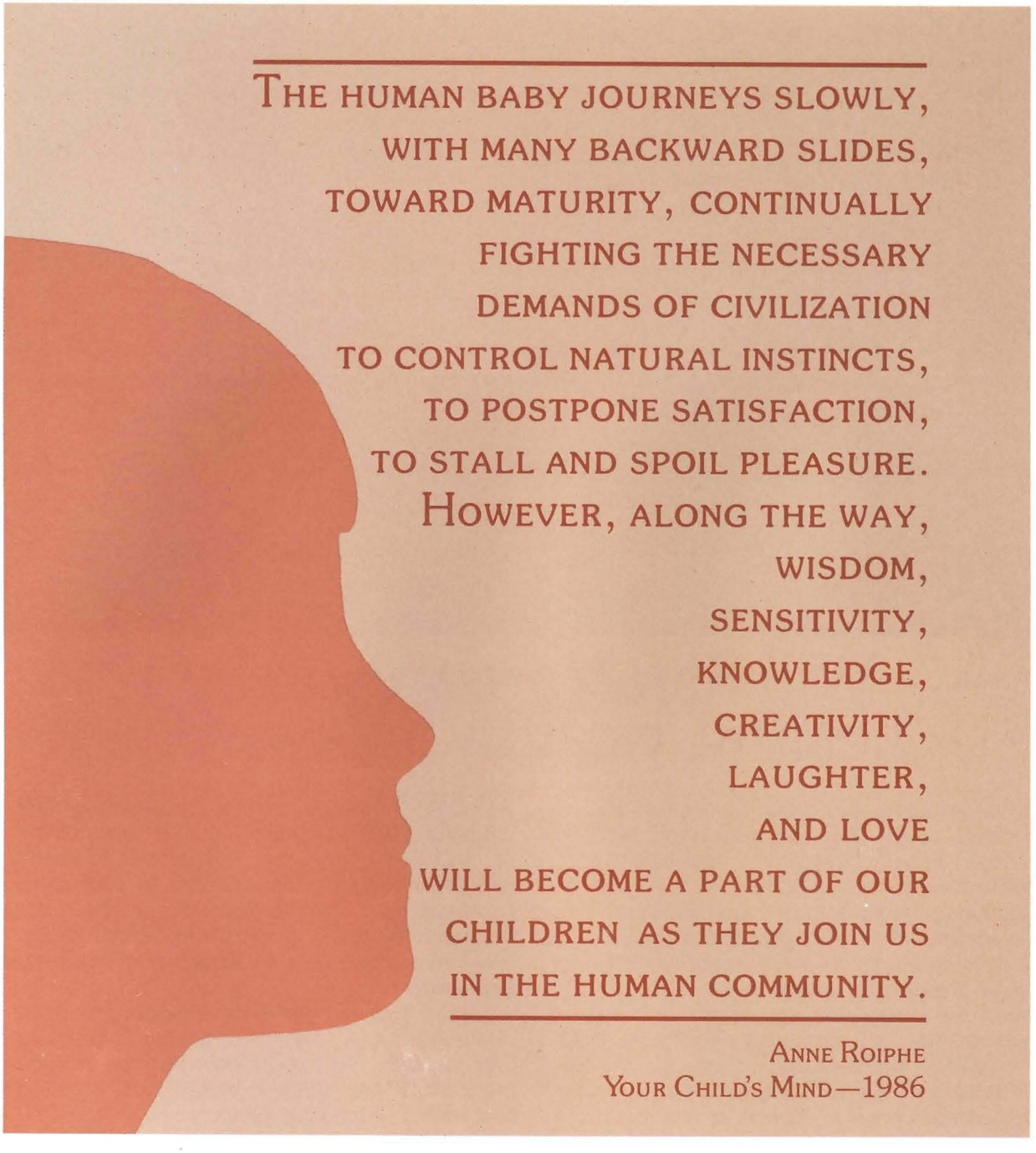
ASDC AMERICAN SOCIETY OF DENTISTRY FOR CHILDREN

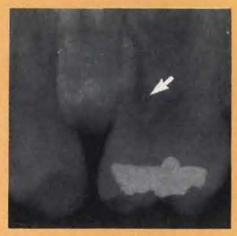
SEPTEMBER-OCTOBER 1987

JOURNAL OF DENTISTRY FOR CHILDREN



TO UNDERSTAND ALL
IS TO FORGIVE ALL
—Madame de Staël

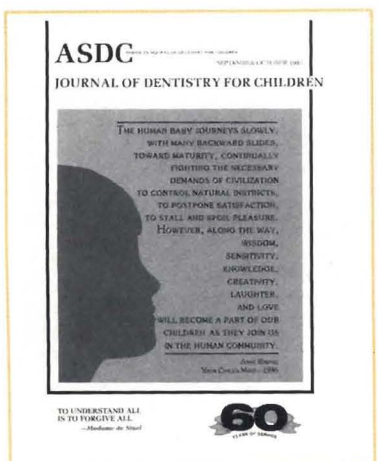
60 YEARS OF SERVICE



ASDC AMERICAN SOCIETY OF DENTISTRY FOR CHILDREN

JOURNAL OF DENTISTRY FOR CHILDREN

Volume 54 Number 5 September-October 1987

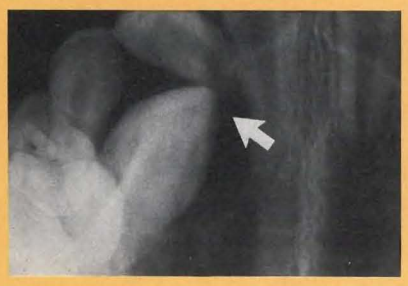


We learn to love and respect our children; and to appreciate their individuality and uniqueness.

Art and design by Sharlene Nowak.

DEVELOPMENT

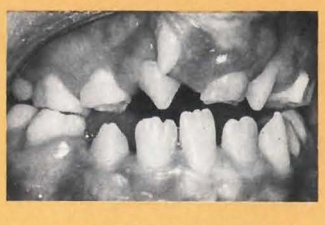
CLINIC



335

368

365



For the busy reader

Does epidermal growth factor control tooth eruption?—page 321

Despite active research for more than several decades the factors responsible for tooth eruption have remained obscure. For a tooth to emerge in the oral cavity, to reach occlusion, and to continue eruption in adulthood, many changes in the tooth and its surrounding tissues occur; these changes must be synchronized. Epidermal growth factor appears to be involved in the eruptive process.

Requests for reprints should be directed to Dr. Irma Thesleff, Institute of Dentistry, University of Helsinki, Mannerheimintie 172, SF-00280 Helsinki, Finland.

Developmental enamel defects of the primary dentition in a group of Californian children—page 330

The parent of each three- to six-year old child with enamel defects, seen at the Loma Linda University School of Dentistry between 1981 and 1982, was interviewed to find an explanation for the defect. The most frequently given was hyperbilirubinemia.

Requests for reprints should be directed to Dr. Wilfred A. Nation, Loma Linda University, School of Dentistry, Department of Pediatric Dentistry, Loma Linda, CA 92350, USA.

Early diagnosis and prevention of impaction of the maxillary canine—page 336

If there is no evidence of a canine bulge, and the tooth appears to be tipped medially in the frontal radiograph, with the crown medial to the lateral border of the nasal cavity, a future impaction of the maxillary canine is a significant possibility.

Requests for reprints should be directed to Dr. Peter W. Ngan, Department of Orthodontics, Ohio State University, College of Dentistry, 305 West 12th Avenue, Columbus, OH 43210.

Effect of etchant time on microleakage—page 339

This study supports the growing evidence that satisfactory marginal sealing can be achieved with reduced enamel etch-times. It is possible to create good marginal sealing in enamel after a fifteen-second etch—especially beneficial in young patients.

Requests for reprints should be directed to Dr. Gary A. Crim, Department of Restorative Dentistry, School of Dentistry, University of Louisville, Louisville, KY 40292.

Complications related to surgical removal of anterior supernumerary teeth in children—page 341

The purpose of this study was to investigate frequency of loss of vitality and sensitivity, resorption of root tissue, and disturbance of the root development of adjacent teeth, after the removal of a supernumerary tooth in the maxilla.

Requests for reprints should be directed to Dr. Lars Andersson, STP Oral Surgery, Central Hospital, S-721 89 Västerås, Sweden.

Another perspective on children's dental needs and demand for services during the 1980s—page 344

In 1984, more than one million acute dental conditions were reported for children younger than five years old, with 630,000 for children between five and seventeen years old. These represent almost five million restricted-activity days; more than 1.6 million days in bed; and 1.7 million missed school days.

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

The diet-heart question: how good is the evidence?—page 349

Cardiovascular diseases (CVD) are the number-one killer in most industrialized countries of the world. A total risk-factor-intervention approach on an individual patient basis would seem to have greater potential for success than a generalized dietary recommendation.

Reprints are not available.

AIDS: children with HIV infection and their families—page 353

Surgeon General Koop charged the group attending a conference workshop at the Children's Hospital of Philadelphia with making recommendations to help set national policy regarding AIDS.

Requests for reprints should be directed to Dr. Linda P. Nelson, Dental Division, Children's Hospital of Philadelphia, 34th and Civic Center Blvd., Philadelphia, PA 19104.

Delayed eruption due to overlying fibrous connective tissue—page 359

Treatment consisted of surgically removing the overlying soft tissue, exposing the crown of the molar.

Requests for reprints should be directed to Dr. Curt Goho, McDonald Army Hospital Dental Clinic, U.S. Army Dental Activity, Ft. Eustis, Virginia 23604.

Root resorption in association with ectopic eruption: report of case—page 361

This patient showed extensive root resorption close to the cementoenamel junction. It is likely that impacted teeth anywhere in the dental arch can participate in the destruction of root surfaces of neighboring teeth.

Requests for reprints should be directed to Dr. Lena Omnell, Department of Orthodontics, University of Washington, Seattle, Washington 98195.

Impacted primary incisor: report of case—page 363

Impaction of a primary incisor is very rare, with only one previous report in the dental literature. After surgical exposure, the case reported here had a normal course of eruption during follow-up.

Requests for reprints should be directed to Dr. L. Bodner, Department of Oral Surgery, The Maurice and Gabriela Goldschleger School of Dental Medicine, Tel Aviv University, Tel Aviv, Israel.

Aplasia of primary canines and anlagen of permanent canines: a cleft-specific condition?—page 365

The patient is a girl with bilateral cleft lip and palate. It appears that one or more specific teratogens that induce definite malformations are also able to disturb the development of the dentition in an unpredictable manner.

Requests for reprints should be directed to Dr. Eva A. Holtgrave, Fachbereich Kieferheilkunde, Freie Universität Berlin (BRD, Germany) Assmanshauser Str. 4-6 1000 Berlin 33.

Dental and oral manifestations of Rubenstein-Taybi syndrome: report of case—page 368

Characteristic features have given some researchers reason to believe that Rubenstein-Taybi syndrome is caused by a chromosomal abnormality. No common etiological factor or pathognomic criterion has been established for this syndrome.

Requests for reprints should be directed to Dr. Moorean A. Baker, 1341 Pennsylvania Avenue, S.E., Washington, D.C. 20003.

Does epidermal growth factor control tooth eruption?

Irma Thesleff, DDS, Dr Odont

Despite active research over several decades the factors responsible for tooth eruption have remained obscure. This is obviously due to the complex nature of the eruptive process, which makes it a very difficult subject for experimental research. There are at least three major complicating aspects that cause problems in the design of experiments as well as in the interpretation of results. First, tooth eruption is a long-lasting process composed of several overlapping phases.[1] It is possible that different control mechanisms operate in these different phases of eruption. For example, the movement of the erupting tooth through the bone obviously implies different tissue reactions as compared with the active eruption of teeth reaching occlusion. Second, the type of eruption varies among different teeth and according to the eruption type, teeth can be grouped in various categories.[1] Notably, the eruption of continuously growing rodent incisors which have been used in numbers of eruption studies may be differently controlled than that of human teeth.[2] The third complicating fact is that the tooth and its surrounding tissues constitute such a wide spectrum of different tissues and cell types that it is virtually impossible to design experiments where only a single tissue component or cell type would be affected. Hence, almost all tissues in and around the tooth have been implicated as having a central role in tooth eruption. On the other hand, the authors who put forward the different theories on the

Dr. Thesleff is with the Department of Pedodontics and Orthodontics, Institute of Dentistry, University of Helsinki, Helsinki, Finland.

mechanism of tooth eruption usually concluded that tooth eruption seems to be a multifactorial process.[2-9]

It is clear that in order for a tooth to emerge in the oral cavity, to reach occlusion and even to continue eruption in adulthood, a multitude of changes in the tooth and its surrounding tissues occur and that these must be synchronized. For example, the roots develop as the tooth with its follicle moves through the bone, and the periodontal ligament is being organized at the same time. The oral epithelium disrupts upon emergence, and the vessels and nerves of the tooth must also adjust to the changes in tooth position. But, despite this multitude of changes which accompany eruption, would it still be possible that there exists only one or possibly a couple of factors that control the process? Such factors could affect responsive cells at different stages of eruption, while most tissue reactions during eruption would occur secondarily to the primary trigger. One candidate for such a controlling element is epidermal growth factor.

EPIDERMAL GROWTH FACTOR

In the early 1960's, Stanley Cohen identified epidermal growth factor as the active component in a submandibular gland extract that caused precocious incisor eruption and eyelid opening in newborn mice.[10] Because of this discovery and continued extensive studies on the structural and functional aspects of epidermal growth factor, Cohen was granted the Nobel Prize in 1986.

Epidermal growth factor is a small molecular weight polypeptide (6045 daltons) that is a potent mitogen for a number of cell types, also nonepithelial cells.[11,12] Despite very active research over a long time, the physiological functions of epidermal growth factor have remained obscure. Physiological roles for epidermal growth factor have been proposed in wound healing, in mammary gland development and function, and in male reproductive function.[13-15] Epidermal growth factor affects hair development in mice and other species and hence a role in epidermal maturation has been suggested.[16-19] Besides cell division, epidermal growth factor affects other cellular functions such as differentiation, synthesis of various macromolecules, and bone resorption.[20-23] Furthermore, epidermal growth factor is identical to urogastrone, which has a gastric cytoprotective function.[24,25] Associations have been revealed between oncogene activation and epidermal growth factor and its receptors.[26,27] Hence, the possible roles of epidermal growth factor and molecules that resemble it in transformation are currently being extensively examined.[28,29]

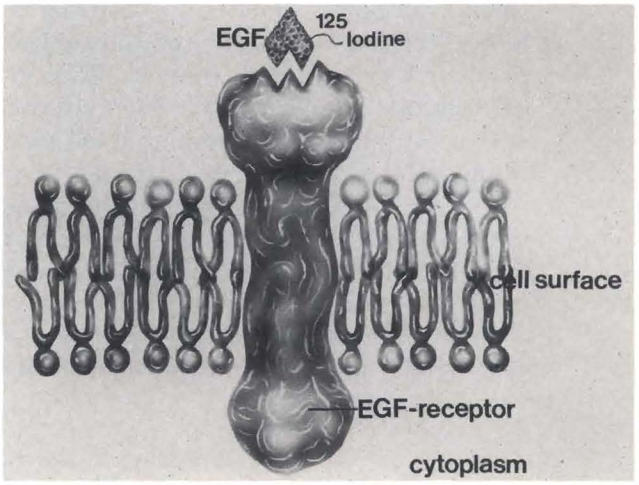
Taken together, epidermal growth factor is a growth regulatory substance that potentially affects many different cell types at various locations in the body. Besides being a potent mitogen it affects various cellular functions according to the target tissue.

Epidermal growth factor and tooth development

Although epidermal growth factor was discovered more than twenty years ago because of its ability to accelerate tooth emergence, its role in tooth formation and eruption have not been studied until recently. It was suggested by the first researchers of epidermal growth factor that the precocious eruption of mouse incisors was due to enhancement of epithelial cell proliferation and shedding of the oral mucosa overlying the emerging tooth.[11] This hypothesis has never been tested, but it appears that besides having an effect on epidermal maturation, epidermal growth factor affects other tissues in and around the erupting tooth.

We have shown that when the eruption of mouse incisors is accelerated by epidermal growth factor, the proliferation of preodontoblasts and preameloblasts is stimulated in the root sheath region.[30] Furthermore,

Figure 1. Schematic illustration of the binding of epidermal growth factor (EGF) to its receptor. The receptor is a large molecule (150,000 daltons), and it extends through the plasma membrane. Binding of EGF activates a protein kinase in the cytoplasmic part of the receptor which leads to the cellular response. EGF- receptors in tissues can be localized by examining the binding of radioactively (e.g. ^{125}Iodine) labelled EGF in autoradiographs (Figures 3-6).



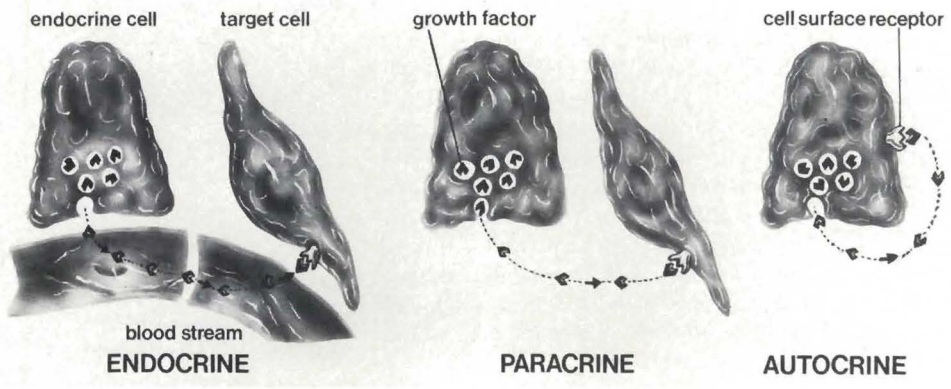


Figure 2. Three different mechanisms of hormone and growth factor action. The classical hormones act by the endocrine mechanism, where the hormone is carried with the blood stream to the target tissue. The growth factors are believed to function locally via paracrine and autocrine mechanisms. In the paracrine mechanism, the growth factor affects a neighboring cell, which has receptors to it. Autocrine mechanism is typical for malignant cells, which stimulate their own growth by responding to growth factors that they have secreted themselves.

tissue culture experiments from our laboratory indicated that high doses of the factor inhibit early tooth morphogenesis while stimulating vascular growth in the surrounding tissue.[31] We have also shown that the responsiveness of the different tissue components in a developing tooth varies according to the stage of development, thus suggesting that tissue interactions are involved in the system.[32,33] The distribution of epidermal growth factor-receptors in developing and erupting teeth is discussed in detail below.

Epidermal growth factor-receptor

Epidermal growth factor belongs to the family of small molecular weight growth factors such as platelet derived growth factor, fibroblast growth factor, and nerve growth factor.[34-36] A common feature of all growth factors is that they affect cells through binding to specific cell- surface receptors (Figure 1). The receptor for epidermal growth factor has been well characterized.[12,37] It is a membrane glycoprotein with a molecular weight of 150,000 daltons and consists of extracellular and intracellular parts and a transmembrane portion connecting them. Binding of epidermal growth factor to its receptor activates a protein kinase in the intracellular part of the receptor, which phosphorylates tyrosine residues of proteins, leading to the cellular response. After binding of epidermal growth factor to its receptor, the receptor complex is internalized in the cell and is ultimately degraded in lysosomes.

This receptor-mediated system implies that only cells that have receptors for a growth factor are able to respond to that particular factor. Epidermal growth factor-receptors are expressed in a variety of different cell types, in varying amounts, and this apparently accounts for the differences between cells in their responsiveness to the factor.

The mechanism of epidermal growth factor action

Although growth factors are hormone-like agents, they do not seem to act by an endocrine mechanism like the classic hormones (Figure 2). Instead of being produced by special endocrine cells in a defined endocrine gland, growth factors are produced by many tissues, and their blood concentrations are generally low. It is believed that growth factors exert their actions mainly by paracrine or autocrine mechanisms (Figure 2). The target cell in a paracrine-type situation is influenced by a growth factor produced by a neighbor cell. The autocrine mechanism has so far been demonstrated in cancer cells, which in this way are able to stimulate their own malignant growth; that is, they produce large numbers of growth factors for which they also have receptors.[28,38]

Epidermal growth factor is detected only in minor amounts in blood, but it is present in varying amounts in body fluids such as saliva, urine and milk.[39,41] The exact sites of epidermal growth factor production are not known; but it is apparent that a wide variety of tissues are able to produce it. In fact, almost all mouse tissues that were tested by Rall *et al* were able to synthesize small amounts of prepro epidermal growth factor, the precursor for epidermal growth factor.[42] The latter has

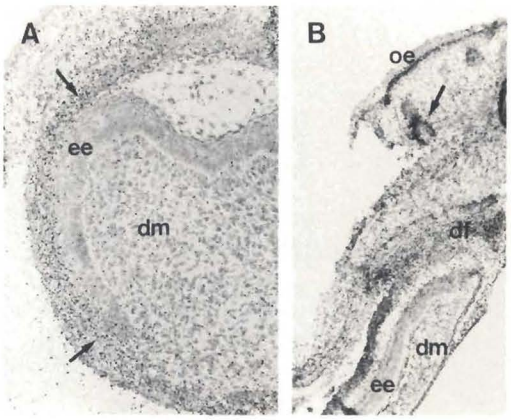


Figure 3. Autoradiographs showing the distribution of bound ^{125}Iodine-labelled epidermal growth factor (^{125}I-EGF) in dental and surrounding tissues of a sixteen-day-old mouse embryo. 3A. Molar tooth germ at the bell stage of development. Abundant binding (black grains) is seen in the dental follicle surrounding the tooth (arrows), whereas the dental papilla mesenchyme (dm) and enamel epithelium (ee) show only background labelling. 3B. Tissues overlying the incisor tooth germ. The dental mesenchyme (dm) and enamel epithelium (ee) show no binding. Abundant labelling is evident in the dental follicle (df), in the basal epithelial cells of the oral epithelium (oe) and in the hair follicles (arrow).

been localized in many human tissues, most notably in salivary and duodenal glands, but also in skin and kidney.[43,44]

It is obvious that the paracrine and autocrine mechanisms of growth factor action provide possibilities for localized stimulation of growth and other cellular functions. Thus, the important issue in a discussion of the biological functions of growth factors is, which tissues produce them and which are responsive to them. We have shown that during the course of tooth development, the tissue distribution of epidermal growth factor-responsive cells varies.[32] It is possible that variations exist also in the distribution of cells that produce epidermal growth factor during tooth development and eruption, and that this interplay controls some steps in these developmental events.

EVIDENCE SUPPORTING AN IMPORTANT ROLE FOR EPIDERMAL GROWTH FACTOR IN TOOTH ERUPTION

Demonstration of epidermal growth factor receptors in developing and erupting teeth

We studied the occurrence of epidermal growth factor receptors in mouse and human teeth by localizing the binding sites of radioactively labelled epidermal growth factor. The freshly dissected tissue pieces (whole embryonic mouse tooth germs, apical ends of mouse incisors, pieces of dental follicle, apical pulp and gingiva of human erupting teeth) were incubated for ninety minutes in the presence of ^{125}Iodine-labelled epidermal growth factor (Figure 1). After extensive washing (to get

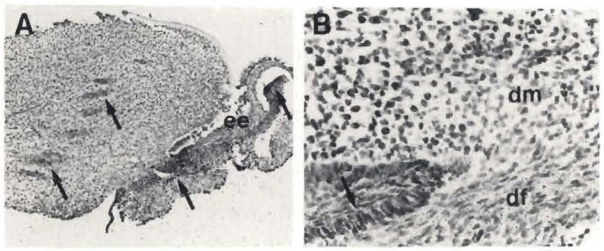


Figure 4. Distribution of ^{125}I-labelled epidermal growth factor in the apical tissues of an erupting incisor from a twelve-day-old mouse. 4A. Longitudinal section of the apical tissue piece that was incubated with ^{125}I-EGF. Abundant binding is seen in the mesenchyme of the dental follicle beneath the apical foramen and under the enamel epithelium (ee), as well as around the blood vessels (arrows). 4B. A higher magnification of the apical area. The mesenchymal cells are labelled in the follicle (df) but not in dental pulp (dm). Also the outer cells of the cervical loop epithelium are labelled (arrows).

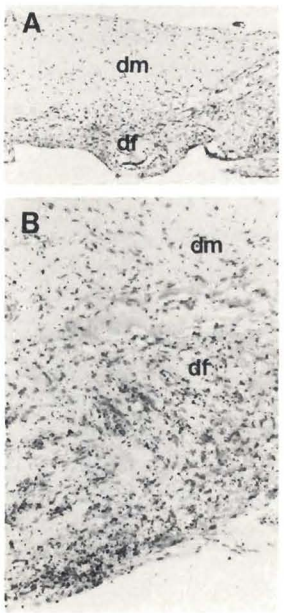


Figure 5. Distribution of ^{125}I-EGF in the apical tissues of an erupting human premolar. 5A. Photomicrograph showing the composition of the tissue that was dissected: dm dental pulp mesenchyme, df dental follicle. 5B. Autoradiograph at a higher magnification shows that the dental follicle (df) is more intensely labelled than the dental pulp mesenchyme (dm).

