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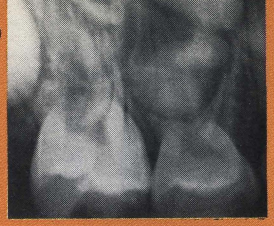
TRADE UNIONISM FOR DOCTORS

"The facts suggest that the workers' basic protection against exploitation is market forces; in other words, the alternatives that are open to any person possessing scarce and valuable attributes."
—W.H. Hutt

THE HEALTH PROFESSIONS ARE CONFRONTED WITH A MAJOR ISSUE

ASDC AMERICAN SOCIETY OF DENTISTRY FOR CHILDREN



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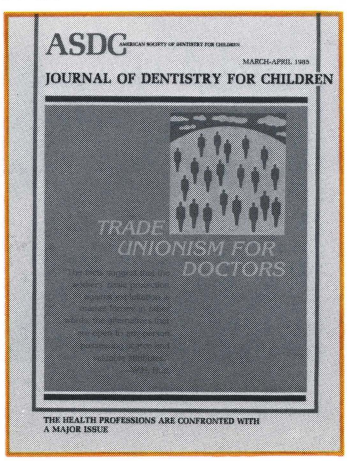
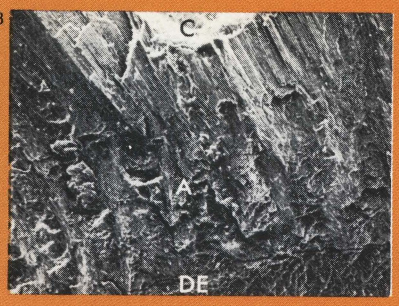
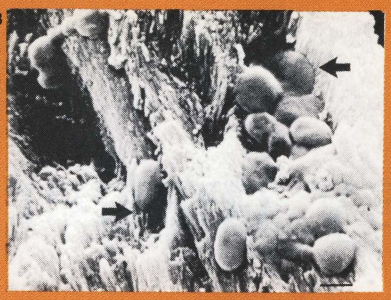
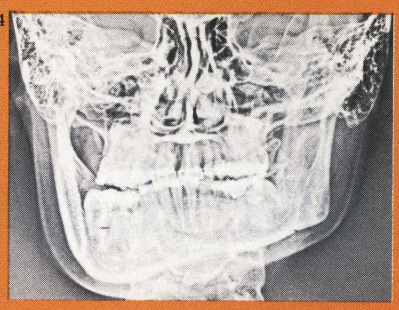


The cover highlights the issue of trade unionism and the health professionals, discussed and debated in the article reprinted from the New England Journal of Medicine and in the ASDC forum published in this issue of JDC. The quotation by professor W. H. Hutt is from his book on the economic consequences of collective bargaining, *The Strike-Threat System*, published by Arlington House. The quotation appears on page 22 of the book.







Deep bacterial penetration of early proximal caries lesions in young human premolars

Clinic

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Although it is generally accepted that the presence of bacteria is necessary for the development of dental caries, the role of microorganisms in the progress of the early caries lesion is not completely understood.[1] In general, observers are of the opinion that dissolution of enamel by acid produced by bacteria on the enamel surface creates the incipient lesion. According to Darling, the destruction of the integrity of the enamel surface precedes the entry of microorganisms; and Silverstone suggests further that bacteria will be found in the dentin only after cavitation has occurred.[2,3]

Frisbie and Nuckolls, as early as 1947, however, using decalcified enamel sections stained for bacteria, found coccus-like microorganisms in the enamel before cavitation occurred.[4] It was also suggested that bacteria, by penetrating enamel, may contribute to the initiation of enamel caries.[5] More recently, Brännström *et al* investigated incipient caries lesions in human teeth, by observing the lesions with the aid of the scanning electron microscope (SEM).[6,7] Bacteria were found replacing enamel prisms, and occasional lateral spreading was observed at the dentinoenamel junction, despite the absence of surface cavitation. Similar observations were reported in SEM-studies of rat fissure caries.[8] Seppä

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examined clinically sound human third molars using SEM.[9] Fractures were made through the fissure area and bacteria were found within the enamel under the apparently intact enamel surface.

The dynamics of the caries process may involve active phases as well as phases of repair and consolidation, when no or very few bacteria are present within the hard tissue. If the lesion is in an active and advancing phase, how deeply into enamel and dentin do bacteria penetrate? The aim of this study was to investigate the frequency and extent of bacterial invasion in incipient caries lesions on proximal surfaces of young human premolars.

MATERIAL AND METHODS

Premolars extracted for orthodontic reasons, from children twelve to fifteen years old, were examined under a stereomicroscope. Seventeen proximal surfaces of the teeth, from eight children, with incipient caries lesions (white spots) or lesions with microcavities (100 to 300 μm deep) were prepared for SEM. The material included twelve surfaces with incipient lesions and five with microcavities. In addition, one surface with an isolated tube-like cavity reaching the dentin (as assessed by stereomicroscope) served as a positive control. Eight proximal surfaces of four unerupted premolars, surgically removed from three teenagers, served as negative controls. Between extraction and preparation for SEM, the teeth were stored in 10 percent neutral formalin. The root of the tooth was cut off and the crown split longitudinally through the middle of the occlusal surface. The surfaces were examined under the stereo-microscope and the outlines of the lesions were marked with a fine bur. This was done for orientation purposes. Surfaces without microcavities were cleaned by rubbing with a cotton pellet soaked with a detergent containing a surface active component combined with 0.2 percent EDTA.* This was done to remove bacterial plaque and reduce the risk of contaminating the fractured surface. The surfaces with microcavities were cleaned with waterspray only. Grooves were prepared on the occlusal, cervical and pulpal aspects to locate the fracture in what was judged to be the center of the lesion. The crown half was split longitudinally with a tubecutter.+ The depth of the chaulky area and any microcavity were assessed in the stereomicroscope. Care was taken not to touch or otherwise contaminate the cut surfaces. Both specimens were mounted for SEM. The technique was the same as described earlier.[6] Photographs were taken from areas with structures that differed from the normal seen on unerupted teeth and on the actual specimen lateral to the lesion. The photographs were examined and evaluated by the two investigators.

Criteria for evidence of bacteria were as follows: Isolated as well as clustered coccus-like, rod-like, or filament-like structures, similar to those seen in the positive control and in earlier studies.[6,7,10,11] Studies dealing with

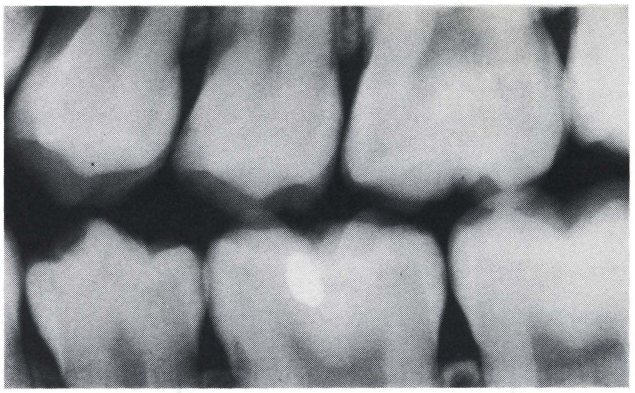

Figure 1. Bitewing radiograph from a twelve-year-old girl. The distal surface of the maxillary left second premolar showed a microcavity; the mesial surface, a white spot.

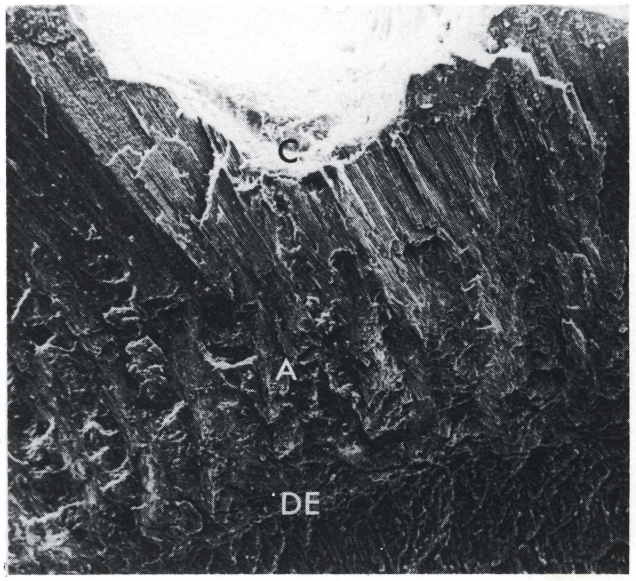

Figure 2. Fracture through the center of micro-cavity (c) on the distal surface of the premolar seen in Figure 1. There was a step-like formed cavity about 0.3 mm deep, free from pellicle formation and plaque (the surface has been cleaned with water-spray only). The prisms were relatively intact to a distance of about 0.2 mm from the bottom of the cavity. From a depth of about half the enamel and to the dentino-enamel junction (DE), local areas could be seen where the prisms were completely destroyed and replaced by a smear of microorganisms. Detail of area A is seen in Figure 3 × 65.

*Tubulicid®; Dental Therapeutics AB, Nacka, Sweden.

+ Oxwall Workshop Tools, Japan.

Tubulicid, Blue Label, Dental Therapeutics AB, 13145 Ektorp, Sweden.

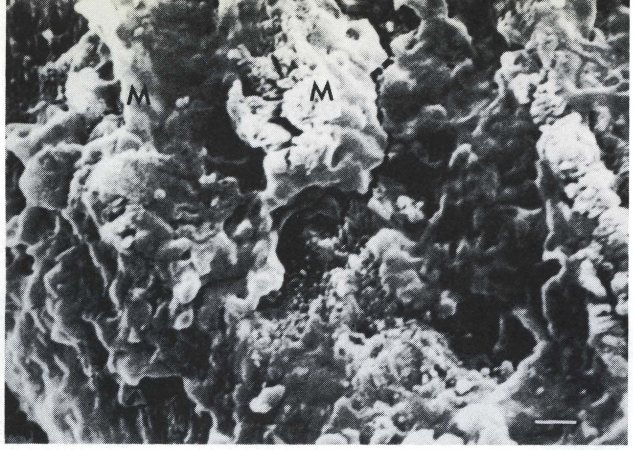

Figure 3. Detail of area A in Figure 2. Smear of microorganisms (M) is seen replacing prism structure. Bar = 1 μm.

Figure 4. Detail from an area approximately 0.7 mm from the surface of the mesial white spot lesion seen in Figure 1. Microorganisms have replaced parts of an enamel prism and they appear integrated with the prism structure (right arrow). One microorganism moved during penetration, disclosing an underlying lacuna (left arrow). Bar = 1 μm.

Figure 5. Same lesion as seen in Figure 4. Detail of well-defined rectangular area at the dentinoenamel junction, possibly tufts. In this area, many microorganisms were seen "floating around" and dividing. Bar = μm.

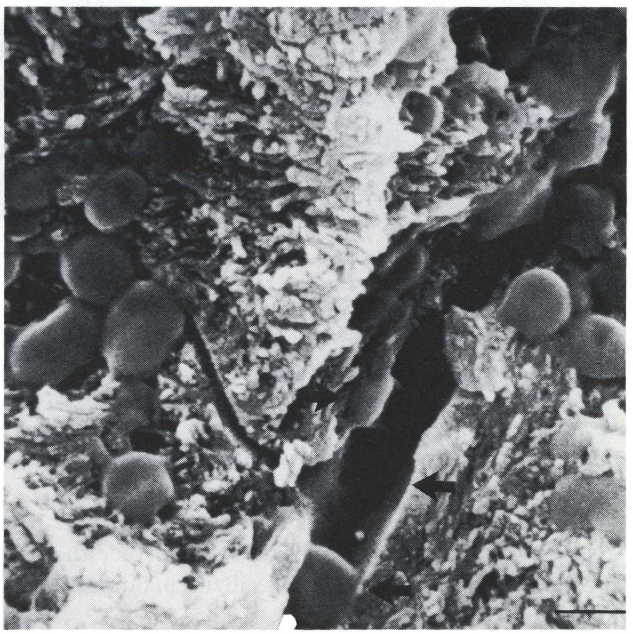

Figure 6. Same lesion as in Figures 4 and 5. Dentin near dentinoenamel junction. Dentinal tubules with depressions in the peritubular wall (arrows) and a canaliculus can be seen. Microorganisms have penetrated intertubular dentin. Bar = 1 μm.

fissure enamel, carious or clinically intact, have revealed, in illustrations, bacteria in free positions on the fracture surface, extending through the fissure.[8,9] During fracturing, such bacteria might have emanated from the contents of the fissure. In our study of smooth enamel lesions, we wanted to avoid such contamination. Bacteria should appear, therefore, in more than one place and be seen replacing tooth structure in some areas.

RESULTS

The depth of chaulkiness in the enamel of the teeth with caries varied from the outer one-fifth of enamel to its entire thickness. All fractured areas from surfaces with microcavities, except one, showed clear evidence of the presence of bacteria in the enamel, at least to the dentinoenamel junction (Figures 1-3).

In the twelve specimens with incipient caries, clear evidence of bacteria could be seen in two and was observed in both enamel and dentin (Figures 4-7). In one of these two specimens, bacteria could be detected penetrating the dentin to a depth of 1.5 mm from the dentinoenamel junction. In both lesions, the chaulkiness extended to about half of the enamel thickness. In the remaining ten lesions, configurations resembling bacteria were seen in eight; but the appearance did not completely fulfill the criteria. Extensive destruction of

Figure 7. Same lesion as seen in Figures 4, 5, 6 and on Figure mesial surface. Area about 1.5 mm from the dentinoenamel junction measured along the dentinal tubules, i.e. halfway to the pulp. Peritubular dentin appears intact, but bacteria are depressed into the tubular walls (arrow). Between the two tubules cavernous destruction of intertubular dentin can be noticed. From the white spot surface to this area, microorganisms were present at all levels. Bar = 1 μm.

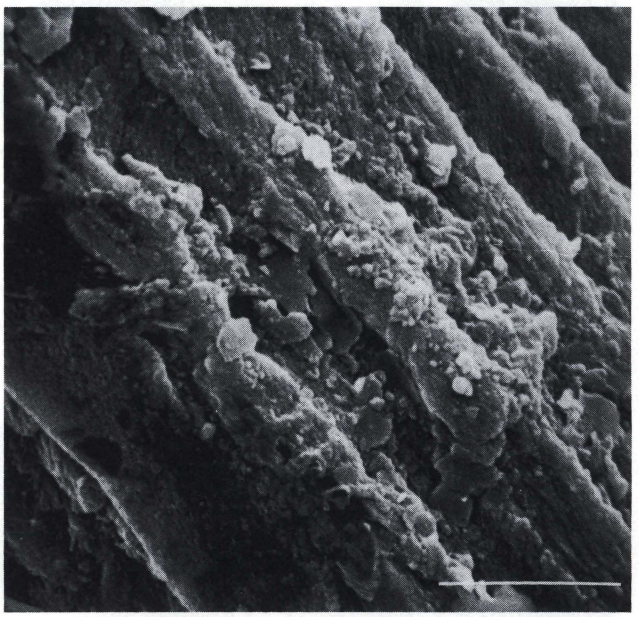

Figure 8. An area under a white spot lesion with microcavity. In the center of the photograph, the prism structure is destroyed and replaced by dense laminar structures of various sizes. In the area under this cavity, no bacteria were seen and the enamel prisms appeared fairly intact. Bar = 10 μm.

enamel was accompanied in all specimens by either the presence of bacteria or by mineralized material replacing prism structure (Figure 8).

The positive control tooth showed coccal, rod-shaped, and filamentous bacteria on the cavity walls of the lesion. Bacteria were also observed in the gap at the dentinoenamel junction and in the enamel and dentin at a distance lateral to the cavity, under a clinically intact surface. The fractured specimens from the unerupted premolars showed no evidence of bacteria.

Evidence of remineralization was seen in all specimens except two and the controls. Remineralization appeared as separate small sheets or as laminated, smooth plates of various sizes distributed irregularly (Figure 8). Such structures were mostly present near the surface, but could also be seen in deeper parts of the lesions.

DISCUSSION

As pointed out earlier, the SEM technique has its limitations, in evaluation of a caries lesion.[7] Only one plane can be examined in each specimen. Moreover, artifacts occur during fracturing and the subsequent preparation of the specimen. For instance, due to dehydration, a solution of the organic part may appear as "droplets" resembling bacteria. This was observed in one tooth in the present study, and dictated the need for strict criteria, for evaluation of the photographs.

