If patients of this category are always to be treated by the same team, far-reaching protective measures must be taken to prevent the increased risk of contamination. If several teams, however, make use of a special treatment room, it is possible that unfamiliarity with its appointments may lead to mistakes.
- In most nonhospital dental practices, it is not feasible to treat this category of patients in a separate room.
- A separate room, completely appointed and equipped, is very expensive. Doing without a separate room, however, does not imply that other supplementary hygienic measures should not be taken. On the contrary, the range of hygienic measures already described should be extended to include additional precautions.

Aerosol restriction

Although most aerosols can be removed with the aid of a high-velocity suction system, aerosol formation should, nevertheless, be avoided as far as possible. This means that patients should be treated without water cooling, ultrasonic devices for removal of dental calculus, and multifunction syringes.

Instead, the green hand-piece is to be used in preparation, calculus must be removed with manual instruments, and a manual insufflator should be used, instead of the multifunction syringe.

Moreover, cofferdam protection should be used in the treatment of the patient, whenever possible.

Aftercare of clothing and instruments

Not only mouth- and nose masks and caps, but also gloves should be discarded immediately after treatment. All disposables should be placed in a separate wastebag, which should be sealed immediately and marked with a sticker. Injection needles are the exception: they are removed in needle containers especially designed for this purpose. Uniforms should not be worn after treating a patient: they must be sent to the laundry. The plastic apron must be carefully cleaned with a disinfectant.

Used instruments should be clearly marked as contaminated, using stickers, before sterilization, to ensure special care in handling. It is not necessary to sterilize these instruments in a special way.

Aftercare of the treatment unit

After treatment of a patient, the treatment chair, unit, cabinets, operating lamp and floor must be disinfected. The suction system must be flushed with a disinfectant fluid especially prepared for this purpose, and the disposable suction bag should be disconnected and removed in a sealed and stickered plastic wastebag.

Timing of treatment

Treatment of patients whose anamnestic data indicate a distinct risk of contamination should be done at the end of the day, so that treatment can be performed without haste and afford ample opportunity afterwards to clean the entire treatment unit.

Measures after accidental perforation or other contaminating accidents

In cases of accidental perforation sustained by nonvaccinated persons and thus exposed to the blood and saliva of a HBsAg-positive patient, it is advisable to administer antihepatitis B immunoglobulin (anti-HBIG) as soon as possible, but in any case within forty-eight hours, preferably by intramuscular injection. Adults need 5 ml and children 0.08 ml/kg body weight.^{17,51}

It is also advisable to vaccinate after two months, and if necessary, supplemented by a second injection of immunoglobulin. Persons whose immune status is either unknown or insufficient (< 10 MIU/ml) should also receive anti-HBIG and a HBsAg booster after accidental perforation. A perforation accident with blood or saliva from an HIV-positive patient requires immediate thorough disinfection. At this time the value and necessity of having the serum checked for antibodies is still debatable.

HYGIENIC MEASURES TO PROTECT PERSONNEL INDIRECTLY INVOLVED IN DENTAL TREATMENT

Because contamination need not be limited to the area surrounding the treatment chair, it is necessary to describe general hygienic measures for all personnel who may be indirectly in touch with patients or instruments. These would be mainly personnel in large group-practices and in dental schools. More specifically:

- X-ray technicians.
- Personnel in the central sterilization unit.
- Maintenance personnel.
- Dental technicians.
- Cleaning service personnel.

X-ray technicians

Apart from conventional measures, such as handwashing and cleaning the X-ray unit, it is advisable that X-ray technicians also wear rubber gloves, according to instructions discussed earlier in this paper. As to the X-ray head, cleaning or disinfection by hand methods is not enough. After each patient, this system must be sterilized, using the procedure already described.

Central sterilization unit personnel

These people should write the word *hygiene* in capital letters: here, after all, is the center of all dental activities. Apart from general principles of cleanliness, the

people in this area should wear rubber gloves. Moreover, strict separation, both in terms of space and in terms of personnel, should be observed between:

- Intake and processing of soiled or used instruments.
- Issuing of clean and sterilized instruments and materials.

Maintenance personnel

The activities of maintenance personnel in any way related to treatment of patients can be divided into:

- Maintenance or repair of small, detachable components.
- Repairs within the unit, during treatment of a patient.
- Maintenance or repair of a unit not in use for patient treatment.

For maintenance or repair of small, detachable components, the components should at least be cleaned in the dishwasher. If possible, sterilization should take place before maintenance personnel begin repairs. Repairs within the unit during treatment must be restricted to real emergencies. All work to maintain or repair treatment units should be done wearing rubber gloves.

Dental technicians

Personnel in the dental laboratory should wear rubber gloves, whenever handling components that have been in or in contact with the patient's mouth. Extramural dental laboratories working for a dental school should be advised accordingly.

Cleaning-service personnel

Cleaning-service personnel should be informed by the head of that department that a treatment room has been used for treating contaminated or infected patients, and that proper hygienic precautions should be observed, therefore, during cleaning work to protect themselves. This means that protective gloves should be worn whenever possible, and certainly during removal of used disposables. The latter should be removed in the wastebag into which they were discarded during treatment. In no case must only the contents of the wastebag be removed.

Mandatory information and vaccination against hepatitis B

Those who have treated patients infected with a specific disease entity are responsible not only for the aftercare of instruments and treatment unit, but for all persons directly or indirectly at risk of contamination. These people should be informed of the risk in advance. This responsibility applies in particular to the personnel discussed above. To protect this personnel to a level beyond that guaranteed by the above mentioned hygienic measures, vaccination against hepatitis B is advisable.

DISCUSSION

The question may arise, whether it is really necessary to take all the measures described in this publication. The risk of infection of dental personnel or cross-infection between patients is not too great, at least for hepatitis B and AIDS. Yet there are two reasons for stricter hygienic measures as part of dental performance in general.

- Infection with HBV or HIV may prove fatal. Moreover, the risk of transmission of such microorganisms is much graver in dental practice than in many other conceivable situations.
- It has been shown in actual practice that a dentist may be held responsible, when patients prove to have been infected. This liability arose from the allegation that insufficient measures were taken to prevent transmission of pathogenic microorganisms. The criteria to be met in general dental practice, however, are still uncertain. No consensus has been reached. In The Netherlands, the guidelines formulated by the Health Council are generally regarded as acceptable minimal requirements. Introduction of measures as described in this article would cause essential changes in the dental treatment of patients. The physical environment in which dental treatment would be conducted would resemble a hospital setting. This would require considerable adaptation, not only by the dentist and other dental personnel, but also by the patient. The patient could become accustomed to the new situation, if properly informed by the dentist. The inconveniences resulting from such measures as wearing rubber gloves will most readily be accepted by future dentists, if introduced in the earliest possible stage of training and applied consistently. These hygienic measures should be included, therefore, in the dental curriculum at the earliest possible moment. Adaptation to the entire

range of measures, however, will not always be possible. A disadvantage, for instance, is the reduced identifiability of a dentist who is wearing mouth- and nose mask, protective glasses and cap. Many patients will not find this an insurmountable obstacle, but in some cases communication, and, therefore, treatment, may be seriously impaired. To be mentioned in this context are children, highly anxious patients, and mentally deficient patients. In such cases, the mouth- and nose mask in particular is undesirable, and whenever indicated, the range of hygienic measures should be adapted to such situations.

As to the financial consequences of the introduction of all these new hygienic measures, it can only be noted that in some way they will have to be charged to the patient. The amount involved depends entirely on the dentist's personal choice and the hygienic rules and regulations accepted in the country where he practices.

SUMMARY

On the basis of the description of potential pathogens and possibilities of contamination during dental treatment in Part I, Part II discusses several measures to be taken, in order to reduce the risk of contamination for patients as well as for dental personnel.

The hygienic measures mostly concern the treatment of patients, including some unidentified infected patients. In this context, direct and indirect behavior of dental personnel, protective measures, domestic cleaning, disinfection, and sterilization are discussed in detail.

In addition, possible supplementary measures to be taken in the treatment of identified infected patients are discussed, with special reference to hepatitis B and HIV.

Finally, hygienic measures to protect personnel not directly involved in dental treatment are discussed.

REFERENCES

1. Amerongen, W.E. van and Graaff, J. de: Hygiene in dental practice. Part I. Potential pathogens and possibilities of contamination. *J Dent Child*, 55:00-00, January-February, 1988.
2. Grandy, J.R.: Enamel aerosols created during use of the air turbine handpiece. *J Dent Res*, 46:409-416, 1967.
3. Kramer, R. von: The dentist's health: high-speed rotary equipment as a risk factor. *Quint Internat*, 16:367-371, 1985.
4. Holbrook, W.P.; Muir, K.F.; MacPhee, J.T. *et al*: Bacterial investigation of the aerosol from ultrasonic scalers. *Brit Dent J*, 144:245-247, 1978.
5. Rosen, S.; Scheid, R.C.; Kin, C.K. *et al*: Potential pathogens in dental aerosols. *Clin Prev Dent*, 5:17-20, 1983.