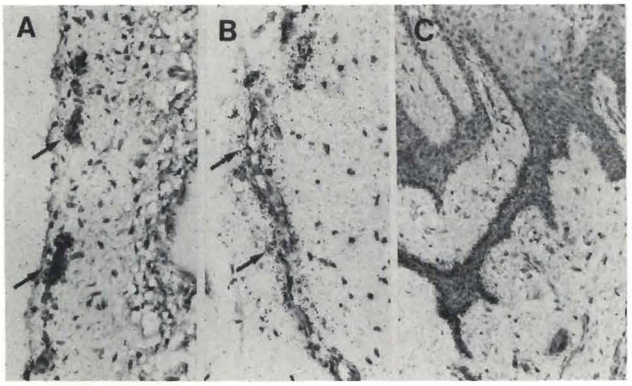


Figure 6. Autoradiographs showing the localization of ^{125}I-EGF binding cells in the tissues associated with an impacted human premolar. 6A. In the dental follicle from the middle third of the root, the epithelial cell rests of Malassez are very heavily labelled (arrows). 6B. In the apical follicle, abundant labelling is associated particularly with blood vessels (arrows). 6C. Autoradiograph of the palatal mucosa overlying the tooth shows that the basal epithelial cells are heavily labelled. Only background binding is seen in the stromal cells.

rid of unbound factor) the tissues were fixed, embedded in paraffin, sectioned, and processed for autoradiography. The binding sites of ^{125}Iodine-epidermal growth factor presumably represent epidermal growth factor receptors and they are seen as black grains in the autoradiographs (Figures 3 to 6).

Binding of epidermal growth factor was evident in all tissue explants studied, thus indicating that the teeth of both mouse and human origin are potentially able to respond to the factor. There were marked changes in the tissue distribution of epidermal growth factor receptors in the mouse teeth, during advancing development.[32] High numbers were expressed during the bud and cap stages of development, first in the dental epithelium and then in the mesenchyme, suggesting a role for epidermal growth factor in morphogenesis. In the bell-stage, however, the receptors disappeared from other dental tissues, except the follicle, where they were localized in a dramatically high density (Figure 3A).[32] A high density of receptors was present also in the apical and coronal parts of the dental follicle of mouse incisors (Figures 3B and 4). The dental follicle of human erupting premolars was also shown to bind epidermal growth factor (Figures 5 and 6).[45] The smooth muscle cells of small arterioles in the dental follicle and in the apical mesenchyme of the dental pulp bound epidermal growth factor abundantly (Figures 5 and 6). Furthermore, in the human tooth follicle, the epithelial cell rests of Malassez were labelled more intensely than any other cells (Figure 6A). The basal epithelial cells of the oral mucosa also stained intensely (unpublished, Figures 3B and 6C). These findings indicate that the dental follicle, blood vessels, the oral epithelium, and the epithelial cell rests of Malassez are the most likely candidates for the target tissues of epidermal growth factor, in erupting teeth.

The mediatory role of epidermal growth factor in hormonal control

The rate of tooth eruption is abnormal in several pathological conditions and syndromes and it can be experimentally altered in animals (Table). The examination of the different factors associated with abnormal eruption rates shows that eruption is largely under endocrine control. Tooth eruption is accelerated in patients with hyperfunction of various endocrine glands, and in experimental animals that have been given the corresponding hormones.[3,7,46-48] It is interesting that, besides epidermal growth factor, hormones and antidiuretic drugs appear to be the only exogenous factors that unequivocally accelerate eruption.[50] Delayed eruption is associated with hypofunction of the thyroid, pituitary and adrenal glands, as well as in animals after removal of the glands.[2,3,7,49] In addition, eruption is delayed, for

Table ☐ Summary of factors associated with abnormal eruption.

	Accelerated	Delayed
Experimentally induced (animals)	epidermal growth factor thyroid hormone growth hormone cortisone insulin antidiuretic drugs	thyroidectomy hypophysectomy adrenalectomy x-rays cyclophosphamide lathyrogens
Pathologic conditions (human)	hyperthyroidism hyperpituitarism hyperinsulinemia hemifacial hypertrophy	hypothyroidism hypopituitarism
Syndromes	Donohue syndrome pachyonychia congenita II	cleidocranial dysplasia osteopetrotic animals Down syndrome

example, by x-rays or cytotoxic drugs, or when collagen fiber organization is impaired with lathyrogens.[2,48,51]

There is increasing evidence that the biological effects of many classic hormones are mediated by growth factors.[52] In fact, Stanley Cohen suggested in his first paper on epidermal growth factor that it possibly was involved in the hormonal effects of thyroxine and cortisone, because these had been reported to have similar effects on eyelid opening and incisor eruption as had his new factor.[10] Later studies confirmed that epidermal growth factor is involved in mediation on the effects of thyroid hormone, growth hormone, insulin and corticosteriods.[52] The interaction between thyroid hormone and epidermal growth factor has been most extensively studied and it is now believed that the factor is the mediator of most, if not all, biological effects that were previously thought to result from direct action of thyroid hormone.[52] Thyroid hormone increases concentrations of epidermal growth factor in urine and in several mouse tissues, including submandibular gland, eye and skin.[17,53,54] The mechanism by which thyroid hormone and other hormones increase the content of the factor in tissues is not known at present, but there are at least three possibilities:

- ☐ The number of epidermal growth factor-receptors may be increased.
- ☐ Epidermal growth factor synthesis may be stimulated either locally or systemically.
- ☐ The degradation of the factor may be slowed. There is evidence that thyroid hormone affects the level of epidermal growth factor receptors in mammary glands and liver.[55,56] It appears that the regulation of the level of epidermal growth factor by thyroid hormone is tissue-specific. Whether thyroid hormone affects its content in teeth is not known at present.

The correlation between a high level of epidermal growth factor and early tooth development in a patient

Abnormal rates of tooth development or eruption are seen in many syndromes, some of which are listed in the Table. The causes of the defects in eruption are generally not known, although in some instances with delayed eruption, the dense structure of bone appears to be the cause.[57,58] Tooth eruption is accelerated in both Donohue syndrome (leprechaunism) and pachyonychia congenita; and in these syndromes, epidermal tissues are generally affected.[58,59] The symptoms of Donohue syndrome include hyperinsulinism, epidermal keratinization and early tooth development, and it is the first disease associated with increased levels of epidermal growth factor.[59] The urinary content of the factor of this particular patient, a five-year-old girl, was five times higher than normal, and her dental age was fourteen years. It will be interesting to see in the future whether epidermal growth factor-levels can be correlated with abnormal tooth development in other patients.

Synthesis of epidermal growth factor by erupting teeth

So far there is one report in the literature where dental tissues were shown to synthesize epidermal growth factor. Rall *et al* localized the synthesis of the precursor of epidermal growth factor, prepro epidermal growth factor, in longitudinal sections of whole adult mice, and demonstrated that the major sites of synthesis were the submandibular glands, kidneys, and the root tips of the continuously erupting incisors.[42] This interesting finding still needs to be confirmed and extended to other types of teeth, before final conclusions can be made about the capacity of the dental tissues to synthesize epidermal growth factor. The modern *in situ* hybridization techniques will also make it possible to localize synthesis of the factor in specific cells and tissues.

EPIDERMAL GROWTH FACTOR AND THE THEORIES OF THE MECHANISM OF TOOTH ERUPTION

It is intriguing to analyze how well the hypothesis that epidermal growth factor is a controlling agent in eruption fits to the various theories that have been introduced on the mechanism of tooth eruption. There are four major tissues in and around the tooth, suggested to be the most important sites of activity during eruption:

- ☐ *The dental follicle.* Cahill and Marks have suggested that bone resorption and apposition adjacent to the erupting tooth are due to activities in the dental follicle, which thus would have a regulatory role during the early stages of eruption.[8,60,61]
- ☐ *The roots.* The theory that growth of the roots causes a tooth to erupt has been criticized, because teeth without roots also erupt, occasionally. This theory, like the first one, cannot explain the continuing eruption beyond the stage where root development has been completed.
- ☐ *Blood vessels.* Changes in vascular and tissue pressures have frequently been suggested to generate the eruptive force.[3,4,50,62] More recently, Sutton presented the "blood-vessel thrust theory", in

which he suggests that the blood flow, which is forced to change its direction in the pulp, is responsible for the eruptive force.[9]

- *The periodontal ligament.* Recently, the periodontium has most frequently been suggested as the site for the eruptive force, and both fibers and special cell types in the ligament have been proposed to generate the force.[6,63-66]

In the light of our current knowledge on the physiology of epidermal growth factor and its receptors in the erupting teeth, it appears that the assumption that the factor controls tooth eruption does not lead to the rejection of any of the eruption theories: First, the hypothesis that epidermal growth factor would affect the dental follicle is supported by several observations. Epidermal growth factor stimulates bone resorption in culture through the prostaglandin-mediated pathway, and it also affects the differentiation of osteoblasts.[21,67,68] It has, in fact, been suggested that it would be involved in bone remodelling.[69] We also localized epidermal growth factor receptors in the dental follicle. Hence, it is possible, that the factor controls the bone remodelling activity of the dental follicle. The assumption that it would control especially the early stages of eruption gets support from observations on unerupted teeth, in hypothyreotic children. The teeth in their sockets are located deep in the bone, although their formation stage is not much retarded (S. Myllärniemi, personal communication). As discussed above, the effect of thyroid hormone is probably mediated by epidermal growth factor.

Second, epidermal growth factor may control root growth. It is a mitogen for many cell types, and our studies on mouse incisor development suggested that cell proliferation was stimulated in the roots of epidermal growth factor treated animals.[30]

Third, the blood vessels are also a potential target for epidermal growth factor. We showed that epidermal growth factor receptors are particularly enriched in the smooth muscle cells of arterioles in the dental follicle and in the apical pulp.[45] Studies on the effect of epidermal growth factor on sheep skin suggest that the blood flow to the skin is increased by epidermal growth factor.[18] Furthermore, epidermal growth factor has been shown to stimulate vascular development.[70,71]

Fourth, epidermal growth factor receptors can be assumed to be present also in the periodontal ligament. Besides their presence in blood vessels, they are found in high numbers in the epithelial cell-rests of Malassez in the dental follicle. How epidermal growth factor would affect the cell-rests and how this could affect eruption is a matter of speculation, but as long as the function of these cell-rests is not known, the possibility remains that they have a role in eruption.

All present knowledge on the functions of epidermal growth factor in general and its associations with tooth eruption in particular, support the assumption that it is involved in the eruptive process. Also, there is no evidence that would contradict this hypothesis. The drawing of more definite conclusions must, however, wait until more studies are available on the metabolism of epidermal growth factor in erupting teeth. If it turns out to be an important controlling factor in tooth eruption, it is probable that our understanding of the mechanism of tooth eruption as well as of the generation of the "eruptive force" will considerably deepen in the near future.

REFERENCES

1. Steedle, J.R. and Proffit, W.R.: The pattern and control of eruptive tooth movements. Am J Orthod, 87:56-66, January, 1985.
2. Ness, A.R.: Movement and forces in tooth eruption. Adv Oral Biol, 1:33-75, 1964.
3. Massler, M. and Shour, I.: Studies in tooth development: Theories of eruption. Oral Surg, 27:552- 576, October, 1941.
4. Magnusson, B.: Tissue changes during molar tooth eruption. Transactions of the Royal School of Dentistry, Stockholm and Umeå, 13:1-122, 1968.
5. Ten Cate, A.R.: The mechanism of tooth eruption. In: Melcher, A.H.; Bowen, W.H. (eds): Biology of the periodontium. New York: Academic Press, 1969, pp 91- 103.
6. Berkovitz, B.K.B.: The effect of root transection and partial root resection on the unimpeded eruption rate of the rat incisor. Arch Oral Biol, 16:1033-1043, September, 1971.
7. Shulman, J.: Causes and mechanisms of tooth eruption. A literature review. Period Abstr, 24:162-171, Winter, 1977.
8. Marks, S.C. and Cahill, D.R.: Experimental study in the dog of the non-active role of the tooth in the eruptive process. Arch Oral Biol, 29:311-322, April, 1984.
9. Sutton, P.R. and Graze, H.R.: The blood-vessel thrust theory of tooth eruption and migration. Med Hypotheses, 18:389-395, November, 1985.
10. Cohen, S.: Isolation of a mouse submaxillary gland protein accelerating incisor eruption and eyelid opening in the newborn animal. J Biol Chem, 237:1555-1562, May, 1962.
11. Cohen, S.: The stimulation of epidermal proliferation by a specific protein (EGF). Dev Biol, 12:394-407, December 1965.
12. Carpenter, G. and Cohen, S.: Epidermal growth factor. Ann Review Biochem, 48:193-216, 1979.
13. Turkington, R.W.: The role of epidermal growth factor in mammary gland development *in vitro*. Exp Cell Res, 57:79-85, September, 1969.
14. Okamoto, S. and Oka, T.: Evidence for physiological function of epidermal growth factor: Pregestational sialoadenectomy of mice decreases milk production and increases offspring mortality during lactation period. Proc Natl Acad Sci USA, 81:6059-6063, October, 1984.
15. Tsutsumi, O.; Kurachi, H.; Oka, T.: A physiological role of epidermal growth factor in male reproductive function. Science, 233:975-977, August, 1986.
16. Hoath, S.B.; Lakshmanan, J.; Fisher, D.A.: Differential hormonal response of epidermal growth factor concentration in the developing mouse: synergism of triiodothyronine and dexamethasone in epidermal maturation. Life Sci, 32:2709-2716, June, 1983.
17. Moore, G.P.M.; Panaretto, B.A.; Robertson, D.: Epidermal

growth factor delays the development of the epidermis and hair follicles of mice during growth of the first coat. Anat Rec, 205:47-55, January, 1983.
18. Moore, G.P.M.; Panaretto, B.A.; Carter, N.B.: Epidermal hyperplasia and wool follicle regression in sheep infused with epidermal growth factor. J Invest Dermatol, 84:172-175, March, 1985.
19. Green, M.R.; Basketter, D.A.; Couchman, J.R.: Distribution and number of epidermal growth factor receptors in skin is related to epithelial cell growth. Dev Biol, 100:506-512, December, 1983.
20. Chen, L.B.; Gudor, R.C.; Sun, T-T *et al*: Control of a cell surface major glycoprotein by epidermal growth factor. Science, 197:776-778, August, 1977.
21. Hata, R-I.; Hori, H.; Nagai, Y. *et al*: Selective inhibition of type I collagen synthesis in osteoblastic cells by epidermal growth factor. Endocrinology, 115:867-876, September, 1984.
22. Turley, E.A.; Hollenberg, M.D.; Pratt, R.M. *et al*: Effect of epidermal growth factor/urogastrone on glycosaminoglycan synthesis and accumulation *in vitro* in the developing mouse palate. Differentiation, 28:279-285, 1985.
23. Chan, C.P. and Krebs, E.G.; Epidermal growth factor stimulates glycogen synthase activity in cultured cells. Proc Natl Acad Sci USA, 82:4563-4567, July, 1985.
24. Gregory, H.: Isolation and structure of urogastrone and its relationship to epidermal growth factor. Nature, 257:325-327, September, 1975.
25. Skov Olsen, P.; Poulsen, S.S.; Kirkegaard, P. *et al*: Role of submandibular saliva and epidermal growth factor in gastric cytoprotection. Gastroenterology, 87:103-108, 1984.
26. Downward, J.; Yarden, Y.; Mayes, E. *et al*: Close similarity of epidermal growth factor receptor and V-erb-B oncogene protein sequences. Nature, 307:521-527, February, 1984.
27. Bravo, R.; Burckhardt, J.; Curran, T. *et al*: Stimulation and inhibition of growth by EGF in different A431 cell clones is accompanied by the rapid induction of c-fos and c-myc proto-oncogenes. EMBO J, 4:1193-1197, May, 1985.
28. Heldin, C-H. and Westermark, B.: Growth factors: Mechanism of action and relation to oncogenes. Cell, 37:9-20, May, 1984.
29. Kurachi, H.; Okamoto, S.; Oka, T.: Evidence for the involvement of the submandibular gland epidermal growth factor in mouse mammary tumorigenesis. Proc Natl Acad Sci USA, 82:5940-5943, September, 1985.
30. Rihtniemi, L. and Thesleff, I.: An autoradiographic study on the effect of epidermal growth factor on cell proliferation in erupting mouse incisors. Arch Oral Biol (submitted).
31. Partanen, A-M.; Ekblom, P.; Thesleff, I.: Epidermal growth factor inhibits morphogenesis and cell differentiation in cultured mouse embryonic teeth. Dev Biol, 111:84-94, September, 1985.
32. Partanen, A-M. and Thesleff, I.: Localization and quantitation of 125-I-epidermal growth factor binding in mouse embryonic tooth and other embryonic tissues at different developmental stages. Dev Biol, 120:186-197, March, 1987.
33. Thesleff, I. and Hurmerinta, K.: Tissue interactions in tooth development. Differentiation, 18:75-88, 1981.
34. Heldin, C-H; Westermark, B.; Wasteson, A.: Specific receptors for platelet derived growth factor on cells derived from connective tissue and glia. Proc Natl Acad Sci USA, 78:3664-3668, June, 1981.
35. Gospodarowicz, D.; Brown, K.D.; Birdwell, C.R. *et al*: Control of proliferation of human vascular endothelial cells: Characterization of the response of human umbilical vein endothelial cells to fibroblast growth factor, epidermal growth factor and thrombin. J Cell Biol, 77:774-788, June, 1978.
36. Levi-Montalcini, R.: Developmental neurobiology and the natural history of nerve growth factor. Annu Rev Neurosci, 5:341-362, 1982.
37. Schlessinger, J.; Schreiber, A.B.; Levi, A. *et al*: Regulation of cell proliferation by epidermal growth factor. Crit Rev Biochem, 14:93-111, 1983.
38. Sporn, M.B. and Todaro, G.J.: Autocrine secretion and malignant transformation of cells. New Eng J Med, 303:878-880, October, 1980.
39. Moran, J.R.; Courtney, M.E.; Orth, D.N. *et al*: Epidermal growth factor in human milk: Daily production and diurnal variation during early lactation in mothers delivering at term and at premature gestation. J Pediatr, 103:402-405, September, 1983.
40. Dagogo-Jack, S.; Atkinson, S.; Kendall-Taylor, P.: Homologous radioimmunoassay for epidermal growth factor in human saliva. J Immunoassay, 6:125-136, 1985.
41. Mattila, A-L.; Perheentupa, J.; Pesonen, K. *et al*: Epidermal growth factor in human urine from birth to puberty. J Clin Endocrinol Metab, 61:997-1000, November, 1985.
42. Rall, L.B.; Scott, J.; Bell, G.J. *et al*: Mouse prepro- epidermal growth factor synthesis by the kidney and other tissues. Nature, 313:228-231, January, 1985.
43. Hirata, Y. and Orth, D.N.: Epidermal growth factor (urogastrone) in human tissues. J Clin Endocrinol Metab, 48:667-672, April, 1979.
44. Kasselberg, A.G.; Orth, D.N.; Gray, M.E.: Immunocytochemical localization of human epidermal growth factor/urogastrone in several human tissues. J Histochem Cytochem, 33:315-322, 1985.
45. Thesleff, I; Partanen, A-M; Rihtniemi, L.: Localization of epidermal growth factor receptors in mouse incisors and human premolars during eruption. Eur J Orthod, 9:24-32, February, 1987.
46. Thesleff, I. Epithelial cell rests of Mallassez bind epidermal growth factor intensely. J Periodont Res, vol 22, 1987 (in press).
47. Parmer, L.G.; Katonah, F.; Angrist, A.A.: Comparative effects of ACTH, cortisone, corticosterone, desoxycorticosterone, pregnenolone on growth and development of infant rats. Proc Soc Exp Biol Med, 77:215-218, June, 1951.
48. Moxham, B.J. and Berkovitz, B.K.B.: Interactions between thyroxine, hydrocortisone and cyclophosphamide in their effects on the eruption of the rat mandibular incisor. Arch Oral Biol, 28:1083-1087, December, 1983.
49. Myllärniemi, S.; Lenko, H.L.; Perheentupa, J.: Dental maturity in hypopituitarism, and dental response to substitution treatment. Scand J Dent Res, 86:307-312, September, 1978.
50. Litvin, P.E. and De Marco, T.J.: The effect of a diuretic and antidiuretic on tooth eruption. Oral Surg, 35:294-298, February, 1973.
51. Taverne, A.A.R.; Lemmens, I.G.; Tonino, G.J.M.: Lathyrogens and the role of collagen in the eruption of rat incisors. Arch Oral Biol, 31:127-131, February, 1986.
52. D'Ercole, A.J. and Underwood, L.E.: Regulation of fetal growth by hormones and growth factors. In: Human growth, Vol 1, eds., F. Falkner and J.M. Tanner. New York: Plenum Press, 1986, pp 327-338.
53. Lakshmanan, J.; Perheentupa, J.; Macaso, T. *et al*: Acquisition of urine, kidney and submandibular gland epidermal growth factor responsiveness to thyroxine adminstration in neonatal mice. Acta Endocrinol, 109:511- 516, August, 1985.
54. Walker, P.: Thyroxine increases submandibular gland nerve growth factor and epidermal growth factor concentrations precociously in neonatal mice: Evidence for thyroid hormone-mediated growth factor synthesis. Pediatr Res, 20:281-284, April, 1986.
55. Vonderhaar, B.K.; Tang, E.; Lyster, R.R. *et al*: Thyroid hormone regulation of epidermal growth factor receptor levels in mouse mammary glands. Endocrinology, 119:580-585, August, 1986.
56. Mukku, V.R.: Regulation of epidermal growth factor receptor levels by thyroid hormone. J Biol Chem, 259:6543-6547, May, 1984.
57. Marks, S.C.: Tooth eruption and bone resorption: experimental investigation of the ia (osteopetrotic) rat as a model for studying their relationships. J Oral Pathol, 5:149-163, May, 1976.

58. Salinas, C.F.: Orodental findings and genetic disorders. Birth Defects: Orig Article Series 18:79-120, 1982.
59. Frindik, J.P.; Kemp, S.F.; Fiser, R.H. *et al*: Phenotypic expression in Donohue syndrome (leprechaunism): A role for epidermal growth factor. J Pediatr, 107:428-430, September, 1985.
60. Cahill, D.R. and Marks, S.C.: tooth eruption: evidence for the central role of the dental follicle. J Oral Pathol, 9:189-200, July, 1980.
61. Marks, S.C.; Cahill, D.R.; Wise, G.E.: The cytology of the dental follicle and adjacent alveolar bone during tooth eruption in the dog. Am J Anat, 168:277-289, November, 1983.
62. Bryer, L.W.: An experimental evaluation of the physiology of tooth eruption. Int Dent J, 7:432-478, September, 1957.
63. Berkovitz, B.K.B.; Moxham, B.J.; Newman, H.N.: the periodontal ligament in health and disease. Oxford: Pergamon Press, 1982.
64. Beertsen, W.; Everts, V.; van den Hooff, A.: Fine structure of fibroblasts in the periodontal ligament of the rat incisor and their possible role in tooth eruption. Arch Oral Biol, 19:1087-1098, December, 1974.
65. Zajicek, G.: Fibroblast cell kinetics in the periodontal ligament of the mouse. Cell Tissue Kinet, 7:479-492, September, 1974.
66. Bellows. C.G.; Melcher, A.H.; Aubin, J.E.: Contraction and organization of collagen gels by cells cultured from periodontal ligament, gingiva and bone suggest functional differences between cell types. J Cell Sci, 50:299-314, August, 1981.
67. Tashjian, A.H. and Levine, L.: Epidermal growth factor stimulates prostaglandin production and bone resorption in cultured rat calvaria. Biochem Biophys Res Commun, 85:966-975, December, 1978.
68. Ng, K.W.; Partridge, N.C.; Niall, M. *et al*: Stimulation of DNA synthesis by epidermal growth factor in osteoblast-like cells. Calcif Tissue Int, 35:624-628, July, 1983.
69. Kumegawa, M.; Hiramatsu, M.; Hatakeyama, K. *et al*: Effects of epidermal growth factor on osteoblastic cells *in vitro*. Calcif Tissue Int, 35:542-548, July, 1983.
70. Gospodarowicz, D.; Bialecki, H.; Thakral, T.K.: the angiogenic activity of the fibroblast and epidermal growth factor. Exp Eye Res, 48:501-514, May, 1979.
71. Thesleff, I.; Ekblom, P.; Keski-Oja, J.: Inhibition of morphogenesis and stimulation of vascular proliferation in embryonic tooth cultures by a sarcoma growth factor preparation. Cancer Res, 43:5902-5909, December, 1983.

AIDS IN UGANDA

In southern Uganda, on the shores of Lake Victoria, is a nondescript village called Kasensero. It is somewhat unusual in that its population consists largely of fishermen, black marketeers and bar girls. Its only other claim to fame is that it is the village which has probably suffered more than any other in Africa, and possibly the whole world, from the ravages of AIDS. Its people have already coined their own nickname for the disease. They call it "Slim."