Deep microbial invasion was obvious in two lesions without cavitation, supporting earlier findings.[4,6-8] The microorganisms seemed to be able to destroy enamel and dentin by direct lacunar resorption (Figures 4,6). In these figures, the microorganisms appear as integral parts of the mineralized tissue, resembling osteoclasts resorbing bone. When bacteria reached the dentinoenamel junction, a spread along this border was also seen in some teeth. Presumably, the microorganisms do not get sufficient food from the enamel structure for the production of acid and enzymes which destroy enamel. One may speculate, therefore, that these microorganisms obtain nourishment from increased outward flow of tissue fluid from the pulp and also from the tooth surface, due to an increase in enamel permeability and diffusion of substrate from the oral cavity.

In several incipient lesions, bacteria could not be observed with any certainty. This could be one example of the limitation of the SEM technique. An active bacterial attack might have been absent on the fractured surface, but present in an area beneath the surface. In this context it should also be emphasized that in several lesions where no bacteria were detected, remineralization was marked. A lesion without bacteria, therefore, could have been in a stage of consolidation. A culturing method would offer the possibility of examining a larger, three-dimensional portion of the lesion and also of identifying the microorganisms that enter the lesion at various stages of development. Such studies are in progress.

The irregular pattern of caries lesions often seen with various histologic methods is more easily explained and understood, if the caries process is considered in terms of phases of destruction alternating with phases of remineralization, as observed in the present study.[12,13]

Many researchers believe that demineralization in the early caries process is a simple physicochemical event controlled from the tooth surface. We suggest that products from the plaque are important also in maintaining channels for nutrition to microbes within the enamel, and that these may directly contribute to hard tissue destruction.

We must consider the possibility that under a white spot lesion, bacteria may occasionally be present at the dentinoenamel junction and even deep in some dentinal tubules, in mineralized dentin. Perhaps, this is the case, when the caries process in a later stage is judged as "acute". One such lesion, the positive control, was seen in the present study. There was a narrow, tube-like cavitation through the enamel, with an extensive lateral spread of the microorganisms along the dentinoenamel junction into dentin, as well as outward, into intact enamel. From a clinical point of view, however, it should be stressed that the presence of bacteria within the enamel or even dentin under a smooth white spot lesion does not mean that the lesion has lost its potential for healing. Blocking communication from the oral cavity to the actual microorganisms (for instance, by stimulating the development of a calcified pellicle, or by resin sealant) may result in the death of these microbes, and thus permit remineralization to occur. An analogous situation is seen in fissure sealing. If only small quantities of bacterial toxins have reached the pulp, pulpal cells may have the capacity to produce irregular dentin. Such a blockage may also contribute to limiting the food supply to bacteria, and an arrested lesion. An analogous situation is seen when superficial dentin has been exposed and left unprotected. Bacteria always invade the tubules, but after production of irregular dentin and calcification of the surface, their opportunity to survive is limited.

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Cyclic neutropenia: a literature review and report of case

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James E. Fleming, DMD

Cyclic neutropenia is a rare hematologic disorder characterized by the disappearance of neutrophils from the blood and bone marrow, at regular intervals. In most cases, these neutropenic cycles occur every twenty-one days, although cases of cycles occurring every fifteen to thirty-five days have been reported.[1,2] Between cycles, the neutrophil count rises, but seldom does it exceed 50 percent of the differential count. In some patients, a compensatory monocytosis and/or eosinophilia permits the total white blood cell count to remain at a low normal level during the neutropenic cycles. In other patients, the white blood cell count may fall to low levels during the neutropenic cycle and rise to near normal levels between cycles.

The neutropenic phase of each cycle is characteristically associated with clinical symptoms such as fever, malaise, chills, anorexia and ulcers of the oral mucous membrane.[2-16] Additional symptoms in some patients include: intermittent arthralgia, abdominal pain, sore throat, lymphadenitis, ischiorectal infections, mental depression, conjunctivitis and cutaneous ulcers.[2] The clinical manifestations usually appear one to three days prior to any changes in the differential blood count and may persist from three or four days up to ten days. The patient is generally healthy between the periods of neutropenia.

Although cyclic neutropenia has been reported to develop at any age, the majority of patients have reported symptoms prior to the age of ten years. Either sex may be affected and the suggested mode of inheritance is autosomal dominant.[17,18] The similarity between reported cases is striking. In most reported cases, once the symptoms are recognized complete remission seldom occurs.

A variety of therapy modalities such as steroids, hormones, splenectomy, nucleic acids, blood transfusions, irradiation, prophylactic tonsillectomies and dental extractions, have been used unsuccessfully in the treatment of cyclic neutropenia.[4,6,11,19-21,23-29] To date, antibiotic therapy is the only treatment that has realized predictable success in the management of systemic infections in these patients. Granulocyte transfusion methods may, however, offer promise in the treatment of these patients in the future.[30]

The purpose of this paper is to present the oral man-

Table □ Serial blood cell counts of approximately one month, showing the periodicity of the neutropenia and its relationship with the monocytosis and eosinophilia.

		Hematologic values			
Day	WBC/mm^3	Neutrophils Percent	Lymphocytes Percent	Eosinophils Percent	Monocytes Percent
0	11,000	0	82	3	15
8	21,600	17	57	4	12
13	5,400	13	60	6	15
19		0	60	2	23
23	9,900	17	54	8	16

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ifestations and dental treatment of a cyclic neutropenia patient over a five-year period of observation. The bone marrow defect, therapy modalities, and suggested etiology will be reviewed and compared with cases in the literature.

MEDICAL HISTORY

The patient, a small four-year-old female, was referred to the University of Minnesota Department of Pediatric Dentistry for evaluation and management of inflamed gingivae and mobile teeth. Because of a family history of cyclic neutropenia, the patient had been monitored since birth via serial blood counts. Consistently, these hematologic evaluations had demonstrated depressed neutrophil counts in the absence of any abnormality among the other blood components. At three months of age it was recorded that the patient's absolute neutrophil count had fluctuated between 0 and 500 cells/mm^3 since birth. This persistent neutrophil deficit was consistent with a diagnosis of chronic neutropenia.

In spite of her hematologic disorder, the patient appeared relatively healthy during the early months of her life. Unlike most chronic neutropenia patients, she did not experience recurrent bacterial infections (for example, pneumonia, recurrent pharyngitis, meningitis, or skin abcesses). She did, however, experience episodes of high fever associated with mouth sores, fatigue and lassitude. These fevers lasted four to five days; then the symptoms decreased and subsequently recurred every sixteen days. On occasion, to prevent secondary infection during these periods, the patient had received supportive antibiotics, in conjunction with steroid therapy.

At approximately sixteen to twenty months of age, the patient experienced a failure to thrive as a result of an unidentified form of liver disease. After months of persistent hepatomegaly and elevated liver enzymes, exploratory surgery revealed a liver abcess and the right lobe was resected. Following surgery, the patient's hematologic condition improved markedly, with postsurgical neutrophil levels significantly elevated in comparison to presurgical levels and cycling at twenty-one-day intervals. A similar improvement in the patient's hematologic condition was recorded following abdominal surgery, one year later. It appeared that the patient's neutrophils were capable of responding to stress, although at a subnormal level.

Shortly after the patient's sixth birthday, serial blood tests confirmed that the neutrophil counts were now

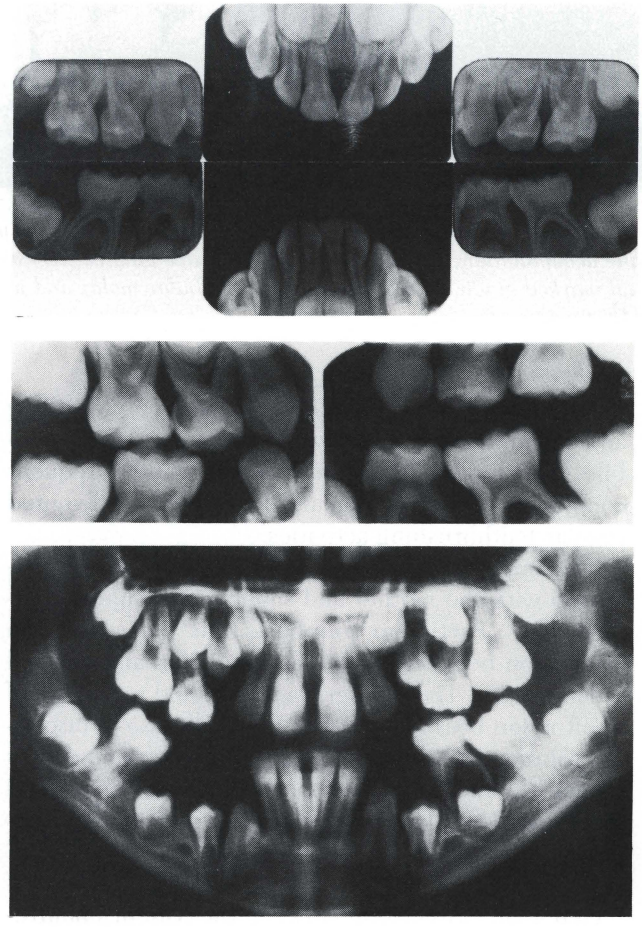

Figure 1. Periodic radiographic evaluations.

cycling consistently at twenty-one day intervals. As recorded previously and shown in Table 1, periods of profound neutropenia were accompanied by a mild monocytosis and moderate eosinophilia. Bone marrow specimens drawn during such a neutropenic phase revealed normal cellular bone marrow with neutrophil maturation arrested at the myelocytic state.

FAMILY HISTORY

The patient's mother and maternal grandfather have classic twenty-one-day cyclic neutropenia. Hematologic evaluations of other family members were within normal limits.

DENTAL HISTORY

The initial dental examination at age four revealed an intact primary dentition, very inflamed and edematous gingival tissue, and moderate to heavy plaque accumulation. Radiographic examination revealed pronounced alveolar bone loss in the anterior regions of the mouth and the mandibular primary molar region (Figure 1). Severe gingival recession was noted in the

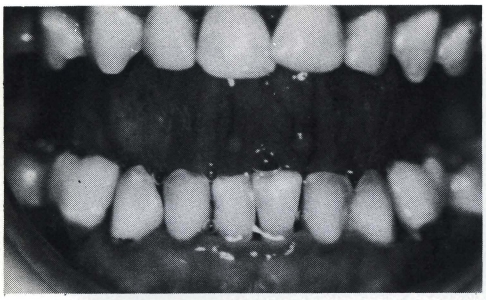

Figure 2. Age four years, six months. Moderate plaque accumulation, edematous gingival tissue and marked gingival recession of mandibular first primary molars.

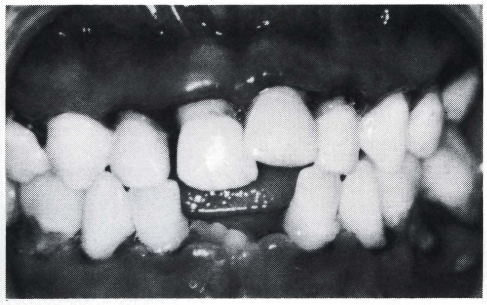

Figure 3. Age five years, six months. Progressive gingival recession particularly in mandibular molar and maxillary anterior region.

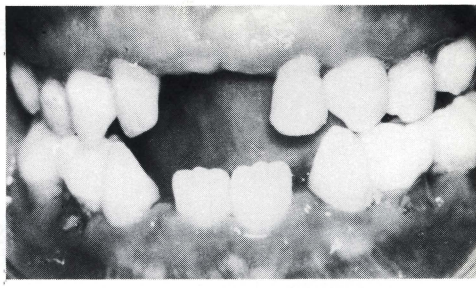

Figure 4. Age six years, three months. Mild plaque accumulation. Severe gingival recession and furcation involvement of mandibular primary molars. Recession is also apparent in maxillary anterior region.

mandibular primary molar region (Figure 2). Maxillary and mandibular anterior teeth were excessively mobile. The mother reported that the child's frequent complaints of "sore gums and loose teeth" prevented regular toothbrushing activities.

The importance of thorough oral hygiene was discussed with the mother and suitable techniques demonstrated. Particular emphasis was placed on the association between plaque accumulation and gingival inflammation. The mother was instructed in the use of a soft nylon brush and dental floss. The use of disclosing solution to evaluate complete plaque removal was demonstrated. A thorough prophylaxis was performed and the patient placed on a six-month maintenance regimen.

Over the first five years, particular attention was directed at any association between the patient's neutropenic cycles and the severity of her gingival condition. It was noted that characteristically during neutropenic phases, the patient experienced severe gingival inflammation (Figure 3). Also, during these phases, all oral hygiene procedures were discontinued, due to the discomfort associated with any manipulation of her oral tissue. Between cycles, gingival inflammation and swelling were reduced and oral hygiene was markedly improved (Figure 4).

Although the degree of gingival inflammation did appear cyclic, alveolar bone loss and gingival recession were progressive. Gingival recession which was first noted in the mandibular primary molar region (Figure 2) progressed to the involvement of all primary teeth (Figures 3,4,5). Alveolar bone resorption originally appeared most significant in the anterior regions and the mandibular molar area (Figure 1). By age seven, significant alveolar bone loss was noted in the maxillary molar region. During the following year, it was necessary to extract maxillary and mandibular first primary molars, because of excessive mobility and irritation to the surrounding oral tissues.

At age eight years, six months, it was noted radiographically that the newly erupted permanent incisors, particularly the mandibular incisors, had diminished alveolar bone support (Figure 1). Clinically, the gingival tissue remained inflamed and edematous (Figure 5). Approximately one year later, there was clinical evidence of gingival recession associated with the mandibular permanent incisors (Figure 6).

Current therapy for this patient involves self-administration of 0.2 percent NaF mouthrinse every other day, constant meticulous oral hygiene techniques, and regular maintenance visits. Although the patient's plaque control has characteristically ranged between average and poor, she has maintained a caries-free dentition. Space-maintaining appliances have been placed with particular care, to insure access to all areas for cleaning and maintenance by the mother.

DISCUSSION

Neutrophil physiology

Neutrophilic granulocytes comprise the body's major initial cellular defense response to tissue damage and invading foreign organisms. The neutrophil's phagocytic response is particularly important in retarding the growth of bacterial infections.[19,31-33] As might be expected, neutropenia increases susceptibility, particularly to staphylococcal and other pyogenic and enteric bacterial infections.[28,33,34] In comparison, these patients do not experience similar reactions to viral or fungal infections.[33,35]

Infections flourish in the neutropenic patient in spite of a compensatory monocytosis or eosinophilia. Results of studies comparing the phagocytic response of eosinophils and monocytes to that of neutrophils offer an explanation of this observation.[36-38] These studies have shown that eosinophils ingest and subsequently kill bacteria less effectively and efficiently than neutrophils. The phagocytic and bactericidal activity of the monocyte is similarly reduced in comparison with the neutrophil.[38]

Numerous investigations have shown that the neutrophil is prominent in both clinically healthy gingival tissues and tissues with gingival inflammation.[39,40] Abundant neutrophils are present within the connective tissue, junctional epithelium and gingival crevice. It is hypothesized that in this position, the neutrophil is a defensive cell which provides continuous protec-

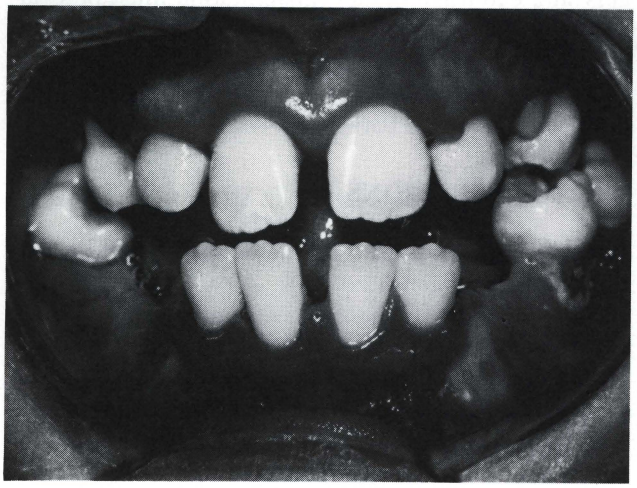

Figure 5. Age eight years, six months. Edematous gingival tissue in anterior region. Gingival recession involving maxillary and mandibular primary molars.

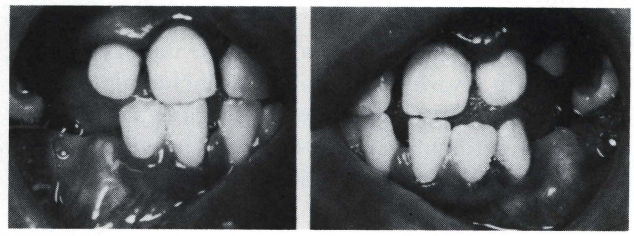

Figure 6. Age nine years, six months. Inflamed gingival tissue with loss of gingival attachment in mandibular anterior region.

tion for the periodontal tissue, including phagocytosis and killing of bacterial microorganisms.[41-43] Thus it is suggested that inadequate neutrophil function, in conjunction with factors which promote plaque accumulation, subjects the periodontium to rapid and severe breakdown, such as noted in this patient.