6. Just, H.M. and Michel, R.L. Infektionsgefährdung durch Bakterien, Pilze und Amöben in Kühl- und Spülwasser zahnärztlicher Einheiten. *Dtsch Zahnärztl Z*, 39:60-64, 1984.
7. White, S.C. and Glaze, S.: Interpatient microbiological cross-contamination after dental radiographic examination. *J Am Dent Assoc*, 96:801, 1978.
8. Allen, A.L. and Organ, R.J.: Occult blood accumulation under finger nails: a mechanism for the spread of blood-borne infection. *J Am Dent Assoc*, 105:455-459, 1982.
9. Miller, J.B.: Herpes simplex virus infection of the finger of a dentist. *J Dent Child*, 43:99-102, 1976.
10. Barker, L.J.: Transmission of serum hepatitis. *J Am Med Assoc*, 211:1509-1512, 1970.
11. Mitchell, R.; Cumming, G.T.; Mac Lennan, W.D. *et al*: The use of operating gloves in dental practice. *Brit Dent J*, 154:372-376, 1983.
12. Hartley, J.L.: Eye and face injuries resulting from dental procedures. *Dent Clin North Am*, 22:505-516, 1978.
13. Cooley, R.L. *et al*: Ocular injuries sustained in the dental office: methods of detection, treatment and prevention. *J Am Dent Assoc*, 97:985-988, 1978.
14. Brooks, S.L.; Rowe, N.H.; Drach, J.C.: Prevalence of herpes simplex virus disease in a professional population. *J Am Dent Assoc*, 102:31-34, 1981.
15. Rowe, N.H.; Heine, C.S.; Kowalski, C.J.: Herpetic whitlow: an occupational disease of practicing dentists. *J Am Dent Assoc*, 105:471-473, 1982.
16. Manzella, J.P.; McConville, J.H.; Valenti, W. *et al*: An outbreak of Herpes simplex virus type I gingivostomatitis in a dental hygiene practice. *J Am Med Assoc*, 252:2019-2022, 1984.
17. Gezondheidsraad: Advies inzake hepatitis B. Den Haag, 1983.
18. Gezondheidsraad: Advies inzake herziene richtlijnen preventie en bestrijding ziekenhuisinfecties. Den Haag, 1976.
19. Martini, L.S.; McDougal, J.S.; Loskoski, S.J.: Desinfection and inactivation of the human T lymphotropic virus type III/lymphadenopathy-associated virus. *J Infect Dis*, 152:400-403, 1985.
20. Council on Dental Materials etc.: Infection control in the dental office. *J Am Dent Assoc*, 97:673, 1978.
21. Academisch Centrum Tandheelkunde Amsterdam (ACTA): Werkgroep uitgifte en sterilisatie. Richtlijnen ter bevordering van de hygiëne. Internal report, 1985.
22. Gezondheidsraad: Tweede advies inzake de problematiek van het verkregen immunodeficiënciesyndroom (AIDS) in Nederland. Klinische, psychosociale en ethische aspecten. Den Haag, 1985.
23. Berghuis, L.P.J.; Schotman, M.D.; Graaff, J. de *et al*: De betrouwbaarheid van de kogelsterilisator. *Ned Tijdschr Tandheelkd*, 91:199-204, 1984.
24. Dayoub, M.B. and Devine, M.J.: Endodontic dry heat sterilizer effectiveness. *J Endod*, 2:343-344, 1976.
25. Windeler, A.S. and Walter, R.G.: The sporadic activity of glass bead sterilizers. *J Endod*, 1-8:273-276, 1975.
26. Dienstag, J.L. and Ryan, D.M.: Occupational exposure to hepatitis B virus in hospital personnel: infection or immunization? *Am J Epidemiol*, 115:26-39, 1982.
27. Favero, M.S.: Detection methods by study of the stability of hepatitis B antigen on surfaces. *J Inf Dis*, 129:210-212, 1974.
28. Cottone, J.A.: Hepatitis B virus infection in the dental practice. *J Am Dent Assoc*, 110:617-621, 1985.
29. Kane, M.A. and Lettau, L.A.: Transmission of HBV from dental personnel to patients. *J Am Dent Assoc*, 110:634-636, 1985.
30. Baker, C.H. and Hawkins, V.L.: Law in the dental workplace: legal implications of hepatitis B for the dental profession. *J Am Dent Assoc*, 110:637-642, 1985.
31. Weiss, S.H.; Saxinger, W.C.; Rechtman, D. *et al*: HTLV-III infection among health care workers. *J Am Med Assoc*, 254:2089-2093, 1985.
32. CDC Update: Evaluation of human T-lymphotropic virus type III/lymphadenopathy-associated virus infection in health-care personnel - United States. *MMWR*, 34:575-578, 1985.
33. Leads from the MMWR 35 Update: Acquired immunodeficiency syndrome U.S. *J Am Med Assoc*, 255:580-593, 1986.
34. Centers for Disease Control: ACIP recommendations for protection against viral hepatitis. *MMWR*, 34:313-335, 1985.
35. Centers for Disease Control: U.S. Department of health and human services. Current Trends. Summary: Recommendations for preventing transmission of Infection with Human T-lymphotropic Virus Type III/lymphadenopathy-associated virus in the workplace. *MMWR*, 34:681-695, 1985.
36. Windecker, D.: Schutz des Zahnarztes und seines Personals vor Hepatitis B und AIDS. *Praxisführung Zahnärztliche Mitteilungen Heft*, Vol. 75, 1985.
37. Palenstein Helderman, W.H. van; Wijngaarden, J.K. van: AIDS en de risico's voor de tandarts. *Ned Tand Blad*: 210-213, 1985.
38. Cottone, J.A. and Goebel, W.M.: Hepatitis B: The clinical detection of the chronic carrier dental patient and the effects of immunization via vaccine. *Oral Surg*, 56:449-454, 1983.
39. Mori, M.: Status of viral hepatitis in the world community: its incidence among dentists and other dental personnel. *Int Dent J*, 34:115-121, 1984.
40. Ahtone, J. and Goodman, R.A.: Hepatitis B and dental personnel: transmission to patients and prevention issues. *J Am Dent Assoc*, 106:219-222, 1983.
41. Sanger, R.G.; Bradford, B.A.; Delaney, J.M.: An inquiry into the sterilization of dental handpieces relative to transmission of hepatitis B virus. *J Am Dent Assoc*, 96:621-624, 1978.
42. Cottone, J.A.: Delta hepatitis: another concern for dentistry. *J Am Dent Assoc*, 112:47-49, 1986.
43. Szmuness, W.; Stevens, C.E.; Harley, E.J. *et al*: Hepatitis B vaccine. Demonstration of efficacy in a controlled clinical trial in a high risk population in the United States. *N Engl J Med*, 303:833-841, 1980.
44. Maupassant, Ph.; Chiron, J.P.; Barin, F. *et al*: Efficacy of Hepatitis B vaccine in prevention of early HBsAg carried state in children. *Lancet*, 1:289-292, 1981.
45. Francis, D.P.; Hadler, S.C.; Thompson, S.E. *et al*: The prevention of hepatitis B with vaccine. Report of the centers for disease control multi-center efficacy trial among homosexual men. *Ann Intern Med*, 97:362-366, 1982.
46. Maynard, J.E.: Prevention of hepatitis B through the use of vaccine. *Ann Intern Med*, 97:442-444, 1982.
47. Coutinho, R.A.; Lelie, N.; Albrecht-van Lent, P. *et al*: Efficacy of a heat inactivated hepatitis B vaccine in male homosexuals: outcome of a placebo controlled double blind trial. *Br Med J Clin Res*, 286:1305-1308, 1983.
48. Levin, M.L.; Maddrey, W.C.; Wands, J.R.: Hepatitis B transmission by dentists. *J Am Med Assoc*, 228:1139-1140, 1974.
49. Vogel, C.: Hepatitisbetragung durch Zahnärzte und ihre forensischen Folgen. *Med Welt*, 29:1978-1979, 1978.
50. Hadler, S.C.; Sorley, D.L.; Acrel, K.H. *et al*: An outbreak of hepatitis B in a dental practice. *Ann Intern Med*, 95:133-138, 1981.
51. Centers for Disease Control: Immune globulins for protection against viral hepatitis. *MMWR*, 52:425-428, 441-442, 1977.

Laurel M. Mellin, MA, RD

The recent national concern about adolescent obesity, including a National Institutes of Health workshop, congressional hearings and media attention, stems from a variety of origins.¹ First, it emanates from the recent recognition that pediatric obesity has increased dramatically in the last fifteen to twenty years. Gortmaker, Dietz and colleagues analyzed nationally representative data and found that obesity in adolescents increased 39 percent and affected 21.9 percent of twelve- to seventeen-year-old children.² Second, since obesity in adults is refractory and intractable, prevention appears prudent. Third, obesity in adolescence has been associated with greater risk of persistence than obesity in younger children.

The clinical determination of obesity in adolescents is arbitrary; however, accepted definitions are a relative weight of 120 percent or more (i. e., 20 percent above the mean weight for height, age and sex) and a triceps skinfold percentile above the 85th.³ Obesity in adolescence is distinct from obesity in other life stages in that the psychosocial and biological changes heralded by puberty profoundly influence obesity care. For instance, the degree to which the adolescent has separated from the family directly influences the extent to which parents should be involved in the adolescent's treatment.