Local citizens say that since the first case in 1983, two hundred people, or a quarter of Kasensero's population, have died of Slim. John Ziwa, a fisherman, is more specific. He claims that, since he arrived in the village two years ago, there have been 112 victims. I ask John how he thinks one gets the disease, and he replies: "Through sex - that's the only way we know here." Have people changed their sexual habits then? "Some have stopped and some are carrying on. Some people die and some live. We follow the situation."

But the most worrying aspect of all is the rate of spread of the disease. Dr. Wilson Carswell is a Scottish doctor who was working at the main government hospital of Mulago for nineteen years until his recent unexplained expulsion from the country by the Ugandan authorities. He comments: "Look at the pattern, In 1984 there was one case a month; in early 1985 one case a week; by late 1985 there was one case a day; and there are currently four cases a day. Draw that on a graph and see where it takes you." Where it takes one is to the conclusion that the number of AIDS cases at Mulago Hospital has been doubling every five months for the last three years. This compared to a current doubling of cases every two years or more in the USA.

Hooper, E.: AIDS in Uganda. Swiss Review of World Affairs, 37:24-27, July, 1987.

Developmental enamel defects of the primary dentition in a group of Californian children

Wilfred A. Nation, DDS
Lars Matsson, LDS, Odont Dr
John E. Peterson, DDS, MS

Several case-control studies have been performed on the occurrence and etiology of developmental enamel defects in the primary dentition. From these studies it was established that a number of prenatal, perinatal, and postnatal disorders affecting the child can cause enamel defects.[1]

Only a few cross-sectional studies on the prevalence of developmental enamel defects in the primary dentition have been reported. These studies, conducted in different parts of the world, revealed a prevalence of hypoplasia varying from 0 to 60 percent.[2-7] Children from developing countries generally were found to display a high incidence of enamel hypoplasia, while a lower incidence was found in children from developed countries.[2-7]

In a study of children from Nigeria, a strong association between socioeconomic status and prevalence of developmental defects was found.[4] This, and the difference in frequency of hypoplasia between developing and developed countries, suggest possible differences in nutritional status, or obstetric and pediatric care, as the principle etiological factors in the variation in frequency of hypoplasia, seen in the epidemiological studies. These studies have not, however, ruled out the possible existence of a difference in predisposition to enamel developmental disturbances between different ethnic groups.

In the Nigerian study, comparisons were made regarding socioeconomic status between two extreme groups.[4] The children in one of the groups came from academic families who lived under optimal conditions, while the children in the other group came from communities with poor standards of living. In a developed country with a high socioeconomic status and a comparably narrow socioeconomic span, a correlation between occurrence of developmental enamel defects in the primary dentition and socioeconomic status may not be found. This, however, seems not to have been investigated.

The present study reports the occurrence of developmental enamel defects in the primary dentition of a group of children who were randomly brought to a dental school for a screening evaluation. Attention was focused on variations in prevalence of enamel defects between different ethnic and socioeconomic groups. In addition, the distribution of enamel defects in relation to the stage of mineralization, as well as to different groups of teeth, were studied.

MATERIAL AND METHODS

All children between three to six years of age coming to Loma Linda University School of Dentistry for an initial

Dr. Nation is Assistant Professor and Dr. Peterson is Associate Professor, Department of Pediatric Dentistry, Loma Linda University Dental School; and Dr. Matsson is Associate Professor, Department of Pedodontics, University of Umeå, Sweden.

Table 1 ☐ Number and percentage of children with developmental enamel defects in relation to ethnic group.

	Number of children	Affected children					
		Total		Hypoplasia		Opacities	
		n	%	n	%	n	%
Total	300	99	33.0	62	20.7	37	12.3
Caucasian	197	74	37.6	45	22.8	29	14.7
Hispanic	73	13	17.8	7	9.6	6	8.2
Black	20	12	60.0	10	50.0	2	10.0
Asian	10	0					

screening evaluation, during the school years 1981 and 1982, and from whose primary dentition no more than two teeth were missing, took part in the study.

The dental examination was performed by one of the authors (WN) using the overhead dental light, mirror and explorer, after careful drying of the teeth with either compressed air or cotton rolls. Plaque was removed, if necessary, for the examination. Each examination was done in a systematic manner by checking first the buccal or facial, and then the occlusal and lingual surfaces from tooth A (upper right molar) through T (lower right molar).

All enamel defects except those resulting from caries were recorded. When the examiner was unsure whether a defect was developmental or carious in nature, the latter diagnosis was used and the defect was not included in the study. Developmental enamel defects were classified in two groups:

- ☐ Hypoplasia, defined as a clear lack of continuity of the enamel surface.
- ☐ Opacity, defined as a white, opaque or discolored area with an intact enamel surface. When a hypoplastic and an opaque spot were seen together, the defect was recorded as hypoplastic.

The chart for reducing data allowed defects to be classified according to the portion of the tooth involved. Defects were recorded as either buccal or lingual. The buccal portion of the proximal surfaces was recorded as belonging to the buccal surface, and the lingual portion of the proximal surfaces as belonging to the lingual. Defects situated on the occlusal surfaces were recorded as either buccal or lingual depending on which half it was closest to.

For all individuals, information about sex, ethnic group, and socioeconomic level was collected. The socioeconomic status was estimated indirectly by the use of the mother's and father's educational levels.[8] The parent having the highest level of education determined to which socioeconomic group the child belonged. The following levels were used: <H = less than high school, H = High school, C = some college, cc = completed college.

The parent of each child with enamel defects was interviewed in an effort to find an explanation for the defect. To standardize the interview, a check list was used which included many plausible causes for disturbances of enamel mineralization. Of the explanations offered by the parent, only those disorders to induce defects in enamel mineralization were accepted.

Table 2 ☐ Number and percentage of children with developmental enamel defects (hypoplasia and/or opacities) in relation to educational level of the parents. (CC = completed college, C = some college, H = High School, <H = less than High School)

	Number of children examined	Affected children	
		n	%
Total			
CC	86	30	34.9
C	89	27	30.3
H	88	33	37.5
<H	37	9	24.3
Caucasian			
CC	61	23	37.7
C	65	20	30.8
H	59	26	44.1
<H	12	5	41.7
Hispanic			
CC	11	2	18.2
C	17	4	23.5
H	25	6	24.0
<H	20	1	5.0
Black			
CC	9	5	55.6
C	6	3	50.0
H	2	1	50.0
<H	3	3	100.00

Based on Logan and Kronfeld's description of tooth development as recorded by McDonald and Avery, developmental enamel defects were subgrouped according to the stage of mineralization (prenatal, perinatal, postnatal).[9] This was determined by the location of the defect. In case of a defect belonging to more than one stage, it's grouping was determined by the highest number of teeth affected at that stage.

Statistical comparisons of the number of affected children between different ethnic groups (Tables 1 and 3) and between different subgroups of educational level (Table 2) were performed by means of the x^2 - test or the

Table 3 ☐ Number and percentage of children with developmental enamel defects (hypoplasia and/or opacities) in relation to stage of mineralization.

	Number of children	Affected children					
		Prenatal		Perinatal		Postnatal	
		n	%	n	%	n	%
Total	300	10	3.3	69	23.0	20	6.7
Caucasian	197	10	5.1	49	24.9	15	7.6
Hispanic	73	0	–	9	12.3	4	5.5
Black	20	0	–	11	55.0	1	5.0

Table 4 ☐ Number of children with developmental enamel defects arranged according to possible explanation for the defects.

	Number of children			Possible explanation
	Caucasian	Hispanic	Black	
Prenatal defects	1			Maternal diabetes
	1			Maternal hypocalcemia
	8			No explanation
Perinatal defects	1			Hypocalcemia
	5	2	1	Hyperbilirubinemia*
	1			Surgery
	1			Pneumonia
	1			Asphyxia
	40	7	10	No explanation
Postnatal defects	2			Pneumonia
	1			Severe diarrhea
	2			Milk intolerance
	1			High fever
	9	4	1	No explanation

*No information available about presence of hemolytic disease

Table 5 ☐ Number of children with developmental enamel defects (n = 99) arranged in groups according to number of teeth affected.

	Number of teeth affected									
	1	2	3	4	5	6	7	8	9	10-20
Number individuals	39	32	8	7	3	4	2	2	0	2

Fisher's exact test. Differences at the 5 percent level of probability were considered to be statistically significant.

RESULTS

No difference in occurrence of developmental enamel defects between sexes was noted; male and female results, therefore, are combined.

Of the 300 children examined, ninety-nine (33 percent) showed some kind of developmental enamel defect (Table 1); sixty-two children (21 percent) showed hypoplasia; and thirty-seven (12 percent) showed opacities only. Black children showed the highest rate of defects (60 percent), followed by Caucasians (38 percent), and Hispanics (18 percent). Of ten Asian children examined, none showed defects. The differences in the occurrence of all types of defects (hypoplasia and/or opacities) between Caucasian and Hispanic children and between Hispanic and Black children were statistically significant at the 0.5 percent and 0.1 percent levels of probability, respectively. The differences in the occurrences of hypoplasia between Caucasian and Hispanic children, between Caucasian and Black children, and between Hispanic and Black children were statistically significant at the 5 percent, 2 percent and 0.1 percent levels.

Only minor differences in the frequencies of enamel defects and no clear pattern were found when the results were subgrouped according to educational levels of the parents (Table 2). The lowest rate of defects was found in the group of Hispanic children whose parents had the lowest levels of education.

In all ethnic groups most defects were located at a level corresponding to the perinatal stage of enamel mineralization (23 percent). This was followed by the postnatal (6.7 percent) and prenatal (3.3 percent) areas (Table 3). All children with defects corresponding to the prenatal area were Caucasian. The incidence of defects in the area corresponding to the perinatal stage ranged from a high of 55 percent in Blacks and 24.9 percent in Caucasians to a low of 12.4 percent in Hispanics. The differences between Caucasian and Hispanic children, Caucasian and Black children, and Hispanic and Black children were statistically significant at the 5 percent, 1 percent and 0.1 percent levels, respectively. The postnatal stage showed a similar incidence among all groups.

In seventy-nine of the ninety-nine children with enamel mineralization defects no explanation for the defects was found in the patient's history (Table 4). Among the remaining twenty children, the parents provided a wide assortment of possible explanations, the most frequent being hyperbilirubinemia.

Seventy-one of the ninety-nine children with enamel

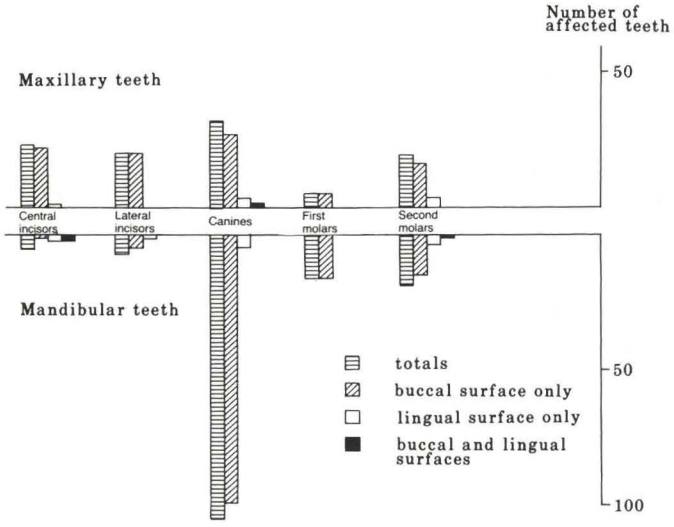


Figure. Affected teeth arranged according to types of teeth and surfaces.

mineralization defects averaged between one and two affected teeth (Table 5). Of a total of 250 teeth with defects the majority were mandibular canines (Figure). Least affected were the mandibular incisors; most defects were found in the buccal surfaces.

DISCUSSION

The material in the present study consisted of children from an area in Southern California surrounding Loma Linda University. They came to the School of Dentistry for a dental screening evaluation. Thus, the selected character of the sample and limited number of children in each subgroup imply that the results should be interpreted with care. Nevertheless, the results from this study may form a basis for future directed studies on larger groups of children.

In the present study 33 percent of the children examined displayed developmental enamel defects (hypoplasia and opacities) in one or more primary teeth. Enamel hypoplasia was found in 21 percent of all children. Compared to the prevalence of hypoplasia found in studies carried out in other developed countries, this figure is high. Holm and Arvidsson reported a prevalence of 5 percent in Swedish preschool children; and Murray and Shaw, a prevalence of 4 percent in English children.[6,7] The prevalence of enamel defects of all types, such as hypoplasia and opacities, was similar in the present study, to that reported by Murray and Shaw; but considerably higher than reported by Holm and Arvidsson.[6,7]

In the studies performed in developing countries, opacities were not recorded; but in most communities studied, the prevalence of hypoplasia far exceeded that found in the present study.[2-5] The differences between the various investigations may partly be explained by differences in the diagnostic criteria. This may be true especially for minor opaque disturbances, but the disparity in prevalence of hypoplasia is difficult to explain merely by differences in the criteria used. The results from the present study indicate, therefore, that there are differences in occurrence of developmental enamel defects not only between children from developed and developing countries; but also between children from different developed countries.

Enwonwu, in a study of children from Nigeria, reported a strong association between socioeconomic status and prevalence of developmental enamel defects in the primary dentition.[4] His material represented a wide variation in socioeconomic status, and the children from low socioeconomic communities in general had a poor nutritional status. The present study was performed in a country with a high socioeconomic level and a comparatively narrow socioeconomic span. Accordingly, no similar correlation between presence of enamel defects and socioeconomic level was found. On the contrary, the present study revealed a lower prevalence of enamel mineralization defects in Hispanic children compared to Caucasian children, in spite of the fact that a comparably higher number of the Hispanic children came from families with low educational levels. The difference between Hispanic children and Caucasian children, therefore, seems to be ascribed to factors directly or indirectly related to ethnic groups. This is further strengthened in the comparisons between Caucasians and Hispanics with similar socioeconomic backgrounds, where a lower prevalence of defects was noted in the Hispanic children in all groups of parental educational levels. For example, there may be general differences between various ethnic groups as to a predisposition to develop enamel disturbances, due to variations in susceptibility to trauma in the cells taking part in amelogenesis, or to a difference in the tendency to develop general disorders or diseases indirectly affecting amelogenesis. Another explanation of the differences between ethnic groups may be variations in ways of living, such as dietary habits.

In the present study, most of the enamel defects were located at a level of the teeth corresponding to the perinatal stage of mineralization, followed by the postnatal stage. This is in agreement with the results from the studies by Massler *et al* and Massier and Schour.[10,11] These authors reported that prenatal defects were very rare. In the present study, however, ten out of

ninety-nine children with enamel defects displayed prenatal disturbances. This variance may be partially explained by differences in diagnostic criteria, but in the present study most of the prenatal defects were very evident. In fact five children had more than four affected teeth, and four children had hypoplasia.

The tendency to develop enamel defects during the postnatal period was about the same in all ethnic groups. A difference was seen, however, during the prenatal and perinatal periods. Thus all prenatal defects were found in the Caucasian children; and during the perinatal period, defects were twice as common in the Caucasians as in the Hispanics. It has been shown that a number of disorders, for example, nutritional deficiencies and different diseases affecting the mother during the pregnancy, can cause developmental enamel defects in the primary dentition. The comparatively high prevalence of developmental defects in the Caucasian children found at the level corresponding to the prenatal period of tooth development, therefore, might indicate a higher rate of nutritional deficiencies or diseases during the pregnancies of Caucasian mothers. In the same way, the higher prevalence of enamel defects corresponding to the perinatal period may reflect a higher prevalence of disorders relating to the delivery. On the other hand, the higher prevalence found in the Caucasian children may also be a result of a higher susceptibility to trauma in the cells taking part in the amelogenesis.

In the present study, the lowest prevalence of defects was found in the mandibular incisors. This is in agreement with the findings by Murray and Shaw, and may be explained by the fact that the major parts of the crowns of these teeth are formed during intrauterine life, and during a comparatively short period of time.[7] The most frequently affected teeth were the mandibular canines. In the study by Murray and Shaw, however, the mandibular second molars were the most commonly affected teeth.[7] We have no explanation of the high prevalence of defects in the mandibular canines found in the present study. As most of the defects of the mandibular canines were located on the buccal surfaces and at a level corresponding to the perinatal period of tooth formation, the defects may hypothetically be ascribed to the protruded location of these teeth during tooth formation. During the act of birth, furthermore, trauma of the canines is induced by compression of the lower buccal alveolar wall by pressure from the maxilla. Skinner and Tat Wai Hung also suggests a relation between minor trauma to the canines of the neonate and the hypoplastic defect.[13] This, however, needs further investigation.

REFERENCES

1. Pindborg, J.J.: Aetiology of development enamel defects not related to fluorosis. Int Dent J, 32:123-134, July 1982.
2. Sweeney, E.A. and Guzman, M.: Oral condition in children from three highland villages in Guatemala. Arch Oral Biol, 11:687-698, December, 1966.
3. Sweeney, E.A.; Cabrera, J.; Urrutia J. *et al*: Factors associated with linear hypoplasia of human deciduous incisors. J Dent Res, 48:1275-1279, November- December, 1969.
4. Enwonwu, C.O.: Influence of socio-economic conditions on dental development in Nigerian children. Arch Oral Biol, 18:95-107, January, 1973.
5. Infante, P.F. and Gillespie, G.M.: An epidemiologic study of linear enamel hypoplasia of deciduous anterior teeth in Guatemalan children. Arch Oral Biol, 19:1055- 1061, November 1974.
6. Holm, A-K. and Arvidsson, S.: Oral health in pre- school Swedish children. 1. Three-year-old children. Odontol Rev, 25:81-98, January, 1974.
7. Murray, J.J. and Shaw, L.: Classification and prevalence of enamel opacities in the human deciduous and permanent dentition. Arch Oral Biol, 24:7-13, January, 1979.
8. Johnsen, D.C.; Pappas, L.R.; Cannon, D. *et al*: Social factors and diet diaries of caries-free and high- caries 2- to 7-year-olds presenting for dental care in West Virginia. Pediatr Dent, 2:279-286, December, 1980.
9. McDonald, R.E. and Avery, D.R.: Eruption of the teeth: Local, systemic and congenital factors that influence the process. In: "Dentistry for the child and adolescent", 3rd ed. McDonald R.E., Avery, D.R., eds. St. Louis: The C.V. Mosby Co., 1978, p 71.
10. Massler, M.; Schour, I.; and Poncher, H.G.: Development pattern of the child as reflected in the calcification of the teeth. Am J Dis Child, 62:33-67, January, 1941.
11. Massler, M. and Schour, I.: Growth of the child and the calcification pattern of the teeth. Am J Orthod Oral Surg, 32:495-517, September, 1946.
12. Skinner, M.F. and Tat Wai Hung, I.: Localized enamel hypoplasia of the primary canine. J Dent Child, 53:197- 200, May-June, 1986.

Early diagnosis and prevention of impaction of the maxillary canine

Clinic

Peter W. Ngan, DMD
Terry Wolf, BS
Gerald Kassoy, DDS, MS

LITERATURE REVIEW

Impacted maxillary canines are often treated in the orthodontic office. Moyers noted the frequency of impacted teeth in descending order: mandibular third molar, maxillary canine, mandibular and maxillary second premolars, maxillary central incisor and so on.[1] It is prudent to distinguish between labially unerupted canines and palatally impacted canines. The palatal impaction is usually detected after the age of thirteen; surgical treatment is required, because palatally impacted canines very rarely erupt spontaneously. In this paper, the term impaction has been used for the palatally impacted canines.

The etiological factors of maxillary canine impaction are rarely mentioned in the professional literature. Moyers writes: "The maxillary cuspid follows a more difficult and tortuous path of eruption than any other tooth. At the age of three years, it is high in the maxilla, with its crown directed mesially and somewhat lingually.

Dr. Ngan is Assistant Professor, Ohio State University College of Dentistry.

Mr. Wolf is a Junior Student, Ohio State University College of Dentistry.

Dr. Kassoy is Clinical Assistant Professor, Ohio State University College of Dentistry.

It moves towards the occlusal plane, gradually uprighting itself until it seems to strike the distal aspect of the lateral incisor root, apparently becoming deflected to a more vertical position."[1] Figure 1 shows the position of the maxillary canine with respect to the roots of the lateral incisor and the premolar in a nine-year-old child. Moyers said that although there are hereditary patterns leading to impacted teeth, the etiological factors of most concern are prolonged retention of primary teeth, localized pathological lesions, and shortening of the length of the arch.

According to McBride, the failure of permanent teeth to erupt into their normal positions in the dental arches is usually due to a discrepancy between tooth size and over-all arch length.[2] Where such a disharmony exists, the teeth that erupt later in the series are either impacted or diverted from their normal eruption paths. In another study, Jacoby found, however, that 85 percent of the palatally impacted canines have sufficient space for eruption.[3] He explained that the bud of the maxillary canine is wedged between the nasal cavity, the orbit, and the anterior wall of the maxillary sinus. The bud of the lateral incisor and the premolar are located behind the canine's palatal surface. An arch-length deficiency will not allow the maxillary canine to "jump" the buds, the nasal cavity, or the sinus, in order to reappear in the palate. On the other hand, a canine can be palatally impacted, if extra space is available in the maxillary bone. This space can be provided by

- ☐ Excessive growth in the base of the maxillary bone.
- ☐ Space created by agenesis or peg-shaped lateral incisors.
- ☐ Stimulated eruption of the lateral incisor or the first premolar. As the lateral incisor has already erupted, the canine normally erupts along the distal margin of this tooth. When the lateral incisor root is misplaced or small, the canine may become impacted, providing there is enough space between the lateral root and the premolar.

Periapical or orthopantomographic radiographs are important for the detection or determination of the position and inclination of the impacted canines. They might provide erroneous information, however, concerning the available space for the canine in the maxillary bone. The topographic occlusal radiographs are necessary supplements. Moreover, the periapical and orthopantomographic films give information about the mesiodistal dimension of the maxillary bone. They ignore the labiopalatal dimension in which the impacted maxillary canine is involved. Oriented lateral and anteroposterior radiographs as advocated by Broadbent can

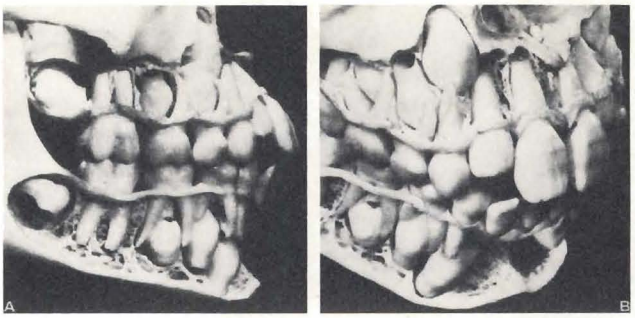


Figure 1. Dissected skull of a nine-year-old child, showing the position of the maxillary canine with respect to the roots of the lateral incisor and the premolar (From Frans P.G.M. van der Linden: Transition of the Human Dentition. Center for Human Growth and Development, The University of Michigan, Ann Arbor, Michigan 48109, 1982, p 103.)

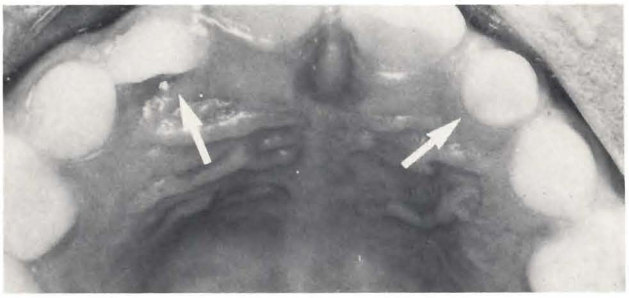


Figure 2. Intraoral photograph of the patient showing spacing of the maxillary incisors and the peg-shaped lateral incisors. The maxillary right lateral incisor is in distolingual version.

be used to locate the relative position of a maxillary canine.[4] Between eight and nine years of dental age, the maxillary canines are easily located in the lateral radiograph. The crown should be near the apex of the primary canine root, inclined mesially in relation to its root. The long axis of the canine should be somewhat parallel to the long axes of the incisors. In the frontal radiograph, the canines should be inclined only slightly medially, with the crowns below the level of the apices of the lateral incisors and well below the lateral border of the nasal cavity. The canine roots should superpose or lie laterally to the nasal cavity.

Clinically, a bulge should be palpable high on the alveolar process above the primary canine, approximately at the age of ten. If there is no evidence of a canine bulge, and the tooth appears to be tipped medially in the frontal radiograph, with the crown medial to the lateral border of the nasal cavity, a future impaction of the maxillary canine is a significant possibility.