Etiology

The suggested etiologies for cyclic neutropenia are perhaps as varied as the clinical symptomatology of this disorder. They range from Reimann's hypothesis of a disturbance in the inherent biological rhythm, to an early hypothesis of endocrine dysfunction.[10,15] These hypotheses as well as those of reticuloendothelial dysfunction, allergy and hypersplenism have depended upon the particular features of individual cases for substantiation. For example, several authors have suggested a relationship between secretion of female sex hormones, the menstrual cycle and the neutropenic cycle as proof that an endocrine dysfunction is manifest in this disorder.[6,13,15,27,44] Thompson reported in his study of forty patients that in seventeen of the eighteen females under forty-five years of age, the neutropenic cycle occurred within one or two days of the menstrual period.[15] This relationship, however, has not been consistently reported in menstruating women suffering cyclic neutropenia.[2,10] Finally, observations of disturbed endocrine function in these patients are sporadic and no consistent hormonal imbalance can be identified.

Investigations into the etiology of this patient's cyclic neutropenia have been futile. An indium chloride bone scan and granulocyte kinetic tests indicated no abnormal leukocyte destruction. These tests also confirmed that persistent splenomegaly was not the cause of this patient's neutropenia.

Bone marrow defect

A stem cell defect or a maturation arrest is the suggested bone marrow defect in this disease.[2,9,11,14,16,18-20,28,44,46,47,49,50] The hypothesis of a maturation arrest has been supported by Haghshenas in his study of a twenty-one-year-old male with cyclic neutropenia. He recorded an increase in early myeloid precursors, but very few mature neutrophils in samples taken from the patient's marrow.[20] Telsey reported similar findings in a fourteen-year-old female patient.[14] Scott, in his studies of cyclic neutropenia in the grey collie dog, also reported that few mature neutrophils were seen in the marrow, despite the presence of undifferentiated stem cells. He hypothesized that the stem cell differentiation was blocked either by a neutrophil associated inhibitor of stem cell differentiation or by inhibition of a normal circulating factor which drives the stem cell differentiation.[51] Dresch, studying labelled granulocytes in human cyclic neutropenia, concluded that an intrinsic stem cell defect caused the production of abnormal mature granulocytes. He hypothesized that these abnormal granulocytes secrete a factor which inhibits the cellular proliferation of granulocyte precursors.[48] Other authors have suggested that cyclic neutropenia merely reflects an accentuation of a normal fluctuation in granulocyte proliferation.[2,10,22,50]

Similar to the patients studied by Hagshenas and Telsey, bone marrow specimens taken from our patient indicated neutrophil maturation arrest.[14,20] These specimens showed that neutrophil maturation was halted at the myelocytic stage. This defect was noted in the presence of otherwise normal cellular bone marrow.

Treatment

Historically, cyclic neutropenic patients have been subjected to a variety of treatments including therapy with various drugs and a host of surgical procedures. Of these treatments, splenectomy has provided symptomatic relief for some patients suffering hypersplenism, but does not eliminate the neutropenic cycles.[10,12,16,20,45-47] Progesterone, estrogen and testosterone injections have provided symptomatic relief for these patients with varying success.[28,29] Current treatment for these patients involves supportive measures to reduce infection, including antibiotics for the control of systemic infections and careful attention to preventive dentistry measures.

Oral manifestations and therapy

Oral manifestations are a significant and consistent clinical symptom of cyclic neutropenia. The spectrum of oral manifestations include multiple ulcers, inflamed gingivae, rapid periodontal breakdown and alveolar and supporting bone loss.[2-5,8-13,14,16,19,24,25,27,29,33,34,43,45,47,52] The ulcerative lesions, which have a reddened raised border and a base covered by a yellowish-gray membrane, may involve the tongue, soft palate, uvula, lips and cheek. These ulcers usually heal with scarring. The gingiva is characteristically described as inflamed, hyperemic and enlarged, with destruction of the interproximal papillae. Extensive loss of alveolar and supporting bone, particularly in the permanent molar and mandibular anterior regions, has been recorded. Cohen and Gorlin have reported similar alveolar bone destruction in the primary dentition in their patients.[8,43] The severe periodontal breakdown frequently causes occlusal dysfunction and early loss of teeth.

The primary goal of dental treatment for these patients is prevention and control of the dental infection before surgical intervention is required. It is recommended that daily fluoride gels or mouthrinses be used to reduce the possibility of dental infection secondary to a caries lesion. A rigorous oral hygiene program, including scaling and prophylaxis, should be implemented to reduce plaque accumulation, which would expedite the periodontal breakdown. As the severity of this disorder will vary according to such factors as, age of onset, level of neutropenia and other compromising health factors, treatment beyond the recommended supportive therapy must be evaluated on an individual basis. That is, the efficacy of periodontal surgery and extensive dental rehabilitation must be evaluated individually. Finally, antibiotic coverage to prevent postoperative infections and bacteremia, is generally recommended prior to performing any dental procedure for these patients.

RECOMMENDATIONS

When treating these patients the dentist should be alert to the following considerations:

- ☐ These patients should receive antibiotic coverage prior to any dental procedure which requires stimulation or manipulation of the gingival tissue. The dosage for the antibiotic regimen should be established through consultation with the physician.
- ☐ When performing restorative therapy, the gingival margins should be placed above the tissue whenever possible.
- ☐ The design of any intraoral appliance should allow for ready access to all surfaces for easy cleaning and maintenance by the patient.
- ☐ Fluoride rinses or gels should be components of a rigorous daily preventive program.

CONCLUSION

Cyclic neutropenia is characterized by the complete disappearance or drastic reduction of neutrophils from the peripheral blood, at approximately twenty-one-day intervals. The oral manifestations, which are a significant component of the clinical features, include oral ulceration, gingival inflammation and rapid alveolar bone destruction.

As the symptoms associated with the oral cavity may be one of the primary debilitating facets of cyclic neutropenia, it is imperative that the practicing dentist be knowledgeable in the oral manifestations of this disease.

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Aberrations of development

Acquired unilateral condylar hyperplasia and facial asymmetry: report of case

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Robert L. Campbell, DDS

Cases of mandibular hyperplasia associated with facial asymmetry are documented in the medical literature as early as 1836.[1] From this condition the patient exhibits a progressive, unilateral elongation of the face with deviation of the chin away from the affected side and a severe malocclusion. This may or may not be accompanied by pain in the affected joint.[2] More than 200 documented cases appear in the dental literature, featuring certain characteristics of the disease. The abnormality is most commonly associated with trauma during the developmental years but localized growth disturbances (including neoplasm) are also causes of condylar hyperplasia.[3-5] Blomquist and Hogeman state that there is an equal sex distribution; other authors, however, feel that condylar hyperplasia is most commonly seen in females.[1,6,9] Unilateral mandibular hyperplasia, which has been reported more commonly than the bilateral disease, can also be classified as deviation prognathism.[4] In the pediatric patient the treatment of deviation prognathism requires a combination of orthodontic, surgical and general dental care. The sequencing of the treatment often depends upon the severity of the hyperplasia, the eruption pattern of the maxillary teeth, and the response of alveolar bone to the abnormal growth of the mandible. The following case demonstrates the complexity of evaluation of a

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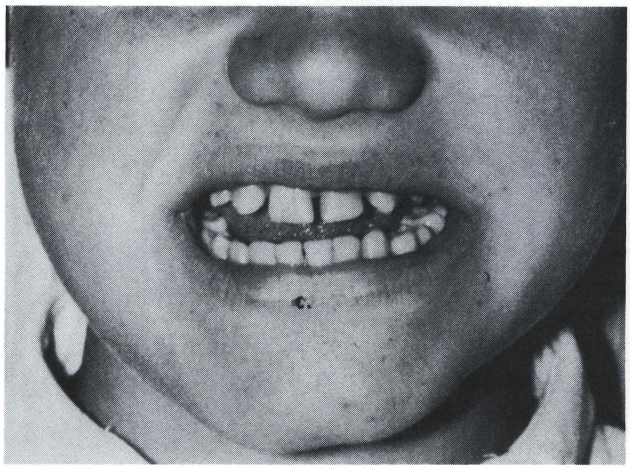

Figure 1. Preoperative full lower face view showing maxillary and mandibular midlines deviated to the patient's right, repaired fracture of the maxillary incisors, and transverse plane malalignment.

patient with facial pain, mandibular asymmetry, and growth compensations in the maxilla.

CASE REPORT

A twelve-year-old female, with a chief complaint of bitemporal headaches, left temporomandibular joint pain, and noticeable facial asymmetry, presented to the Medical College of Virginia, Temporomandibular Joint Pain Clinic in May, 1981. After appropriate referral to a local orthodontist, consultation was sought from the Department of Pediatric Dentistry and the Department of Oral and Maxillofacial Surgery. The medical history was essentially noncontributory, with the exception of a bicycle accident at the age of eight. The patient sustained a laceration of the chin and what was described to be class II fractures of the maxillary incisors.

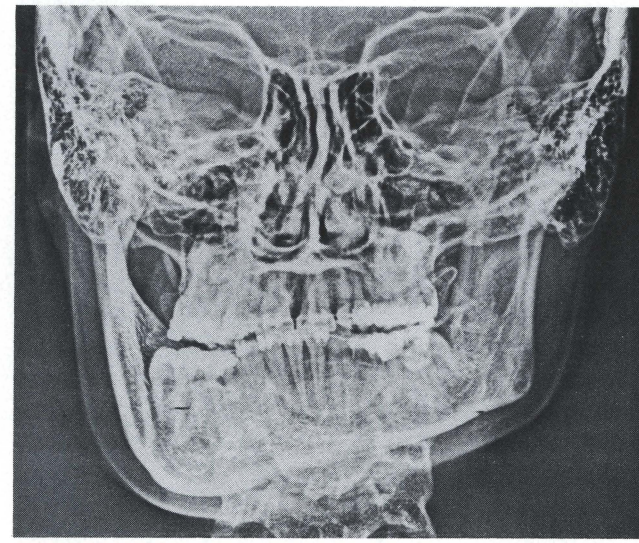

Figure 3. Preoperative anterior view from xeroradiograph, showing the facial asymmetry, excessive condylar growth on the patient's right, and severe transverse cant of the occlusion.

A facial asymmetry with the chin deviated to the left and a full-rounded contour of the right body of the mandible with a somewhat flat appearance in the left mandibular area were observed. The mandibular opening did not appear restricted in functional, lateral or protrusive movements. No pain was elicited during mandibular excursions at the time of the initial examination. In centric relation, the mandibular midline was 4.5 mm to the left of the maxillary midline (Figure 1).

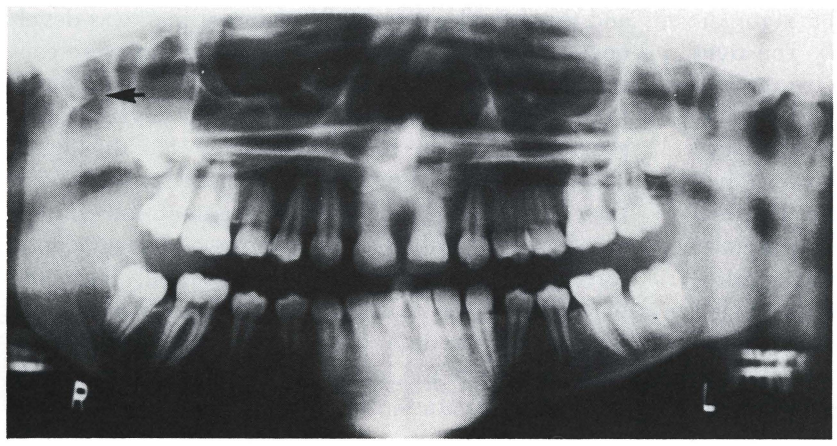

Figure 2. Preoperative panoramic view of the mandible, showing congenitally missing maxillary lateral incisors, large discrepancy between the alveolar and basalar bone in the mandibular posterior areas, and the growth deformity in the right condylar head and neck (arrow).

Figure 4. Preoperative tomogram of right condylar head and neck, showing beaked growth deformity (arrow). Tomogram of left condyle (not shown) showed no abnormalities.

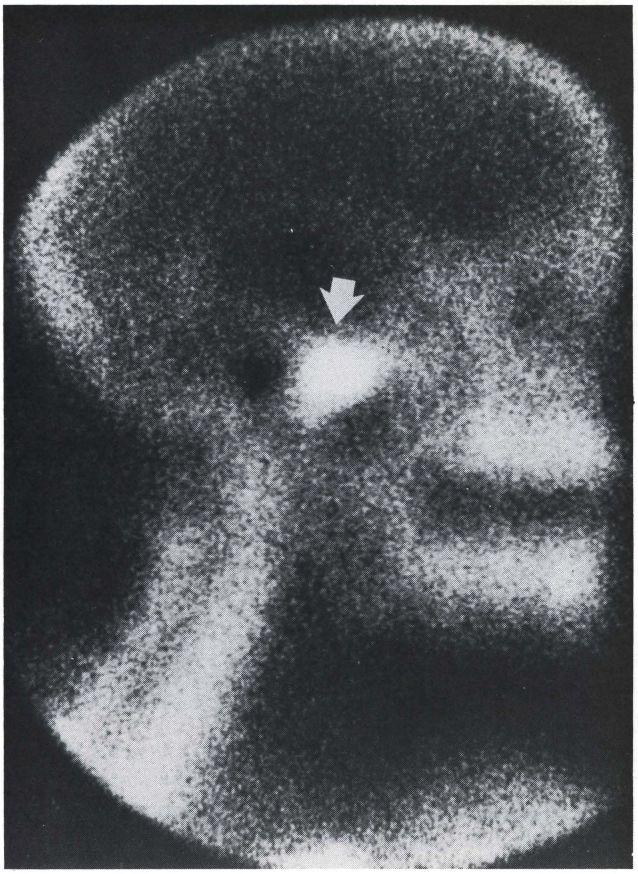

Figure 5. Preoperative lateral scintigram showing increased activity in the right temporomandibular joint (arrow). The dark area posterior represents the external auditory canal.

The maxillary midline was approximately 3 mm to the right of the maxillary lip-soft-tissue midline (a vertical line from the glabella to the nasal tip, bisecting Cupid's bow of the lip). The cant of the occlusion appeared as though the maxillary right premolars and molars had extruded approximately 5 mm on the right side in comparison to the left. Rather than representing extrusion of the teeth, it was most likely associated with compensatory maxillary alveolar growth. Radiographic evidence of an elongated right condylar neck and hyperplastic, deformed condylar head were evident on the temporomandibular joint panorex, the xeroradiogram, and the tomograph (Figures 2,3,4). The right body of the mandible appeared to have an overgrowth of the cortical plates where the normal antegonial curvature should be present. The inferior alveolar canal in the right body of the mandible was approximately 5 mm from the inferior border of the mandible. The total alveolar height, from the roof of the inferior alveolar canal, to the cusp tip of the first molar was approximately 5 mm greater than a similar measure on the left side. It appeared as though the alveolar process of the mandible had also increased in height in comparison to the left side (Figure 1). The xeroradiogram from an anterior posterior direction revealed a significant maxillary deformity. A technetium bone scan, utilizing technetium^{99} dihydrophosphate showed a markedly abnormal uptake in the right mandibular condyle indicating continued growth or destructive activity.[8] Calcium and phosphate ion turnover is responsible for the white in the lateral and anteroposterior views (Figures 5,6).