CAUSES

The etiology of adolescent obesity is diverse and multifactorial. Although the initial weight gain may be caused by a singular factor, the increase in adiposity itself typically induces other changes that contribute further to weight gain. The adolescent is often caught in a downward spiral of inactivity, overeating, social isolation, depression, low self-esteem and weight gain.

Genetic factors appear to influence body weight, with recent data from analysis of adoptees by Stunkard and colleagues supporting the role of genetics in human obesity.⁴ Other data as summarized by Garn reveal the variability of weight throughout the life cycle, supporting the role of environment.⁵ Although the precise role of genetics is not well understood, it is believed that genetic factors are important and yet environmental factors serve an important role in mediating body weight.

Organic conditions cause obesity in fewer than 2 percent of cases. These rare conditions, such as Prader-Willi syndrome and Turner's syndrome, are associated with short stature or growth problems.⁶ Certain medications, such as steroids and insulin, can contribute to obesity in young people. Often medical problems, particularly those that influence physical activity, such as asthma and orthopedic problems, cause adolescent weight gain.

Although the roles of genetics and family behavioral patterns in the etiology of obesity have long been appreciated, the contribution of the family system has recently received recognition. For instance, Harkaway describes the functions of the symptom of excessive adiposity in the family.⁷ It can delay the adolescent's separation and emancipation and thus maintain the family, serve as a method of getting parental attention, especially when the parent is depressed or suffers a loss, provide a distraction from more serious or painful family problems or convey an affirmation of family loyalty or membership in an obese family.

Little is known about the role of psychological variables in the onset or maintenance of adolescent obesity.⁸ Although some data suggest that obese adolescents may have a more negative self-concept and lower self-esteem and may be more depressed, these differences may be consequences rather than causes of the obesity.^{9,10}

Contrary to popular belief, obese adolescents do not appear to consume more calories than their non-obese peers.¹¹⁻¹³ The primary distinction between the eating behaviors of the obese and non-obese is the frequency of eating, with the obese more likely to skip meals and to

eat less frequently.¹⁴ It is unclear whether this distinction is a cause or consequence of the obesity.

The obese adolescent appears to be significantly less physically active and less physically fit than the normal-weight adolescent.^{15,16} This may be related to the poor and declining physical fitness of the nation's youth.¹⁷ Television viewing, a passive, sedentary activity has been linked to increased fatness and decreased fitness.^{18,19}

DISADVANTAGES

The disadvantages of adolescent obesity are often severe.²⁰ From a health perspective, the major risk is persistence into adulthood. As many as 70 percent of obese adolescents will become obese adults.⁵ Adolescence appears to be a pivotal time in determining adult fatness. Even if the obese youth normalizes weight during adulthood, having gone through adolescence in the obese state may increase risk of hypertension. In addition, growth and development are affected by obesity as obese adolescents enter puberty earlier and have a shorter period of long bone growth.^{21,22} This results in an adult stature less than the individual's genetic potential. The immediate health risks of adolescent obesity include increased risk of hyperinsulemia, hypertension, hyperlipidemia, and respiratory, orthopedic and menstrual problems.¹⁹

The psychosocial disadvantages of adolescent obesity are often severe. These children may be delayed in their psychosocial development, perhaps a consequence of peer discrimination or a family system that discourages emancipation. Obese adolescents are more frequently discriminated against by peers, parents, teachers and employers.¹⁵ This prejudicial treatment can contribute to a more negative self-concept and body-image disparagement.

TREATMENT

Until the last five years, treatment for adolescent obesity has demonstrated poor effectiveness, high recidivism and excessive drop-out rates.²³ Virtually none of the approaches evaluated demonstrated long-term effectiveness. Due to the increased prevalence of adolescent obesity and the heightened interest in the prevention of adult obesity, a broader range of treatment options has recently emerged. Effectiveness varies considerably; some of these approaches, however, have demonstrated more positive outcomes.

High-risk treatments

Very low-calorie diets for treating adolescent obesity have proved ineffective and hazardous, by significantly decreasing lean body mass and growth velocity.^{24,25} Surgical approaches have shown significant mortality, morbidity and recidivism.²⁶

Commercial programs

Summer camps and commercial diet programs offer obesity interventions to youth. Camps may be costly and stress excessive weight loss. Commercial diet programs typically focus on nutrition exclusively and recommend rigid or overly restrictive diets. The primary disadvantages of commercial programs are that they include no significant family intervention or follow-up care, focus excessively on diet and exercise, may be nutritionally inadequate to support normal growth and development, and involve lay staff members who may be insufficiently trained in the biopsychosocial aspects of adolescent obesity. Recidivism and dropout rates with these approaches are excessive.

Comprehensive low-risk programs

Although low-risk programs in general continue to report poor outcomes, highly developed comprehensive weight-management programs conducted by health professionals have shown impressive short- and long-term effectiveness.^{27,28} These interventions promote long-term changes in weight, blood pressure, diet and exercise behaviors, self-esteem and knowledge. They share these characteristics:

Comprehensive assessment

Because of the diversity of causes and consequences of adolescent obesity, the obesity needs to be comprehensively assessed prior to treatment.²⁹ Biological, behavioral, psychological and family interactional contributors are identified and the possibility of organic obesity should be assessed. The interventions are capable of being individualized to meet the needs of the family and adolescent.

Nutritionally protective

During this sensitive period of growth and development, the nutritional recommendations are consistent with an adequate diet for age and sex, based on the Recommended Dietary Allowances and a preventive diet according to the U.S. Dietary Guidelines. The nutritional component does not include severe food restrictions or avoidances. Weight loss averages no more

than one to two pounds per week and is related to development. Less weight loss is advised during periods of rapid growth and development.

Physical activity

Because of the beneficial effect of exercise on weight loss, health and mood, physical activity is a core component of good care. Physical activities that improve endurance, flexibility and strength are included in the adolescent's comprehensive exercise program. Activities that have the potential to be continued in adulthood are emphasized.

Extensive family intervention

Extensive, separate parental sessions have been shown to be critical to the long-term success of adolescent obesity interventions.²⁸ Sessions are conducted with the support of a mental health professional and focus on family changes in diet, exercise, communication, interaction, and attitudes.

Psychologically beneficial

These approaches enhance self-esteem and improve body image. Techniques that are potentially psychologically distressing, such as aversion therapy and rigid or restrictive diets, are avoided.

Coordinated with health care

The adolescent obesity intervention is coordinated with medical care. The physician assesses the adolescent's health prior to the intervention and monitors health indices during treatment. Other health professionals are available to the adolescent and family, including a mental health professional, registered dietitian, and licensed exercise physiologist.

Long-term, follow-up care

The maintenance of changes in life-style, diet and exercise often require continued support for an extended period. Supportive counseling should be available to the adolescent and family for at least one year after the intervention.

SUMMARY

The prevalence of adolescent obesity is increasing at an alarming rate. Obesity during adolescence is likely to persist into adulthood. The etiology and consequences of adolescent obesity are diverse, necessitating a biopsychosocial assessment and a comprehensive, individualized approach to treatment by a team of health professionals.