CASE REPORT

T.N., a ten-year, one-month-old female presented to the Ohio State University pedodontic clinic for a consultation. Clinically, she had a mixed dentition with spacing of the maxillary permanent incisors. The maxill-

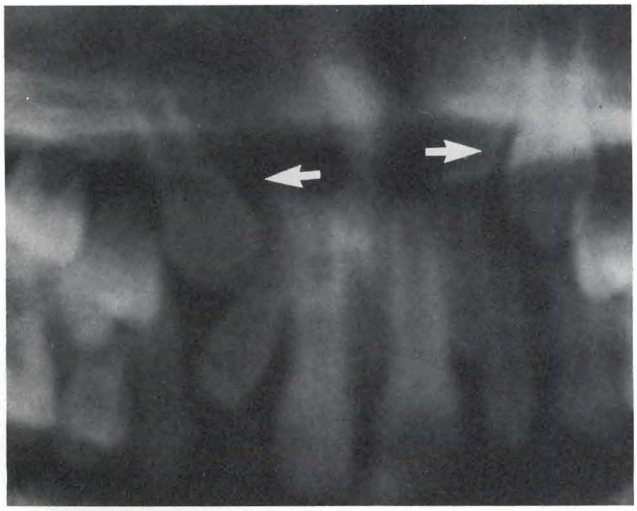


Figure 3. Orthopantomograph of the patient showing that the right maxillary canine was tipped mesially and was in a more medial position than the left canine.

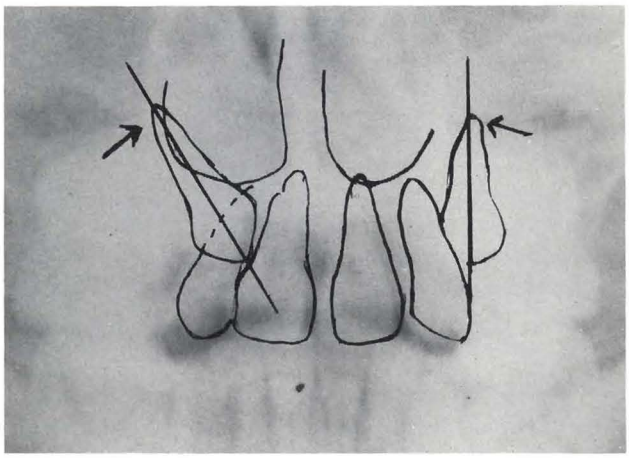


Figure 4. Frontal radiograph of the patient showing the medial tilting of the long axis of the maxillary right canine in relation to the lateral wall of the nasal cavity.

ary lateral incisors were peg-shaped and the right one was in distolingual version (Figure 2). All posterior primary teeth were present; and the dental age was estimated to be nine years and five months. Canine bulge could not be palpated on the right side. The mother reported that the maxillary primary incisors were extracted at approximately the age of four, because of extensive caries. There was no history of trauma.

The orthopantomograph made at the initial examination showed that the maxillary right canine was tipped toward the midline and more medially positioned than the left canine (Figure 3). The frontal radiograph also showed medial tilting of the long axis of the canine in relation to the lateral wall of the nasal cavity (Figure 4). The occlusal radiograph confirmed that the maxillary right canine is behind the lateral incisor and had displaced the root of the lateral incisor in a medial direction (Figure 5). The periapical film showed a surprising root dilaceration of the right lateral incisor. In contrast to the corresponding side, no resorption of the maxillary primary right canine was in evidence (Figure 6).

In light of the palatal and medial position of the maxillary right canine, it was decided to extract the maxillary

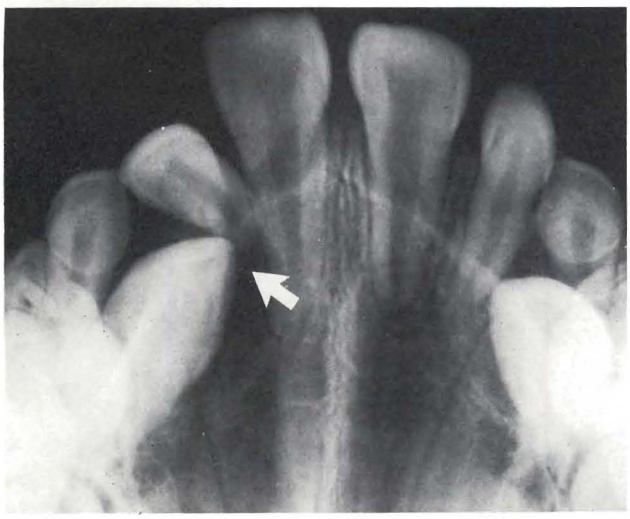


Figure 5. Occlusal radiograph of the patient showing that the position of the maxillary right canine is behind the lateral incisor displacing the lateral incisor in medial direction.

Figure 6. Periapical radiograph of the patient six months after the removal of the right primary canine. Note the position of the maxillary canine with respect to the root of the lateral incisor, which has a tortuous and dilacerated root.

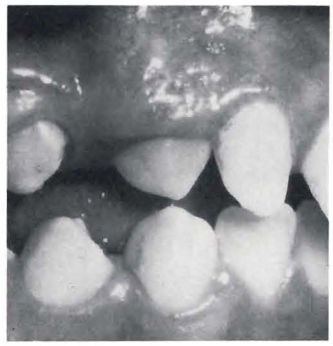


Figure 7. Intraoral photograph of the patient eighteen months after the removal of the primary canine.

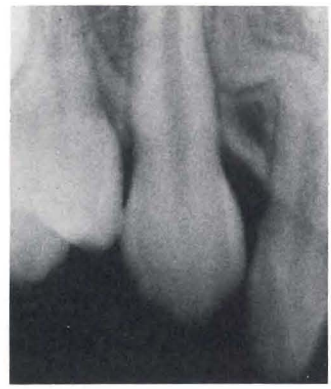


Figure 8. Periapical radiograph of the patient, eighteen months after the removal of the primary canine.

primary right canine and observe the path of eruption of the permanent canine, every six months with radiographs. After eighteen months, the canine erupted into a position that required only minor orthodontic treatment to guide it into a functionally correct position in the dental arch (Figures 7 and 8).

DISCUSSION

If the etiology of palatally impacted canines is related to the availability of excessive space in the maxillary bone, then the presence of spacing and of peg-shaped maxillary incisors should warn clinicians to observe the eruption of the permanent canines in children, between the ages of eight and ten. The absence of guidance by the lateral incisor could open a new course for a downward path on the palatal side. In this case, the presence of the tortuous dilacerated roots is responsible for the abnormal position of the lateral incisor as the canine strikes the distal aspect of the root of the lateral incisor. Together with the availability of space, the canine may be deflected to a more palatal course of eruption, instead of resorbing the primary canine. The removal of the primary canine as advocated by Williams, in cases with suspected canine impaction, can redirect the path of eruption of the permanent canine, since erupting teeth travel in the path of lease resistance.[5] It is recommended, therefore, in a growing child:

- ☐ Observation of the intrabony movement of the maxillary canine between the ages of eight and ten.
- ☐ Abnormal displacement of the lateral incisors with absence of canine bulges at the approximate age of ten should be followed by appropriate radiographs, including an orthopantomographic, occlusal, and periapical radiographs. If palatal impaction is suspected, frontal and lateral radiographs could clarify the mesiodistal and the labiolingual positions of the impacted canine.
- ☐ Early extraction of the primary canines may redirect the path of eruption of the permanent canines; and in the case of palatal impaction, early intervention may prevent a long period of orthodontic and surgical therapy.

REFERENCES

1. Moyers, R.E.: Handbook of orthodontics, 3rd ed. Chicago: Year Book Medical Publisher, Inc. 1973, pp 232, 526.
2. McBride, L.J.: Traction - A surgical/orthodontic procedure. Am J Orthod, 76:287-299, September, 1979.
3. Jacoby, H.: The etiology of maxillary canine impactions. Am J Orthod, 84:125-132, August, 1983.
4. Broadbent, B.H.: Ontogenic development of occlusion. Angle Orthod, 11:223-241, October, 1941.
5. Williams, B.: Diagnosis and prevention of maxillary cuspid impaction. Angle Orthod, 51:30-40, January, 1981.

TRANSFUSION-ASSOCIATED HEPATITIS AND AIDS

Patients and physicians need to understand that an absolutely safe blood supply is an unattainable goal, but that current approaches to donor selection and testing are highly effective in minimizing the risk of transfusion-transmitted infection.

Bove, J.R.: Transfusion-associated hepatitis and AIDS. N Engl J. Med, 317:242-245, July 23, 1987.

Effect of etchant time on microleakage

Gary A. Crim, DMD, MSD
Jeffrey S. Shay, DMD

The first reported use of an acid conditioning agent for enamel was by Buonocore in 1955. Since that time, this procedure has become a standard technique in the placement of composite restorative materials, fissure sealants, resin and porcelain veneers, orthodontic attachments, and acid-etched fixed partial dentures. Etching of the enamel is used to remove residual contaminants, raise the surface energy of enamel, and create micropores, into which a resin may flow. The result is an increase in the bond-strength of resins to enamel and a reduction in marginal leakage.

The conditioning of enamel for one minute is customary.[1-5] The necessity of etching for this length of time is questioned. Tensile bond investigations have shown no reduction in bond-strengths, when the etch time was limited to fifteen to thirty seconds.[6-8] Further, a study using the scanning electron microscope showed no differences in enamel morphology with reduced etchant time.[8]

The purpose of this study was to assess the effect of reduced etchant time on the microleakage of a composite resin utilizing a dentin bonding agent in class V preparations.

Dr. Crim and Dr. Shay are Associate Professors in the Department of Restorative Dentistry, School of Dentistry, University of Louisville, Louisville, KY 40292.

MATERIALS AND METHODS

The materials used were the visible, light-activated, composite resin, Silux; and the dentin adhesive, Scotchbond VLC[a].

Extracted noncarious human molar teeth were hand-scaled, cleaned with a slurry of flour of pumice, and stored in tap water at room temperature. Class V cavities were prepared in the middle third of the teeth, using No. 170 plain tungsten carbide burs in a high speed handpiece equipped with a water spray. New burs were frequently employed. The margins of all preparations were surrounded by enamel and the cavosurface margin was finished with a small bevel, using a No. 170 bur.

The cavities were washed with tap water and dried with compressed air. The teeth were randomly divided into three groups of ten specimens each, and etched with an etchant gel for fifteen seconds, thirty seconds, and sixty seconds, respectively. Ten additional specimens were not etched and served as the control group. The etched preparations were washed with tap water for sixty seconds, to ensure removal of the reaction products, and the cavities were dried with oil-free compressed air.

The preparations were primed with one layer of Scotchbond, which was blown to a thin film and cured fifty seconds with a Coe-Lite.[b] Using a syringe, Silux

[a]3M Company, St. Paul, MN 55144
[b]Coe Laboratories, Chicago, IL 60658

was inserted in the cavity and contoured with an anodized aluminum instrument. The restoration was cured for forty seconds with the Coe-Lite.

The gross excess of composite resin was removed after fifteen minutes, with a No. 170 bur. The restoration was finished with a medium silicon carbide disk, lubricated with cocoa butter. The restored teeth were placed in water at room temperature for twenty-four hours. The specimens were coated with one application of a clear nail polish, leaving 1 mm around the restoration, free of polish. Aluminum foil was adapted over the coated roots and a second application of polish was used to seal the edges of the foil.

The restorations were then subjected to 100 temperature cycles. A cycle consisted of twenty-three seconds at 37°C, four seconds at 12°C, twenty-three seconds at 37°C, and four seconds at 54°C. Following the cycling procedure, the teeth were placed in an aqueous solution of basic fuchsin dye for twenty-four hours. The teeth were retrieved, the foil and nail polish were removed, and the teeth were lightly pumiced to remove superficial dye. The teeth were sectioned longitudinally in a labiolingual direction through the center of the restoration. The sections were examined under a 25 X microscope and the extent of dye penetration at the occlusal and gingival margins was categorized as:

0 = no leakage.

1 = dye penetration short of the dentinoenamel junction.

2 = dye penetration to the dentinoenamel junction.

3 = dye penetration short of the axial wall.

4 = dye penetration to and along the axial wall. The data were analyzed by the nonparametric chi-square test.

RESULTS

The three different etch-times were equally effective in eliminating microleakage. No margin of any of these specimens presented measurable leakage. A significant difference ($p < 0.001$) existed between the etched groups and the nonetched control group. The following scores were recorded for the nonetched restorations: occlusal margins: three margins scored 0, four margins scored 1, and three margins scored 3; gingival margins: one margin scored 0, six margins scored 1, two scored 3, one margin scored 4.

DISCUSSION

This study supports the growing evidence that satisfactory marginal sealing can be achieved with reduced enamel etch-times. Specifically, it is possible to create good marginal sealing in enamel, following a fifteen-second etch. This shorted etching time will speed up the restoration of teeth and minimize the loss of enamel. The conservation of time is especially beneficial in the treatment of young patients.

An interesting observation is that the dentin bonding agent was not totally effective in minimizing leakage that had proceeded beyond the dentinoenamel junction in nonetched cavities. The reason is unknown, but might be due to compositional and structural differences, occurring in deep dentin. When etching was employed, however, this bonding agent provided a superior seal to that achieved by conventional resin bonding agents, investigated in this laboratory.[9-11]

CONCLUSION

Under the experimental conditions of this *in vitro* study, it would appear that it is not necessary to employ the extended acid etching times that have been generally accepted in the past. Etching enamel for fifteen seconds with an acid gel was as effective in eliminating microleakage as thirty or sixty-second etch-times.

REFERENCES

1. Ibsen, R.L. and Neville, K.: Adhesive restorative dentistry. Philadelphia: W.B. Saunders Co., 1974, p 43.
2. Sturdevant, C.M.: The art and science of operative dentistry. St. Louis: The C.V. Mosby Co., 1985, p 296.
3. Baum, L.; Phillips, R.W.; and Lund, M.R.: Textbook of operative dentistry. Philadelphia: W.B. Saunders Co., 1981, p 211.
4. Rupp, N.W.: Dental composites: state-of-the-art in dentin bonding. Ohio Dent J, 58:23-25, November, 1984.
5. Ford, T.R. Pitt: The restoration of teeth. Boston: Blackwell Scientific Publications, 1985, p 131.
6. Brannstrom, M.; Malmgren, O.; and Nordenvall, K.J.: Etching of young permanent teeth with an acid gel. Am J Orthod, 82:379-383, November, 1982.
7. Bates, D.; Retief, D.H.; Jamison, H.C.; *et al*: Effects of acid etch parameters on enamel topography and composite resin-enamel bond strength. Pediatr Dent, 4:106-110, February, 1982.
8. Barkmeier, W.W.; Gwinnett, A.J.; and Shaffer, S.E.: Effects of enamel etching time on bond strength and morphology. J Clin Orthod, 19:36-38, January, 1985.
9. Crim, G.A. and Mattingly, S.L.: Microleakage and the class V composite cavosurface. J Dent Child, 47:333-336, September-October, 1980.
10. Crim, G.A. and Mattingly, S.L.: Evaluation of two methods for assessing marginal leakage. J Prosthet Dent, 45:160-163, February, 1981.
11. Crim, G.A.; Esposito, C.J.; and Chapman, K.W.: Microleakage with a dentin-bonding agent. Gen Dent, 33:232-234, May-June, 1985.

Complications related to surgical removal of anterior supernumerary teeth in children

Åsa Högström, DDS
Lars Andersson, DDS

Supernumerary teeth are found most frequently in the anterior region of the maxilla. The single midline supernumerary tooth is most common, and the single unilateral supernumerary tooth is next in frequency.[1-4] Multiple bilateral occurrences have also been reported.[4]

The etiology is essentially unknown, but hyperactivity of the dental lamina is the most widely supported theory.[4] Males are affected twice as often as females.[1]

Enlarged follicular sacs, cystic formations, and eruptions of the teeth into the nasal cavity are described as complications associated with unerupted supernumeraries.[4-6]

Interferences with the adjacent teeth are reported as disturbances of normal eruption: displacement and formation of the diastema, root resorption, malformation, and loss of vitality.[4,5,7,8] When any of these complications occurs or is anticipated, removal of the supernumerary tooth is indicated. Opinions differ, however, concerning the optimal time for surgical intervention. Some recommend immediate removal, while others favor postponement until the root development of neighboring incisors is completed.[1,2] The possible disadvantages of immediate intervention are:

- ☐ Damage to adjacent teeth, which could lead to devitalization or malformation.
- ☐ Inability of a young child to tolerate the surgical procedure psychologically and thus create a potential dental anxiety.[4] The possible disadvantages of delayed intervention are:
- ☐ Loss of the eruptive force of adjacent teeth.
- ☐ Loss of the anterior arch.
- ☐ Midline shift.[4]

The purpose of the present investigation was to study the frequency of loss of vitality and sensitivity, resorption of root tissue, and disturbance of the root development of adjacent teeth, after removal of a supernumerary in the maxilla. In addition, the psychological effect of surgery at an early age was elucidated.

MATERIAL AND METHODS

Records of operated patients

Information was taken from records and radiographs of all forty patients who had anterior maxillary supernumerary teeth surgically removed in the department of oral surgery in Västerås hospital, during the years 1979 to 1983. The patients were classified in two age groups. In group 1, surgery had been performed before the age of eleven; in group 2, at the age of eleven or after. In group 1 patients, none of the teeth adjacent to the supernumerary teeth had completed root development; while in group 2, all of the adjacent teeth had completed root development. Table 1 shows the distribution by age and sex.

The indications for surgical removal are presented Table 2. According to the case histories, methods of anesthesia and preoperative sedation were individually

The authors are with the Department of Oral and Maxillofacial Surgery, Central Hospital, Västerås, Sweden.

Table 1 ☐ Distribution of study population by age and sex.

Group	Age	Girls	Boys	Number of patients
1*	6		1	1
	7		1	1
	8	2	7	9
	9	2	8	10
	10	1	1	2
2**	11	1	5	6
	12		1	1
	13	1		1
	14	2	1	3
	15	1	2	3
	16	1		1
	18	2		2
Totals		13	27	40

*Patients less than eleven years of age at time of surgery.
**Patients eleven years of age or older at time of surgery.

Table 2 ☐ Indications for surgical removal of supernumerary teeth.

	Group 1*	Group 2**	Number of patients
Disturbance of eruption	13	1	14
Orthodontic treatment	2	4	6
Formation of diastema	5		5
Other displacement	3	3	6
Enlarged follicular sac		3	3
Periodontal prophylactic		6	6
Totals	23	17	40

*Patients less than eleven years of age at time of surgery.
**Patients eleven years of age or older at time of surgery.

chosen for each patient (Table 3). A history of anxiety was one indication for sedation (N_2O or Diazepam) or general anesthesia.

The majority of the supernumeraries in this material were located palatally in the dental arch. Palatal approach was used in twenty-six patients, labial in ten, and both palatal and labial approaches in four patients.

The vertical position of the supernumerary tooth as it related to the adjacent incisor tooth is seen in Table 4. In the majority of cases, the supernumerary teeth were located in close relationship to the incisors. When bone had to be removed, a conventional bur technique, using a saline solution for cooling, was used. The removal was generally performed with forceps. In cases where separation of the supernumerary tooth was necessary, a groove was made in it before separation was completed with an elevator.

Postoperative records

These records were made during a three-year period beginning a year after surgery and ending three years later. The following conditions were recorded as positive or negative.

- ☐ Sensitivity: tested with an electric pulp tester.
- ☐ Loss of vitality: when periapical radiolucencies or endodontic treatment of the neighboring incisors were present.
- ☐ Root resorption: when radiographic signs of external or internal root resorption of neighboring teeth were present.
- ☐ Disturbance of root development: when radiographically observed disturbances of postsurgical root development of neighboring teeth were present.

Interview

The existence of dental anxiety was determined by interviewing the family dentist, the parents and the child. Cooperation during dental treatment, both before and after surgery was investigated. Also, the child's personal opinion of the operation was sought.

RESULTS

Loss of sensitivity and vitality

In three patients, a temporary loss of sensitivity was noted: two of these patients belonged to group 1, in one of whom the vertical position of the supernumerary was superior to the apex and, in the other, inferior to the cervical level. In the third patient, belonging to group 2, a color change of the crown was also noted. The color and sensitivity were normalized within the first year.

In only one patient, a permanent loss of vitality of an adjacent incisor occurred. The supernumerary was, in this case, situated close to the apex. This patient belonged to group 2. All the other teeth, in both groups, had a normal periodontal space.

Root resorption

On the root surface of one adjacent incisor, external resorption was noticed. The resorbed area did not show any progressive pattern, during follow-up examinations, and the periodontal space returned to normal width. Percussion sounds and mobility of this tooth were also normal, at the follow-up examinations. In three patients, exposure of adjacent roots was noted during surgery. None of these teeth, however, showed root resorption, during the follow-up period.

Disturbance of root development

In group 1, all the roots were found to develop normally after surgery.

Creation of dental anxiety

Eleven children, seven in group 1, and four in group 2, reported slight anxiety at the time of surgery. Two children from group 1 described the surgical operation as a "very negative experience".

According to interviews of the family dentists, the children, and the parents, later disturbances of behavior during dental treatment were noted in five children,

Table 3 ☐ Methods of anesthesia and type of sedation.

	Group 1*	Group 2**	Number of patients
Local anesthesia	4	13	17
Local anesthesia and diazepam	4	2	6
Local anesthesia and N_2O	1	1	2
General anesthesia, intubation	14	1	15
Totals	23	17	40

*Patients less than eleven years of age at time of surgery.
**Patients eleven years of age or older at time of surgery.

Table 4 ☐ Vertical position of supernumerary tooth as related to adjacent tooth.

	Group 1*	Group 2**	Number of patients
Superior to apex level	4	9	13
Between apex and cervix level	9	7	16
Inferior to cervix level	10	1	11
Totals	23	17	40

*Patients less than eleven years of age at time of surgery.
**Patients eleven years of age or older at time of surgery.

three from group 1 and two from group 2. In four of these, two from each group, lack of cooperation during dental treatment was noticed even before surgery.

DISCUSSION

This study showed that the surgical removal of a supernumerary tooth in the premaxilla did not cause any permanent complications, such as loss of sensitivity or vitality, root resorption or disturbance of root development, to the adjacent teeth with incomplete root development.

Although this study showed low frequencies of complications related to the surgery, it should be remembered that all supernumerary teeth should not necessarily be removed. Koch suggests that no immediate removal is necessary, if pathological conditions are absent, if no orthodontic treatment involving the region of the supernumerary tooth is planned, and if the child is seen regularly by a dentist.

It was found that dental pulps subjected to interruption of blood supply after luxation, will revascularize to a lesser extent, if the root development is completed at the time of trauma.[14,15] Teeth with fully developed roots may thus be regarded as more vulnerable to luxation at the time of surgery: for example, removal of the supernumerary tooth that is in close proximity to an adjacent tooth.

The findings of a temporary loss of sensitivity in the young age-group further supports this assumption. Consequently, early removal of a supernumerary tooth may possibly decrease the risks of interfering with the vitality of adjacent teeth.

Root development was not found to be disturbed in adjacent incisors, when surgical removal of the supernumerary tooth was performed before root development of the incisors was completed.

Root resorption was seen in only one case. Follow- up showed that this resorption was not progressive in nature, but of the "surface" type described by Andreasen.[16] This type of root resorption is related to a limited area of injury to the periodontium and is known to heal spontaneously without treatment.

The majority of children, regardless of age-group, did not experience the surgical operation negatively. The few children reported cooperating poorly during conventional dental treatment after surgery were reported cooperating poorly even before surgery.

On the condition that a careful history is taken and suitable methods of anesthesia and sedation are selected, this study does not indicate an increased risk of creating dental anxiety by performing surgery at an early age. It is the authors' opinion that sedation or general anesthesia as a complement to local anesthesia should be considered in children showing a history of anxiety.

CONCLUSION

If there are indications for early surgical removal of an anterior supernumerary tooth, the operation, according to the results of this investigation, neither impairs the prognosis for the adjacent teeth nor increases the risk for dental anxiety, compared to postponement of surgery until the root development of adjacent incisors is completed.

REFERENCES

1. Bergstrom, K.: An orthopantomographic study of hypodontia, supernumeraries and other anomalies in school children between the ages of 8-9 years – an epidemiological study. Sw Dent J, 1:145-157, 1977.
2. Grahnen, H. and Lindahl, B.: Supernumerary teeth in permanent dentition. Odont Revy, 12:290-294, 1961.
3. Luten, J.: The prevalence of supernumerary teeth in primary and mixed dentitions. J Dent Child, 34:346-353, September, 1967.
4. Primosch, R.: Anterior supernumerary teeth in primary and mixed dentitions. Pediatr Dent, 3:204-215, 1981.
5. Bodin, I.; Julin, P.; and Thomsson, M.: Hyperdontia III. Supernumerary anterior teeth. Dentomaxillofac Radiol, 10:35-42, 1981.
6. Thawley, S.E. and La Ferriere, K.A.: Supernumerary nasal tooth. Laryngoscope, 87:1770-1773, 1977.
7. Billberg, B. and Lind, V.: Medfödda antalsvariationer i permanenta dentitionen. Odont Revy, 16:258-272, 1965.
8. Jokela, M.: Rapid root resorption in central incisor caused by a supernumerary tooth – a case report. Proc Finn Dent Soc, 72:56-59, 1976.
9. Weber, F.N.: Supernumerary teeth. Dent Clin N Am, July, 1964, pp 509-517.
10. Day R.C.B.: Supernumerary teeth in the premaxillary region. Brit Dent J, 116:304-308, 1964.
11. Thoma, K.H.: Oral surgery. St. Louis: The C.V. Mosby Company, 1969, p 381.
12. Hertz, R.S.; Saunders, B.; and Wolk, R.S.: Dentoalveolar surgery. In Saunders, B. (ed) Pediatric Oral and Maxillofacial Surgery. St. Louis: The C.V. Mosby Company, 1979, p 173.
13. Koch, H.; Schwarz, O.; and Klausen, B.: Indications for surgical removal of supernumerary teeth in the premaxilla. Int J Oral Maxillofac Surg, 15:272-281, June, 1986.
14. Andreasen, F.M. and Vestergaard Pedersen, B.: Prognosis of luxated permanent teeth – the development of pulp necrosis. Endod Dent Traumatol, 1:207-220, December, 1985.
15. Andreasen, F.M.; Jhijie, Yu; and Thomsen, B.L.: Relationship between pulp dimensions and development injuries in the permanent dentition. Endod Dent Traumatol, 2:90-98, June, 1986.
16. Andreasen, J.O.: Traumatic injuries of the teeth. Copenhagen: Munksgaard, 2nd ed, 1981.