Course of Treatment

After consultation with a pedodontist, an oral surgeon, and the orthodontist, a full treatment plan was developed. Because the maxillary lateral incisors were congenitally missing, the maxillary canines were recontoured to the shape of the lateral incisors. The maxillary first premolars were also reshaped by removal of the lingual cusps to appear as canine teeth. These six teeth were then considered as the anterior segment and consolidated as a six-tooth segment. During early orthodontic care, the maxillary occlusal cant was partially corrected by intruding the maxillary right premolar and molar teeth. A 2.5 mm discrepancy still remained when comparing the right and left sides. During the orthodontic process, while attempting to consolidate the posterior

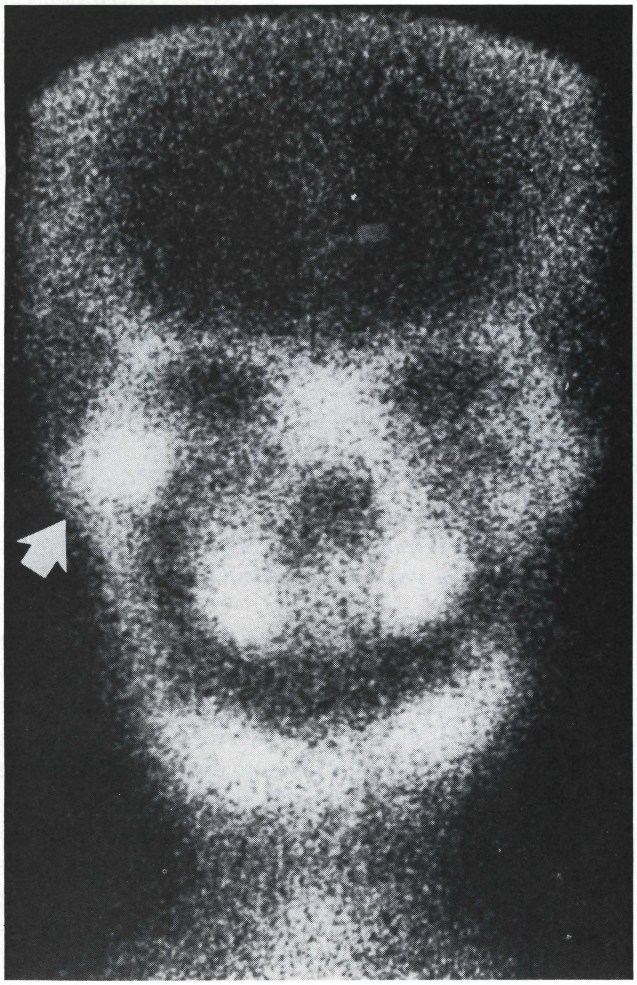

Figure 6. Preoperative anteroposterior view showing the increased uptake of the technetium in the patient's right condyle (arrow). There is no comparable activity on the contralateral side.

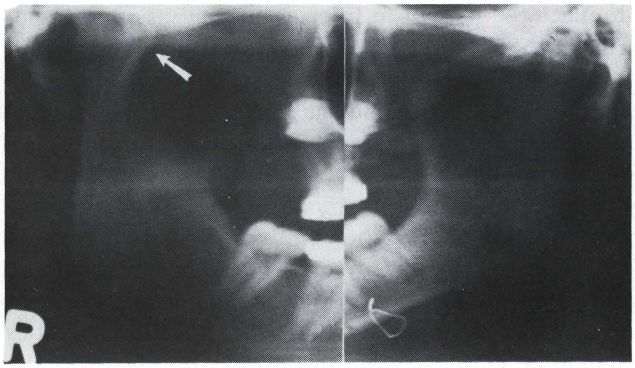

Figure 7. Postoperative panoramic view of mandible showing left and right condyle. Right condylar head is normal in size and shape, after condylectomy and recontouring (arrow). Wire on left mandibular border is stabilization for the proplast augmentation.

maxillary teeth, align the mandibular teeth, and correct dental rotations, the patient underwent a fairly rapid growth spurt of the right condyle. The facial asymmetry became more pronounced. The patient also

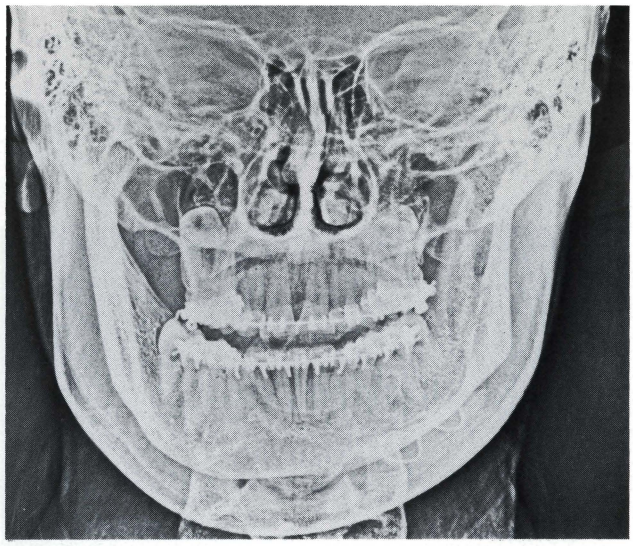

Figure 8. Postoperative anterior view from xeroradiograph, showing facial symmetry and normalized transverse cant of occlusion.

developed more severe pain and muscle spasm in the left temporomandibular joint (no obvious pathology was seen radiographically).

Because of the rapid growth, it was decided to change the original surgical plan (which would have corrected the malocclusion, the condylar hyperplasia, and the maxillary cant in one operation) to a condylectomy on the affected side, to correct the facial asymmetry. After anteroposterior cephalometric drawings were made, it was decided to remove 5 mm of the inferior border of the right mandible, 5 mm of condylar head, and augment the left inferior border of the mandible with Proplast®* by 5 mm. Two months prior to the surgical intervention, all active orthodontic forces were terminated. The maxillary teeth on the right side were then allowed to relapse. The surgical procedures as outlined were performed and the postoperative esthetics were considered more than satisfactory. The panorex taken postoperatively showed a more normal contour of the right condylar head and neck (Figure 7). The biopsy of the right condylar head showed no evidence of neoplastic disease.

Approximately six months from the first surgery, a second technetium bone scan was done in order to evaluate the activity of the right temporomandibular joint. The scan showed evidence of decreasing activity compared to the original scan. The clinical examination, including opening and lateral excursions, did not appear to be affected by condylar surgery. A second surgical procedure was then planned to correct the maxillary cant after consolidation of the posterior teeth with the six anterior maxillary teeth. A one-piece LeFort I maxillary osteotomy was done to intrude the right maxilla approximately 3 mm. Since the left inferior bor-

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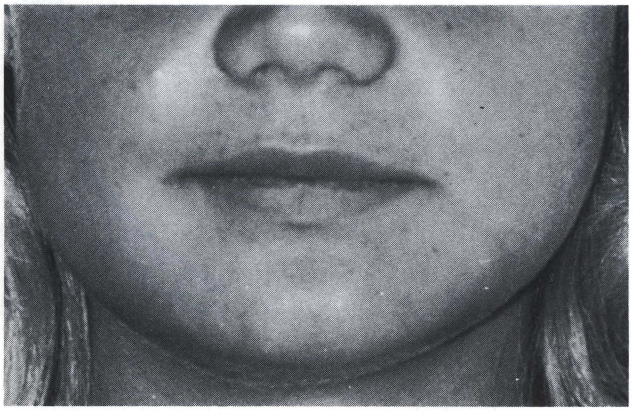

Figure 9. Postoperative full lower face view showing facial symmetry and acceptable aesthetics.

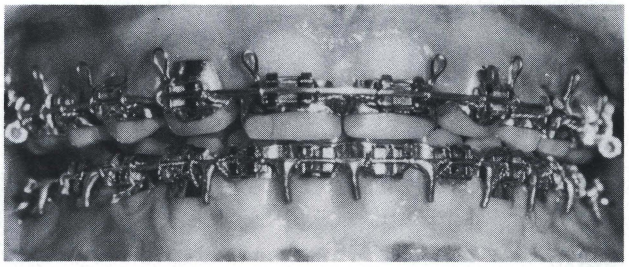

Figure 10. Postoperative intraoral frontal photograph showing corrected transverse cant of occlusion, corrected midlines and final stages of orthodontic treatment.

der of the mandible had an alloplastic implant (Proplast), it was decided to perform an extraoral closed subcondylar osteotomy on the left mandible and a conventional intraoral sagittal split osteotomy on the right. The closed subcondylar was performed through two small incisions in front and back of the condylar neck in order to prevent an intraoral incision from encroaching on the proplast implant at the inferior border of the mandible. The maxilla and mandible were stabilized in a normal fashion with interdental and skeletal fixation. At the nine-month follow-up, the patient was asymptomatic from the standpoint of facial or temporomandibular joint pain. There was no evidence of continued growth from the condyle, as demonstrated by a normal repeatable occlusion and esthetic facial symmetry (Figures 8,9,10).

DISCUSSION

The case history of this young female is not atypical of that found in patients who have unilateral hyperplasia of the mandible.[5] The bicycle accident occurred at age eight, when significant condylar growth was taking place. Whether the patient suffered a fracture of the head of the right condyle or of the right condylar neck is difficult to determine. Since not only the size of the condylar head but also the neck was significantly increased, it is possible that the injury was at the junction of the neck and head of the right condyle. We propose that this injury led to the acquired condition of mandibular hyperplasia, since there was no other genetic or metabolic disorder which would be associated with unilateral condylar hyperplasia.

The initial treatment after the accident consisted of radiographs in the emergency room for a possible right wrist fracture as well as the closure of lip lacerations. After the emergency room visit, temporary treatment for the fractured maxillary incisor teeth was given. At no time were radiographs taken to evaluate the patient for possible condylar or mandibular fractures.

In addition to a dental evaluation, it is appropriate to evaluate the patient for symptoms and signs of mandibular fracture. These would include: pain during mandibular function, malocclusion of the teeth on attempts to close, midline deviation during opening, and the presence of swelling in the preauricular area.[5] If occlusion is repeatable without significant pain, it is not necessary to apply intermaxillary fixation. If the occlusion is not repeatable, despite the absence of a condylar fracture on the radiograph, it is appropriate to place the patient in intermaxillary fixation for a period of five to seven days. This would allow the patient to become more comfortable, regardless of whether the diagnosis is a high condylar fracture that may not be picked up on a radiograph, a hematoma of the upper or lower temporomandibular joint compartments, or spasm of the lateral pterygoid muscle. After relatively firm intermaxillary fixation for five days, loose interarch elastics can be utilized to correct any deviations on opening. The decision to put one or more elastics on the appropriate side to allow the mandible (elastics should be placed on the unaffected side to correct the deviation to the affected side) to open in a straight up and down direction is important. Follow-up radiographs should be taken, one year after the trauma. After this point, regular evaluation of the occlusion as well as the transverse maxillary plane is important to rule out clinically condylar hyperplasia in its early stages. It is generally thought that the condyle can produce an abnormal growth response up to the final growth spurt which is normally achieved within one year after the onset of menarche. Deviation of the mandible rather

than a straight opening pattern is also a very early clinical sign of condylar disease or meniscus dysfunction. In this case there were no signs of clicking, popping, grating or intermittent locking of the temporomandibular which would normally necessitate an arthrogram.

Previous fracture of the mandibular condylar process has been estimated to have occurred in up to 10 percent of all those patients with severe mandibular deficiencies or asymmetry problems.[5] Other studies, associated with mandibular fractures in young people with subsequent mandibular deformities, are nonconclusive. Eight percent to 78 percent of patients with mandibular condylar fractures also experience growth disturbances after the injury.[1,5] In our patient, compensatory left temporomandibular joint pain and facial asymmetry were first seen at age eleven, three years after the original trauma. This latent period of two to three years after the injury is commonly seen and well documented.[7,9,10] The condition worsened after the onset of menarche and seemed to spurt between ages eleven and fourteen (the time of the first surgical intervention). It is generally accepted that if a long bone is fractured during puberty, accelerated growth at the epiphyseal plates and elongation may occur.[11] In our patient, this probably occurred at the junction of the condylar neck and the condylar head; perhaps in the area of endochrondral bone formation. It may be more desirable, therefore, to intervene surgically in a hyperplastic growth area, prior to the full-growth period, since more extensive surgery may be needed to correct the more advanced deformity at a later date, as illustrated in our case. When maxillary dental compensations, in the form of alveolar growth, have occurred secondary to hyperplastic growth of the mandibular condyle, a combined maxillary and mandibular surgical procedure should be planned by intruding the maxilla and shifting the mandible toward the pathological side. With this treatment plan, a good surgical result could have been obtained. In this case, however, an attempt was made to correct the maxillary cant orthodontically. The initial treatment was directed at alignment of the arches and correction of the tipping or rotational movements, followed by surgical correction of the maxillary cant and mandibular hyperplasia. At the time of the first surgical intervention, since part of the maxillary compensations had been reduced orthodontically, the maxilla could only have been moved up 2.5 mm to correct the discrepancy in the transverse plane; but the mandibular occlusal plane needed to be moved up 5 mm or more to produce a Class I molar occlusion. An alternative plan, therefore, had to be developed. The first surgical procedure was done to arrest the rapid condylar growth and correct the laterognathism. Augmentation of the inferior border of the mandible was necessary, however, to correct the remaining facial asymmetry. Proplast, a microporous synthetic carbon and polytetrafluoroethylene material, which allows ingrowth of fibrous tissue was used in lieu of silastic, for better stabilization.[12] An autologous rib would have required an additional surgical site, leaving a residual scar. Homologous, freeze-dried rib from another donor was not used, because of the possibility of severe, unpredictable bone resorption and infection.[13]

Making the assumption that removing the active orthodontic forces would allow another 2.5 mm extrusion of the eight maxillary molar and premolar teeth, it was planned through cephalometric analysis and tracing of the xeroradiogram of both the hard and soft tissues, that a second procedure could be done to correct the cant of the maxilla and the mandible. Although it may have been more desirable to avoid biarch surgery and intrude the maxillary teeth from the right side and extrude the maxillary teeth on the left side, this is a potentially unstable move, orthodontically, and subject to relapse. In order to prevent surgical relapse, maxillary, mandibular and condylar surgery (high condyle shave) could have been done in one surgical procedure. However, this would necessitate intermaxillary fixation for a minimum of four to six weeks. During this period of fixation, immobility of the condyle would lead to either fibrous or bony ankylosis. For this reason, orthodontics and surgical intervention were done in stages. This case illustrates the necessity of coordinating the orthodontics, surgery, and general dental health of the patient.

CONCLUSION

Unilateral condylar hyperplasia, which is most commonly thought to be caused by trauma, presents a problem that is best managed by a team. Often the general dentist or the pediatric dentist is the first to see these patients after the patient is seen in the emergency room. If a fracture of the condyle is ruled out after initial evaluation, it is incumbent upon the dentist to evaluate the patient over a long-term period. Emergency dental care should be administered to patients who have dental fractures but a high degree of suspicion should be employed for the long-term management of the patient. Because of the severe consequences

of this type of injury, it is important to evaluate the patient radiographically and clinically at intervals during the normal growth and development period. Although the oral surgeon may provide emergency treatment, the primary health care practitioner is responsible for the overall management and diagnosis of a growth deformity. Interceptive orthodontics may be instituted, if any suspicion of compensatory maxillary dental growth seems to be occurring. A flat-plane, night-type appliance covering the occlusal surfaces of the maxillary teeth could prevent the supraeruption of the teeth on the affected side. Even though this would not influence the mandibular teeth and their alignment, the deformity would be confined to one arch. Definitive orthodontic support can then be sought. The proper planning of both the orthodontics and the surgery is paramount, as illustrated by this case.

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DENTISTRY AND THE LAW

Business law affects the practice of dentistry. For example, touching on each of several aspects of practice is contract law. Contract principles run not only through each of the above umbrella areas, but also throughout a dentist's professional life and the typical business day. For example, whether you practice as an independent contractor or as an employee, your position is governed by contract law. When acquiring space to practice via lease you enter a contract. Employees from hygienist to receptionist, represent a dentist's practice pursuant to a contract: an employment agreement. Other commonly encountered contracts include supply purchases, patient care, hiring temporary help, and agreements for equipment repairs. Contracts affected by government regulation, labor relations, and consumerism exist virtually from the first day of practice to the last. Yet, usually only in special circumstances does a dentist seek the aid of the attorney to interpret contractual terms and to assist in securing the optimum negotiated legal position.

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Hyperdontia in children below four years of age: a radiographic study

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Sigurd Humerfelt

According to most accounts, supernumerary teeth occur more frequently in the permanent dentition than in the primary dentition.[1] In studies that discriminated between the primary and permanent dentitions, hyperdontia occurred in the primary dentition in a range of 0.03 percent to 1.9 percent.[1,2] Differences in materials and methods may account for this wide range. In particular, studies of older age-groups may fail to record primary supernumerary teeth lost by normal exfoliation, because they often erupt normally, are often normally shaped, and frequently appear to be in proper alignment.[1,3,4] As a result, the abnormality goes unnoticed by patients or their parents.

Hyperdontia shows a predilection for the premaxillary sites: in the primary dentition, in the region of the lateral incisors; in the permanent dentition, in the central incisor region.[4-7]

The objectives of our investigation were to present characteristics of hyperdontia in young children, in whom both dentitions were accountable, to permit evaluation of the differences in this anomaly between the primary and the permanent dentitions.

MATERIAL AND METHODS

The study population was comprised of sixty-six children of Norwegian origin (41 boys and 25 girls), ranging in age from nine months to three years, eleven months; the mean was two years, five months. They were the youngest of 900 patients with a diagnosis of premaxillary hyperdontia, admitted to the Department of Oral Radiology, University of Oslo, during recent years. None of the patients was afflicted with a predisposing congenital disorder, such as cleft lip and palate or cleidocranial dysostosis.