REFERENCES

1. Science, 232:20-21, 1986.
2. Dietz, W.H. and Gordon, J.E.: Obesity in infants, children and adolescents in the United States. *Nutr Res*, 1:193, 1981.
3. Johnson, R.E.: Validity of triceps skinfold and relative weight as measures of adolescent obesity. *J Adolescent Health Care*, 6:185, 1985.
4. Stunkard, A.H.; Sorensen, T.I.A.; Hanis, C. *et al*: An adoption study of human obesity. *N Engl J Med*, 314:193-198, 1986.
5. Garn, S.M.: Continuities and changes in fatness from infancy through adulthood. *Current Problems in Pediatrics*, 15:1-47, 1985.
6. Malloy, M.J. and Kane, J.P.: Obesity. In: *Basic and Clinical Endocrinology*. Edited by F. Greenspan and P.H. Forsham. Los Altos, CA: Lange Medical Publications, 1983.
7. Harkaway, J.E.: Family intervention in the treatment of childhood and adolescent obesity. In: *Eating disorders. The family therapy collections*. Edited by J.E. Harkaway, Vol. 20. Rockville, MD: Aspen Publishers, Inc., 1987.
8. Wadden, T.A. and Stunkard, A.J.: Social and psychological consequences of obesity. *Annals of Inter Med*, 103:1062-1067, 1985.
9. Sallade, J.A.: Comparison of the psychological adjustment of obese vs non-obese children. *J Psychosomatic Res*, 17:89-96, 1973.
10. Werkman, S.L. and Greenberg, E.S.: Personality and interest patterns in obese adolescent girls. *Psychosomatic Med*, 29:72-80, 1967.
11. Rolland-Cachera, M.F. and Bellisele, F.: No correlation between adiposity and food intake: Why are working class children fatter? *Am J Clin Nutr*, 44:779-787, 1986.
12. Hampton, M.C.; Hueneman, R.L.; Shapiro, L.R. *et al*: Caloric and nutrient intakes of teenagers. *J Am Dietetic Assoc*, 50:385-396, 1987.
13. Johnson, M.L.; Burke, B.S.; Mayer, J.: Relative importance of inactivity and overeating in energy balance of obese high school girls. *Am J Clin Nutr*, 44:779-787, 1986.
14. Kaufman, N.A.; Pozanski, R.; Guggenheim, K.: Eating habits and opinions of teenagers on nutrition and obesity. *J Am Diet Assoc*, 66:264-268, 1975.
15. Waxman, M. and Stunkard, A.J.: Caloric intake and expenditure of obese boys. *J Pediatr*, 96:187-193, 1980.
16. Bullen, B.A.; Reed, R.B.; Mayer, J.: Physical activity of obese and non-obese adolescent girls appraised by motion picture sampling. *Am J Clin Nutr*, 14:218-221, 1964.
17. Summary of Findings from the National Children and Youth Fitness Study. *J Phys Ed Rec Dance*, 1:43, 1985.
18. Dietz, W.H.: Do we fatten our children at the television set? Obesity and television viewing in children and adolescents. *Pediatrics*, 75:807, 1985.
19. Tucker, L.A.: The relationship of television viewing to physical fitness and obesity. *Adolescence*, 21:797-806, 1986.
20. Dietz, W.H.: Obesity in infants, children and adolescents in the United States. 1. Identification, natural history and aftereffects. *Nutr Res*, 1:117-134, 1981.
21. *Control of the Onset of Puberty*. Edited by M.M. Grunback, D. Grave and F.F. Mayer. New York: John Wiley & Sons, 1974.
22. *Growth at Adolescence*. Edited by J.M. Tanner (2nd edition), Lippincott, 1962.
23. Coates, T.J.: Treating obesity in children and adolescents: a review. *Am J Pub Health*, 68:144, 1981.
24. Archibald, E.H.; Harrison, J.E.; Pencharz, P.B.: Effect of weight-reducing high-protein diet on body composition of obese adolescents. *Am J Dis Child*, 137:658, 1983.
25. Dietz, W.H. and Hartung, R.: Changes in height velocity of obese preadolescents during weight reduction. *Am J Dis Child*, 139:705, 1985.
26. Andersen, A.E.; Soper, R.T.; Scott, D.H.: Gastric bypass for morbid obesity in children and adolescents. *J Pediatr Surg*, 15:876, 1980.
27. Mellin, L.M.; Slinkard, L.A.; Irwin, C.E.: Validation of the SHAPEDOWN adolescent obesity intervention. *J Am Diet Assoc*, 87:333, 1987.
28. Brownell, K.D.: Treatment of obese children with and without their mothers: changes in weight and blood pressure. *Pediatrics*, 71:515, 1983.
29. Mellin, L.M.: Youth evaluation scale: a biopsychosocial assessment instrument for adolescent weight problems. Larkspur, CA: Balboa Publishing, 1986.

Reprinted from *Contemporary Nutrition* by permission of General Mills Nutrition Department.

THE FUTURE

In the past ten years, significant changes in the style of dental practice have occurred. Fluoridation of water supplies, the increase in number of dental professionals, and dental health education have all had an impact on the incidence of caries. New emphasis is placed on "cosmetic dentistry" and the biotechnology of dental implants. The practice setting is more varied and advertising is now accepted as a normal part of many dental practices. An increasing number of women are entering the profession, and joining the faculties of dental schools. It will be interesting to observe how their presence affects the educational process and the practice of dentistry.

As the turn of the century approaches, dental professionals will be challenged by new events, new research, new discoveries. They will have the opportunity, by being better informed and better educated, not only to respond to trends, but to set them.

Jong, A.W.: *Community Dental Health*,
St. Louis: The C.V. Mosby Company, 1988, p 37.

Emergency care in pediatric dentistry

MaryAnn Ready Battenhouse, DMD

M.M. Nazif, DDS, MDS

T. Zullo, PhD

Emergency care is an essential component of dental services. Improved prognosis is often linked to availability and quality of treatment. Numerous surveys have been published on traumatic injuries to teeth, dental caries, and other pathological entities. Yet, other vital information such as seasonal variations, time of day, and geographic factors are still lacking.

The objective of this study is to investigate the incidence and classification of all pediatric dental emergencies presenting to the emergency room at Children's Hospital of Pittsburgh, during an entire calendar year.

METHODS AND MATERIALS

The emergency room records of 1,456 children who presented to Children's Hospital of Pittsburgh with any oral complaint during a single calendar year were reviewed. The following data were collected: the date and time of visit, age and gender of patient, diagnostic classification and its anatomical involvement, and etiology. All data were analyzed on the University of Pittsburgh's DEC 10 System using the SPSS Statistical Program.

Patients ranged in age from a month to eighteen

Dr. Battenhouse is Assistant Professor, Department of Pediatric Dentistry, Medical College of Georgia; Dr. Nazif is Chief of Dental Services, Children's Hospital of Pittsburgh; Dr. Zullo is Professor, Department of Learning Resources, School of Dental Medicine, University of Pittsburgh.

Table 1 □ Frequency of emergency visits by gender and etiology.

Etiology	Gender		Totals
	Male	Female	
Trauma	440	236	676
Nontrauma	396	384	780
Totals	836	620	1456

$\chi^2 = 30.37$
 $p < .001$

Table 2 □ Frequency of emergency visits by age and etiology.

Etiology	Age				Totals
	≤3	4-6	7-12	13-18	
Trauma	325	106	192	53	676
Nontrauma	97	185	336	162	780
Totals	422	291	528	215	1456

$\chi^2 = 233.57$
 $p < .001$

Table 3 □ Frequency of emergency visits by season and etiology.

Etiology	Season				Total
	Winter	Spring	Summer	Fall	
Trauma	127	176	206	167	676
Nontrauma	179	195	205	199	778
Total	306	371	411	366	1454

$\chi^2 = 5.48$
 $p < .139$

Table 4 □ Frequency and percent of trauma diagnoses.

	Frequency	Percent
Lacerations and abrasions	638	58
Dental		
Displacements and subluxations	229	21
Fractures	156	14
Bony or TMJ	30	3
Other		
Burns, bites, foreign bodies	29	3
Complications	11	1
Total	1,093	100

years. Patients with more than one emergency complaint received additional diagnostic entries as needed.

Trauma-related diagnostic categories included: abrasions and lacerations, dental fractures, dental displacement and subluxation, bone fractures and TMJ injuries, complications secondary to treatment, and other injuries that included bites, burns, and foreign body related accidents.

Nontrauma diagnostic categories included: dental caries without abscess formation, dental caries with abscess formation, eruption-related complications, dental anomalies, inflammatory changes of nondental origin, neoplastic lesions, and orthodontic or prosthodontic appliance-related complications.

RESULTS

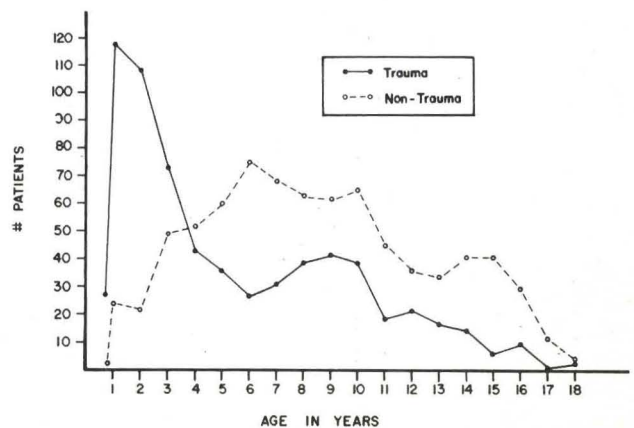
Trauma was responsible for 676 (46 percent) of the 1,456 recorded emergency visits. Table 1 depicts the relationship between gender and nature of the emergency visit. The percentage of trauma visits for males (53 percent) was significantly greater than the percentage of trauma visits for females (37 percent) at the 0.001 level of significance.

To examine the relationship between age and nature of the emergency (trauma vs nontrauma), four age-cate-

gories were established: birth to three years; four to six years; seven to twelve years; and thirteen to eighteen years of age. The Chi Square Test revealed a statistically significant relationship between age and nature of the emergency ($\chi^2 = 233.57$; $p < 0.001$). The number of trauma emergencies was significantly higher for the youngest age-group than for any of the others (Table 2, Figure).

No statistically significant relationship ($\chi^2 = 5.48$; $p < 0.139$) (Table 3) was found between seasons (winter,

Figure. Graphic illustration of the frequency of trauma and nontrauma cases in relationship to age.



spring, summer, fall) and nature of the emergency.

The 676 trauma-related visits yielded a total of 1,093 trauma diagnoses. Table 4 summarizes the distribution of the various diagnoses encountered. Lacerations and abrasions represented the most frequently observed trauma diagnosis (58 percent); while dental trauma, which included displacement and subluxation, and crown and root fractures, accounted for 385 (35 percent) of the trauma diagnoses. Bony fractures and TMJ injuries accounted for only 3 percent.

Table 5 presents a breakdown of the laceration diagnoses according to anatomical sites. The most common lacerations were those of the lip (39 percent).

Etiology of trauma was attributed primarily to falls (62 percent), while athletic activities contributed to only 20 percent. Fighting or other violent acts accounted for the remaining 18 percent.

Nontraumatic events were responsible for 830 of the total emergency diagnoses. The most commonly encountered nontraumatic diagnosis was dental caries with abscess formation (45 percent), followed by eruption-related complications and dental anomalies (24 percent) (Table 6).