Epidemiology

Another perspective on children's dental needs and demand for services during the 1980s

H. Barry Waldman, DDS, MPH, PhD

Historically, the continuing need for dental services by children generally has focused on the results from various national and local studies that report oral conditions as observed by trained personnel. Another perspective on children's dental needs and demand* for services is provided by the National Center for Health Statistics in its annual report from the National Health Interview Survey. This series of reports is based upon perceptions of lay respondents. The expanded dental data presentation for 1983 and 1984 provides an opportunity to consider the current dental state (in terms of acute conditions**) of younger children (under five years of age) and older children (aged five to seventeen) as perceived by parents, guardians, and others.***[1,2]

Such an evaluation, to a greater or lesser degree, is subjective in nature and may be based upon factors related to economics, culture, and levels of education. (Data from professionally performed clinical studies will be presented to support some of these subjective parental views.) The use of these subjective evaluations does provide an opportunity, however,

- ☐ To review the need and demand for dental services from the perspective of the individual(s) who will be requesting dental services for the child.
- ☐ To evaluate the evolving use of dental services by children since the last recession. And further, the use of National Health Interview Survey data permits a review of some of the consequences of dental

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

Table 1 ☐ Total number of acute dental conditions, number per 100 persons, percent treated by age and gender: 1984.[2]

	Under 5 Years			5-17 Years		
	Total number	Number per 100 persons	Percent of conditions treated	Total number	Number per 100 persons	Percent of conditions treated
Male	437,000	4.7*	55.8*	154,000	0.7*	33.1*
Female	628,000	7.1*	74.7*	476,000	2.2*	22.9*
Total	1,064,000	5.9	66.9*	630,000	1.4*	25.4*

*Relative standard error of more than 30 percent.

Table 2 ☐ Dental treatment needs per 100 children age five by gender and race: 1979-1980.[3]

	White		Black and all others		Total	
	Male	Female	Male	Female	Male	Female
Primary surface restorations	122.4	117.1	139.1	168.4	125.4	125.9
Permanent surface restorations	5.5	4.1	0.0	5.3	4.3	4.4
Primary teeth extractions	10.1	6.4	14.9	3.8	10.9	5.9
Primary teeth need crowns	7.5	7.5	13.6	9.4	8.5	7.8
Permanent teeth and crowns	0.5	0.0	0.0	0.0	0.4	0.0
Pulpal treatment of permanent teeth	0.5	0.0	0.0	0.0	0.4	0.0
Gingival treatment needs*	12.8	13.7	17.6	16.3	13.7	14.2

*Gingival inflation adjacent to one half or more of the teeth present, and/or bleeding occurs around one or more teeth when the affected tissue is pressed gently with the side of a mirror head, and/or periodontal treatment by a dentist is indicated.

disease for different age children: Specifically, restricted activity, bed days and loss of school days.

NEED AND DEMAND FOR DENTAL SERVICES

In 1984, over a million acute dental conditions were reported for children under five years of age; 630,000 for children between five and seventeen years. Girls had more service needs per 100 persons than boys. While two thirds of these needs were treated for children under five years of age; for older children, one quarter of the needs were treated.† In addition, a greater percentage of the dental needs of girls under five years of age, as compared to boys, were treated (Table 1).

The reported difference in the percentage of conditions treated is borne out in national clinical examination survey data. By age five, young boys, as compared to young girls, are in need of more extractions of, and crowns for the primary teeth and more frequent treatment of the pulp of permanent teeth (Table 2).

The differences in dental needs by gender for five-year-old children are even more pronounced, when dental needs are considered by race: male black and "all

*Generally, need is defined as a biological and psychological state of health as perceived by trained health personnel. However, for purposes of the National Health Interview Survey, need is defined in terms of the respondent's perceptions.

A demand is related to market behavior as related to consumer wants, prices of health services, price of other goods and financial resources.

**A condition is considered acute if a) it was first noticed no longer than 3 months before the reference date of the interview and b) it is not a condition that is considered chronic regardless of the time of onset.[1]

***Changes in age cohort presentations and lack of particular data in earlier reports preclude comparisons over time.

†It should be noted that, for the most part, the ratio and percent data (e.g. acute dental conditions per one hundred persons and percent of acute conditions treated) used in the National Health Interview Survey report, have relative standard errors in excess of 30 percent and, therefore, should be used with extreme care.

The relative standard error of an estimate is obtained by dividing the standard error (i.e. primarily a measure of sampling variation) by the estimate itself and is expressed as a percentage of the estimate.[1]

other children" require more services than young white children. One particular exception from the generally greater need for dental services by young boys is the far greater need for restorations of the primary surfaces by female minority children than by their male counterparts (Table 2).

The difference in dental service needs occurs despite the slightly greater number of dental visits reported for two- to-five-year-old boys, as compared to young girls (Table 3).

It is of interest to note that, although male children under five years of age have fewer of their dental service needs treated, there are only minor differences by gender in the level of treatment provided to young children for all other acute medical conditions (Table 4).

Finally, parent and guardian respondents reported some variations by race and income in the number of dental visits and the percent of acute dental conditions treated for all children by race and income‡ (Tables 3 and 5). Of particular note was the progressive increase in the percentage of dental needs treated, associated with increasing family income.

Table 3 ☐ Number of dental visits per person from age two to less than five years by gender, race and family income: 1983.[4]

	Number of visits
Male	0.7
Female	0.6
Total	0.7
Race	
White	0.7
Black	0.5*
Other	0.5*
Income	
Less than $10,000	0.6
$10,000 - $19,999	0.5
$20,000 - 34,999	1.0
$35,000 +	0.7

*Relative standard error of more than 30 percent.

Table 4 ☐ Percent of acute conditions medically treated by age and gender: 1984.[2]

	Under 5 years		5-17 years	
	Male	Female	Male	Female
All acute conditions	75.9	77.5	49.1	47.3
Infective and parasitic	73.6	76.2	58.5	54.4
Respiratory	66.0	68.7	33.8	35.1
Digestive system	60.8*	84.9*	18.1*	25.6
Injuries	94.1	95.4	88.4	82.5
Selected other	87.4	88.8	58.0	74.8

*Relative standard error of more than 30 percent.

Table 5 ☐ Total number of acute dental conditions for persons under eighteen years, number per 100 persons, percent treated by race and family income: 1984.[2]

	Under 18 years		
	Total number	Number per 100 persons	Percent of conditions treated
Race			
White	1,404,000	2.7	49.6*
Black	239,000	2.5*	52.3*
Income			
Less than $10,000	172,000	1.6*	39.5*
$10,000-$19,999	774,000	5.7*	39.8*
$20,000-$34,999	448,000	2.4*	66.5*
$35,000 +	146,000	1.1*	100.0*

*Relative standard error of more than 30 percent.

RESTRICTED ACTIVITY DAYS

In 1984, children under eighteen years of age had almost five million restricted activity days,* associated with acute dental conditions. In total number of days and number per person, boys under five years of age had more restricted days, resulting from dental conditions. The situation was reversed in the five to seventeen-year cohort: girls had more restricted days, associated with dental conditions (Table 6).

In addition, black children and children in families with smaller incomes, had more restricted activity days, associated with acute dental conditions, than did white children and children from higher income families (Table 7).

NUMBER OF BED DAYS

In 1984, younger and older children had a total of more than 1.6 million days in bed,** associated with acute dental conditions. More bed days were reported for

- ☐ Boys under five years of age than young girls.
- ☐ Girls between five and seventeen years than older boys (Table 8).
- ☐ Black children and children in families with lower incomes than white children and those from higher income families (Table 9).

‡Based upon the greater number of restricted days, bed days and school days lost (see below) and the lower number of visits per person reported for black children, questions may be raised regarding the greater percentage of reported treated acute dental conditions for black children than for white children.

*Refers to a relatively short-term reduction in a person's activities below his or her normal capacity.[2]

**A day during which a person stayed in bed more than half of a day because of illness or injury.[2]

Table 6 ☐ Total number of restricted activity days associated with acute dental conditions, number per 100 persons by age and gender: 1984.[2]

	Under 5 years		5-17 years	
	Total number	Number per 100 persons	Total number	Number per 100 persons
Male	2,429,000	26.3*	257,000	1.1*
Female	543,000	6.2*	1,719,000	7.9*
Total	2,972,000	16.4*	1,976,000	4.4*

*Relative standard error of more than 30 percent.

Table 7 ☐ Total number of restricted activity days associated with acute dental conditions for persons under eighteen years, number per 100 persons, by race and family income: 1984. [2]

	Under 18 years	
	Total number	Number per 100 persons
Race		
White	3,513,000	6.9*
Black	1,436,000	15.1*
Income		
Less than $10,000	1,088,000	10.2*
$10,000-$19,999	2,112,000	15.5*
$20,000-$34,999	1,444,000	7.8*
$35,000 +	52,000	0.4*

*Relative standard error of more than 30 percent.

Table 8 ☐ Total number of bed days associated with acute dental conditions, number per 100 persons by age and gender: 1984.[2]

	Under 5 years		5-17 years	
	Total number	Number per 100 persons	Total number	Number per 100 persons
Male	450,000	4.9*	51,000	0.2*
Female	331,000	3.8*	799,000	3.7*
Total	781,000	4.3*	850,000	1.9*

*Relative standard error of more than 30 percent.

Table 9 ☐ Total number of bed days associated with acute dental conditions for persons under eighteen years, number per 100 persons, by race and family income: 1984.[2]

	Under 18 years	
	Total number	Number per 100 persons
Race		
White	1,169,000	2.3*
Black	1,142,000	4.9*
Income		
Less than $10,000	68,000	0.6*
$10,000-$19,999	1,201,000	8.8*
$20,000-$34,999	200,000	1.1*
$35,000 +	—	—

*Relative standard error of more than 30 percent.

Table 10 ☐ Total number of school days lost and days per 100 persons associated with acute dental condition for children age five through seventeen by gender, race and family income: 1984.[2]

	5-17 Years	
	Total number	Number per 100 persons
Male	257,000	1.1*
Female	1,505,000	6.9*
Total	1,762,000	4.0*
Race		
White	1,368,000	3.7*
Black	394,000	6.0*
Income		
Less than $10,000	53,000	0.8*
$10,000-$19,999	842,000	8.9*
$20,000-$34,999	757,000	5.8*
$35,000 +	52,000	0.5*

*Relative standard error of more than 30 percent.

NUMBER OF SCHOOL DAYS LOST

Dental conditions affect school attendance. In 1984, school age children missed more than 1.7 million school days, as a result of acute dental conditions.♣

As with the reports on restricted activity and bed days associated with dental conditions, blacks and children in families with lower incomes missed more school days than white children and children from higher income families. In addition, females, as compared to males, missed more schools days as a result of dental conditions (Table 10).

Throughout these data, it was reported that children from families with an income between $10,000 to $19,999 have had more restricted and bed days, more lost school days and, generally, a lower percentage of conditions treated than children from families in other income brackets. This difference may reflect economic difficulties faced by those families with incomes slightly above Medicaid income limits. Thus, children in low income families may receive needed dental services that are unavailable to children in slightly higher income families.

OVERVIEW

The substantial decrease in the prevalence of dental caries (in each age between five and seventeen years) has been reported repeatedly in lay and professional publications. Reports from the 1979/1980 National Dental Caries Prevalence Survey indicate that there is now half

♣A day in which a student five to seventeen years of age missed more than half a day from school in which he was enrolled.[2]

as much untreated dental caries in children (between five and seventeen years) as there was in the early 1970's.

Yet parent and guardian respondents in the National Health Interview Survey perceive that approximately one third of acute dental conditions of young children are untreated; approximately three quarters for children between five and seventeen years. In addition, they are aware of the association between the loss of activity days, bed days and school days and acute dental condition.

Again, it should be noted that some of the data used in this presentation are derived from subjective evaluations and should be interpreted carefully, because of the associated large relative standard errors. Nevertheless, such information does provide some awareness of the general perceptions of the individual who will be requesting dental services for the child and some indication of the potential future demand for dental services for children.

Since the last recession, there have been dramatic changes in the demand for the delivery of dental services to the general population. Results from the National Health Interview Survey indicate continuing perceived need for dental services for children of all ages, and an awareness of serious consequences of lack of treatment (including the restricted and bed days and lost school days, noted above).

The sum total of changing disease patterns, the evolving use of dental services and an awareness of the consequences of lack of dental treatment would seem to indicate that, although dental services for children have changed, there will be a continuing demand by parents and guardians for high levels of dental services for children in our communities.

REFERENCES

1. U.S. Department of Health and Human Services. Current Estimates from the National Health Interview Survey: United States, 1983. Series 10, No. 154, DHHS Publication No. (PHS) 86-1582. Washington, D.C.: Government Printing Office, June, 1986.
2. U.S. Department of Health and Human Services. Current Estimates from the National Health Interview Survey: United States, 1984. Series 10, No. 156, DHHS Publication No. (PHS) 86-1584. Washington, D.C.: Government Printing Office, July, 1986.
3. U.S. Department of Health and Human Services. Dental Treatment Needs of United States Children: 1979- 1980. NIH Publication No. 83-2246. Washington, D.C., Government Printing Office, December, 1982.
4. U.S. Department of Health and Human Services. Use of Dental Services: United States, 1983. NCHS Advance Data, #122. Washington, D.C., Government Printing Office, August 8, 1986.

A TIME FOR CAUTION

The AIDS epidemic frightens us all. But we should not allow our fear to cloud our judgement. Hasty and indiscriminate screening for antibody to HIV is imprudent and potentially dangerous, whether we suggest the tests to young women, require them of engaged couples, or impose them on our veterans. Although screening of blood donors and military recruits appears to have generated few false positive results, we do not know whether this performance can continue if the testing programs are expanded. Standardization and quality control should come first. These will take time and money; monitoring laboratory performance will require continuing effort, expenditure, and regulation.

If we want to test each other, we should make a deliberate choice of the threshold probability of infection above which we will screen. We should make explicit the tradeoffs implicit in any testing program. How many engagements should end to prevent one infection? How many jobs should be lost? How many insurance policies should be canceled or denied? How many fetuses should be aborted and how many couples should remain childless to avert the birth of one child with AIDS?

Meyer, K.B. and Pauker, S.G: Screening for HIV: Can we afford the false positive rate? N Engl J Med, 317: 238-241, July 23, 1987.

The diet-heart question: how good is the evidence?

Nutrition

Donald J. McNamara, PhD

Cardiovascular diseases (CVD) are the number one killer in most industrialized countries of the world. This multifactorial disease has a number of known risk factors which include cigarette smoking, high blood pressure and an elevated blood cholesterol level. While there is little debate relating these risk factors to CVD, there exists substantial debate regarding the role of dietary fat and cholesterol as contributing factors to blood cholesterol levels and CVD risk.[1,2] The problem facing health professionals is the extent to which high quality, nutritionally dense foods should be restricted in the diet of the average American based on the saturated fat and cholesterol content of these foods.

This paper will examine some of the questions which exist regarding the diet-heart disease relationship, potential problems associated with generalized dietary recommendations to the public and aspects of the mass intervention versus individualized treatment approaches to CVD risk reduction.

Plasma cholesterol and CVD risk: Whom to treat?

The initial question facing a health professional is what is a sufficiently high plasma cholesterol level as to warrant dietary intervention. Even this point, whom to treat, is debated within the biomedical community. To answer this question on an individual patient basis, one must consider the patient's overall risk profile, since an over-

Dr. McNamara is with the Department of Nutrition and Food Science, 309 Shantz Building, University of Arizona, Tucson, AZ 85721.

weight, hypertensive, cigarette-smoking male will be at a higher risk at a lower cholesterol level than a patient whose only risk factor is a high plasma cholesterol level.[3]

The most rational approach is to treat all the apparent risk factors with a goal to reduce the plasma cholesterol level to less than 230 mg/dl.[4] There is some indication that lower levels are advisable but the evidence indicating substantial CVD risk reduction by lowering plasma cholesterol below 200 mg/dl is still debatable.[3] In treating a hypercholesterolemic patient, attaining a cholesterol level of less than 230 mg/dl should achieve a significant reduction in CVD risk.

Which dietary goals to follow?

The American Heart Association's recommendation to lower plasma cholesterol levels includes reducing dietary cholesterol intake to less than 300 mg/day, reducing dietary fat to 30 percent of total calories and having dietary fat composed of equal parts of saturated, monounsaturated and polyunsaturated fat.[5] These recommendations apply to not only hypercholesterolemic individuals but every adult and child over the age of two years.[4] For those individuals with normal plasma cholesterol levels and in young children, it would seem reasonable to be more concerned about a balanced and nutritious diet than with an exclusionary policy towards animal product foods which play such an important role in our diets.[6] Prior to recommending that everyone consume such a "prudent diet," it would seem reasonable to consider the evidence that a dietary change will effectively reduce plasma cholesterol levels and CVD incidence. This evidence and related questions are discussed below.

Does epidemiological evidence support dietary change?

Numerous cross-cultural studies have demonstrated a relationship between plasma cholesterol levels and the dietary intake of saturated fats and cholesterol; however, studies within populations have not documented a clear relationship between dietary factors and plasma cholesterol levels. There are numerous reasons why such a relationship may not be demonstrable within a population; however, it may be that such relationships do not exist. At the present time, intrapopulation studies continue to confound a clear diet-heart disease relationship. Confusing the problem still further is the fact that there is evidence that in some populations increases in dietary saturated fat and cholesterol intakes have occurred without corresponding changes in CVD incidences.[7] Epidemiological evidence for a relationship between dietary patterns and disease incidence can only show trends and interactions and does not constitute proof of a causal relationship.

Do intervention trials support dietary change?

To date, there have been no conclusive dietary intervention trials supporting the proposed benefits in CVD risk reduction from initiation of a prudent diet.[3] While there are numerous reasons for the lack of definitive evidence from such trials, the absence of such evidence poses a serious question in the diet-heart disease debate. The recent Lipid Research Clinics Trial does provide evidence of CVD risk reduction in severely hypercholesterolemic men treated with the plasma cholesterol-lowering drug cholestyramine; however, extrapolation from a drug study in high-risk men to dietary interventions in the general public may not be justified since the interventions in the general public may not be justified since the interventions lower plasma cholesterol levels by very different mechanisms.[9]

Dietary and plasma cholesterol: are they synonymous?

For most of the public, there is a conceptual relationship between dietary cholesterol and plasma cholesterol, and the first dietary change made is a reduction in egg consumption. The question of whether dietary cholesterol contributes to plasma cholesterol levels has been extensively investigated without a clear resolution.[10]

The evidence for a dietary cholesterol effect, when considered within the ranges of dietary cholesterol most people consume, would suggest that if one exists, it is minimal. The reasons for this small effect are due to three factors: dietary cholesterol absorption varies from subject to subject with a mean fractional absorption of 60 percent; the rate of endogenous cholesterol synthesis is three-fold the amount obtained from an average diet; and, finally, most individuals express fairly precise feedback homeostatic regulation which reduces cholesterol synthesis in the body to compensate for an increased dietary cholesterol intake.[11] The combined effects of these regulatory factors may negate any substantial cholesterol-lowering benefit from a reduction in dietary cholesterol intake from the current 450 mg/day to the recommended 300 mg/day.[12] It should be noted,

however, that approximately one-third of the population lacks precise regulatory mechanisms and is sensitive to dietary cholesterol; these individuals can gain plasma cholesterol-lowering benefits from a reduction in dietary cholesterol intake.[13]

Does the quantity of fat have an impact on plasma cholesterol?

Dietary recommendations have proposed that individuals reduce their total fat intake from the current 40 percent of calories to 30 percent of total calories and that the fats consumed be 10 percent each of saturated, monounsaturated and polyunsaturated fats.[5] This reduction in total fat calories is to be compensated for by an increase in complex dietary carbohydrates. Two questions can be raised regarding the advisability of this recommendation: first, will such a change effectively reduce plasma cholesterol levels and, second, is the proposed shift in caloric intake free of any potentially harmful side effects.

Unfortunately, there is limited scientific evidence that a reduction in total fat calories will reduce plasma cholesterol levels in most people. The available studies do suggest that some individuals have a cholesterol-lowering benefit from reducing total fat calories; yet even in these studies, there is the complicating factor that often two variables have been changed, fat quantity and quality, and, in many instances, an increase in dietary carbohydrates is associated with an increase in plasma triglyceride levels.[14]

What is the impact of increasing CHO?

The second point regarding potential side effects of a shift in calories from fat to carbohydrates has been reviewed by Reaven.[14] The author points out that an increase in carbohydrate calories would be expected to increase plasma triglycerides, plasma insulin and plasma glucose. Some researchers believe that any one of these could be associated with an increased risk for CVD. Reaven also noted that the shift in calories could be especially dangerous to the elderly since carbohydrate metabolism is less well-regulated and there is a potential for glucose intolerance.

Does the quality of fat affect plasma cholesterol?

Numerous metabolic ward studies have demonstrated that plasma cholesterol levels can be reduced by shifting the dietary fat quality from a low to high polyunsaturated to saturated fat (P/S) ratio.[5] It should be noted, however, that changes in fat P/S similar to the dietary recommendations do not provide as consistent cholesterol- lowering benefits in all subjects tested. Many studies indicate that, just as with dietary cholesterol, there is a large degree of variability in response to an increase in dietary fat P/S ratio – some patients obtain a cholesterol-lowering benefit while others are relatively insensitive to the change.[15] While most of the studies over the past fifteen years have concentrated on the P/S ratio of dietary fat and its relationship to plasma cholesterol levels, recent studies have shown that monounsaturated fats, previously thought to be neutral in terms of plasma cholesterol levels, can significantly reduce plasma cholesterol levels and can play an important role in dietary therapy for those who need intervention.[16] Recent studies have also shown the plasma cholesterol- lowering benefits of incorporating high levels of omega-3 fatty acids in the diet.[17] These various studies raise a very important question which has yet to be answered: do we really know what dietary fat changes will effectively and safely lower plasma cholesterol levels in hypercholesterolemic patients?

How does caloric intake affect plasma cholesterol?

Probably one of the most overlooked aspects of the dietary recommendations to reduce CVD risk is the recommendation that people attain and maintain ideal body weight.[18] There is consistent evidence that obesity is a factor in hyperlipidemia and that weight loss can significantly reduce plasma cholesterol levels.[19] Weight reduction not only lowers plasma cholesterol levels but also reduces the elevated rates of cholesterol and lipoprotein synthesis commonly found in obese patients.[19] Weight reduction can be one of the most effective modalities for lowering plasma cholesterol levels.

How good is the evidence for individuals?

The data supporting a relationship between dietary fat and cholesterol intake and elevated plasma cholesterol levels must be evaluated not as mean values but on the basis of individual patient responses. There is no doubt that some individuals can lower their plasma cholesterol levels by decreasing dietary cholesterol or saturated fat intake; however, there is persuasive evidence that there are those for whom a dietary change within the range of the dietary recommendations will have little benefit.[19] Clinically, the problem is to determine, first, who needs

intervention and, second, how effective is the intervention. There is no component in generalized dietary recommendations to screen for hypercholesterolemia or to carry out the follow-up evaluation to determine the efficacy of the intervention.

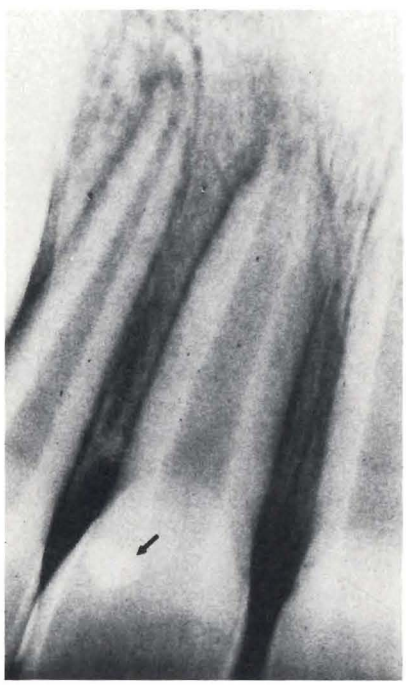
Summary

There is little doubt that dietary intervention should be the first approach to treating the hypercholesterolemic patient in an attempt to reduce his or her risk of heart disease. The questions raised in this review deal with the advisability of generalized dietary recommendations to the public and their potential benefit in reducing CVD incidence. A total risk factor intervention approach on an individual patient basis would seem to have greater potential for success than a generalized dietary recommendation.