Radiographs included occlusal and profile views of the premaxillary region. Whenever desirable, supplemental radiographs were taken, to improve the accuracy of localizing the supernumerary tooth. Standard films, equipment, exposure technique, and processing procedures were employed, and the radiographs were interpreted under optimal lighting conditions.

Distinction between primary and permanent supernumerary teeth was based primarily upon their topographical relation with either of the dentitions, and with due regard to their shape and state of development. The reliability of this radiographic assessment was checked by histological evaluation of neonatal lines in the dentin of twelve extracted supernumerary teeth, randomly selected. Agreement was achieved in all cases.

The data were coded and transferred to a computer.

Professor Hanna S. Koppang, Department of Oral Pathology, University of Oslo, was the histological consultant for this study.
Dr. D. Humerfelt is acting head of the Department of Oral Radiology; Dr. Hurlen is associate professor, Department of Oral Surgery and Oral Medicine, Dental Faculty, University of Oslo. Mr. S. Humerfelt is software engineer.

Table 1 ☐ Distribution of supernumerary teeth with regard to location and the dentition from which they emanated.

	Region				
12/52	11/51	21/61	22/62	Total	
Primary dentition	20	5	4	16	45
Permanent dentition	11	17	14	8	50
Total	31	22	18	24	95

Table 2 ☐ Distribution of patients with premaxillary hyperdontia by sex and origin of supernumerary teeth. Number of supernumerary teeth in parenthesis.

Origin	Male	Female	Total
Primary dentition	11 (12)	10 (12)	21 (24)
Permanent dentition	15 (18)	12 (13)	27 (31)
Both dentitions	15 (33)	3 (7)	18 (40)
Total	41 (63)	25 (32)	66 (95)

Table 3 ☐ Position of the supernumerary teeth in relation to the dental arch and the dentition from which they are derived.

	Position relative to the dental arch					
Origin	Labial	Labial/ within*	Within	Palatal/ within*	Palatal	Total
Primary dentition	2	1	35	2	5	45
Permanent dentition	1	0	5	2	42	50
Total	3	1	40	4	47	95

*Part of the tooth located within the dental arch

RESULTS

Distribution of supernumerary teeth

The sixty-six children had ninety-five supernumerary teeth, or 1.4 supernumerary teeth per individual (1.5 per boy and 1.3 per girl). The distribution of primary and permanent supernumerary teeth by location is shown in Table 1. The dentitions were about equally represented, and in eighteen cases the primary supernumerary tooth had a successor in the permanent dentition (Table 2). These eighteen children had twenty-one primary and nineteen permanent supernumerary teeth.

Two of the patients also had supernumerary teeth outside their premaxillary regions.

Location

Although of small significance, the supernumerary teeth were found more frequently on the right side of the median suture than on the left side, fifty-three and forty-two respectively, in both dentitions. Primary supernumerary teeth were predominantly seen in the lateral incisor region, whereas those in the permanent dentition were predominantly found in the region of the central incisors (Table 1).

Position

All the primary supernumerary teeth and 54 percent of the permanent ones were in a vertical position with the crown of the tooth pointing downwards. Approximately 76 percent of supernumerary teeth in this position were situated in the regions of the lateral incisors. Twenty-one permanent supernumerary teeth (22 percent) were in a vertical, but inverted position, and in the central incisor regions. Only two supernumerary teeth were in a horizontal position, both belonging to the permanent dentition and in the central incisor regions.

From the sagittal views, the primary supernumerary teeth were predominantly within the dental arch; whereas, permanent supernumerary teeth were predominantly seen palatally to the arch (Table 3). Two of the latter were found in close proximity to the nasopalatine bone.

Eruption

Thirty-three (73.3 percent) of the primary supernumerary teeth had erupted before the first examination was made. The remaining primary and all the permanent supernumerary teeth were unerupted at this time.

Rate of tooth development

Judging from the radiographs, root development was completed in forty-five supernumerary teeth (one permanent and forty-four primary). In fifty teeth, development was incomplete (one primary and forty-nine permanent). The state of development of the supernumerary relative to that of the corresponding normal tooth could be assessed only when root development was incomplete, at least in one of them. This comparison could not be made for forty-three of the primary teeth. For the remaining fifty-two teeth, the comparison showed that the state of development of the su-

pernumerary teeth equaled that of the corresponding normal tooth in thirty-two cases, was advanced in fifteen cases, and delayed in five cases.

Malformation

Malformation occurred in fifty-six teeth and in forty patients. Cleavage of the tooth-bud, resulting in twin-tooth formation, occurred in twenty-eight patients; followed by fusion; concrescence; and gemination. In this context, the expressions employed are those previously discussed by Tannenbaum and Alling.[8] *Dens invaginatus* was seen in one permanent supernumerary tooth, and in another, the root was extremely hook-shaped. Malformation occurred most frequently in the primary dentition: 70 percent of the twin-tooth formation, and with one exception, in the lateral incisor region. Malformation was seen in both dentitions, in eleven of twelve cases.

Interference with the normal dentition

The supernumerary teeth interfered with the normal dentition in several ways. In nine cases, permanent supernumerary teeth caused displacement of the median suture. In five cases, supernumerary teeth in the central incisor region caused diastema or displacement of neighboring teeth. One of these was a primary supernumerary tooth. Rotation of permanent teeth adjacent to supernumerary teeth was observed in nine cases of permanent supernumerary teeth, obviously due to the presence of the extra tooth. Impaction was seen in five cases, four of them in the permanent dentition. Follow-up radiographs one to six years later(mean: two years, nine months) showed that the hinderance was not temporary—the contralateral unhindered tooth had advanced, the impacted one had not.

DISCUSSION

Disagreement in previous reports regarding the prevalence of primary supernumerary teeth, may in part be ascribed to differences in methods employed; the age composition of the study population, however, seems to be more important. The highest frequency was found in the youngest age-groups. There is no doubt that the reason for this age-dependent discrepancy is loss by exfoliation or extraction of supernumerary teeth during childhood. A reliable assessment of hyperdontia in the primary dentition, therefore, should be based on a representative group of children well below the age of normal tooth shedding. A careful history, furthermore, should be taken to disclose any possible removal of supernumerary teeth prior to the examination. The present study, however, did not meet the latter requirement, and the deductions made did not include the assessment of prevalence.

It is noteworthy that nearly half of the supernumerary teeth found in our investigation occurred in the primary dentitions. This suggests that the prevalence of primary supernumerary teeth has been considerably underestimated, at least concerning the premaxillary region. In the present study, both dentitions were present and recorded on radiographs. Most supernumerary teeth develop in conjunction with the corresponding normal teeth; some a little earlier; but very few, later. Followup radiographs of twenty-two of the youngest children did not unveil additional supernumerary teeth. Late development of supernumerary teeth has been reported, but it is rare.[9]

There were several differences in the characteristics of primary and permanent supernumerary teeth. According to previous reports, a low percentage of supernumerary teeth erupt; but in the present study as high as 73.3 percent of the primary supernumerary teeth erupted. As expected, none of the permanent supernumerary teeth had erupted, at the time of examination.

The predominance of boys among the children with hyperdontia in both the primary and the permanent dentitions was astonishing. In children with primary supernumerary teeth only, no sex difference could be seen. This is in agreement with the finding of Randle Luten, but not with that of Järvinen and Lehtinen, who found a male/female ratio of 4:1 in Finnish children.[3,5]

In the primary dentition, supernumerary teeth occurred most often in the lateral incisor regions, as opposed to permanent supernumerary teeth which prevailed in the central incisor regions. The preponderance of supernumerary primary lateral incisors may reflect a locally disturbed odontogenesis caused by the biological activity connected with the fusion of the premaxilla with the maxillary process, forming the incisive suture. Another theory suggests that supernumerary teeth in this region represent a microform of cleft palate: that the dental lamina on both sides of the fissure possesses the potential of forming a lateral incisor tooth.[10] If so, these germs normally have to unite and proliferate as one tooth unit. Possibly then, gemination may represent an abortive union. The occurrence of supernumerary teeth, as well as malformed teeth, away from the sites of fusion of fetal processes, indicates that pro-

liferation of extra tooth buds may be initiated by various factors.

Furthermore, the lateral incisor regions seem to be the preferred areas for twinning of teeth. They were the sites of twinning in twenty-seven of the twenty-eight patients in whom this phenomenon occurred. Both dentitions were represented, but mostly the primary dentition. Only one single permanent supernumerary occurred in the central incisor region.

There were differences between the two dentitions not only in the locations, but also in the positions of the supernumerary teeth. All inverted teeth were permanent, situated in the central incisor region and palatally to the dental arch. The reason for this inversion is obscure, but a possible explanation of both the inversion and the palatal position might be an occasional posterior folding of the dental lamina in this area.

The immediate clinical importance of supernumerary teeth in the present age-group was not as obvious as its occurrence at older ages. The main complications were various types of interference with the normal dentition, including malposition and delayed or prevented eruption; but less frequently than in older populations. This may be explained by the high rate of normal eruption of the primary supernumerary teeth, and possibly by a greater ability of adjustment in this age-group. Moreover, the impact of permanent supernumerary teeth could hardly be fully unveiled until the time of normal eruption. The present study, however, showed that early radiography could help us predict maleruption of permanent teeth even in the youngest children. In our experience, hyperdontia in the primary dentition seldom warrants surgical intervention; and generally, the removal of permanent supernumerary teeth should await at least the time of normal eruption of the corresponding normal teeth. Whether and when removal is necessary should be decisions made for the individual case. The frequency of hyperdontia involving both dentitions in the present material emphasizes the importance of careful radiographic follow-up in cases of primary supernumerary teeth, particularly when the anomaly occurs in the lateral incisor region.

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WHY RADIOGRAPH THE PRIMARY TEETH

Radiographs show not only the condition of the primary teeth but also the developing permanent teeth. The retention of primary teeth until the proper time for shedding them is important to the future dental health of a child. Demonstration radiographs can show conditions such as the tipping of permanent teeth due to early loss of a primary tooth, a missing permanent tooth requiring extra care to protect and retain the primary tooth, and abnormal resorption of the primary tooth root with associated malpositioning of the permanent tooth requiring extraction of the primary tooth. Early identification and treatment of these problems can often avoid expensive orthodontic correction at a later date.

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Hereditary agenesis of ten maxillary posterior teeth: a family history

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Among nordic populations, the prevalence index of hypodontia is 6 to 8 percent in the permanent dentition, excluding the third molars.[1] In the primary dentition, it is 0.5 to 0.9 percent.[2-4] Agenesis of the first and second permanent molars and the primary molars is extremely rare.[1-5]

Agenesis of teeth is often unilateral. Approximately half the congenitally missing teeth are missing unilaterally.[6] This fluctuating asymmetry results from the failure of the organism to duplicate perfectly a bilateral structure.[7]

Many studies on families and twins have shown the importance of genetic factors in the occurrence of dental agenesis.[5] Some investigators consider hypodontia to be the result of a single dominant gene; although others have opted for a recessive hypothesis.[8,9] Some recent investigators favor the hypothesis of a polygenically determined, quasi-continuous type of inheritance.[10-13] Combined genetic and environmental influences should be considered as etiological factors.[13]

Hypodontia is not an isolated phenomenon, but is linked to a complex of dental changes. It is related to the frequency of missing teeth, the size and form of the remaining teeth, and the overall rate of dental development.[14-17] Moreover, hypodontia is a very common anomaly in children with cleft lip and palate, in trisomy 21, in ectodermal dysplasia, and in many other syndromes.[18-22]

The purpose of this paper was to describe a family where the mother and her sons had agenesis of all maxillary permanent molars, and the second primary molars and their succedaneous premolars.

REPORT OF THE CASES

The clinical and radiographic examinations were done in the Department of Dentistry, The Finnish Red Cross Cleft Center.

Mrs. S.Y. was born in September, 1946. She said that none of her teeth had been extracted. Clinically and radiographically, the upper second premolars, and the first, second and third molars were congenitally missing (Figure 1A). The size, shape and form of the remaining teeth were within normal limits. The alveolar process in the posterior area of the maxilla was totally absent. The lower first and second molars were elongated and the third molars impacted. She had a slight nasality of speech and adhesive otitis media, but was otherwise healthy. Mrs. S.Y. reported that her parents and other near relatives had no hypodontia similar to hers.

Mr. M.Y. was born in February, 1945. He and his near relatives had no history of hypodontia.

Their children

Master J.Y. was born in July, 1973. Radiographs revealed the congenital absence of the following maxil-

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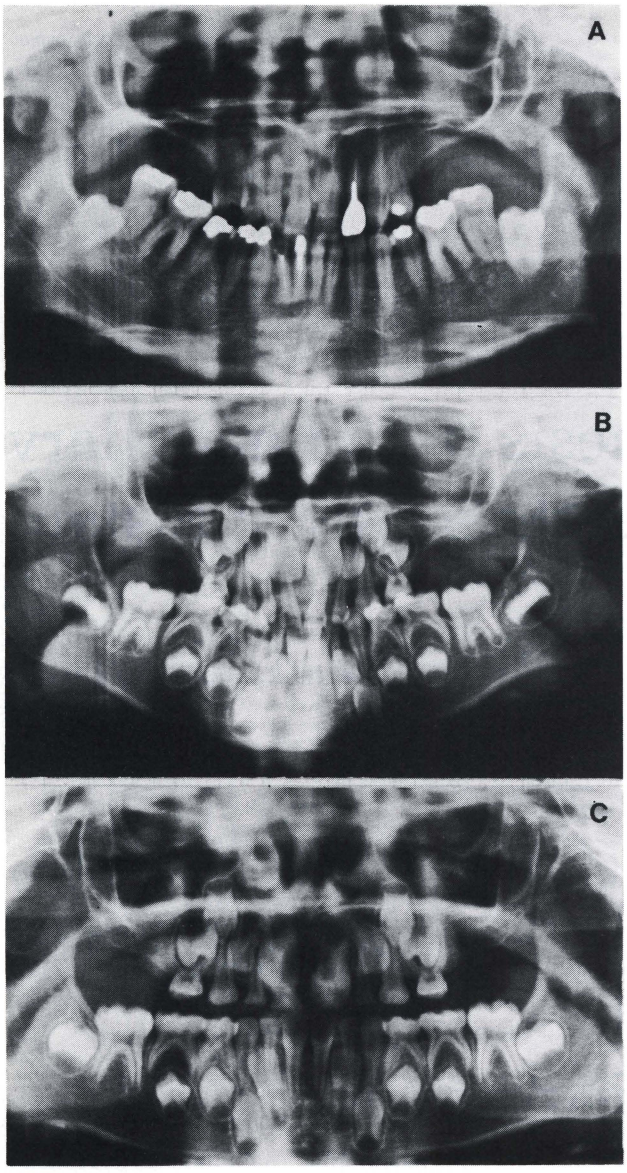

Figure 1. Radiographs of the patients. Mother S.Y. (A), Master J.Y. at the age of 6.2 years (B) and Master T.Y. at the age of 8.1 years (C). Note the similarity of the patterns of the hypodontia and of the upper posterior alveolar processes, in the three radiographs.

lary teeth: the second primary molars, the second premolars, the first and second molars and possibly the third molars. The maxillary alveolar process of the posterior region was totally absent (Figure 1B). He, like his mother, had a slight nasality of speech and adhesive otitis media, but was otherwise healthy. His dental age and the size, shape and formation of his teeth were within normal limits.

Master T.Y. was born in March, 1976. Radiographs revealed the following congenitally absent maxillary teeth: the second primary molars, the second premolars, the first, second and possibly third molars. The alveolar process in the area of hypodontia was absent (Figure 1C). His speech and overall somatic growth were normal. He also had adhesive otitis media.

DISCUSSION

This family history strongly supports the argument that the etiology of hypodontia has an important hereditary component. The mother and her two sons had the same teeth congenitally missing, in symmetric patterns. In general, familial hypodontia has marked variation in relation to the missing teeth, and the teeth are missing quite often only unilaterally.[5,6,13] The size, shape and formation of the developing teeth of the mother and the two boys were within normal limits. Interesting features were a slight nasality of speech of the mother and one boy, and adhesive otitis media, in the mother and both boys.