Time of emergency visits was recorded as either day (7:30 AM - 4:30 PM), evening (4:30 PM - 11:30 PM) or night (11:30 PM - 7:30 AM). The majority (65 percent) of emergency visits occurred during the day, while only 1.5 percent occurred between 11:30 PM and 7:30 AM (Table 7).

DISCUSSION

The findings of this survey are in general agreement with available literature, in regard to the higher incidence of traumatic injuries between birth and three years of age.¹⁻³ This significant trend is frequently due to falls that occur when infants learn to walk, their increased mobility and activity, and limited coordination. It could also be to some extent, a reflection of heightened anxiety of parents of younger children after minor traumata, which may not be the case with older children. Another area of agreement with existing literature is the higher incidence of traumatic injuries among males and the higher incidence of soft tissue injuries in conjunction with dental injuries.^{1,4}

The etiology of traumatic injuries was mostly due to falls in the youngest age-groups, while older children's injuries were secondary to athletic activities or fighting. This is in agreement with the work of Magnusson and others.³⁻⁶

Table 5 □ Frequency and percent of laceration sites.

Site	Number	Percent
Lip	175	39
Extraoral	82	18
Oral mucosa (including frenum tears)	81	18
Gingiva (including deglovements)	62	14
Tongue	45	10
Total	445	100

Table 6 □ Frequency and percent of nontrauma diagnoses.

	Number	Percent
Caries with abscess	373	45
Eruption-related complications and dental anomalies	199	24
Caries without abscess	135	16
Morbidity-inflammatory or neoplastic	96	12
Orthodontic, prosthodontic	27	3
Total	830	100

Table 7 □ Frequency and percent for time of emergency room visit.

Hour of visit	Number	Percent
7:30 A.M. - 4:29 P.M.	922	65
4:30 P.M. - 11:29 P.M.	481	34
11:30 P.M. - 7:29 A.M.	21	1
Total	1,424	100

This survey shows a trend for injuries to occur more frequently during the summer season, although not statistically significant. This finding is contrary to the work of Meadow *et al* who reported the highest peak during early fall.⁷

Child abuse was not suspected in any of the patients examined in this survey; suspicious injuries, such as bites or burns should be carefully investigated, however, and appropriately documented and reported.

Repeated emergency visits occurred in 5 percent of the total sample. This may be an indication of parental neglect or the child's needs for regular dental care. Appropriate counseling and encouragement should be carefully attempted. Failure to achieve positive results may necessitate the involvement of appropriate social agencies.

This survey illustrates the dental emergency needs of a community the size of Southwestern Pennsylvania, where the population is approximately two million. The area's predominant terrain is rolling hills. The climate of the region is generally temperate with mild spring and fall, snowy winter, and a hot summer.

The availability of emergency dental services around the clock in a central location is an excellent service to any large community. Area physicians and dentists benefit from reliable backup coverage during evening hours, weekends, and holidays, and the sponsoring institution receives a steady flow of new patients, who may have multiple health care needs. The positive public relation image created by a well operated emergency service has an enormously favorable impact on the viability and success of the hospital dental services.

The results of this survey illustrate the continued need for a national dental trauma prevention program emphasizing improved safety features in children's bicycles and other athletic equipment as well as increased utilization of currently available head and mouth protection.

Finally, it is evident from the high incidence of soft tissue injuries that postgraduate programs in pediatric dentistry should place increased emphasis on the surgical care of such injuries, which complements the traditional emphasis on the care of dental injuries.

CONCLUSIONS

The following conclusions can be drawn from the data presented in this paper:

- Trauma cases contributed 46 percent of the total emergency visits.
- Males represented a significantly greater incidence of trauma visits than females.

- The youngest age-group (birth to three years) had a significantly higher percentage (77 percent) of traumatic injuries than other age- groups.
- Soft tissue injuries represented a substantially higher percentage of trauma cases (58 percent) as compared to other types of injuries.
- There was no significant relationship between seasonal change and the nature of emergency visits.
- Approximately a third of all emergency visits occurred during evening hours.

REFERENCES

1. Andreasen, J.O.: Etiology and pathogenesis of traumatic dental injuries. A clinical study of 1,298 cases. *Scand J Dent Res*, 78:329-342, 1970.
2. Andreasen, J.O. and Ravn, J.J.: Epidemiology of traumatic dental injuries to primary and permanent teeth in a Danish population sample. *Int J Oral Surg*, 1:235-239, 1972.
3. Johnson, J.E.: Causes of accidental injuries to the teeth and jaws. *J Pub Hlth Dent*, 35:123-131, Spring, 1975.
4. Hedegard, B. and Stalhane, I.: A study of traumatized permanent teeth in children aged 7-15 years. Part I. *Swed Dent J*, 66:431-450, 1973.
5. Magnusson, B. and Holm, A.: Traumatized permanent teeth in children - a follow-up. I. Pulpal complications and root resorption. *Svensk Tandlak*, 62:61-70, 1969.
6. O'Mullane, D.M.: Some factors predisposing to injuries of permanent incisors in school children. *Brit Dent J*, 134:328-332, April, 1973.
7. Meadow, D.; Linder, G.; and Needleman, H.: Oral trauma in children. *Pediat Dent*, 6:248-251, December, 1984.

Presented at the American Association for Dental Research Meeting, Washington, D.C., 1986.

CONGENITAL MALFORMATIONS IN DIABETES

It is now well established that insulin-requiring diabetes during organogenesis doubles or triples a woman's risk for producing a malformed infant. It appears that women with metabolic abnormalities of some type during organogenesis are the population at risk. Reports of malformations linked to gestational diabetes (where carbohydrate tolerance during organogenesis is normal) are of great interest. If they can be verified, additional investigation of the role of genetic factors would be appropriate.

Many organ systems are affected by diabetes. High rates of cardiac malformations, neural tube defects, and renal malformations are noted. In addition, some rare defects such as sacral dysgenesis and holoprosencephaly are found. It is difficult to identify the teratogenic agent in diabetes because most women are not under observation at the appropriate time. Retrospective data on glycosylated hemoglobin show an association between poorly controlled diabetes and increased malformation rates.

Mills, J.L.: Congenital malformations in diabetes, In *Infant of the Diabetic Mother*. Columbus, Ohio: Ross Laboratories, 1987, p 18.

Case reports

Oral manifestations of Crohn disease: update of the literature and report of case

Robert A. Boraz, DDS

Crohn disease is a segmental transmural intestinal disease that may involve one or more segments of the gut from the mouth to the anus.^{1,2} The distal ileum and the colon are the most commonly affected areas.^{2,3} The inflammatory process consists of noncaseating granulomas with regional lymphatic involvement.^{1,2} Early in the course of the disease, biopsied tissue may fail to reveal granulomas and may be identical to the mucosa of a child with ulcerative colitis.¹ An increase in incidence of Crohn disease occurred in Western Europe and North America in the last decade, but has now plateaued.⁴ The cause of Crohn disease is unknown, but the disease is commonly found among Jews, first degree relatives of patients, and patients with ankylosing spondylitis.^{1,4,5} Approximately 25 percent of the cases begin before the age of twenty years. Onset in infancy is extremely rare.^{1,2}

The initial phase of the Crohn disease begins with subtle clinical manifestations. Frequently, many months will pass between the first symptoms and the establishment of a correct diagnosis.¹ Abdominal pain associated with cramps is the most common initial complaint, followed by diarrhea. Other common early complaints include nonintestinal problems such as fever, anorexia, growth failure, general malaise, and joint pain.^{1-3,5-7} Any teenager with chronic malaise or persisting growth problems, particularly with a fever, should be suspected

Dr. Boraz is Associate Professor of Surgery and Pediatrics; Diplomate, American Board of Pediatric Dentistry; Director, Dental Service, University of Kansas Medical Center, 39th and Rainbow Boulevard, Kansas City, KS 66103.

a victim of this disease.^{1,8} Chronic perianal lesions may be another sign.⁹ The abdominal pain associated with Crohn disease is often periumbilical or in the right lower quadrant rather than confined to the lower abdomen as in ulcerative colitis.¹ Blood is seen in stools less frequently than in ulcerative colitis, but bleeding can be massive.^{1,9} The arthritis associated with the disease is frequently found in large joints and nearly 20 percent of Crohn patients develop this problem.^{1,2,10} Erythema nodosum, iritis, hepatitis, and phlebitis occur, but are rare manifestations.¹

The diagnosis of Crohn disease is based on clinical and laboratory findings. The clinical symptomatology indicates the presence of an inflammatory bowel disease. The laboratory results indicating active disease include an elevated erythrocyte sedimentation rate, mildly depressed hemoglobin levels, and, frequently, reduced serum albumin levels.^{1,11,12} If the disease is suspected, barium contrast roentgenograms of the small and large bowel are used to define the segments involved. Crohn involvement is frequently characterized by irregular mucosa or a cobblestone-like pattern, thickened bowel, and enteric fistulae; but it is the segmented distribution of the lesions that is diagnostic.¹ Biopsies of the rectal or oral mucosa that reveal granulomas are also diagnostic.^{1,10}

Curative medical therapy for Crohn disease is not available. Operative resection is not usually feasible and is not curative. Prednisone is indicated for acute exacerbations. The usual course of prednisone is 1-2 mg/kg/24 hours for six weeks, with gradual tapering of the dose over another four to eight weeks.¹ The concomitant use of azathioprine in difficult cases reduced the use of the steroid.¹³⁻¹⁵ Surgery is indicated when the disease leads to massive hemorrhage, intestinal perforation, or persistent bowel obstruction. Since medications are palliative, family support measures are very important in dealing with the disease.