REFERENCES

1. Samuel, P.; McNamara, D.J. and Shapiro, J.: Ann Rev Med, 34:179-194, 1983.
2. Food and Nutrition Board, National Research Council: Guidelines toward healthful diets. Washington, D.C.: National Academy of Sciences, 1980.
3. Stamler, J.; Wentworth, D.; and Neaton, J.D.: JAMA 256:2823-2828, 1986.
4. The Pooling Project Research Group. J Chron Dis, 31:201-306, 1978.
5. Grundy, S.M.; Bilheimer, D.; Blackburn, H. *et al*: Circ, 65:839A-854A, 1982.
6. American Academy of Pediatrics. Committee on Nutrition, Pediatrics, 78:521-525, 1986.
7. Marmot, M.G.; Booth, M.; and Beral, V.: Atherosclerosis Reviews, 9:19-27, 1982.
8. Mann, J.E. and Marr, J.W.: In: Metabolic aspect of cardiovascular disease. Edited by Miller, N.E. and Lewis, B. New York: Elsevier, 1981, pp 197-210.
9. The Lipid Research Clinics Program. JAMA, 251:351- 374, 1984.
10. McGill, H.C.: Am J Clin Nutr, 32:2664-2702, 1979.
11. McNamara, D.J.: Am J Clin Nutr, 41:657-658, 1985.
12. Lipid Research Clinic's Population Studies Data Book, Vol. 2, NIH Publication No. 82-2014. National Institutes of Health, Bethesda, MD, 1982.
13. Katan, M.B.; Beynen, A.C.; DeVries, J.H.M. *et al*: Am J Epidemiol, 123:221-234, 1986.
14. Reaven, G.M.: J Nutr, 116:1143-1147, 1986.
15. Wolf, R.N. and Grundy, S.M.: J Nutr, 113:1521-1528, 1983.
16. Grundy, S.M.: N Eng J Med, 314:745-748, 1986.
17. Goodnight, S.C.; Harris, W.S., Connor, W.E. *et al*: Arteriosclerosis, 2:87-113, 1982.
18. Barrett-Connor, E.L.: Ann Intern Med, 103:1010-1019, 1985.
19. McNamara, D.J.: Ann Rev Nutr, 7:273-290, 1987.

Reprinted from Contemporary Nutrition, with permission of General Mills Nutrition Department.



In the case report by Shulman and Corio, published in the May-June issue, arrows were mistakenly placed in Figure 5. The corrected figure is shown here.

Figure 5. Postorthodontic radiograph in November, 1982, showing presence of radiopacity with a halo of radiolucency.

Conference report

AIDS: children with HIV infection and their families

Linda P. Nelson, DMD, MScD
Manual M. Album, DDS

The first meeting to discuss pediatric AIDS took place in 1984; the second, in 1986. But the severity of the situation was not really brought to light until April, 1987, when the Surgeon General invited 150 health care providers, researchers, media representatives, and lay people to Children's Hospital of Philadelphia for a conference workshop. Surgeon General Koop charged the group with making recommendations to help set national policy regarding this growing problem. This review is based on the participation of the two pediatric dentists who were invited to the workshop and co-authored this manuscript.

DEFINITION

Childhood-acquired immunodeficiency syndrome (AIDS) is defined by the Centers for Disease Control (CDC) for the purposes of public programs, and health care planning and policy.[1]

Infants and children with perinatal infection up to fifteen months of age

Infection in infants and children up to fifteen months of age, who were exposed to infected mothers in the per-

Dr. Nelson is an Assistant Professor of Clinical Pediatric Dentistry in the Department of Pediatric Surgery, University of Pennsylvania School of Medicine. She is also director of Pediatric Dentistry and senior dentist at Children's Hospital of Philadelphia. Dr. Album is a Clinical Professor of Pediatric Dentistry at University of Pennsylvania School of Dental Medicine. He is also a senior dentist at The Children's Hospital of Philadelphia.

inatal period, may be defined by one or more of the following:

- ☐ The identification of the virus in blood or other tissues.
- ☐ The presence of HIV (human immunodeficiency virus) antibody as indicated by a repeatedly reactive screening test (for example, enzyme immunoassay) plus a positive confirmatory test (for example, Western blot, immunofluorescence assay) in an infant or child with abnormal immunological test results indicating both humoral and cellular immunodeficiency and meeting the requirements of one or more of the subclasses listed under P-2 (Table 1).
- ☐ Any child who meets the previously published CDC case definition for pediatric AIDS.[2,3]

The infection status of other perinatally exposed seropositive children up to fifteen months of age who lack the above immunological and clinical criteria is indeterminate, and these infants should be observed regularly for HIV-related illness and tested at regular intervals for persistence of antibody to HIV. Infants and children who become seronegative, who are virus-culture-negative (if performed), and continue to have no clinical or laboratory abnormalities associated with the HIV infection, are unlikely to be infected.

Older children with perinatal infection and children with other modes of transmission

HIV infection in these children is defined by one or more of the following:

- ☐ The identification of virus in blood or tissues.
- ☐ The presence of HIV antibody (positive screening test plus confirmatory test) regardless of whether immunological abnormalities or signs or symptoms are present.
- ☐ Any child who meets the previously published CDC case definition for pediatric AIDS.[2,3] These definitions apply to children under thirteen years of age. It is recommended that HIV infection in adolescents be classified according to the adult classification systems.[4]

EPIDEMIOLOGY

Data through mid-April, 1987, compiled by CDC showed the following statistics: of 35,397 total cases of AIDS in the United States, there were 600 cases of pediatric AIDS that fit the CDC definition in the United States; of these, 471 cases of AIDS were in children under the age of thirteen years, and 139 cases in children between the ages of thirteen and nineteen years. Sixty percent of these children have died. An additional 2,000 children had symptoms of disease that do not fit the CDC criteria for classification. It is expected that by 1991, over 3,000 children will have the disease.[5] Epicenters of the disease have been identified in the United States: New York City has the highest total number of cases of AIDS, followed by San Francisco, and Los Angeles. Of the total number of AIDS cases, 66 percent are homosexual, bisexual males, 17 percent have a positive history of IV drug abuse, 8 percent are homosexual males and have a positive history of drug abuse, 2 percent have a history of blood transfusions, 1 percent are hemophiliacs, and 3 percent of the total cases reported have no risk factor.[6]

Since 1981, 47,000 cases of pediatric and adult AIDS have been reported to the World Health Organization with most cases reported from central equatorial Africa. Fifteen to 20 percent of pediatric patients hospitalized

Table 1 ☐ Summary of the classification of HIV infection in children under 13 years of age.

Class P-0. Indeterminate Infection
Class P-1. Asymptomatic Infection
Subclass A. Normal Immune Function
Subclass B. Abnormal Immune Function
Subclass C. Immune Function Not Tested
Class P-2. Symptomatic Infection
Subclass A. Nonspecific Findings
Subclass B. Progressive Neurologic Disease
Subclass C. Lymphoid Interstitial Pneumonitis
Subclass D. Secondary Infectious Diseases
Category D-1. Specified secondary infectious diseases listed in the CDC surveillance definition for AIDS
Category D-2. Recurrent serious bacterial infections
Category D-3. Other specified secondary infectious diseases
Subclass E. Secondary Cancers
Category E-1. Specified secondary cancers listed in the CDC surveillance definition for AIDS
Category E-2. Other cancers possibly secondary to HIV infection
Subclass F. Other Diseases Possibly Due to HIV Infection

Table 2 ☐ Clinical abnormalities in infants with acquired immunodeficiency syndrome.[7]

Findings reported in >90 percent
Poor growth/failure to thrive
Chronic interstitial pneumonitis
Hepatosplenomegaly
Findings reported in 50 percent to 90 percent
Diffuse adenopathy
Findings reported in 10 percent to 50 percent
Protracted or recurrent diarrhea
Thrombocytopenia
Birth weight <2500 g
Eczematoid rash
Recurrent otitis media
Developmental failure
Microcephaly
Findings present in <10 percent
Kaposi's sarcoma
Chronic parotid swelling

in Zaire have AIDS-related symptoms. Brazil and the Carribean countries have increasing numbers of AIDS patients.

Twenty to 60 percent of children born to infected mothers will have the disease. Half of these congenitally infected infants are Black and one quarter are Hispanic. All will probably die. In many cases, the neonate is the index case leading to the diagnosis of the asymptomatic mother. On the other hand, it has been found that pregnancy may accelerate HIV expression in some affected women. Possible risk factors for the transmission of HIV infection from the mother to the infant include viral factors and IV drug abuse. False positive antibody testing of infants is possible, due to maternal antibody. Eighty percent of the infected children have at least one parent with the disease. HIV children are often orphaned or abandoned by both their natural parents and society. Foster parents are needed to help these children lead "normal, dignified" lives.

Presently, approximately 80 percent of the cases of pediatric AIDS have been the result of transmission by infected/or at-risk mothers to the child during birth, 12 percent of the cases have been transmitted by transfusion, 5 percent of the children have been diagnosed as Factor VIII or IX hemophiliacs, and 3 percent have no risk factors determined.

Approximately 17 percent of the children with HIV infection became infected by blood products. The incubation period from exposure to seroconversion is from six weeks to six months. From incubation time to symptoms appearing in the child is two to seventy-six months, with a mean of thirty-eight months. The mean age of perinatal AIDS is 2.9 years, predominantly in males. None of the reported cases of HIV infection in hemophiliacs was the result of screened heat-treated blood. AIDS is rarely seen in pediatric oncology patients or children on dialysis; but there is an increased risk of infection with increased transfusions. Transplacental transmission of HIV has been demonstrated, as well as infection of infants through breast milk infected with HIV from a postnatally infected mother.

CLINICAL PRESENTATION

A voluminous amount of information has been accumulated concerning the clinical manifestations of pediatric AIDS. The clinical picture of seropositive AIDS in the infant includes fever, failure to thrive, weight loss, diarrhea, diaphoresis, lymphadenopathy, hepatosplenomegaly, and chronic cough.[5] A summary of the clinical symptoms of pediatric AIDS is presented in Table 2. Other symptoms from infection with opportunistic pathogens include persistent oral esophageal candidiasis, despite appropriate therapy; lymphoid interstitial pneumonitis; encephalopathy; cardiomyopathy; protonemia; hepatitis; salivary gland enlargement; and recurrent herpetic lesions. Karposi's sarcoma and hairy tongue, although often seen in the adult AIDS patients are rarely seen in infants with AIDS.[6] Another characteristic of childhood AIDS is a shorter incubation time between HIV acquisition and opportunistic infection. It appears that the infant may develop symptomatic AIDS in three or four months from the time of viral infection.[7] Chronic lymphocytic interstitial pneumonitis (LIP), diagnosed from its histological characteristics, is often seen in infant AIDS and has been well documented in adults. In a seropositive infant, LIP is a diagnostic feature for AIDS.[8] Common bacterial infections occur frequently in childhood AIDS and account for significant morbidity and mortality; these include otitis media, pneumonia, and gram-negative sepsis.

The spectrum of clinical HIV infection is extensive, as it ranges from the asymptomatic, seronegative, but infected, child; through the symptomatic, seronegative child; to the asymptomatic, but seropositive child, including children with infectious mononucleosis-like illness, reflecting systemic viremia.[7] The natural history of the disease is still unclear.

Dr. Robert Marion of the Albert Einstein College of Medicine reported that newborns infected with HIV have identifiable facial features, which can be used to help identify the disease. The "fetal AIDS syndrome", as the pattern of facial characteristics is called, includes a small head, prominent forehead, a flattened nose, and very prominent eyes with a bluish tint in the sclera. It was reported that the traits can be found in up to 75 percent of the children infected with HIV. It should be noted that the presence of these facial characteristics is still being debated in the pediatric community.

THERAPEUTIC MODALITIES

Opportunistic pathogens are frequent finding in children with HIV. Compiled pediatric AIDS cases from CDC in January, 1986, documented the following data: Pneumocystis in 58 percent of the cases, disseminated cytomegalovirus in 19 percent, esophageal candida infection in fifteen percent, cryptosporidiosis in 6 percent, Karposi's sarcoma in 4 percent, and other opportunistic diseases in 22 percent. Ordinary infection may be life-threatening in these young children. Pneumocystis carinii pneumonia is the most common serious infection in

Table 3 ☐ Control and therapeutic tactics against AIDS.[7]

A. Prevention
  1. Intensive education programs to reduce risk
     Safe sex
     No shared needles
     Safe blood supply
  2. Deferment of pregnancy among high-risk subjects
  3. HTLV-III/LAV vaccine
B. Treatment of HTLV-III/LAV infection: Chemotherapy against retrovirus
C. Therapy of HTLV/LAV infection sequelae
  1. Immunologic reconstitution
     Bone marrow transplantation
     Transfer of immune competent cells
  2. Immunologic enhancement
     Cytokines (e.g. interleukin-2 and interferons)
     Immunologic adjuvants
  3. Aggressive treatment of infection and malignancy
  4. New therapy for refractory infections
     Mycobacterium avium-intracellulare
     CMV
     Cryptosporidiosis
     Pneumocystis carinii
  5. Prophylaxis against pneumocystis with trimethoprim sulfamethoxasole
  6. Gammaglobulin infusion
     ? Treatment for lymphocytic interstitial pneumonitis (LIP)
     ? Protection against bacterial and viral sepsis

Table 4 ☐ Investigational anti-infection therapies.[7]

Infection	Therapy
HTLV-III/LAV	Suramin
	Ribavirin
	HPA-23 (antimoniotungstate)
	Foscarnet
	Imuthiol
	3'-azido-3'-deoxythymidine (BW A509U)
Pneumocystis	DFMG
	Dapsone
CMV	DHPG (Syntex) or
	BW 759u (Burroughs-Wellcome) (acyclovir and analog)
	Foscarnet
Cryptosporidiosis	Spiramycin

children with AIDS, and oral candida or thrush is universal. Tables 3 and 4 summarize the therapeutic modalities currently in use. Vaccines are not currently available. The Public Health Service plan for the prevention and control of AIDS includes the following statement, "It is unlikely that a vaccine or therapy to limit transmission substantially will be generally available before 1990."

Oral candida infection is universal in both pediatric and adult AIDS patients, and extension of disease to the esophagus is common. Treatment in children includes oral antifungal agents such as, nystatin, ketoconnazole, or clotrimazole troches. Intravenous amphotericin has been used, when involvement has been extensive. Candida esophagitis may be complicated by concomitant herpes infection.[7] Herpes simplex infection is effectively managed with intravenous acyclovir.

PSYCHOSOCIAL PROBLEMS AND ATTITUDES

The psychosocial problems encountered by children with AIDS and the frightening picture it elicits set this disease apart from every other public health disorder.[9] Its onset influences everything important and dear to the patient, his family, and friends. Provider institutions, community agencies, and school systems become involved. Ethical issues become a matter of debate and create a schism in the health-care field and in the community itself.

Diagnosis of HIV infection in children creates a state of crisis in the family. It is important to discuss the details of the illness with the family and how to cope with the available resources to treat the illness, as well as the part the family must play in the total care of the patient. The way the crisis is handled will make the family stronger or weaker.[10] Response to the initial diagnosis creates shock and disbelief plus a sense of denial until the parent can gather all of the inner strength necessary to cope with the situation.

AIDS and its therapies are stressful for the young child and the adolescent. The high mortality rate and the young age of the patient are stressful enough, but the debilitating and disfiguring effects compound the stress factors. Patients with a fatal disease can often use denial as a defensive mechanism to control their predicament, but AIDS patients find such denial impossible, because of the constant coverage on radio, television, and in newspapers. Denial is intense when the mother belongs to a risk group and is infected herself.[9,11] This lack of acceptance can last for months and often will interfere with the medical advice, and the child subsequently suffers. Since many parents are carriers, the mother's focus may be on the child's symptoms, while not accepting the illness in herself. It becomes quite difficult to maintain a hopeful attitude about the future and the chances for cure. Young adolescents are often demoralized by newspaper reports. Older children inwardly worry about transmitting the disease to family and friends. Since the general population is somewhat ambivalent about AIDS, patients are denied some of the psychological benefits, such as nurturing, that other seriously ill patients receive.

It has been reported that at diagnosis, many health-care providers fail to recognize the AIDS patient's long-term and immediate need for psychological support.[12] It is important that psychological intervention by a mental health professional begin immediately. Thus, early intervention will help to resolve how medical costs will be paid, and how the patient will communicate with family and friends about AIDS.

Families adopting children with AIDS need to be aware of the implications of the diagnosis. Especially for those families where the parents have succumbed to the disease, foster parents must be found, and they must be helped in complying with the demands of the medical care.[11] The response of the community to the foster

family must also be considered. Ostracism and stigmatization may occur, when the diagnosis is known. Governmental agencies must provide support to the acquired families, if placement is to be successful.[14]

Children with symptoms of AIDS in later stages of the disease need help in eating, bathing, dressing and other basic activities of daily routine living. These simple tasks we normally take for granted have a tremendous impact on the patient and the people close to him.[13] The overwhelming emotional aspects of these simple tasks often interfere with therapy. Fear often causes the intelligent youngster to place excessive demands on the health-care providers and may lead to mistrust.

Feelings experienced by any patient approaching death become aggravated in AIDS children, because of the limited knowledge about the etiology, treatment, and transmission of the disease. Social abandonment, therefore, resolves itself in isolation.[9]

Support systems normally available to medically ill patients, which include the family and a network of friends and neighbors, are less available to the child with AIDS. The AIDS child lives in a "family reconstitution" with close relatives.[9] Young adult patients with illnesses such as leukemia become intensely reinvolved with their families of origin and rely on the family for financial and emotional support. Pediatric cancer patients have psychological intervention to help them regain a sense of autonomy. Their families try to develop ways of being directly involved with the youngster without undermining his sense of identity. In the child with AIDS, there is a wide range of mental status from psychological maturity to psychological dysfunction. These children who have psychological dysfunction require psychosocial intervention to permit them to cope with the ongoing complex problems that ensue. Almost constant staff intervention is required to control the periods of frustration and high anxiety, secondary to changes in medical status.

Because of the stigma of AIDS, and their own guilt feelings, parents often fear social rejection and withdraw from society in anticipation.[9,10,13] They feel that the traditional support system of friends, family, and clergy would not understand and that their confidentiality would not be protected. Parents become adept at answering and avoiding questions regarding the child's illness. This just prolongs the acceptance of the illness by the family. Family members also have other conflicts about the child with AIDS. When therapy is not successful and death is imminent, families usually wish to be close to the patient, yet fear of contagion arises. At times the families may request that the AIDS diagnosis not be indicated on the death certificate. Each crisis forces the family to deal with the death of the child. The parents often question the advisability of treating the HIV infection, when there is no effective antiviral drug available, thus creating untold suffering for the child.[9] Each family must be evaluated individually to identify the relationship's weaknesses and strengths in dealing with the HIV infection. Family resources for the duration of the illness must be ascertained.[10]

Health-care providers are subject to stress and often find they require support services, because of their involvement with the child with AIDS. The enormous demands on their own energies and the anxiety and fear for themselves and their families of the risk of contracting the disease is a daily stress factor. Staff people often become "family" to the child. The Social Work Department at Memorial Sloan-Kettering Cancer Center developed a psychosocial intervention program for patients, friends, and relatives and the center's staff in 1982.[9,15] The study revealed a major lack of home and supportive care resources and a lack of coordination between acute care facilities and community groups. Techniques for relaxation and behavior showed patients how to cope with anxiety related to medical procedures, pain, and stress. Education and programs for physicians and other professionals helped allay misinformation about the disease.

Community response has passed from panic to the establishment of information sharing groups, and mobilization of political groups to monitor services, as well as to advocate for patients.[9] This has resulted in the resolution of many psychosocial problems. With knowledge of AIDS and its probable mode of transmission, fears of health-care personnel about their own health, while treating these patients, were reduced. There is burnout, however, among service providers. This is due to the disproportionate increase in the number of AIDS patients and the limited number of health-care providers. Volunteers to help AIDS patients have decreased in number, and difficult psychosocial problems have not abated.

The sociocultural burden of a diagnosis of AIDS creates untold problems. The social stigma associated with the contagious aspect causes altered behavior in people, including physical and social avoidance. These children become isolated from schools and from other children by frightened parents.[16] The American Academy of Pediatrics has established recommendations for school, day care, and foster care attendance for children and adolescents with HIV infection. Hemophiliacs and recipients of blood transfusions receive sympathetic response, due to the perceived random nature of the exposure.[13] Although there is an increase in the number of people

whose disease is related to transfusions, the tendency is still to blame the victim. Parents of children with transfusion-related illnesses blame themselves for permitting the transfusion as well as the hospital for not allowing the family to give their own blood.

CONFIDENTIALITY

Fear of disclosure of name and identifying data is characteristic of the members of various risk groups.[17] This fear has been grudgingly understood by politicians, researchers, and public health officials.

The parents are confronted with how much must be explained to the child for fear that the child will naively explain to his friends he or she has AIDS. Protecting siblings is impossible, and attempts only increase anxiety and stress. Honest answers according to the child's age and understanding concept will help eliminate unforeseen problems.[10] Professional counseling is important, when the parent cannot handle the situation.

INITIATIVES IN THE PEDIATRIC AIDS AREA

Responding to recommendations developed by conference workshops to remedy some problem areas, the U.S. Surgeon General, C. Everett Koop, announced several action plans to be undertaken in the pediatric AIDS area. In response to the confusion over defining actual pediatric AIDS cases, and citing the new pediatric AIDS definitions (see above), Koop said that the new clinical classifications will be used in comparable longitudinal studies of progression of the disease and in developing multicenter studies of various therapeutic interventions.

In response to the legal and ethical concerns of drug trials in children, Koop said that because many young AIDS patients are under the guardianship of child welfare agencies, these agencies must be educated about pediatric AIDS, as well as the risks and benefits of various treatment protocols. He also noted that child abuse is emerging as one of the means of transmission.

The Surgeon General agreed that pilot studies on the most efficacious methods of newborn detection, including the examination of core blood, newborn assessment and follow-up, and laboratory distinctions between maternal and infant antibodies, must be undertaken. In addressing the implications of breast milk transmission, especially in developing countries, Koop recommended against discouraging breast feeding. He noted that there is an immediate need for more intensified programs for IV drug abusers.

Koop also noted the need to develop a network of nurturing homes for the children with AIDS, in order to ensure their access to comprehensive services. He also said that he will be bringing together representatives from public and private sector organizations to discuss possible partnerships that will guarantee the availability of and access to these services.

Koop cited the need for better minority educational efforts, and indicated that a project for reaching minority leaders is already underway.[18]

The Surgeon General's Workshop on AIDS, The Children's Hospital of Philadelphia, April 6-8, 1987.

REFERENCES

1. CDC-Update: Acquired immunodeficiency syndrome AIDS - United States. MMWR, 32:688-691, 1984.
2. CDC-Classification system for human immunodeficiency virus (HIV) infection in children under 13 years of age. (Draft in press)
3. CDC-Revision of the case definition of acquired immunodeficiency syndrome for national reporting-United States. MMWR, 34:373-375, 1985.
4. CDC-Classification system for human t-lymphotrophic virus type III/lymphadenopathy-associated virus infection. MMWR, 35:334-339, 1986.
5. Koop, C.E.: Charge to participants: the surgeon general's workshop on children with HIV infection and their families. The Children's Hospital of Philadelphia, April 6-8, 1987.
6. Quinn, T.: Global epidemiology: the surgeon general's workshop on children with HIV infection and their families. The Children's Hospital of Philadelphia, April 6-8, 1987.
7. Barbour, S.D.: Acquired immunodeficiency syndrome of childhood. Pediatr Clin N Am, 34:247-268, 1987.
8. Parks, W.P. and Scott, G.B.: An overview of pediatric AIDS: approaches to diagnosis and outcome assessment. From AIDS: Modern concepts and therapeutic challenges edited by Samual Broder. New York: Marcel Dekker, Inc., 1987, pp 245-262.
9. Christ, G.H. and Wiener, L.S.: Psychosocial issues in AIDS. From AIDS, edited by Vincent Devita. Philadelphia: J.B. Lippincott, 1985, pp 275-297.
10. Boland, M.G.: The child with AIDS special concern. From The Person with AIDS, Nursing perspective, edited by J. Durham, F.L. Cohen. New York: Springer Publ, 1987, pp 192-210.
11. Boland, M.G.; Tasker, M.; Evans, P.N. *et al*: Helping children with AIDS: the role of the child welfare worker. Public Welfare, Winter 1987, pp 23-28.
12. Boland, M.: Management of children with HIV infection: the surgeon general's workshop on children with HIV infection and their families. The Children's Hospital of Philadelphia, April 6-8, 1987.
13. Holland, J.C. and Tross, S.: The psychosocial and neuropsychiatric sequelae of the acquired immunodeficiency syndrome and related disorders. Ann Intern Med (Am Col Phys), 103:760-764, 1985.
14. Ginzburg, H.M.: Legal and ethical issues: the surgeon general's workshop on children with HIV infection and their families. The Children's Hospital of Philadelphia, April 6-8, 1987.
15. Siegel, K.: Education to prevent HIV infection: the surgeon general's workshop on children with HIV infection and their families. The Children's Hospital of Philadelphia, April 6-8, 1987.
16. Kusknick, H.G.: A mother's point of view: the surgeon general's workshop on children with HIV infection and their families. The Children's Hospital of Philadelphia, April 6-8, 1987.
17. Cassens, B.J.: Social consequences of the acquired immunodeficiency syndrome. Ann Intern Med (Am Col Phys) 103:768-771, 1985.
18. Brown, J.: Surgeon general Koop reveals multiple initiatives in pediatric AIDS area. The AIDS Record, 1:1- 2, 1987.