In these cases, hypodontia of the maxillary posterior teeth was not associated with delayed development or microdontia of the remaining teeth. Thus, hypodontia in these cases seems to be associated with a developmental disorder of the posterior maxilla. Hypodontia may be a secondary result. Review of the literature on hypodontia in association with specific syndromes disclosed only the Gorlin-Chaudhry-Moss syndrome, in which hypodontia similar to the present cases is reported to occur.[21,22] The specific disorders of the syndrome are craniosynostosis, midface hypoplasia, hypertrichosis, and anomalies of the eye, teeth, heart, and external genitalia.[21] These anomalies and disorders were not diagnosed in the present cases. Thus, the present familial hypodontia associated with nasality of speech and adhesive otitis media may represent a new syndrome. According to Jorgenson "the significance of hypodontia in syndromes is obvious: when a specific pattern of agenesis is observed, a syndrome may be suggested".[22] In this family, the mode of inheritance of hypodontia and the other disorders may be autosomally dominant with complete penetrance.

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MERSYNDOL FOR TMJ PAIN DYSFUNCTION SYNDROME

The analgesic/antihistamine combination known as Mersyndol was selected for a trial in the management of pain associated with TMJPDS, as it has proved to be successful in the management of acute migraine attacks, and a related preparation (Syndol), which contains 30 mg of caffeine, had been reported to be of value in the management of tension headache, pain following dental surgery, and pain following orthopedic operations.

Mersyndol contains two analgesics (paracetamol and codeine phosphate) and an antihistamine (doxylamine succinate). We felt that their combinated effect in a condition generally considered refractory to ordinary analgesics and somewhat responsive to anxiolytics deserved analysis.

The analgesic action of codeine is believed to depend on a central effect, while paracetamol may owe its pain-relieving properties to a peripheral effect antagonizing the action of bradykinin or possibly by inhibiting the synthesis of prostaglandins. The inclusion of the antihistamine, doxylamine, may exert an additive effect by allaying anxiety and in turn decreasing muscle spasm.

The results of this study indicate that Mersyndol was capable of significantly relieving pain associated with TMJPDS and that the benefit was not due to placebo effect alone. From these results it seems reasonable to recommend the use of it as an effective symptomatic, albeit temporary treatment, in the management of TMJPDS while arranging for definitive treatment.

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Dentinal dysplasia type I: report of case

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C. William Snyder, PhD, DDS
Ghada Baka'een, MD, DCH

There is a variety of systemic and genetic reasons for the premature exfoliation of teeth and for dental malformations. Dentinal Dysplasia Type I is a rare hereditary condition of anomalous dentin formation.

Characteristic findings in Dentinal Dysplasia Type I are: Normal enamel formation, defective root formation, periapical radiolucencies, and pulp chambers that are absent or malformed, and a horizontal radiolucent line at the dentinoenamel junction.[1]

Ballschmiede published the first report of this affliction in 1920, followed by Rushton in 1939, who termed it "dentin dysplasia".[2,3]

A complete review of the literature was done in 1982 by Dym *et al.*[4]

CASE REPORT

This eight-year-old male exfoliated malformed teeth since eighteen months of age. He was admitted to King Hussein Medical Center, Amman, Jordan, for investigation of this malady.

He was the son of healthy parents, and was born at full term with no relevant prenatal or postnatal history. There was a history of infantile facial eczema and chronic diarrhea in early childhood. Some of his developmental milestones were delayed: for example, he was two years of age before he walked.

The heights of his parents, brothers, and sisters were within normal ranges and the immediate family had normal dental development.

At the time of hospital admission, routine blood and blood chemistry tests were within normal limits, except for microcytic hypochromic anemia, which was verified by bone marrow biopsy. Skull radiographs showed normal sella turcica; chest radiographs showed normal heart size and clear lungs. His bone age was determined to be about five years (chronological age was eight years).

A panoramic dental radiograph (Figure) showed a mixed dentition with short, conical, misshapen roots. The root canal systems of the primary teeth were obliterated or malformed. The permanent dentition was remarkable in that the root canal systems were absent with anomalous root formation, which was most striking in the lower incisors. Also visible in the radiograph was a follicle-like structure in the right third molar region. The few teeth clinically present were extremely mobile. The permanent incisors had a slightly yellower cast than normal and there was the usual color difference between the permanent and primary dentitions.

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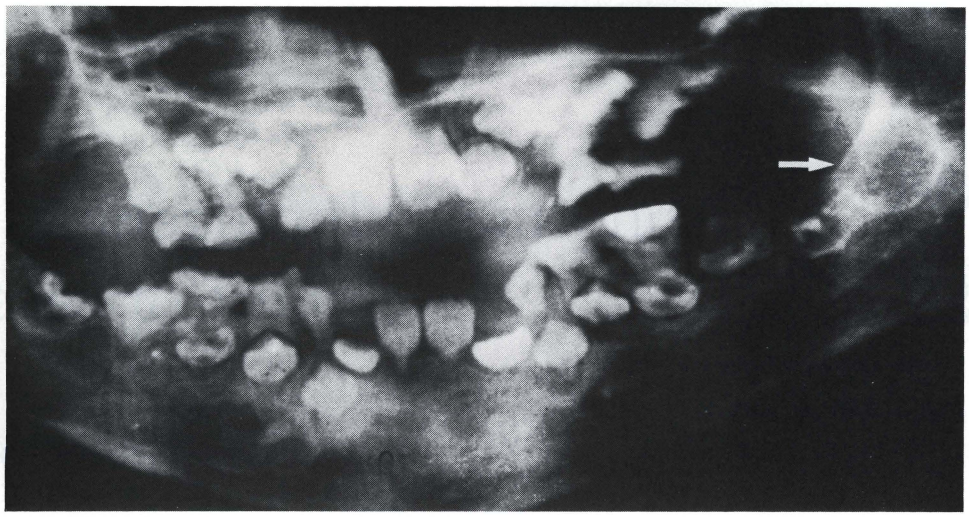

The arrow indicates a follicle-like structure in the third molar region. Note the abnormal dental morphology.

The oral mucosa was clinically normal.

The patient's anemia was thought to be of nutritional origin, due to the inability to chew various foods. He was placed on multiple vitamins with iron therapy; a recent blood workup was within normal limits. At this time his weight and height were in the third percentile for his age.

On physical examination at age five, he appeared pale; chest, heart, abdomen, limbs, lungs, skin, and central nervous system, however, were within normal limits. His weight and height were below the third percentile for his age. No dental examination was performed at this time, but his teeth apparently erupted at the normal times.

DISCUSSION

The genetic inheritance pattern of Dentinal Dysplasia Type I is autosomal dominant. There is usually a familial pattern with this affliction, but in this case and others described in the literature, no such pattern was apparent.[3,5,6]

As a result of poor root formation, these teeth loosen and exfoliate early, even with normal eruption times. The symptomatic treatment is extraction with removable prosthetic devices as the ultimate restorative modality.

This case is unusual in that there is some deviation in the normal bone development pattern and in the existence of the cyst-like follicle in the third molar region.

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Case reports

Abnormal odontogenesis: report of case

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David S. Richardson, DDS, MS, FRDC(C)

Abnormal odontogenesis not related to genetic aberrations or known local factors is uncommon. The causes of most odontogenic aberrations are unclear and may include any abnormal occurrence or element during odontogenesis. Descriptions of these aberrations, therefore, should not be limited to a specific anomaly. The literature on abnormal odontogenesis of the type described in this report is sparse. Features of the case presented here have not appeared in previous reports.

In the case report to follow, no definitive diagnosis could be made; but a differential diagnosis and a possible explanation were explored and discussed.

CASE REPORT

The patient, a twenty-seven-month-old white female, presented for a routine dental examination, in May, 1983. The examination revealed the clinical absence of the maxillary left primary first molar; all other primary teeth were erupted and noncarious. This condition was of unknown duration and represented an incidental finding during the patient's first clinical examination.

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Dr. Thomas Aufdemorte was the consultant oral pathologist.

Family history

The patient was an only child and there was no history of this or a similar condition in her immediate family.

Medical history

The patient was born six weeks premature and the mother was Rh sensitized, but the patient's mental and physical development were normal. The patient had the expected childhood diseases, including occasional mild episodes of bronchitis. All of her immunizations were up to date and she had no previous febrile illnesses. There was no history of trauma or infection in the affected area; the patient did have, however, a discolored maxillary left primary central incisor.

Radiographic findings

Radiographs of the maxillary right and left primary molar regions revealed an impacted maxillary left primary first molar and a radiolucent, partially radiopaque lesion in the area coronal to the impacted primary first molar (Figures 1, 2). The radiographic appearance was reminiscent of a developing tooth, although in an inverted position.

Treatment

An excisional biopsy, performed under local anesthesia, produced a firm, irregularly ovoid piece of gray-tan tissue, measuring 1.0 × 0.8 × 0.7 cm. The biopsy site healed without incident, within seven days; and there has been no recurrence of the lesion to date.

Five months after surgical treatment was performed, follow-up radiographs were taken which revealed the maxillary left primary first molar still to be impacted, with no maxillary first premolar forming apically (Figures 3, 4). All other structures appeared intact and present in the follow-up radiographs.

Histologic findings

The histologic examination revealed cellular collagenous connective tissue, associated with proliferative odontogenic epithelial rests with ameloblastic features. There were also calcific debris and acellular eosinophilic matrix, suggestive of an abnormal attempt to produce dentin matrix. The odontogenic rests were in intimate relation to the calcific debris. The collagenous tissue was focally myxoid and ground substance rich.

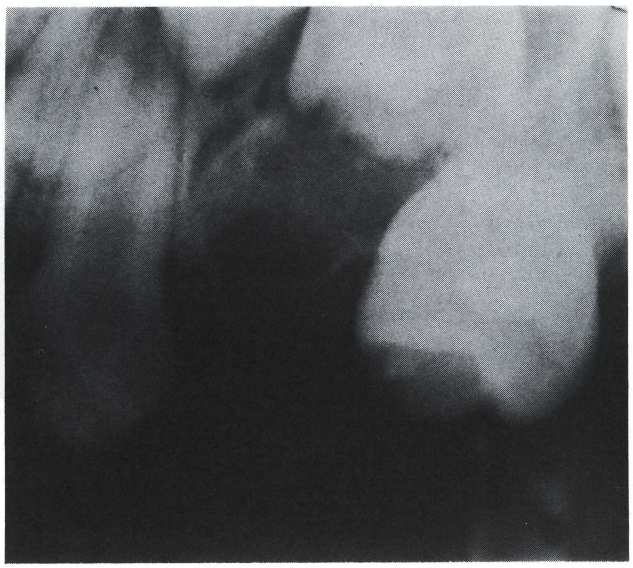

Figure 1. Preoperative periapical radiograph of affected side showing a radiolucent, partially radiopaque lesion.

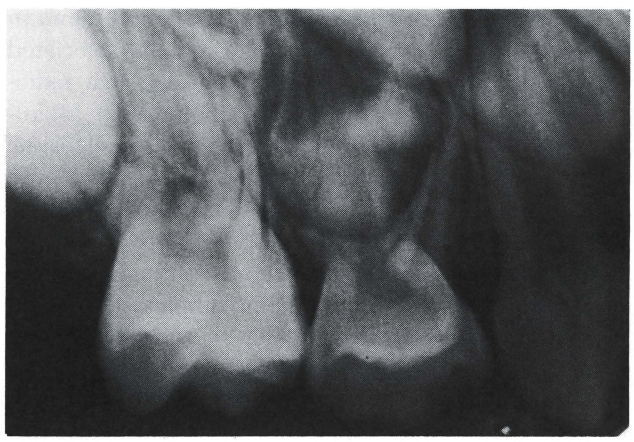

Figure 2. Preoperative periapical radiograph of unaffected side showing normal tooth development.

Remnants of reduced enamel epithelium were also identified (Figures 5, 6).

DISCUSSION

Because this condition was discovered at such an early stage of development, it is difficult to be conclusive concerning the diagnosis. The differential diagnosis, however, can be explored. From the radiographic and histologic findings, the differential diagnosis was limited to:

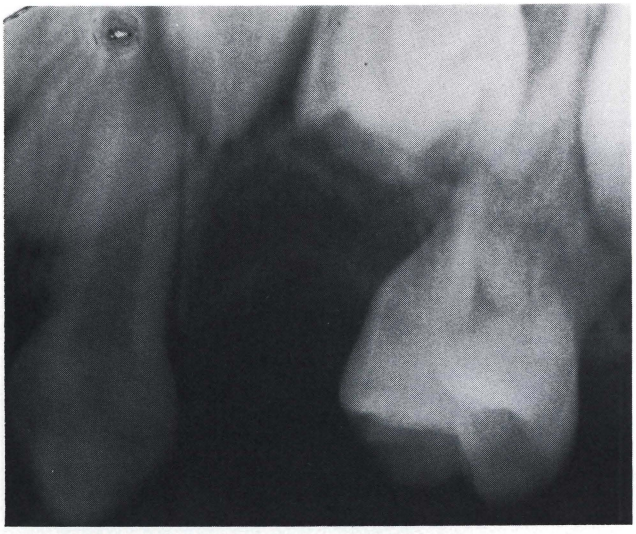

Figure 3. Postoperative radiograph of affected side showing absence of a developing first premolar.

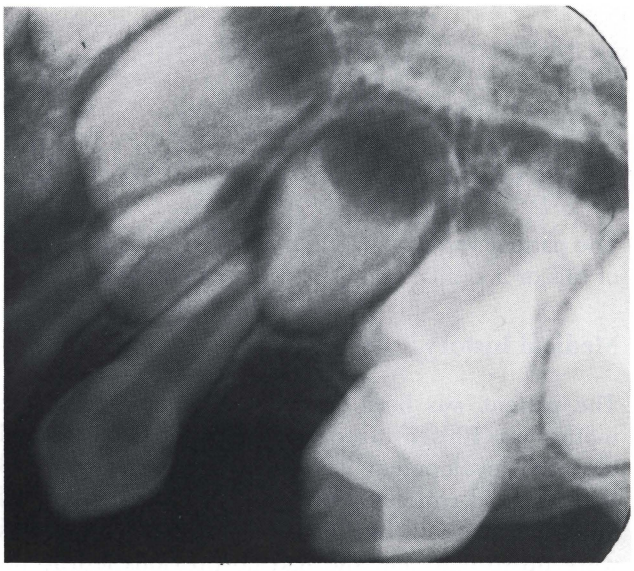

Figure 4. Postoperative occlusal radiograph of affected side showing absence of a developing first premolar and presence of first primary molar.

- ☐ Ameloblastic fibro-odontoma.
- ☐ Compound or complex odontoma.
- ☐ Odontodysplasia.
- ☐ Displaced maxillary first premolar tooth bud.

An ameloblastic fibro-odontoma is a lesion found in either the maxilla or mandible and may be associated with unerupted teeth.[2-4] Radiographically, it is radiopaque with varying degrees of radiolucency. Microscopically, it has areas of dentin and enamel, ameloblastic epithelium, and tissue resembling dental papilla.[4,5] The lesion described in this case does not satisfy the histologic criteria for the ameloblastic fibro-odontoma.

It is possible that the lesion represents an early stage of development of a compound or complex odontoma.[6] The lesion showed the result of an attempt to form "dentinoid;" however, there was no enamel matrix. Odontomas are benign odontogenic tumors containing enamel, dentin, and cementum, which may cause disturbances in the position or eruption of teeth.[3,7-9] Odontomas are usually discovered when a permanent tooth is impacted or a primary tooth is retained beyond the expected time limit of exfoliation.[7,9] Diagnosis has also been made upon observation of symtomless swelling, displaced erupted teeth, or on routine radiographic examination.[7,10] The lesion presented in this case report satisfies some of the characteristics of an odontoma; the absence of an enamel matrix suggests, however, that histodifferentiation did not progress to production of an odontoma.

Odontodysplasia is a rare anomaly which displays deficient and abnormal formation of dentin and enamel.[11] Odontodysplasia has been identified with late eruption and incomplete eruption of primary teeth.[12,13] Many etiologies for odontodysplasia have been suggested, including somatic mutation, early local trauma, local circulatory disorder, Rh incompatibility, local infection, viral infection, genetic transmission, and metabolic disturbances.[12,14,15] Again, some of these features are consistent with the lesion reported here; odontodysplasia, however, most often affects several teeth and usually both the primary and permanent teeth.[11,13,16] In this case report, only a solitary lesion is described.

The literature on displaced teeth is sparse and usually limited to transposition in the permanent dentition. Transposed teeth are considered to be a very rare dental anomaly.[17-19] The etiology is based on theories of how the anomaly could have occurred and include heredity, change in position during development and crowding.[1] The histology report describes many of the early features of a developing tooth, in that the lesion had cells with features of ameloblastoma, calcific debris, acellular eosinophilic matrix and remnants of reduced enamel epithelium.[2,20] Since there was no apparent maxillary left first premolar tooth bud present in the follow-up radiographs (Figures 3, 4) and based on evidence from the preoperative radiographs (Figure 1), the theory of a displaced maxillary first premolar tooth bud that appears to have become inverted in its descent to the alveolar crest is strongly supported.