The prognosis for Crohn disease is that the inflammatory activity will remit and exacerbate through life without a consistent pattern. The involved areas of the body frequently remain unchanged. The incidence of intestinal cancer is increased in Crohn patients, but not as much as in ulcerative colitis.^{1,16}

ORAL MANIFESTATIONS

Included are:

- Ulceration, sometimes aphthous, but more characteristically persistent, deep, and surrounded by hyperplastic margins.

- Diffuse labial, buccal, or gingival swelling.
- Focal areas of mucosal inflammatory hyperplasia and fissuring (cobblestoning).
- Indurated polypoid "tag" lesions on vestibular and retromolar mucosa.
- Indurated fissuring on the midline of the lower lip.
- Cheilitis, granulomatous or angular.
- Pyostomatitis vegetans.^{10,17,25}

Because of the significant oral manifestations and the difficulty in absolute diagnosis of the disease, a thorough understanding of Crohn disease by the dental practitioner is most important.

CASE REPORT

The patient, a Caucasian female, weighed seven pounds, nine ounces at birth. The only complication during pregnancy was a maternal urinary tract infection, two weeks prior to delivery. The patient has two healthy, older siblings, one male and one female. The family history included two spontaneous abortions by the mother and an affliction of cancer of the stomach in the paternal grandfather. The patient was diagnosed to have bilateral retinoblastomas, and underwent bilateral enucleation procedures at age fourteen months. The surgery was followed with chemotherapy, utilizing vincristine and cytosin and low dosage radiation therapy. At age six years, she underwent a tonsillectomy; and at age eight years, a Hickman catheter was placed for hyperalimentation. The patient was first evaluated at the University of Kansas Medical Center for Crohn disease at age eight years, four months. She was complaining of an intermittent, low grade fever of three months duration; and of a constant, dull abdominal pain, for two months. A double-contrast, small bowel radiographic series revealed nodularity of the terminal ileum. Laboratory results included a significant increase in the erythrocyte sedimentation rate and a decreased hemoglobin level. She was dismissed from the hospital with a diagnosis of terminal ileitis and possible early Crohn disease. She was placed on 15 mg of prednisone, three times daily. The patient returned to the hospital two months later with continued fevers, and increasing abdominal pain. She was experiencing decreased activity and tired easily. She was started on a lactose-free, low-fiber diet; and 1/4 grain of codeine, every six hours as necessary, was added to the medication regimen. The status of the patient continued to fluctuate.

The patient was first seen in the Dental Clinic at the University of Kansas Medical Center at age eight years, ten months. She was referred by the pediatric gastroen-

terologist for evaluation of an oral ulceration. The physician strongly suspected the patient had Crohn disease and was seeking a biopsy for a definitive diagnosis. Visual examination revealed a small, undernourished female. The patient was blind as a result of her retinoblastomas. She was cheerful, but apprehensive. She demonstrated a normal, well-developed mixed dentition. The occlusion was adequate. The patient had an aphthous-like oral ulceration, approximately 7 mm in diameter on the right maxillary buccal mucosa, near the vestibule. The lesion had been present for approximately three weeks. No other abnormalities of the soft or hard structures of the orofacial region were noted.

An excisional biopsy of the lesion was performed under local anesthesia. Two 3-0 silk sutures were utilized to facilitate wound healing by primary intention. The biopsied material was placed in 10 percent formalin and sent to the pathology laboratory for evaluation. The pathology report found marked inflammation with numerous histiocytes. Several possible granulomas were noted. The pathologist reported the appearance of the biopsied material to be consistent with the findings of Crohn disease.

DISCUSSION

Crohn disease is a devastating, chronic illness that often develops in the childhood years. Frequently, oral manifestations are present and may be diagnostic of the disease. Many patients with Crohn disease are misdiagnosed. Although no cure is currently available, the proper diagnosis is essential for obtaining symptomatic relief and learning to live with the problem. Because the disease frequently begins in childhood with oral signs and symptoms, it is important for the dentist to be knowledgeable of the problem.

REFERENCES

- Behrman, R. E. and Vaughan, V. C.: Nelson textbook of pediatrics. Philadelphia: W. B. Saunders, 1983, pp 923-925, 979-980.
- Crohn, B. B.; Ginzburg, L.; Oppenheimer, G. D.: Regional ileitis: a pathologic and clinical entity. *JAMA*, 99:1323, October, 1932.
- Potsuma, R. and Moroz, S. P.: Pediatric Crohn's disease. *J Pediatr Surg*, 20:478-482, October, 1985.
- Sandler, R. S. and Golden, A. L.: Epidemiology of Crohn's disease. *J Clin Gastroenterol*, 8:160-165, April, 1986.
- Passo, M. H.; Fitzgerald, J. F.; Brandy, K. D.: Arthritis associated with inflammatory bowel disease in children. Relationship of joint disease to activity and severity of bowel lesion. *Dig Dis Sci*, 31:492-497, May, 1986.
- Wright, R.: Crohn's disease: diagnosis and management. *Compr Ther*, 11:38-44, April, 1985.
- Gee, M. I.; Wensel, R. H.; Thomson, A. B. R.: Nutritional status of gastroenterology outpatients: comparison of inflammatory bowel disease with functional disorders. *J Am Diet Assoc*, 85:1591-1599, December, 1985.
- Mock, D. M.: Growth retardation in chronic inflammatory bowel disease. *Gastroenterology*, 91:1019-1021, October, 1986.
- Ward, C. S.; Dunphy, E. P.; Jagoe, W. S.: Crohn's disease limited to the mouth and anus. *J Clin Gastroenterol*, 7:516-521, December, 1985.
- Frankel, D. H.; Mostofic, R. S.; Lorincz, A. L.: Oral Crohn's disease: report of two cases in brothers with metallic dysgeusia and a review of the literature. *J Am Acad Dermatol*, 12:260-268, February, 1985.
- MacFarlane, P. I.: Laboratory assessment of disease activity in childhood Crohn's disease and ulcerative colitis. *J Pediatr Gastroenterol Nutr*, 5:93-96, January, 1986.
- Brignola, C.; Lanfranchi, G. A.; Campieri, M.: Importance of laboratory parameters in the evaluation of Crohn's disease activity. *J Clin Gastroenterol*, 8:245-248, June, 1986.
- van Hogezaand, R. A.; van Hess, P. A.; Zwanenburg, B.: Disposition of disodium azodisalicylate in healthy subjects. A possible new drug for inflammatory bowel disease. *Gastroenterology*, 88:717-722, March, 1985.
- Peppercorn, M. A.: Current status of drug therapy for inflammatory bowel disease. *Compr Ther*, 11:14-19, December, 1985.
- Friedman, G.: Sulfasalazine and new analogues. *Am J Gastroenterol*, 81:141-144, February, 1986.
- Israel, K. J. and Nissenblatt, M. J.: Association of inflammatory bowel disease (IBD) with indolent soft-tissue sarcomas: report of two cases and review of the literature. *J Surg Oncol*, 32:125-130, June, 1986.
- Shafer, W. G.; Hine, M. Y.; Levy, B. M.: A textbook of oral pathology. Philadelphia: W. B. Saunders, 1983, pp 361, 362, 785.
- McCarthy, F. P.: Pyostomatitis vegetans: report of 3 cases. *Arch Dermatol Syph*, 60:750, June, 1949.
- McCarthy, F. P. and Shklar, G.: A syndrome of pyostomatitis vegetans and ulcerative colitis. *Arch Dermatol*, 88:913, December, 1963.
- Castaldo, E.; Covino, M. C.; Tesone, P. E.: Pyostomatitis vegetans. *Oral Surg*, 52:172, August, 1981.
- Bernstein, M. L. and McDonald, J. S.: Oral lesions in Crohn's disease: report of two cases and update of the literature. *Oral Surg*, 460:234, August, 1978.
- Bottomley, W. K.; Giorgini, G. L.; Julienne, C. H.: Oral extension of regional enteritis (Crohn's disease). *Oral Surg*, 34:47, July, 1972.
- Eisenbud, L.; Katzka, I.; Platt, J.: Oral manifestations in Crohn's disease. *Oral Surg*, 34:770, November, 1972.
- Estrin, H. M. and Hughes, R. W.: Oral manifestations in Crohn's disease: report of a case. *Am J Gastroenterol*, 80:352-354, May, 1985.
- Johnson, D. A.; Cattau, E. L.; Hancock, J. E.: Primary Crohn's disease of the oropharynx. *Ear, Nose, Throat J*, 64:534, 536, November, 1985.