Case reports

Delayed eruption due to overlying fibrous connective tissue

Curt Goho, DDS, CPT, USA, DC

Radiolucent lesions associated with the crowns of unerupted teeth are occasionally found during children's dental examinations. Those which cause excessive delayed eruption of the permanent teeth are of particular importance, due to their alteration of normal eruption patterns, and their indication of other possible pathological entities such as ameloblastoma, ameloblastic fibrous-odontoma, and dentigerous cysts.[1,2] These diagnoses are the ones normally implicated in causing delayed eruption.[3-5] Occasionally, however, nonpathological entities, such as overlying fibrous connective tissue, are also implicated in cases of delayed eruption.[6] Regardless of the final diagnosis of such lesions, they must be promptly treated, to prevent future problems with the child's eruption pattern, and to rule out other pathological entities.

CASE REPORT

A seven-year-old Caucasian girl was seen for a routine dental examination. All permanent first molars were clinically present, except the mandibular right first molar. Radiographs showed the tooth's eruption to be delayed, but within acceptable limits for the child's age. At age eight years, five months, the tooth still had not erupted. Radiographs showed no change in the tooth's position, and a definite radiolucent area around the crown of the unerupted molar (Figure 1). The molar was

Captain Goho is stationed at McDonald Army Hospital Dental Clinic, U.S. Army Dental Activity, Ft. Eustice, Virginia 23604. The patient was treated at Amberg Dental Clinic, Amberg, West Germany.

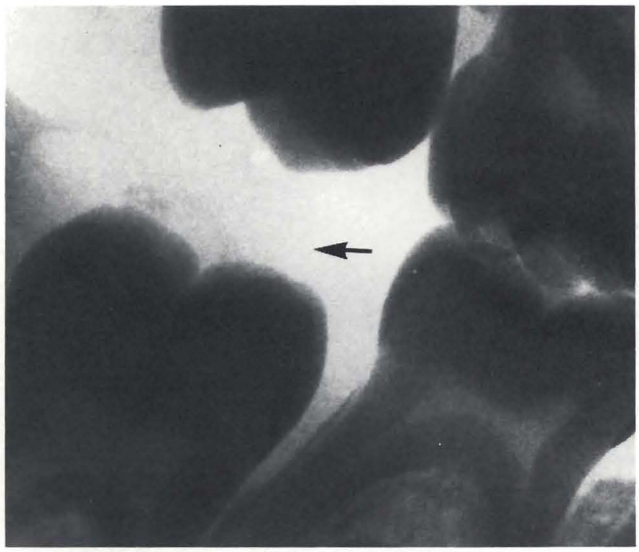


Figure 1. Unerupted right first molar with radiolucent area above the crown of the tooth.

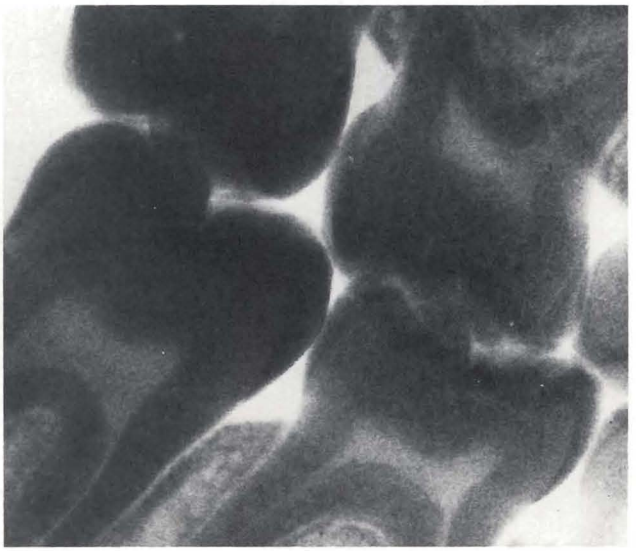


Figure 2. Four weeks postsurgery. Removal of the overlying fibrous tissue resulted in rapid correction of the eruption problem.

in an upright position with no blockage of the eruption path by any other teeth. Clinically, the overlying soft tissue was unremarkable. The patient was asymptomatic. The initial diagnosis was a cystic lesion, possibly a dentigerous cyst. Nonpathological entities were not considered in the differential diagnosis.

Treatment consisted of surgically removing the overlying soft tissue, exposing the crown of the molar. Initial gross examination of the excised mass revealed a very dense, fibrous tissue. Histological examination diagnosed the specimen as a proliferation of dense fibrous connective tissue, containing numerous odontogenic rests. There was no thin-walled lumen or stratified squamous cell lining, thus ruling out the initial diagnosis.

Examination of the patient four weeks later revealed rapid and satisfactory eruption of the first molar to a normal plane of occlusion (Figure 2).

This report shows the potential of a nonpathological and normally innocuous entity, dense fibrous connective tissue, to inhibit normal tooth eruption. Surgical intervention for rapid correction of the eruption schedule, and to rule out other morbidity, is the treatment of choice.

REFERENCES

1. Shafer, W.G.; Hine, M.K.; Levy, B.M.: Oral pathology, 3rd Edition. Philadelphia: W. B. Saunders, 1974, pp 239- 241.
2. Kaban, L.B.; Needleman, H.L.; Hertzberg, J.: Idiopathic failure of eruption of permanent molar teeth. Oral Surg, 41:155-163, August, 1976.
3. McDonald, R.E.: Dentistry for the child and adolescent, 2nd Edition. St. Louis: The C.V. Mosby Co., 1974, p 337.
4. Salzman, J.A.: Orthodontics: principles and prevention. Philadelphia: J.B. Lippincott, 1957, p 297.
5. Johnsen, D.C.: Prevalence of delayed emergence of permanent teeth as a result of local factors. JADA, 94:100-106, January, 1977.
6. Di Salvo, N.A.: Evaluation of unerupted teeth: orthodontic viewpoints. JADA, 82:829-835, April, 1971.

AIDS

Semantic differences aside, the data on AIDS have only recently been organized in such a fashion as to yield clues to the nature of the disease: its incidence, prevalence, epidemiologic features, associated diseases, and precipitating causes of mortality in AIDS patients.

Barr C.E. and Marder, M.Z.: AIDS
Chicago: Quintessence Books, 1987, p 13.

Root resorption in association with ectopic eruption: report of case

Lena Omnell, DDS, MSD
Dennis Sipher, DDS

The patient is a twelve-year-old healthy male who came to the Odessa Brown Children's Clinic for a routine dental examination. The oral hygiene was poor and generalized gingival inflammation was present. He had an Angle Class I molar and canine relationship on the right side and a Class II molar, Class I canine relationship on the left side. The maxillary midline deviated 1 mm to the left. The maxillary left second premolar was impacted lingually. It was decided that one tooth in the maxillary left quadrant had to be removed. the bitewing of the area (Figure 1) showed the impacted second premolar and adjacent teeth. Normally, extraction of the first or the second premolar would have been considered. The radiolucency over the mesial root area of the first molar, however, raised some concern. A second set of radiographs for stereoviewing was obtained (Figure 2) and the radiolucency on the first molar was interpreted as root resorption. Consequently, extraction of the first molar was suggested. A lingual flap was elevated over the impacted premolar and neighboring teeth. The area of root resorption of the first molar was verified with a probe and the tooth was extracted. The flap was sutured back in position and the postoperative healing was unremarkable. Figure 3 shows the extracted first molar with the resorption cavity and pulp exposure (arrow).

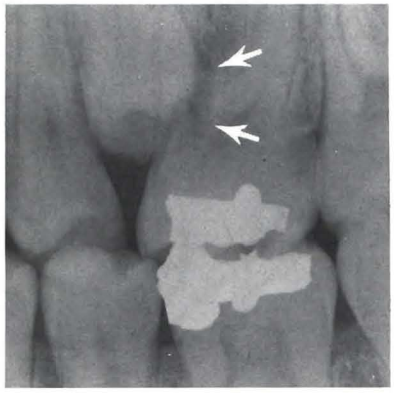


Figure 1. Bitewing radiograph of left premolar-molar area with impacted maxillary second premolar. Note radiolucency (arrow).

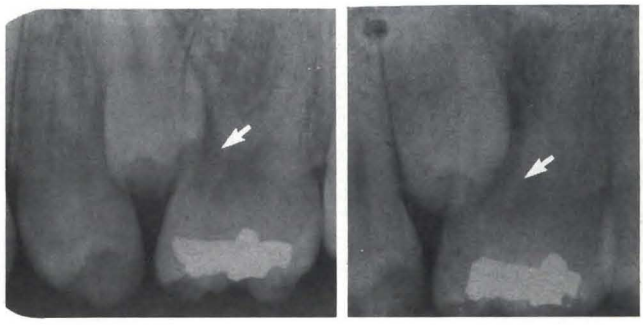


Figure 2. Stereopair of radiographs illustrating root resorption of the mesial root surface of the first molar.

Dr. Omnell is Research Assistant Professor, Department of Orthodontics and Department of Biological Structure, University of Washington, Seattle, Washington 98195; and Staff Orthodontist, Children's Hospital and Medical Center, Seattle, Washington 98105. Dr. Sipher is Director for Pediatric Dental Residency, Children's Hospital and Medical Center, Seattle, Washington 98105.

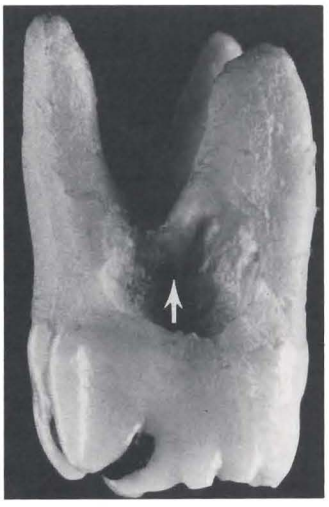


Figure 3. Extracted maxillary left first permanent molar showing resorptive lacunae and pulp exposure (arrow).

Root resorption of permanent teeth is a well- recognized pathologic process during orthodontic treatment, and in association with trauma, tumors and osteitis.[1] A somewhat less well-understood phenomenon is the idiopathic external root resorption that has been reported by Sullivan and Jolly (1957) and Newman (1975).[2,3] In addition, teeth in a normal healthy individual may be resorbed by a neighboring impacted tooth. The pressure exerted by the impacted tooth on the root surface of the adjacent tooth has been assumed to be the main cause of this type of root resorption.[1] Nitzan *et al* (1981) concluded, however, that the pressure from the unerupted tooth was not the only causative factor.[4] They found a marked difference in root resorption between males and females in their sample of 199 cases. Root resorption of a tooth in close proximity to an unerupted tooth was more frequent in males than in females. In both groups root resorption was more prevalent in younger individuals than in older. They also noted that the area close to the cementoenamel junction was the least affected.

Our patient showed extensive root resorption close to the cementoenamel junction. It was impossible to determine, however, where the resorption had started, since previous radiographs were not available. Root resorption induced by gingival connective tissue was recently described and it was assumed that granulation tissue derived from bone can induce root resorption after the integrity of the periodontal membrane has been disrupted.[5-9] The combination of an unerupted crowded premolar in close proximity with the gingiva, and gingival inflammation may have had an effect on the integrity of the periodontal membrane on the mesial root surface of the molar. These circumstances were assumed to be predisposing factors for osteoclastic activity, and to be the main reasons for this unusual site of root resorption, in our patient.

It is likely that impacted teeth anywhere in the dental arch can participate in the destruction of the root surfaces of their neighboring teeth. Maxillary impacted canines have often been linked to resorption of maxillary incisors.[1,10] Impacted mandibular third molars are sometimes assumed to cause resorption of the distal root surfaces of mandibular second molars.[4] In addition, Langford and Sims (1981) reported a case where distal movement of the first maxillary molar led to impaction of the second molar and resorption of the distal root surface of the first molar.[11]

Data from the literature as well as our case report suggest that unerupted, crowded teeth, if not immediately removed, in young individuals, should be followed radiographically in order to detect possible root resorption of adjacent teeth.

REFERENCES

1. Worth, H.: Principles and practice of radiographic interpretation, Ed. 2. Chicago: Year Book Medical Publisher, Inc., 1975, pp 163-172.
2. Sullivan, H.R. and Jolly, M.: Idiopathic resorption. Aust Dent J, 2:193, April, 1957 (abst.)
3. Newman, G.W.: Possible etiologic factors in external root resorption. Am J Orthod, 67:522-539, May, 1975.
4. Nitzan, D.; Keren, T.; and Marmary, Y.: Does an impacted tooth cause root resorption of the adjacent one? Oral Surg, 51:221-224, March, 1981.
5. Nyman, S.; Karring, T.; Lindhe, J. *et al*: Healing following implantation of periodontitis affected roots into gingival connective tissue. J Clin Periodontol, 7: 394- 401, October, 1980.
6. Andreasen, J.O.: Interrelation between alveolar bone and periodontal ligament repair after replantation of mature permanent incisors in monkeys. J Periodont Res, 16:228-235, March, 1981.
7. Boyko, G.A.; Melcher, A.H.; and Brunette, D.M.: Formation of new periodontal ligament by periodontal ligament cells implanted *in vivo* after culture *in vitro*. A preliminary study of transplanted roots in the dog. J Periodont Res, 16:73-88, January, 1981.
8. Karring, T.; Nyman, S.; Lindhe, J. *et al*: Potential for root resorption during periodontal wound healing. J Clin Periodontal, 11:41-52, January, 1984.
9. Magnuson, I.; Claffey, N.; Bogle, G. *et al*: Root resorption following periodontal flap procedures in monkeys. J Periodont Res, 20:79-85, January, 1985.
10. Sasakura, H.; Yoshida, T.; Murayama, S. *et al*: Root resorption of upper permanent incisor caused by impacted canine. Int J Oral Surg, 13:299-306, August, 1984.
11. Langford, S.R. and Sims, M.R.: Upper molar root resorption because of distal movement. Am J Orthod, 79:669-679, June, 1981.

Impacted primary incisor: report of case

Lipa Bodner, DMD
Itzhak Horowitz, DMD, MD

A tooth that fails to erupt into a normal functional position by the time it normally should is considered impacted.[1,2] The majority of impacted teeth are of the permanent dentition and are relatively common at the early mixed-dentition age.[1,3] Impaction of primary teeth is less common, but when it does occur it usually involves the first and second molars.[4] Surgical exposure and elimination of mechanical obstruction is frequently the treatment of choice and spontaneous eruption can then be expected.[5,6]

Impaction of a primary incisor is very rare and review of the literature revealed only one previous report, which occurred in a patient with malformed teeth.[7] The purpose of this article is to present a case of an impacted primary maxillary lateral incisor, its surgical exposure, and follow-up to a normal eruption.

CASE REPORT

A two-and-a-half-year-old Caucasian male was presented by his parents for consultation, because of a missing tooth in the left maxilla (Figure 1). The family and medical histories were unremarkable. No history of trauma to face or mouth was recalled. Extraoral examination was noncontributory.

Intraoral examination revealed that with the exception of the maxillary left lateral incisor, all primary teeth were normal and present in the mouth. No abnormally was noted in the gingiva and alveolar bone of the left maxilla. A periapical radiograph showed an unerupted left maxillary incisor, inclined mesially, with a well-developed crown and partially developed root (Figure 2).

The crown of the lateral incisor was adjacent to the root of the central incisor and was covered by soft tissue. Under premedication (Elixir Pentobarbital, 2.0 mg/Kg) and following local anesthesia (Lidocaine HCl 2 percent

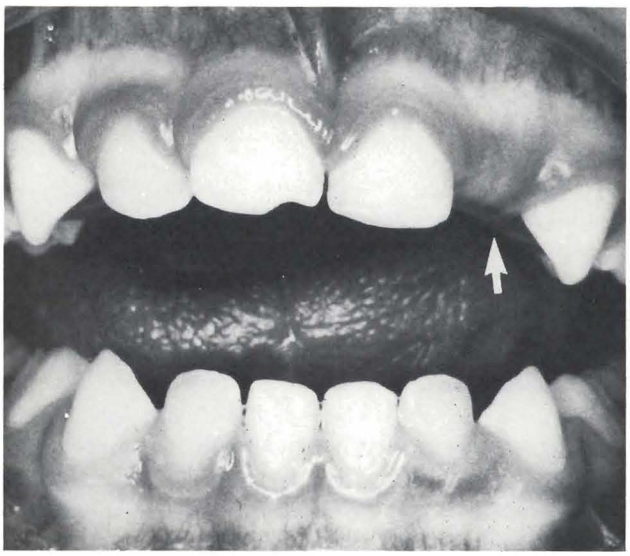


Figure 1. Unerupted lateral primary incisor.

The authors are with the Department of Oral Surgery, The Maurice and Gabriela Goldschleger School of Dental Medicine, Tel Aviv University, and Tel Aviv Medical Center, Ichilov Hospital, Tel Aviv, Israel.

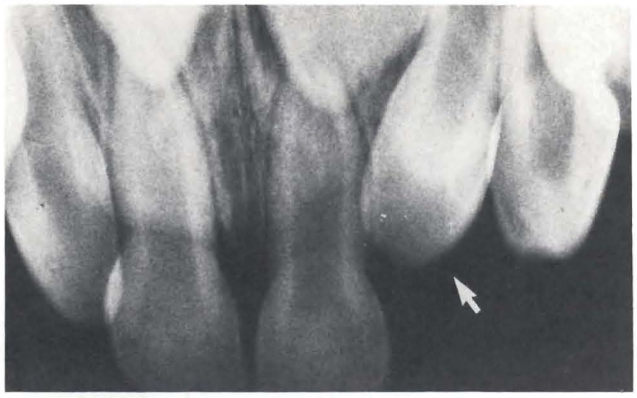


Figure 2. Radiograph showing that the primary maxillary left lateral incisor is impacted, mesially inclined and the root is not completely developed.

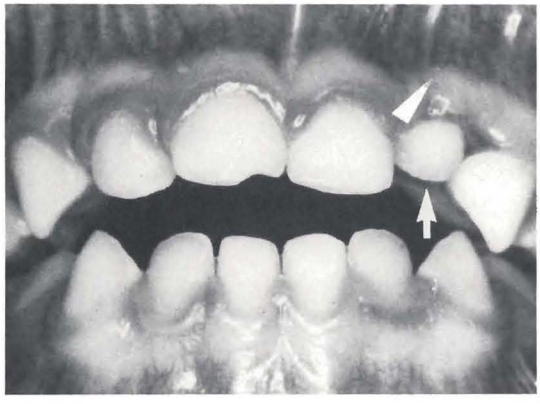


Figure 3. Partial eruption, one week after exposure.

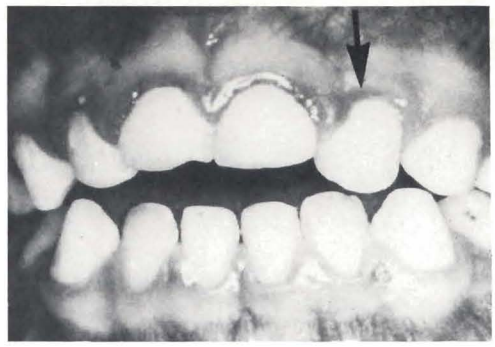


Figure 4. Normal clinical appearance, three years after surgery.

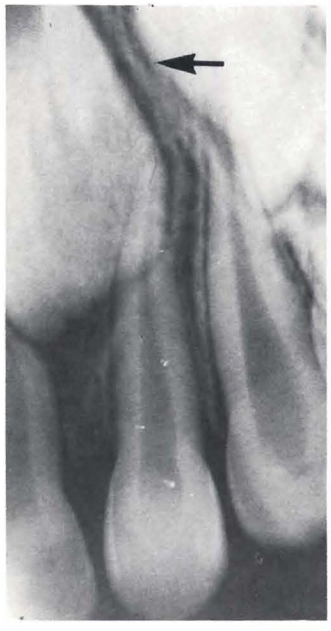


Figure 5. Radiograph showing normal root development of the lateral incisor, three years after exposure. Note that the permanent lateral incisor bud is absent.

and Adrenalin 1:100,000) surgical exposure of the incisal half of the crown was performed. One week after surgery, a slight eruption was noted (Figure 3). Two months later, the eruption was completed. Clinical and radiographic appearance of the impacted tooth, three years after surgery, was normal (Figures 4,5).

Additional radiographs revealed that the patient was missing his maxillary permanent lateral incisors, bilaterally.

DISCUSSION

The failure of permanent incisors to erupt is relatively common. Among the possible reasons for failure may be lack of space, malformation due to early trauma, and mechanical obstruction, such as a supernumerary tooth, odontoma, or scar tissue, due to early loss of primary teeth.[5,6]

In the present case, there was no lack of space, the tooth was normally developed, and no obvious mechanical obstruction was apparent. A possible explanation was the presence of dense scar tissue covering the crown, although it was difficult to penetrate. The scar tissue could have developed, due either to an unwitnessed traumatic incident or as a response of the oral mucosa to mastication, by forming a scar-like tissue. The mesial inclination of the tooth did not appear abnormal and the absence of the permanent lateral incisor bud could not be the cause for the failure to erupt.

According to available data, there is a 75 percent probability of spontaneous eruption, following the surgical exposure of impacted permanent incisors.[6] A surgical approach, therefore, was the treatment of choice. Eruption was completed postoperatively, approximately two years late.

REFERENCES

1. Lytle, J.J.: Indications and contra-indications for removal of the impacted tooth. Dent Clin North Am, 23:333-346, July, 1979.
2. Pindborg, J.J.: Pathology of the dental hard tissues. Philadelphia: W.B. Saunders Co., 1970, p 241.
3. Kramer, R.M. and Williams, A.C.: The incidence of impacted teeth. Oral Surg, 29:237-241, February, 1970.
4. Amir, E. and Duperon, D.F.: Unerupted second primary molar. J Dent Child, 49:365-368, September- October, 1982.
5. Brin, I.; Zilberman, Y.; and Azaz, B.: The unerupted maxillary central incisor; review of its etiology and treatment. J Dent Child, 49:352-356, September-October, 1982.
6. DiBiase, D.D.: Mucous membrane and delayed eruption. Dent Pract Dent Rec, 21:241-249, March, 1971.
7. Bimstein, E.: Root dilaceration and stunting in two unerupted primary incisors. J Dent Child, 45:223-225, May-June, 1978.

Aplasia of primary canines and anlagen of permanent canines: a cleft-specific condition?

Eva A. Holtgrave, DDS, Dr Odont

Aberrations of the primary dentition occur only rarely, in an otherwise normal child. Their prevalence ranges from 0.4 percent to 0.9 percent.[1-4] Absence of primary teeth occurs most frequently in the anterior area.[1,3,4] Aplastic development of canines or molars is even rarer. Grahnen and Granath found aplasia of primary teeth in forty-six individuals, with a less prevalent hypodontia in the permanent dentition.[1] In one case, a mandibular canine was also missing and in seven other cases lateral maxillary and mandibular incisors were absent. In all cases, hypodontia occurred bilaterally; but in two cases also in an additional region. The occurrence of a missing permanent tooth may be an isolated event or it may occur in conjunction with a missing primary predecessor. Schulze pointed out, however, that if a primary tooth is missing, development of the permanent tooth will also be affected.[5] This holds true especially in ectodermal dysplasia, and in cleft lip and palate malformations. The reverse event is very uncommon and, to date, mainly found in cleft lip and palate patients. Ranta presented four cases without evidence of development of primary canines, whereas the primordia of their permanent successors are present; in most cases, however, with conical configuration and accelerated eruption. We can add a similar case; but, unlike Ranta's cases, the absence of a lateral primary incisor was followed by a successor with a conical form. We find this case, therefore, to be of sufficient interest and importance to present it here.

CASE REPORT

The patient is a female minor with bilateral cleft lip and palate. She is the first child of healthy parents with no known family history of malformations. Supposedly,

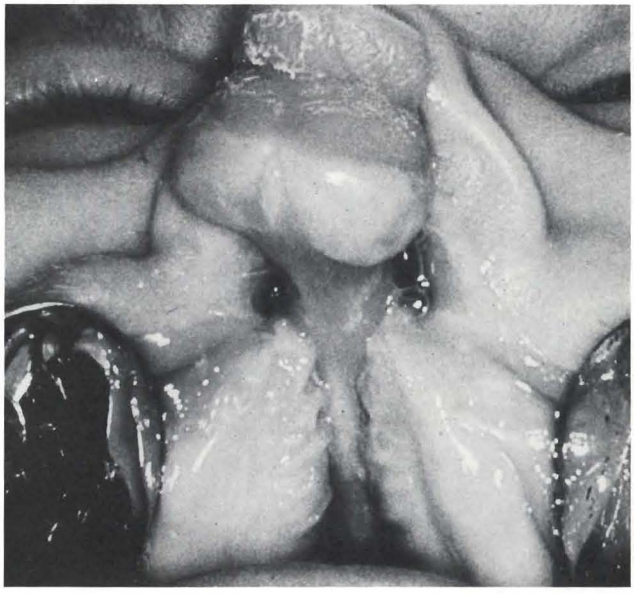


Figure 1. Bilateral cleft lip and palate at birth. Premaxilla was extremely protruded with hypoplastic prolabium and premaxilla.