SUMMARY

All that can be concluded from the available information is that the lesion was a variation on the spectrum of developmental anomalies without a known etiology or a conclusive diagnosis. This lesion is most probably related to an aborted formation of a maxillary first premolar or immature complex odontoma. More information could have been obtained, if the surgery had been delayed until further development of the lesion

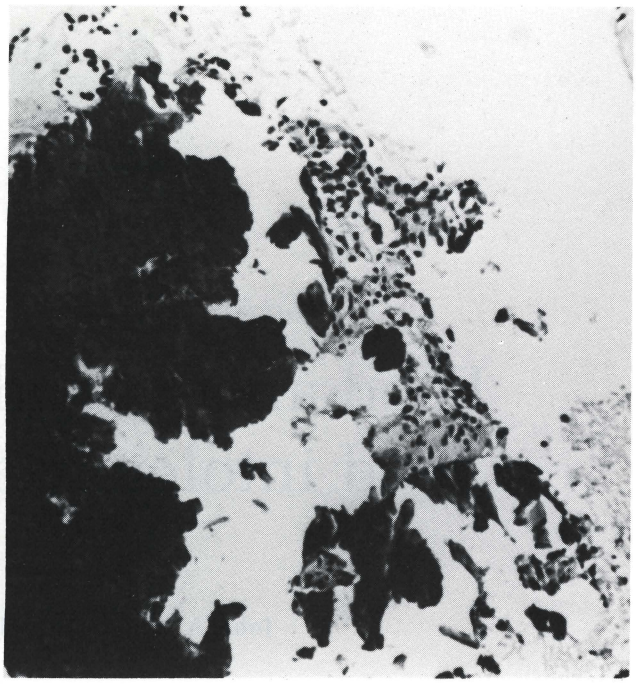

Figure 5. Light micrograph of lesion. This section, stained in hemotoxylin and eosin stain (×40), shows odontogenic epithelium and shattered calcific debris.

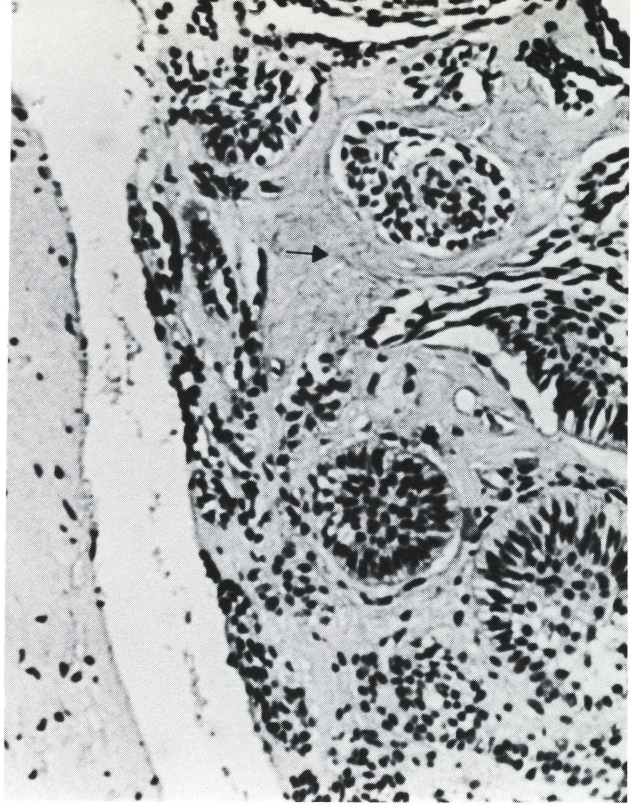

Figure 6. Light micrograph of lesion. This section, stained in hemotoxylin and eosin stain (×40), shows odontogenic epithelium with some peripheral palisading and columnar morphology associated with material resembling primitive "dentinoid" at arrow.

and of the patient's dentition had occurred; but this was not feasible, since definitive treatment was required. Further radiographic and clinical follow-up may assist in confirming the suspicion of a displaced maxillary left first premolar, when the patient's dentition is further developed. Since the position of the impacted primary tooth had not changed by the first follow-up examination, it is anticipated that it will not erupt on its own and will require further treatment, possibly in the form of surgical exposure and orthodontic manipulation or surgical removal and space management.

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Nutrition

Food allergy and food intolerance

John A. Anderson, MD

Adverse reactions to foods and food additives may be broadly classified into two types: (1) immunologic reactions (food allergy) and (2) non-immunologic reactions (food intolerance).[1]

Food allergy includes hypersensitivity reactions that are proved to be caused by or influenced by a food protein (allergens) via an allergen-specific immunoglobulin E. Immunoglobulin E (IgE) is a serum protein that mediates many common types of allergies besides food allergies, including hay fever, bee stings and penicillin hypersensitivities. Food allergy may also include other immune reactions.

Food intolerance is an adverse response to a food involving non-immunologic reactions (i.e., does not involve IgE). These include food poisoning and toxicity, anaphylactoid and other food-mediator release reactions. Food intolerances also include food idiosyncratic reactions, such as those caused as a result of genetically induced enzymatic deficiencies (e.g., lactose intolerance), pharmacologic (drug-like) food reactions and, finally, food reactions that affect the body metabolism.

SIGNS AND SYMPTOMS OF FOOD ALLERGY

Most of the signs and symptoms of food allergy involve the skin (itching, erythema, hives, eczema, edema) or the gastrointestinal tract (vomiting, diarrhea, abdomi-

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nal pain). In the case of systemic anaphylaxis, additional signs and symptoms may include the respiratory system (sneezing, wheezing), the eyes (conjunctivitis), and the cardiovascular system (palpitation, cardiac arrhythmia, shock collapse).[2]

Over the past fifty years, a school of thought has emerged suggesting that occasionally the symptoms of allergy can involve the nervous system, behavior and muscles and joints.[2] Thus, signs and symptoms, such as fatigue, hyperactivity, headache, anxiety, muscle and joint pain and arthritis, also have been attributed to food allergy.[1-3]

Although debate in this area is likely to continue, when well-designed blinded studies have been performed, the relationship between these types of symptoms/signs and diet have not proved to be important.[2,4-9] At this point, it is safe to conclude that in those patients presenting with either skin or gastrointestinal complaints (the signs more typical of allergy) an immunologic reaction to food (food allergy) should be considered. Patients with other symptoms are unlikely to be suffering from food allergy.

INCIDENCE OF FOOD ALLERGY

The exact incidence of all adverse reactions to foods is unknown. Food intolerance reactions, however, far outnumber the immunologic or food allergy reactions. True allergic reactions to foods are most common in infants, the greatest prevalence probably being among those children consuming cow's milk where the incidence may be 1 percent. Genuine food allergy reactions in adults are relatively uncommon.[1,2]

The literature on this subject may be confusing. Surveys among parents of small children have indicated that 5 percent to 10 percent may have gastrointestinal complaints or skin rashes as a result of exposure to a variety of foodstuffs.[10,11] However, Bock, when studying the natural history of food allergy, found that if the offending food was avoided for a short period of time, many of the patients would not react when rechallenged.[11]

In an older group of children with a history of food allergy, May and Bock found, using double-blind food challenge (DBFC), that only a third of the children could be proven to be food sensitive.[12] When specific children who have documented allergic reactions to foods have been followed, some have been shown to regain their clinical tolerance to a specific food despite retaining positive skin tests to food extracts or the presence of food-specific IgE antibodies in the serum.[10] One of the great difficulties in establishing true incidence figures, particularly in adults, relates again to the exaggerated reports of the various behavioral, neurological and somatic complaints related to diet.

ALLERGIC REACTIONS TO FOOD ALLERGENS

According to May, about 90 percent of food allergic reactions as evidenced by DBFC are caused by a relatively few foods, including milk, eggs, legumes, tree nuts and wheat.[2,12] Recent experience utilizing DBFC in children with atopic dermititis have identified the same foods.[13] Well-documented studies involving individual patients with anaphylactic sensitivity have implicated the following additional important foods: fish, seafood (particularly shrimp), and to a lesser degree, citrus fruit, melons, bananas, tomatoes, corn barley, rice and celery.[1]

IDIOSYNCRATIC REACTIONS TO FOOD ADDITIVES

A number of food additives have been implicated in food intolerances but the evidence is inconclusive and several components continue to be researched. This includes tartrazine (yellow #5), sodium benzoate, butylated hydroxyanisole (BHA), butylated hydroxytoluene (BHT), monosodium L-glutamate and sulfites. All of the adverse reactions associated with these food additives are believed to follow non-immunologic mechanisms.[14]

Tartrazine has been reported to induce bronchospasm in as many as 15 percent to 60 percent of aspirin-sensitive asthmatics, in causing or exacerbating chronic urticaria in adults, and being responsible with other colors in causing hyperactivity in children. Despite Feingold's claim that 50 percent of children with hyperactivity would be helped by a diet devoid of colors and preservatives, it has been found that only a few children would be improved on the diet or show an adverse effect in a special learning test on color challenge (predominantly tartrazine).[4]

Although BHA/BHT and sodium benzoate have also been implicated, very little documented evidence relates BHA/BHT and sodium benzoate to either asthma, exacerbation or chronic urticaria. While Scandinavian studies report that food additives are a major cause of chronic urticaria, studies in the United States have not verified this impression.[1]

Monosodium glutamate is reported to be responsible for the "Chinese restaurant syndrome" (headache, facial flush and chest pain). There also has been a single report of monosodium glutamate causing bronchospasm similar to that seen in sulfite-sensitive individuals.[15]

The sulfites, such as sodium metabisulfite ($Na_2S_2O_5$), have been implicated in a new syndrome of acute onset bronchospasm, especially in known asthmatics, as well as shock in some cases.[16] The exact prevalence of these problems is not clear at this time.[1,14] However, some investigators feel as many as 5 percent of all asthmatics could be sensitive.[1,14,17,18] Diets devoid of sulfites, however, may not improve the overall asthma condition.[17] Finally, clinical challenges of suspected patients who react in a typical fashion after a restaurant meal often do not confirm a sensitivity to sulfites, indicating that other factors may be involved.[19]

DIAGNOSIS OF SUSPECTED FOOD ALLERGY

The history of the events surrounding the adverse reaction to the ingestion of a food is the most important aspect of diagnosis.[12,20] The history may give clues to help separate those issues that are probably non-immunologic in nature from those immunologic or allergic reactions. The mainstay of simple office diagnosis in the investigation of allergic reactions to foods is the use of a standard elimination diet devoid of the more common allergic foods (see Allergic Reactions to Food Allergens). The diet is administered for a period of time (two weeks) and is followed by a step-wise, open challenge of single foods added back to the diet over a three- to seven-day period.[1,12,20] For those cases requiring confirmation of the history and open challenge, the DBFC procedure under controlled conditions can be used to verify the relationship between the food or food additive and the symptom complex. Even DBFC has its drawbacks.[2] Food challenge in this manner is not recommended in the case of systemic anaphylaxis because of the risk of the procedure. The results of DBFC do not indicate the mechanism of the adverse food reaction. Finally, since the DBFC procedure is done under "unnatural" conditions, all "negative" food challenges should be confirmed by subsequent open challenges.[20] In the case of suspected sulfite-, tartrazine- and monosodium glutamate-induced bronchial asthma, oral challenge of the suspected food additive can be evaluated using serial pulmonary function testing as an end point.

In food allergy problems, supplemental information concerning the mechanism of reaction can be obtained by the use of immediate-reacting (IgE) prick skin testing, the allergen-specific radio-allergosorbent test (RAST), or an enzyme-linked immunosorbent assay (ELISA). Prick skin testing may be helpful as a screening test to predict which foods the patient has developed an immunoglobulin E-mediated sensitivity.[13] A positive prick test to a food extract can be verified clinically by DBFC in approximately one-third of the cases, whereas a negative food prick test almost always is associated with a negative DBFC. Other immunologic tests may be helpful. The leukocytotoxic test and subcutaneous/sublingual titration and provocative techniques have been shown in several studies to be unreliable indices of adverse food reactions.[1,21]

TREATMENT OF FOOD ALLERGY

The principal treatment for all adverse reactions to foods is avoidance. Following investigations to identify the offending food, the specific food should be either completely eliminated from the diet or avoided to such an extent that clinical symptoms are not a problem. In the case of systemic anaphylaxis to a food, it is often advisable to supply the patient a preloaded syringe of epinephrine (HCl) in case the patient has a reaction to an unrecognized food. Oral experimental lactose-free cromolyn sodium and corticosteroids may be helpful in some allergic gastrointestinal disorders and in Heiner's syndrome.[1]

Available data suggest that it is possible to prevent allergic sensitization in potentially susceptible infants if cow's milk and some solids are withheld from the diet of the infant born to allergic parents.[1,10] It also may be important to eliminate potential allergic foods from the mother's diet in the last stages of pregnancy as well as during the time when she is breast feeding.

Sublingual neutralization treatment has been advocated by some physicians as beneficial in the management of food allergy, but these claims have largely been disrupted. Classic allergen immunotherapy has not been shown helpful in the management of the usual food allergic patient. The Feingold diet may be safe to use in the management of hyperkinesis, but the expectation for success should be limited.[4,21]

SUMMARY

Adverse reactions to foods and food additives include those that involve an immune mechanism of reaction (food allergies) and those that are non-immunological

in nature (food intolerance). The signs and symptoms of food allergy usually involve the skin and gastrointestinal tract and are "classic" allergic symptoms. Classic food allergy is more likely to occur in children. Food intolerance occurs more frequently at all ages. A number of food additives have been implicated in food intolerance, as none involve an immune mechanism of reaction. The role of food additives in food intolerance is not well established in many cases, has been discounted in others and continues to be the subject of current research. Although the history of events concerning an adverse reaction is important and gives clues to the specific type of problem (food allergy versus food intolerance), confirmation of the reaction is sometimes desirable. This can be done either by use of a standard elimination diet of non-allergic foods, followed by open challenge or by DBFC for more difficult situations. Food allergy skin testing and other *in vitro* immunologic tests may be helpful as supplemental information in those cases where food allergy is suspected. The best treatment for an adverse reaction to food is avoidance. Unproven and unapproved diagnostic (e.g., leukocytotoxic test) and therapeutic techniques (e.g., sublingual neutralization) are not recommended in food allergy management.

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SWEET PREFERENCE AND DENTAL CARIES

There is a growing body of evidence that man, as many other mammals, is born with an innate preference for the sweet taste. It seems, however, that environmental factors in their broadest sense play a decisive role in the development of dietary habits of the adult individual. The exact mechanism guiding the complex interrelationship between innate and acquired factors has not been sufficiently clarified.

Epidemiologic studies conducted on "changing populations" such as Australian aborigines, New Zealand Maoris, Eskimos, and others, all indicate that as the lifestyle of these groups became more Westernized, and their diet richer in sugar, caries prevalence showed a clear increase. Combining data on a global scale, it has been demonstrated that developing countries are characterized by less sugar consumption and lower levels of caries prevalence than the more developed countries.

Steiner, J. E. *et al*: Sweet preference and dental caries among Bedouin youth in Israel. Comm Dent Oral Epidemiol, 12: 386-389, December, 1984.

Vitamin use and abuse

William Tyler Jarvis, PhD

VITAMIN—A WONDERFUL WORD

Vitamin is a conjugation of "vital amine" done by Funk when he thought he had discovered the "vital" element in food.[1] The "amine" portion of the word came from a belief that the vital substance of food was a protein. This belief stemmed from work done in 1816 by Magendie, who determined that animals needed "albuminous" foods to survive and Mulder's naming such foods proteins (i.e., "first foods") because they were thought to contain *the essential element for life.*[2] The belief that food contained a basic vital element can be traced back through the alchemists who searched for the *Elixir Vitae*, Galen who espoused the doctrine of vitalism, Hippocrates who thought foods contained a single principle, to primitive beliefs that foods contain "life force".[3,4]

Although nutritional scientists use the term *vitamin* to classify organic substances needed exogenously in the diet, the word in the public's mind seems to conjure up the mystification inherent in the ancient search for the vitalistic element of food.

APPROPRIATENESS OF USE

Nutritional supplementation is a product of scientific inquiry and modern food technology. Supplementation, such as the enrichment of flour, fortification of basic foodstuffs, fluoridation of water and many other practices are scientifically valid and promoted by health professionals. Vitamins are suitably prescribed for pregnant women, infants and those with special needs.

Nutrition surveys show that marginal nutrient intakes occur within some segments of the population. While most of these problems can and should be solved by dietary improvements, there are instances where supplementation is justified. Questionable use and abuse of vitamins are separate issues from these and are covered below.