Oligodontia in the primary dentition with permanent successors: report of case

Takashi Ooshima, DDS, PhD
Keiko Sugiyama, DDS
Shizuo Sobue, DDS, PhD

The congenital absence of teeth is usually referred to by one of two terms: according to Stewart, *oligodontia* is defined as the agenesis of numerous teeth (commonly associated with specific syndromes or severe systemic abnormalities); while *hypodontia* is used in cases where the absence of teeth is limited to one or a few.¹ Stewart defines the term *anodontia* as an extreme expression of oligodontia, and usually indicates the total absence of teeth.¹

Anodontia in the primary or permanent dentition is extremely rare and is commonly associated with other signs of ectodermal dysplasia.² On the other hand, hypodontia in the permanent dentition is so common that it is considered to be a variant of normal. The prevalence has been reported to be in the range of two to ten cases per 100, even excluding the third molars.³ Hypodontia in the primary dentition is uncommon, however, and the frequency ranges between 0.1 percent and 0.9 percent.⁴⁻¹¹ The prevalence of patients with oligodontia is much lower than the prevalence of patients with hypodontia. Hobkirk and Brook demonstrated that six or more congenitally missing teeth were found only in approximately one in fifteen hypodontic patients, giving a population prevalence of the order of 0.3 percent in the

permanent dentition, and being much less in the primary dentition.¹²

Epidemiological surveys demonstrated a high probability that the corresponding permanent successors would be congenitally missing when the primary teeth were missing. According to Grahnen and Granath, individuals with hypodontia in the primary dentition had the same condition in the permanent dentition, in 75 percent of the cases.⁶ Ravn also reported that patients with aplasia in the primary dentition showed aplasia of the succedaneous teeth, in a frequency of 80 percent.⁸ Furthermore, recent studies revealed similar findings; three of three patients, according to Jarvinen and Lehtinen; and sixteen of eighteen teeth according to Gellin.^{10,13} These studies concluded that oligodontia in the primary dentition, though the frequency was extremely low, nearly always showed aplasia of the succedaneous teeth. The purpose of this paper is to describe a child with oligodontia of the primary dentition who has the corresponding permanent teeth.

CASE REPORT

A two-year-old Japanese boy was brought to Osaka University Dental Hospital because he was missing several of his primary teeth. The child was examined with particular attention to hair, nails and skin, all of which appeared to be normal. The mother stated that the pregnancy was normal and she had not taken any special

The authors are with the Department of Pedodontics, Osaka University Faculty of Dentistry, 2-8, Yamada-Oka, Suita, Osaka 565, Japan.

medications. Her son had no history of illness and appeared to be physically normal, following a normal gestation and birth. Furthermore, the family history showed no record of hypodontia.

Examination showed eight primary anterior teeth (four central incisors, two right lateral incisors, the mandibular left lateral incisor and canine) to be missing (Figures 1,2). Also, the maxillary right canine and first molar, and the mandibular first molars appeared small and malformed (Figures 3,4). The maxillary second primary molars had not erupted. According to the mother, no teeth had been lost by trauma, exfoliation, or extraction.

Radiographic examination showed the permanent

tooth buds in the anterior regions of both arches. After the primary molars erupted completely, mesiodistal diameters of the teeth were determined from study models by measuring the most widely spaced points, using slider calipers held as parallel to the occlusal surface as possible. The findings by Ono were used for comparison (Table 1).¹⁴ The primary second molars, the mandibular right canine and maxillary left first molar were of normal size, shape and color. The maxillary left lateral incisor and canine, however, were somewhat smaller and tapering. The maxillary right canine and first molar, and the mandibular first molars also showed reduction in size and were malformed.

An orthopantomogram was obtained, when the pa-



Figure 1. Anterior view. Note eight anterior teeth are missing.

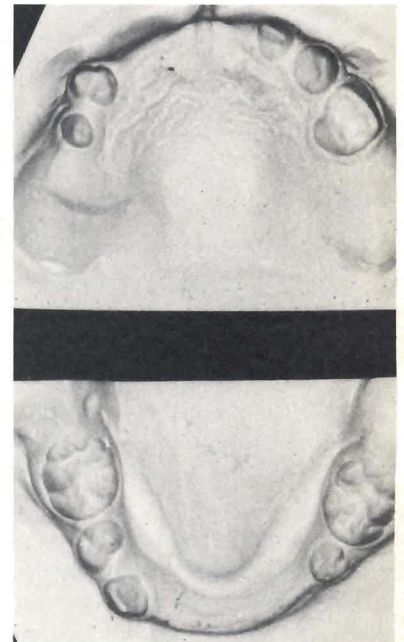


Figure 2. Note the malformed teeth and the number of teeth that are missing.



Figure 3. View of right maxillary region. Note the malformed canine and first molar consisting of a conical cusp. Second molar is normal in shape, size and color.



Figure 4. View of right mandibular region. Note the malformed first molar. Canine and second molar are normal.

Table 1 □ Mesiodistal crown diameters of primary teeth of the patient with oligodontia.

Primary tooth	Diameters of maxillary teeth			Diameters of mandibular teeth		
	Right	Left	Ono	Right	Left	Ono
A	*	-	6.6 mm**	-	-	4.3 mm
B	-	4.6 mm	5.5	-	-	4.8
C	4.5 mm	6.2	6.8	6.3 mm	-	6.0
D	4.1	7.9	7.3	4.8	5.1 mm	8.3
E	9.4	9.3	9.3	10.8	10.7	10.4

* congenitally missing tooth

** Readings are the mean mesiodistal diameters of Japanese children reported by Ono (196).¹⁴

Table 2 □ Prevalence of hypodontia and oligodontia in the primary dentition.

Sample	Age	Hypodontia Number	Hypodontia percent	Oligodontia	Country	Reference
572	2-7	3	0.5	0	Iceland	Magnusson ¹¹
1141	3-4	10	0.9	0	Finland	Jarvinen & Lehtinen ¹⁰
741	3-5	2	0.3	0	England	Brook ⁹
4564	3-3.5	25	0.5	0	Denmark	Ravn ⁸
609	2-7	1	0.2	0	Iceland	Moller ⁷
1173	3-5	5	0.4	0	Sweden	Grahnén & Granath ⁶
7589	3-7	15	0.2	0	Japan	Siato ³
2209	2-7	2	0.1	0	U.S.A.	Menczer ⁴

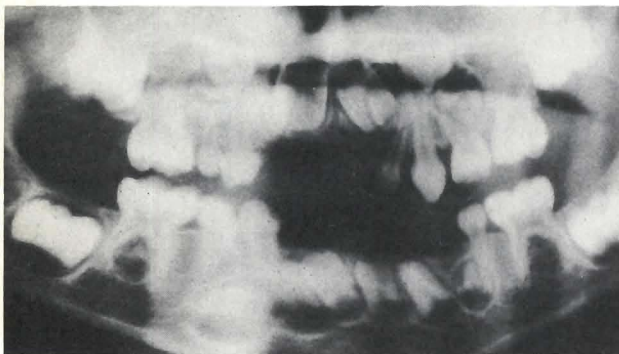


Figure 5. Orthopantomogram at the age of four years. Note that all the permanent buds are present.

tient was four years and ten months of age (Figure 5). At that time, all the permanent tooth germs, including the corresponding successors of the congenitally missing primary teeth were recognized.

Table 2 shows the frequency of hypodontia and oligodontia in the primary dentition, according to surveys made in several countries, during the past three decades.

REFERENCES

- Stewart, R.E.: The dentition and anomalies of tooth size, form, structure, and eruption. In *Pediatric Dentistry*. Eds. Stewart, R.E.; Barber, T.K.; Troutman, K.C. *et al.* St. Louis: The C.V. Mosby, 1982, p 91.
- Dixon, G.H. and Stewart, R.E.: Genetic aspects of anomalous tooth development. In *Oral facial genetics*. Eds. Stewart, R.E. and Prescott, G.H. St. Louis: The C.V. Mosby, 1976, pp 124-150.
- Silverman, N.E. and Ackerman, J.L.: Oligodontia: a study of its prevalence and variation in 4032 children. *J Dent Child*, 46: 470-477, November-December, 1979.
- Menczer, L.F.: Anomalies of the primary dentition. *J Dent Child*, 22:57-63, First Quarter, 1955.
- Saito, T.: A genetic study on the degenerative anomalies of deciduous teeth. *Jap J Hum Genet*, 4:27-53, No. 1, 1959.
- Grahnén, H. and Granath, L.: Numerical variations in primary dentition and their correlation with the permanent dentition. *Odont Revy*, 12:348-357, June, 1961.
- Moller, P.: Oral health survey of preschool children in Iceland. *Acta Odontol Scand*, 21:47-97, February, 1963.
- Ravn, J.J.: Aplasia, supernumerary teeth and fused teeth in the primary dentition. An epidemiologic study. *Scand J Dent Res*, 79:1-6, February, 1971.
- Brook, A.H.: Dental anomalies of number, form and size: their prevalence in British schoolchildren. *J Int Assoc Dent Child*, 5:37-53, December, 1974.
- Jarvinen, S. and Lehtinen, L.: Supernumerary and congenitally missing teeth in Finnish children. *Acta Odont Scand*, 39:83-86, No. 2, 1981.
- Magnusson, T.E.: Hypodontia, hyperdontia, and double formation of primary teeth in Iceland. *Acta Odontol Scand*, 42:137-139, June, 1984.
- Hobkirk, J.A. and Brook, A.H.: The management of patients with severe hypodontia. *J Oral Rehab*, 7:289-298, July, 1980.
- Gellin, M.E.: The distribution of anomalies of primary anterior teeth and their effect on the permanent successors. *Dent Clin N Am*, 28:69-80, No. 1, 1984.
- Ono, H.: Mesiodistal crown diameter of the primary and permanent teeth and the relationship between the dentitions. *J Jap Stomat Soc*, 27:221-234, October, 1960 (in Japanese).