Dr. Holtgrave is with Fachbereich Zahn-, Mund- und Kieferheilkunde der Freien Universität Berlin (BRD, Germany) Poliklinik für Kieferorthopädie.

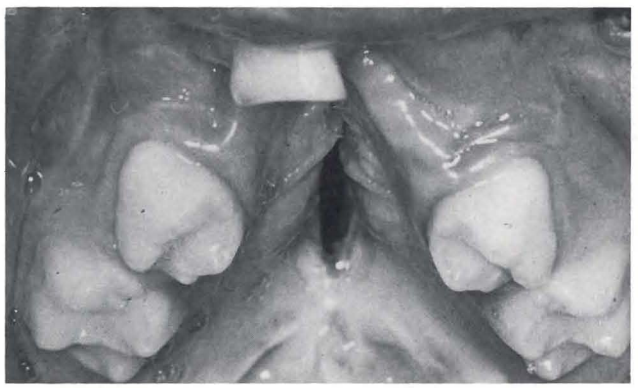


Figure 2. All present maxillary primary teeth are erupted: one tooth in the anterior area and two very large primary molars on each side.

there was no ingestion of drugs, during pregnancy. At birth, the premaxilla was extremely protruded with hypoplastic prolabium and premaxilla. The palatal segments were completely collapsed (Figure 1). Further malformations were not present. The maxillofacial region developed well after surgical closure of the clefts. The eruption times of the primary teeth were within normal limits. At the age of three and a half, all present primary teeth were erupted: an incisor in the anterior maxillary region and two very large primary molars on each side (Figure 2). One mandibular incisor and the left mandibular canine were also missing. The right mandibular canine had a conical configuration. As in Ranta's cases the succedaneous maxillary canines and left mandibular canine were conical in form and erupted prematurely. Unlike Ranta's cases, however, in this case, the

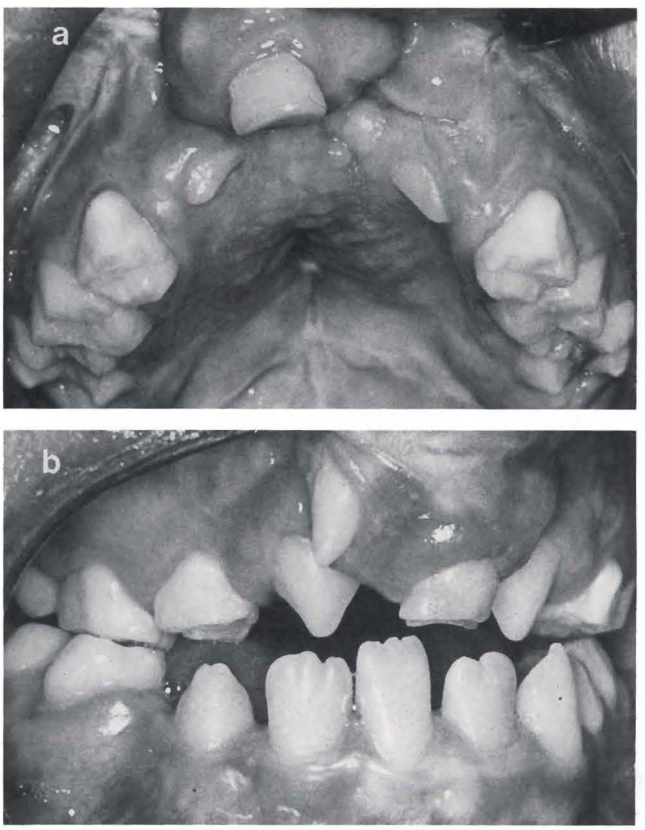


Figure 3a, b. The development of the jaws was normal, in spite of hypodontia.

succedaneous lateral incisor erupted three years late and also had a conical configuration. Additional permanent teeth are missing: two anterior maxillary teeth, the

Table ☐ Cases with missing primary canines and anlagen of permanent successors.

Case No.	Sex	Cleft	Missing primary canines and other teeth		Missing permanent teeth		Supernumerary teeth	Shape of crown (canines)	Remarks
			maxilla	mandible	maxillary	mandible			
1†	m	Bilat. cleft lip and palate	53,63	74		34	12,42	conical	—
2†	m	Bilat. cleft lip and palate	52,63	73,74,84	12,22,15, 13	34,44	—	conical	—
3†	m	Bilat. subcutan. cleft lip	53,36					normal	early exfoliation because of crowding?
4†	m	Bilateral cleft lip and palate	53,63	73				conical	—
5††	f	Bilateral cleft lip and palate	53,63 52,62 61	73	15,25,21,22	34,35 44,45 32		conical	—

† = Ranta's cases, †† = own case

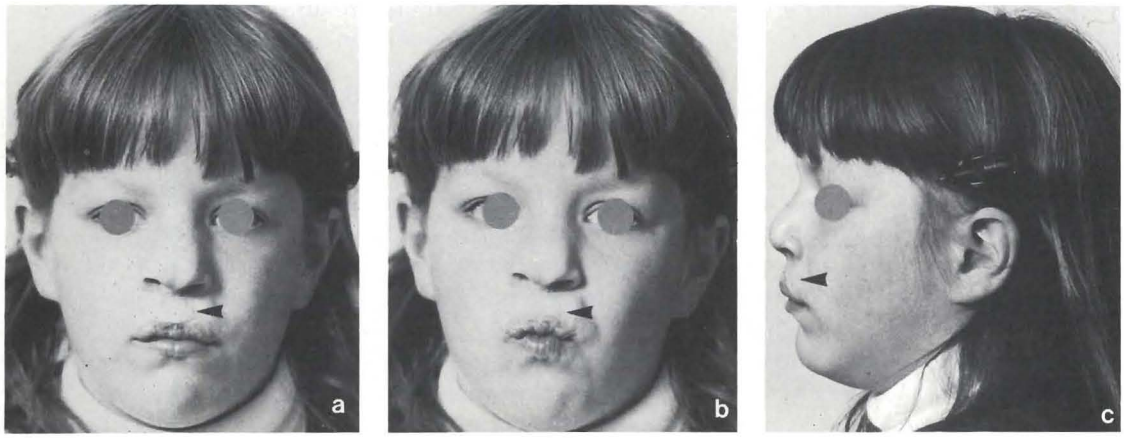


Figure 4a, b, c. The hypoplastic upper lip developed well, with satisfactory function.

second maxillary premolars; and in the mandible, all premolars and a tooth in the anterior area. The development of the jaws occurred in the normal range, in spite of the hypodontia (Figures 3a, b); and the hypoplastic upper lip developed well, with satisfactory function (Figures 4a, b, c).

DISCUSSION

In children affected with cleft lip and palate, hypodontia in the primary dentition occurs most frequently in the cleft region, particularly affecting lateral incisors. In addition, microdontia and delayed development and eruption of permanent teeth is not unusual.[7-9] These anomalies are also present in a noncleft population, but to a lesser extent, as Jordan *et al* pointed out.[10] They assumed that the same factors that are responsible for the cleft will also affect the dentition, but not in a way that can be anticipated. Usually hypodontia and variations in the size and form of the teeth increase with the

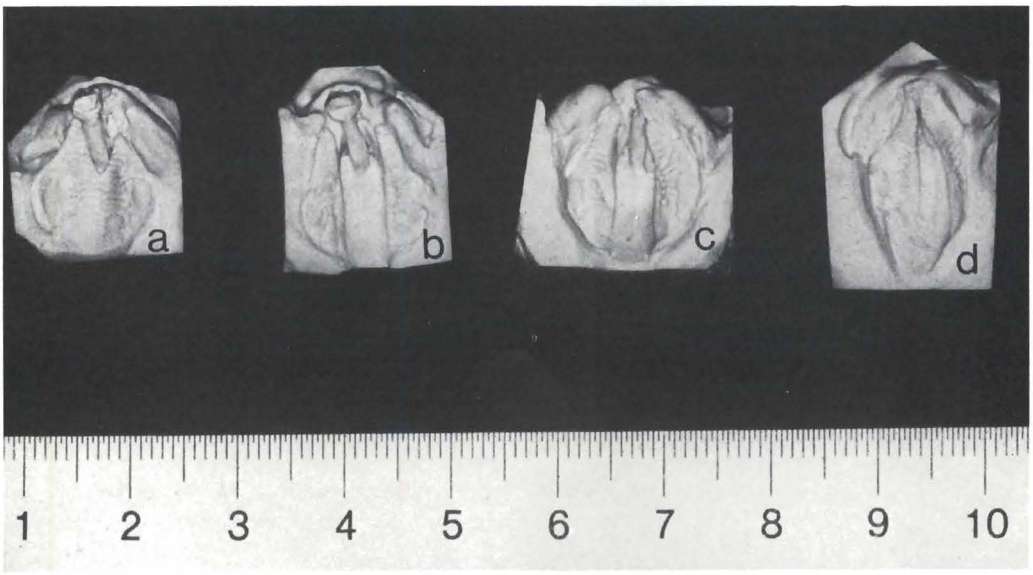


Figure 5a. The application of a low dose of cyclophosphamide at day 11 of gestation resulted in cleft alveolus in rabbits, in most instances. Cyclophosphamide is not the active substance. The effective component results after metabolization. Therefore, not all animals are affected. b,c. Bilateral and unilateral cleft (lip) alveolus and palate in rabbits after application of a higher dose of cyclophosphamide at day 11 or days 11 and 12 of gestation. d. Cleft palate only after application of a low dose of cyclophosphamide at day 12 of gestation.

severity of the cleft, and are, therefore, more prevalent in patients with bilateral clefts. A very uncommon and rare phenomenon is the simultaneous absence of primary teeth and the anlagen of the succedaneous teeth, especially outside the cleft region. This phenomenon is not described in cleft patients with lower lip sinus syndrome, an autosomal dominant, inherited malformation, which is usually associated with a cleft condition, hypodontia, asymmetrical development of tooth pairs, pegshaped upper lateral incisors and retarded development of the teeth.[11] Ranta and Rintala argued that, in this condition, the etiology, attributed to genetic factors is more decisive in dental abnormalities.[11] In cleft cases with dramatically affected dentitions, however, exogenous factors seem to be more prevalent in the multifactorial etiology of the cleft condition. It is especially noteworthy that in Ranta's cases and in our own, only children with bilateral cleft lip and palate malformations are affected. As teratological research has shown, this is a well known effect of teratogenic action of some drugs with dose- and phase- specificities. An application of a given dose of a drug in this defined developmental system may produce a specific malformation. The increase of the dose often results in a more severe defect.[12] For example, with a low dose of 6- mercaptopurine, polydactyly is produced in rats, at a high frequency. When a higher dose is applied, syndactyly, ectrodactyly and phocomelia result; whereas, clefts in rabbits may result after treatment of the dams with cyclophosphamide at the specific stage of gestation in animals.[12] As we could show, when low doses of this drug are given (day 11), in most instances only cleft alveolus occurs. The application of higher doses (day 11 or days 11 and 12) result in unilateral or bilateral clefts of the alveolus and palate in combination with ectrodactyly or phocomelia (Figure 5). This is especially the case, when bilateral cleft conditions are present. The reason is, that in all gestational stages, an organ in the process of differentiation is especially sensitive to interference by toxic substances. On the basis of this information, it may be concluded that similar conditions apply to humans. It appears that one or more specific teratogens that induce definite malformations are also able to disturb the development of the dentition in an unpredictable manner. It is especially noteworthy that bilateral cleft conditions, the most severe malformations, are present in all cases. Unfortunately, in our own case, we were not able to prove this postulated connection. Nevertheless, on the basis of the existing evidence, it would be worthwhile and necessary to collect additional identical cases with severely affected dentitions, especially with aplasia of primary teeth and of the anlagen of the succedaneous teeth.

Further research may establish a more definite relationship between ingestion, during pregnancy, of specific drugs or toxic substances and bilateral cleft conditions.

REFERENCES

1. Grahnen, H. and Granath, L.: Numerical variations in primary dentition and their correlation with the permanent dentition. Odont Revy, 12:348-357, October, 1961.
2. Mencer, L.F.: Anomalies of the primary dentition, J Dent Child, 22:57-62, January-February, 1975.
3. Magnusson, T.F.: Hypodontia, hyperdontia and double formation of primary teeth in Iceland. Acta Odont Scand, 42:137-139, 1984.
4. Ravn, J.J.: Aplasia, supernumerary teeth and fused teeth in the primary dentition. Scand J Dent Res, 79:1-6, January, 1971.
5. Schulze, C.: Über Milchzahnanomalien unter besonderer Berücksichtigung der Anomalien bei Kieferspalten. Stoma (Heidelberg) 6:201-221, 1953.
6. Ranta, R.: Premature mineralization of permanent canines associated with aplasia of their predecessors: report of four cases. J Dent Child, 50:274-277, July- August, 1983.
7. Ranta, R.: A review of tooth formation in children with cleft lip/palate, Am J Orthod Dentofac Orthop, 90:11- 18, July, 1986.
8. Byloff-Clar, H. and Droschl, H.: Zahnzahl and Zahnformen bei Lippen- Kiefer-Gaumenspalten. Fortschr Kieferorthop, 33:417-446, 1972.
9. Weise, W. and Schürholz, B.: Nichtanlage, Verkümmerung, Spätanlage und Überzahl von Zähnen. Dtsch Zahnärztl Z, 25:641-649, 1970.
10. Jordan, R.E.; Kraus, B.S.; Neptune, C.M.: Dental anomalies associated with cleft lip and/or palate. Cleft Palate J, 3:22-55, January, 1966.
11. Ranta, R.; Rintala, A.: Tooth anomalies associated with congenital sinuses of the lower lip and cleft lip/palate. Angle Orthod, 52:212-220, January, 1982.
12. Neubert, D. and Barrach, H.J.: Organotropic effects of dose response relationships in teratology. In: Methods in prenatal toxicology. ed., Neubert, D.; Merker, H.J.; Kwasigroch, T.E.: Thieme, 1977, pp 405-412.

GLOVES IN DENTAL PRACTICE

Does a patient have the right to insist on the operator wearing gloves? The answer must be a definite 'yes', and should the dentist refuse to comply then the patient must have the right to decline treatment.

Editorial. Austral Dent J, 32:140, April, 1987.

Dental and oral manifestations of Rubinstein-Taybi syndrome: report of case

Moorean A. Baker, DDS

Rubinstein-Taybi syndrome is a rare medical disorder first described in 1963, when seven cases were investigated in detail.[1] The main clinical features of the syndrome are mental and motor retardation, small stature, broad terminal phalanges of thumbs (Figure 1) and great toes, ocular abnormalities, a characteristic facies and high-arched palate.[2] Other clinical features include cryptorchidism, heart murmur, gastrointestinal tract abnormalities and recurrent respiratory infections. All of these features were present in this patient.

Many of the afflicted children often resemble neither their parents nor other siblings, but other unrelated children who have the same syndrome.[3] These characteristic features have given some researchers reason to believe that Rubinstein-Taybi syndrome is caused by a chromosomal abnormality.[4,5] To date, however, no common etiological factor or pathognomonic criterion has been established for this syndrome.

CASE REPORT

A ten-year-old black American male with abnormal facial characteristics and short stature was referred for dental treatment. His mother was concerned about "the way his teeth were coming in." At age four, his medical condition was diagnosed as Rubinstein-Taybi syndrome, at Howard University Hospital, in Washington, D.C. The patient is the younger of two children. Both children were delivered by caesarian section without complications. His sister, age twelve, is medically and dentally normal, except for an anterior crossbite involving the permanent maxillary left lateral incisor and permanent mandibular left canine. There is no family history of anomalies and no history of parental consanguinity. The mother is a black American and the father is of Haitian extraction. As previously mentioned, there is no genetic mode of inheritance for this syndrome.[6]

The patient's history contained the following remarkable findings:

- ☐ A short systolic heart murmur.
- ☐ A positive tine test and purified protein derivative test for tuberculosis, at eight months of age.
- ☐ Recurrent ear infections since infancy, which resulted in placement of polyethylene drainage tubes in the external ear, at the age of two years.
- ☐ Grand mal seizure at the age of one year.
- ☐ A repeat of grand mal seizure at the age of twenty months, when the patient was placed on phenobarbital until the age of four years.

The patient was hospitalized at the age of five months, for surgical treatment of a choledochus cyst; and at the age of five years, for unilateral testicular surgery to correct cryptorchidism. Isolated cases of choledochus cysts have been reported in children afflicted with Rubinstein-Taybi syndrome.[3] Incomplete or delayed descent of the testes in males (cryptorchidism) was noted in forty-four of fifty-one cases reported by Widd.[3]

Until initial clinical examination, the patient was fearful and apprehensive, but behaved exceptionally well after the behavior modification technique of tell-show-

Dr. Baker is Instructor, Pediatric Dentistry Department, Howard University College of Dentistry; and in private practice of pediatric dentistry, Capitol Hill, Washington, D.C., Silver Spring, MD.

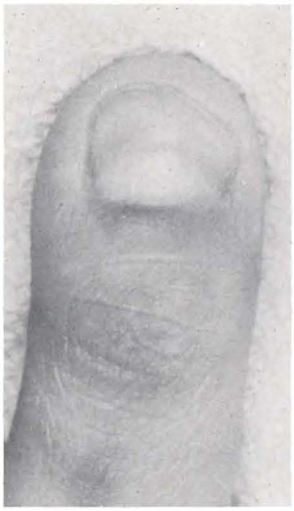


Figure 1. Broad terminal phalange of the thumb, a characteristic of Rubinstein-Taybi syndrome.

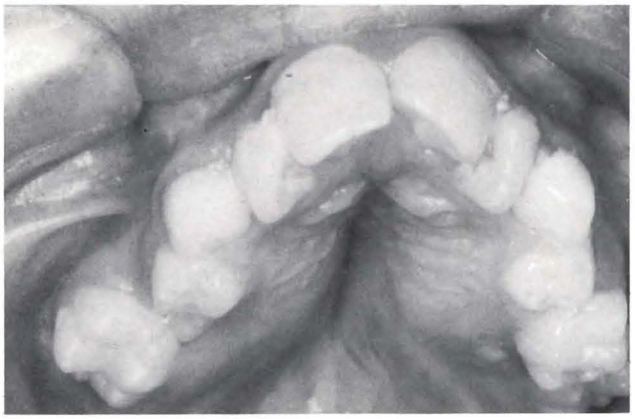


Figure 2. A highly arched, constricted palate, associated with this syndrome.

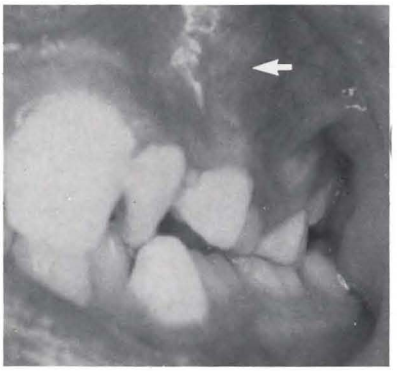


Figure 3. A unilateral crossbite existed.

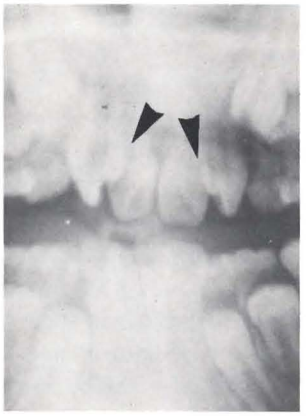


Figure 4. Talon cusps on the maxillary lateral incisors.

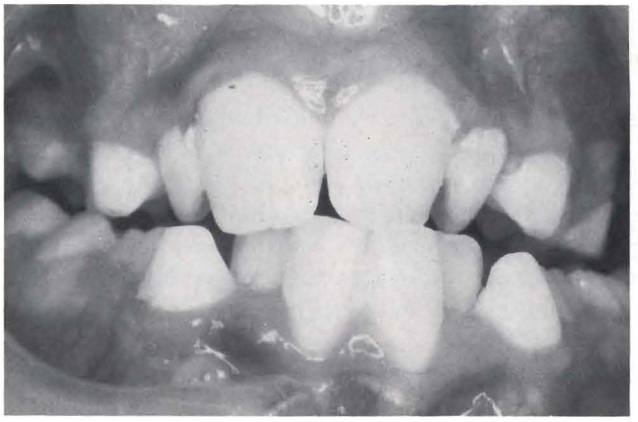


Figure 5. Severe crowding of the anterior teeth occurred.

do was instituted. The palate was highly arched, constricted and without clefting (Figure 2). The palatal slopes were convex and "valleyed in" toward the midline, ending in a midline slit as described by Kinirons (Figure 2).[7] The maxillary arch was narrow, "V" shaped, and in unilateral crossbite with the mandibular arch on the left side (Figure 3). Talon cusps (markedly enlarged cingula) were found on the maxillary lateral incisors and anterior crowding was present (Figures 4,5). The sizes of the teeth appeared normal and the patient was caries-free. A panoramic roentgenograph revealed a supernumerary second premolar in the mandibular left quadrant, and ectopic (mesial) eruption path of both maxillary first permanent molars (Figure 4).

DISCUSSION

In 1963, Rubinstein and Taybi described a rare medical disorder that has no known etiology or pathognomonic criterion. More than 200 reports of Rubinstein-Taybi syndrome have appeared in the literature since 1963; it appears, however, that only two reports have emphasized the oral and dental aspects of Rubinstein-Taybi syndrome.[8] A Canadian report by Gardner *et al* was limited to the association of talon cusps of the incisors, and a German report by Kinirons presented several intraoral changes associated with the syndrome.[7,9] This article would appear to be the first report of the dental and oral aspects of the syndrome, among the American

population. Rubinstein-Taybi syndrome was diagnosed in this patient at the age of four years. The dental and oral manifestations of the syndrome were described in detail. These manifestations are easily discernible and can prove to be very beneficial in the definitive diagnosis of the syndrome. Oftentimes, the pediatric dentist is one of the first health-care practitioners to treat children afflicted with unusual syndromes, such as Rubinstein-Taybi syndrome. It is axiomatic, therefore, that we learn about these cases, to provide the most appropriate and comprehensive dental care possible.

REFERENCES

1. Rubinstein, J.H. and Taybi, H.: Broad thumbs and toes and facial abnormalities. A possible mental retardation syndrome. Am J Dis Child, 105:588-608, June, 1963.
2. Simpson, N.E. and Brissenden, J.E.: The rubinstein- taybi syndrome: familial and dermatoglyphic data. Am J Genet, 25:225-229, 1973.
3. Widd, S.: Rubinstein-Taybi syndrome. Nursing Times, pp 61-65, October, 19, 1983.
4. Padfield, C.J.; Partington, M.W.; and Simpson, N.E.: The rubinstein-taybi syndrome. Arch Dis Child, 43:94-101.
5. Wulfsberg, I.; Klisak, I.; and Sparkes, S.: High resolution chromosome bonding in the rubinstein-taybi syndrome. Clin Genet, 23:35-37, January, 1983.
6. Coffin, G.A.: Bradydactyly, peculiar facies and mental retardation. Am J Dis Child, 108:351, October, 1964.
7. Kinirons, M.J.: Oral aspects of rubinstein-taybi syndrome. Br Dent J, 154:46-47, January, 1983.
8. Barister, M. and Preece, M.A.: The rubinstein-taybi syndrome: occurrence in two sets of identical twins. Clin Genet, 23:318-320, April, 1983.
9. Gardner, D.F. *et al*: Talon cusps: a dental anomaly in the rubinstein-taybi syndrome. J Oral Surg, 47:519-521, June, 1979.

CREATIVITY

Imagination is given to all children. Why it lasts for some into their adult years we do not know. We do know that the imaginative response, the creative drawing, the poem are not products of confusion and unbridled anger. They are, rather, the results of control over desires. Making order for your child is not going to stifle artistic capacities, but permitting chaos to overwhelm the child prevents the ordering and forming work that is the basis of all creative effort. Artists are probably born, not made, and they rarely come from families that expect them; but children can be limited if limitations are not placed on their behavior.

Overpraising a child for his drawing, his poem, or his first song can be destructive. Children recognize how important their work is for you and they can become frightened that they will not continue to please you or that they are not really as good as you say. This can make it hard for them to produce freely and happily.

Parents can help their children best by sharing their own pleasure in art in ways that are appropriate and accessible. Children will enjoy a short trip to the museum to see just a few items. They will appreciate listening to music with you from time to time. They will relish all the stories you tell them and many of the pictures you look at with them. Some children are more original in their responses and perceptions than others; some are more conforming and lack an ability to play with form in color, words, or sound. We do not know why this is so and have to resort to an explanation that rests on the genes. Parents should not worry about the creativity of their child. For most children it is as natural as breathing, not so long-lasting as we would like, but wonderful and satisfying while it is there.

Roiphe, H. and Roiphe, A.: Your child's mind, New York:
St. Martin's/Marek, 1985, pp 403, 404.