We wish to thank Dr. Tong Sui Ming Lily for her helpful advice and for her translation to English.

ABSTRACTS

Pinkham, Jimmy R.; Casamassimo, Paul S.; Levy, Steven M.: Dentistry and the children of poverty. J Dent Child, 55:17-24, January-February, 1988.

Millions of American children are still being raised in poor environments, with no elimination of this problem foreseen. Poor children often have greater dental needs than do children from more fortunate social classes. This need for more dental services is aggravated substantially because the poor often have limited access to professional care, as well as financial obstacles to paying for such care. In addition, poor children, because of circumstances associated with the social consequences of poverty, particularly at younger age levels, often have problems meeting the behavioral expectations of dentistry, especially in terms of preventive practices. The dentist must be sympathetic to their needs and understand the challenges inherent in working with this group of children, which include: overcoming the children's dread of interacting with authority figures; improving communication techniques; understanding the children's emotional needs; and dealing appropriately with their fears.

Pediatric dentistry; Epidemiology; Poverty; Nutrition; Behavior management; Communication

Davila, Jorge M.; Gwinnett, A. John; Robles, Juan C.: Marginal adaptation of composite resins and dental bonding agents. J Dent Child, 55:25-28, January-February, 1988.

The purpose of this *in vitro* study was to determine the bonding qualities of three commercially available dentin bonding agents, namely Dentin Adhesit/Heliomolar, Creation Bond/Visar Fil and Scotchbond/P30. Microleakage tests and a scanning electron microscopy study of the interface between dentin and resin were performed. In eighteen specimens, the penetration of 0.5 percent basic fuchsin solution was studied across the enamel and dentin

interfaces with the resin. Half of the specimens in each group of products were thermocycled. Leakage was evaluated after sectioning under a light optical stereomicroscope at 20x magnification, using a classification reported by Eriksen and Buonocore. The interface between the restorative resin and the dentin axial walls of the preparation and its floor were examined by scanning electron microscopy (SEM). The results showed that leakage was commonly found at the resin/dentin interface and SEM showed gaps between the restoration and walls and floor of the preparation. It was concluded that the dentin bonding agents failed to provide a bond between restorative resin and dental tissue.

Restorative resin materials; Microleakage; Dentinal bridging; Bonding

Fisbein, Sergio; Holan, Gideon; Grajower, Rafael; Fuks, Anna: The effect of VLC Scotchbond and an incremental filling technique on leakage around class II composite restorations. J Dent Child, 55:29-33, January-February, 1988.

The object of this study was to determine the effect of VLC Scotchbond and of an incremental filling technique on microleakage around class II composite restorations *in vitro*. Four groups of 15-16 teeth each, were filled with the resin P-30 by one of the following techniques: A, Scotchbond and incremental filling; B, Scotchbond and bulk filling; C, Enamel Bond and incremental filling; D, Enamel Bond and bulk filling. The teeth were thermocycled, insulated up to 1 mm from the restorations, immersed in 2 percent basic fuchsin, embedded in acrylic resin and ground off to various depths, parallel to the plane through the vertical and the mesiodistal axes. Marginal leakage was assessed from the degree of dye penetration at the sections. Dye penetration at the occlusal margins was similar for all groups. Dye penetration at the cervical margins

for the different groups increased in the sequence: A/C/B/D. Incremental filling resulted in a significantly lower dye penetration at cervical margins for both bonding agents. The differences resulting from the use of Scotchbond were not significant, if the same packing technique had been used. Best results were obtained with Scotchbond and incremental filling.

Class II cavities; Scotchbond; Incremental filling; Microleakage; Composite resins

De Craene, G.P.; Martens, C.; Deraut, R.: The invasive pit-and-fissure sealing technique in pediatric dentistry: an SEM study of a preventive restoration. J Dent Child, 55:34-42, January-February, 1988.

From the dental literature, it is clear that the clinical application of pit-and-fissure sealants is a necessity in pedodontics. The choice between the non-invasive and the invasive techniques remains a matter of debate. In this SEM study, a comparison is made between different types of burs in order to obtain the best fissure preparation before the application of a sealant. The results of this study clearly show that, by applying the invasive sealing technique, the choice of an adequate bur is important to obtain a caries-free fissure, with a minimal loss of tooth substance. Two types of burs were found to perform best, according to the amount of tooth substance removed. Moreover, from a clinical point of view they were also found to be the most efficient ones. A detailed review is given concerning the choice of the actual preventive restorations for use in pedodontics.

Pit-and-fissure sealants; Restorations, preventive; Sealants; Caries

Fos, Peter J.: An alternative approach to prevention: computer-assisted patient education. J Dent Child, 55:43-46, January-February, 1988.

A computer-assisted instruction (CAI)

has been developed as an educational tool for patients' dental education. The health-care profession must educate their patients as to what knowledge is needed to ensure optimal overall health. In addition to traditional teaching methods, the use of computers is beginning to gain acceptance and has caused an impact on health-education techniques. Computers and CAIs can be used to augment and relieve the task portions of patient education, which are the responsibilities of the practitioner and his or her staff.

Computers; Dental education; Patient care

van Amerongen, William E. and de Graaff, Johannes: Hygiene in dental practice - Part I: Potential pathogens and possibilities of contamination. J Dent Child, 55:47-55, January-February, 1988.

Preventive efforts are focused on potentially pathogenic microorganisms in the area of health care. Among the most relevant for the dental practitioner are tuberculosis, gonorrhoea, syphilis, herpes simplex virus infections, viral hepatitis, hepatitis D, AIDS, Legionnaires' disease; type, number, and virulence are important risk factors. Measures to reduce risks are discussed in Part II.

Microorganisms; Transmission, pathogen; Dental practice

de Graaff, Johannes; van Amerongen, William E.; Mulder, Grietje G.: Part II: Measures to reduce the risk of contamination. J Dent Child, 55:56-63, January-February, 1988.

Reduction of the risk of contamination in the treatment of most infected patients - sometimes unidentified - can be achieved using the following methods: direct preventive behavior; protective measures; domestic cleaning; disinfection; sterilization; and indirect preventive behavior. Transmission may take place by way of instruments, aerosols,

hands and face, clothing, and treatment unit.

Microorganisms; Transmission, pathogens; Prevention

Battenhouse, MaryAnn Ready; Nazif, M.M.; Zullo, T.: Emergency care in pediatric dentistry. J Dent Child, 55:68-71, January-February, 1988.

The emergency room records of 1,456 children who were treated at Children's Hospital of Pittsburgh with any oral complaint, during a single calendar year, were reviewed with the data entered into a computer. The data were tabulated and statistically analyzed. Trauma (676 visits) was responsible for 46 percent of the total visits, with boys representing a significantly higher percentage of trauma visits than girls (440 vs 236). The total number of trauma visits was also significantly higher for the youngest age-group (birth to three years). Soft-tissue injuries represented a substantially higher percentage of trauma cases (58 percent), compared with dental injuries (35 percent) and bony injuries (3 percent). Emergency dental services are a viable part of emergency services in major population centers; emergency care is an essential component of overall dental services.

Trauma; Emergencies, dental; Treatment visits

Boraz, Robert A.: Oral manifestations of Crohn disease: update of the literature and report of case. J Dent Child, 55:72-74, January- February, 1988.

Crohn disease, a devastating chronic illness, often develops in the childhood years. The disease is a segmental transmural intestinal disease that may involve one or more segments of the gut from the mouth to the anus. Crohn disease is frequently confused with ulcerative colitis. Frequent oral manifestations include persistent oral ulcerations, diffuse gingival swelling, mucosal hyperplasia and fissuring, cheilitis, and pyostomatitis vegetans. Frequently, oral biopsy is essential in

establishing a correct diagnosis of the disease. Although no cure is currently available for the Crohn disease, the proper diagnosis of the problem is essential for providing symptomatic relief. A case report is presented documenting the value of the proper diagnosis of the disease. The literature is reviewed and updated.

Crohn disease; Oral manifestations; Diagnosis

Ooshima, Takashi; Sugiyama, Keiko; Sobue, Shizuo: Oligodontia in the primary dentition with the permanent successors: report of case. J Dent Child, 55:75-77, January-February, 1988.

A case of oligodontia in the primary dentition with corresponding permanent successors is described. A two-year-old Japanese boy was admitted to the pedodontic clinic with the chief complaint of congenitally missing primary teeth. The boy appeared to be normally developed and was in good physical health. There was no history of trauma, extraction, or exfoliation of primary teeth. Clinical examination showed that eight anterior teeth were missing. Also, a canine and three first molars were much smaller in size and were malformed. However, radiographic examination at age four years, ten months showed that all of the permanent tooth germs - including the corresponding successors of congenitally missing primary teeth - were evident.

Oligodontia; Permanent dentition; Radiography