Self-prescribing based upon advertising promotion, self-help books, health magazines, salespeople and media "talk shows" is likely to involve the inappropriate use of vitamin supplements. Promoters often emphasize certain glamour vitamins, such as C and E. Studies among the elderly show that those taking supplements were no better off nutritionally than nonusers, because, rather than taking supplements that provided the marginal nutrients, they were taking those popularized by promoters.[5-8]

Taking megadoses of vitamins represents the most inappropriate use of these chemical substances. (Megadose refers to routinely ingesting several orders of magnitude greater than the Recommended Dietary Allowances (RDA) for one or more nutrients, usually in a nonfood form.) Whether this is done on the basis of self-prescribing or the advice of a medical practitioner working from less than good scientific evidence, the results can be, at best, economically unjustified and, at worst, a distinct hazard to patient well-being. Thirty-seven percent of all adult Americans are now believed to take megavitamin supplements.[9]

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MEGAVITAMIN THERAPY

The efficacy of megavitamin therapy in treating certain inborn errors of metabolism is well established.[10] However, mavericks and fringe practitioners have been promoting the unproven use of megavitamins for many years.

Herbert points out that the term "megavitamin therapy" is a misnomer since it misinforms about mode of action; it is really megachemical therapy and not nutrient therapy.[11] Vitamins function in small quantities by regulating specific metabolic activity, usually by acting as coenzymes (vitamin D also functions as a hormone). When vitamin *coenzymes* enter the body, they are combined with a protein *apoenzyme* that has been synthesized within the cell to form a *holoenzyme*, which is commonly referred to simply as an *enzyme*.

The quantity of *apoenzymes* any cell can produce per unit of time is the limiting factor in the body's ability to make use of excessive vitamins as nutrients. Regular ingestion of excessive amounts of nutrients act as pharmacological chemicals that may produce a drug or toxic effect. Such effects could prove useful or harmful depending upon the circumstances, but excessive amounts of vitamins would not be functioning as vitamins.

Proponents of megavitamin therapy generally concentrate upon presenting anecdotal evidence of patient benefits. They are frequently closed-minded to the possibility that clinical successes might be due to placebo effects, natural remissions, observer biases, or uncontrolled reactive effects known to confound clinical findings. Proponents have also been relatively silent about possible undesirable effects of continuous megavitamin dosing.

MEGAVITAMIN THEORIES

Megavitamin theories have attracted attention of scientists and stimulated a substantial amount of research on safety and effectiveness. Vitamins are sufficiently benign, inexpensive and easily disguised to make doubleblind and/or cross-over trials with placebos easy to conduct. Megavitamin trials have been noteworthy by their failures in such tests as improving behavior in children with minimal brain dysfunction, performance in the mentally retarded, angina pectoris, resistance to colds, survival of cancer patients, schizophrenia and many more.[12-17] With such a dismal record, it might be asked why proponents persist in their advocacy of megavitamin therapy.

Many other claims for vitamin and other supplementary benefits appear to be based upon the effects of dietary deficiencies. Dose-response patterns common to drugs are inappropriately applied to nutrients. In actuality, nutrient-response curves rise during deficiency states and flatten out, as optimal needs, well within the range of the RDA, are met.

For example, biotin deficiency symptoms include mental depressions; but it does not follow that biotin deficiency is a major cause of depression in our society or that ingesting biotin supplements will protect a person from the effects of stress. Further, it is believed that the biotin needs of all healthy adults are met by a conventional mixed diet.[18]

Another example is that irritability, headaches, sleeplessness, loss of memory and signs of emotional instability often occur as early signs of pellagra, but it does not follow that taking extra niacin will offer protection from similar stress-related symptoms. The author is not aware of any valid evidence that ingesting commercial "stress formulas" have value as advertised.

Sometimes animal study results are inappropriately used to justify vitamin supplementation. Studies have shown that dietary vitamin E protects rats from some chemical toxicants under special laboratory conditions; however, vitamin E supplementation has not been shown to be important to humans.[19]

Cigarette smokers have been shown to have lower ascorbate levels than nonsmokers, but leading people to believe they can offset the effects of this pernicious habit by ingesting extra vitamin C is unwarranted.[20,21]

The idea that extra vitamin B_6 is needed for women on birth control pills is also disputed on the basis that only one of several parameters used to access body levels is affected by oral contraceptive use. Further controlled studies have indicated that needs can be met within the established RDA.[22]

The notion that increased exercise justifies the need for supplementation appeals to common sense. However, the increased food intake normally associated with exercising generally makes up for the additional needs which are primarily extra calories, electrolytes and water.[23] The belief that extra vitamins are useful to athletes is tied to the misconception that *extra vitamins provide people with extra energy*, which was found to be the most widely held misbelief among Americans.[24]

Vitamin pill-taking seems to have paved the way for other unnecessary forms of supplementation. Amino acids easily available in foods (e.g., tryptophan, phenlalanine, arginine) and substances unnecessary in the human diet (e.g., bioflavonoids, para-amenobenzoic acid [PABA], lecithin) are widely promoted. Several prod-

ucts have been speciously marketed as "vitamins." Bioflavonoids, ruten and hesperidin are sometimes called "vitamin P," several concoctions have been labeled "vitamin B_{15}" and laetrile has been promoted as "vitamin B_{17}."[18]

EXTENT OF USE

Studies of the percentage of the general population taking vitamins run from 37 percent to 66.6 percent.[25,26] Among subgroups, vitamin supplementation is reported as university students (56.5 percent), registered nurses (38 percent), male and female athletes (56 percent and 33 percent, respectively), and among the elderly (66 percent to 72 percent).[5,6,27-29]

WHY PEOPLE TAKE VITAMINS

Reasons given for taking vitamin supplements are often vague. General notions about staying healthy predominate (78 percent), but "to prevent colds" (58 percent), "prevent or treat arthritis" (11 percent) or ". . .cancer" (4 percent) are reported.[24] Interestingly, studies find that a majority of the public believe that they should only take supplements on a physician's advice.[30] Most vitamin users (58 percent) cite medical doctors as sources of influence. However, close interviewing found that in some cases patients broach the subject of supplementation and their doctors merely said that it would be "all right."[24] The self-help movement and recent popular interest in health and fitness play a major role in the rapid growth of the supplementation market.

SUMMARY

Patients should be quizzed about what supplements they use and why they take them. Potentially dangerous megadosing should be discouraged and patients should be informed that studies have conclusively shown that vitamin C will not prevent colds, recent dietary recommendations for possible reduction of cancer risk do not recommend supplementation, and state that food supplements are of no value in preventing or treating arthritis.[15,21,34] Patients also need to be warned that not only the fat-soluble vitamins can be dangerous, but water-soluble vitamins, once thought to be safe, also pose dangers when ingested in unnecessarily large amounts.[33]

There is no reason to alarm patients who are merely ingesting a daily multivitamin or multivitamin-mineral supplement. It should be emphasized that supplements do not correct poor dietary habits since micronutrients are only a part of good nutrition and people should recognize that a diet containing a reasonable variety (applying the Basic Five Food Groups) will supply adequate amounts of the necessary nutrients for most people. It will be necessary to instruct patients to take a "buyer beware" attitude toward all food supplements as long as Congress continues to prevent the Food and Drug Administration's regulation of these products.

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Malnutrition and the oral health of children in Ogbomosho, Nigeria

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Malnutrition beginning early in life has been reported to affect adversely various aspects of human growth and development.[1-4] In experimental animals, protein malnutrition at the time of tooth development resulted in smaller teeth, delayed eruption, and increased susceptibility to caries.[5] Linear hypoplasia has been reported in the enamel of primary teeth of malnourished children in poor populations, around the world.[6] These hypoplastic areas are reported to be very susceptible to dental caries.[6]

The oral hygiene of most Nigerians is poor and epidemiological studies in Nigeria indicated a high prevalence of severe periodontal disease and a low prevalence of dental caries.[7-12] Socioeconomic conditions have been shown to influence dental development, and the prevalence of dental caries and periodontal disease, in Nigeria.[1,13]

The present report presents a study of the oral health, with special reference to dental caries, enamel hypoplasia and gingivitis/periodontal disease, of a group of malnourished children in a missionary nutritional center, in a rural area of Nigeria, West Africa.

The authors wish to thank Ruth Womack, RN, Director of the Kearsey Home Center, John Tarpley, MD and the Staff of Baptist Hospital Ogbomosho and the Department of Clinical Pathology of the College of Medicine of the University of Lagos for their help with the clinical and laboratory portions of this study. Drs. Peter Moon and William Gander for help with the statistical analysis of the data.

MATERIALS AND METHODS

A total of fifty-two children (twenty-seven females and twenty-five males), ranging in age from one to five years comprised the study sample. The children belonged to the Yoruba tribe and resided within the boundaries of Oyo State, Nigeria. Forty-five of these children were patients at the Kearsey Home (Nutritional Center) operated by Baptist missionaries with the help of a Nigerian staff. The center itself is located in the bush, approximately six miles outside the village of Ogbomosho. Most of the forty-five children, who were patients at the center, were severely malnourished (Figure 1). Two suffered from cancrum oris (Figure 2) and three (including one of those with cancrum oris) died within a few weeks of the beginning of the study. The remaining seven children, those who comprised the well-nourished group in this study, were the children of the Nigerian staff along with a set of twin orphans, who continue to live at the center.

EXAMINATIONS

Physical examinations were conducted by the staff of the center and Baptist Hospital Ogbomosho. The authors had access to the children's hospital charts.

All dental examinations were made by the authors, with both present. The examinations were thorough and complete, using optimum light and a dental examination kit. The data were recorded on specially pre-

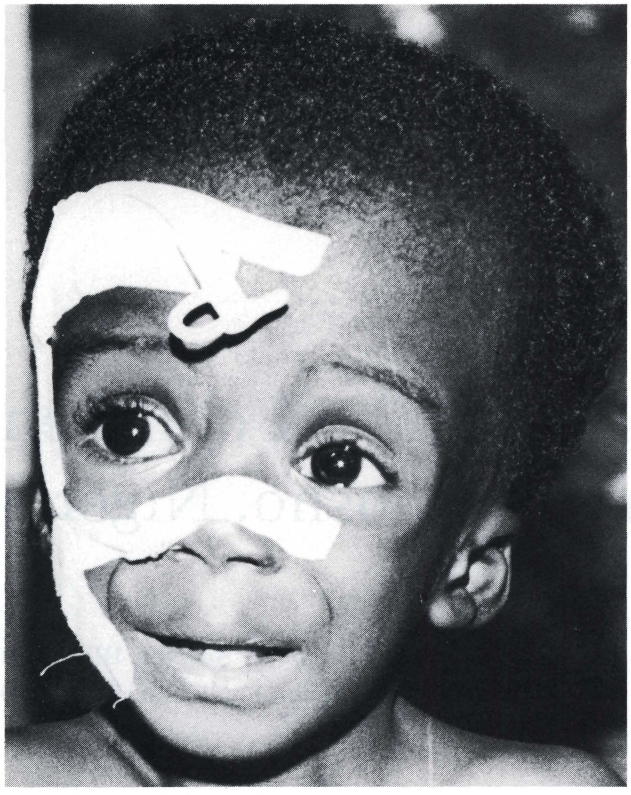

Figure 1. Severely malnourished Nigerian boy. This child was being fed intravenously as he refused to eat.

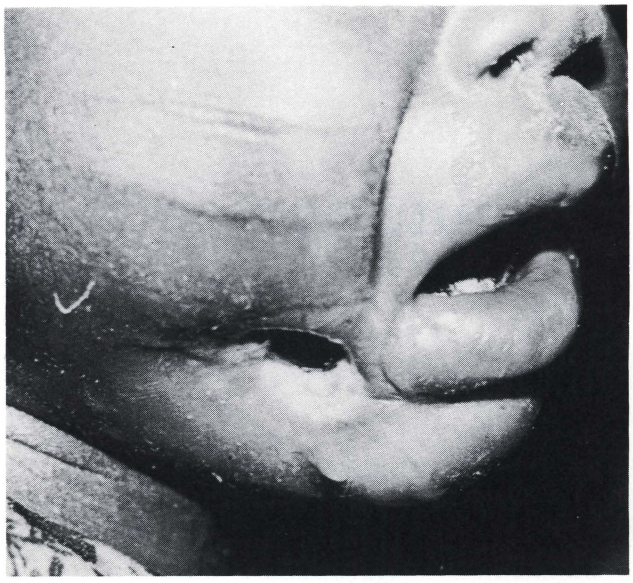

Figure 2. Malnourished Nigerian boy showing extensive loss of soft tissue of the right cheek as the result of cancrum oris. Considerable bone loss eventually occurred in this patient.

pared dental charts. Special attention was paid to the assessment of enamel hypoplasia, dental caries and gingival/periodontal health. A tooth was considered hypoplastic, if the defect of the enamel was readily seen, of palpable depth and characterized by grooved rings or linear lines.

The location and extent of caries lesions were recorded.

Gingivitis was recorded as absent or present, and, if present, classified as mild, moderate, or severe. When evaluated in accordance with the methods and standards of Greene and Vermillion, Russell and Enwonwu, periodontal involvement was not observed in these cases.[13-15] In this report, therefore, we will present data concerning the absence or presence (with degree) of gingivitis. Eleven patients from the malnourished group were observed over a period of six months and then reexamined. These eleven were maintained on proper diets during this period.

BIOCHEMICAL STUDIES

Blood samples were collected from all seven children in the well-nourished group and from the two patients with cancrum oris. Blood samples were drawn from fifteen of the forty-three patients in the malnourished group without cancrum oris. The fifteen were randomly selected after excluding some six children, who were so seriously ill they were approaching death. Serum proteins were measured for the authors at Baptist Hospital Ogbomosho and in the Department of Clinical Pathology at the College of Medicine of the University of Lagos.

RESULTS

Although it was obvious that those patients included in the malnourished group were seriously ill due to protein malnutrition and were clinically diagnosed as such by the experts in Ogbomosho, it was deemed helpful to analyze the serum protein levels to show the degree of protein deficiency. This aspect of the study was facilitated by the fact that blood had to be drawn on many of these patients for other reasons. Table 1 shows the serum protein levels for the three groups studied.

Total serum protein levels were highest in the well-nourished group and lowest in the cancrum oris group. The difference was statistically significant when the well-nourished was compared with the malnourished group ($P<0.05$) and with the cancrum oris group. There was also a statistically significant difference in total serum protein levels ($P<0.05$) between the malnourished and cancrum oris groups. The serum albumin levels were lower in the malnourished and cancrum oris group than in the well-nourished children. The difference was sta-

Table 1☐ Serum proteins in well-nourished children, malnourished children, and children with cancrum oris.

Group	Age	Number Examined	Total Protein (g/100 ml)		Albumin (g/100 ml)		Globulin (g/100 ml)		A/G Ratio	
			Mean	S.D.	Mean	S.D.	Mean	S.D.	Ratio	S.D.
Well-nourished	2-5*	7	7.68	0.48	4.78	0.31	2.91	0.52	1.69:1	0.37
Malnourished	1-5	15	6.76	0.98	3.27	0.73	3.27	0.73	1.01:1	0.43
Cancrum oris	3-5	2	5.21	0.16	2.41	0.57	2.80	0.72	0.92:1	0.44

*Age in years

Table 2☐ Prevalence of enamel hypoplasia, dental caries and gingivitis in well-nourished children, malnourished, and children with cancrum oris.

Group	Age	Number examined	Enamel hypoplasia		Dental caries		Gingivitis		
			N	%	N	%	Degree**	N	%
Well-nourished	2-5	7	0	0.00	0	0.00	Mild	1	14.29
							Moderate	1	14.29
							Severe	0	0.00
							Total	2	28.58
Malnourished	1-5	43	8	18.60	5	11.63	Mild	5	11.63
							Moderate	9	20.93
							Severe	11	25.58
							Total	25	58.14
Cancrum oris	3-5	2	1	50.00	0	0.00	Mild	0	0.00
							Moderate	0	0.00
							Severe	2	100.00
							Total	2	100.00

*Age in years
**Gingivitis was classified as mild, moderate or severe. The severe form was a necrotizing ulcerative gingivitis (NUG)

tistically significant ($P<0.001$) when comparing the well-nourished with the two other groups. The difference in the albumin levels, however, between the malnourished children and those with cancrum oris was not statistically significant ($P>0.05$). The malnourished group had higher serum globulin levels than the other two groups; the cancrum oris group had the lowest levels. These differences, however, were not statistically significant in each case with $P>0.05$. While the albumin/globulin (A/G) ratio was normal (1.69:1) in the well-nourished group, it was abnormal in the malnourished group and reversed in those patients with cancrum oris. When comparing the A/G ratio of the well-nourished group with the other groups the differences were statistically significant ($P<0.005$) for malnourished and $P<0.05$ for cancrum oris). Total serum protein levels in the patients in this study were lower, therefore, in the malnourished group and lowest in the cancrum oris group, due to a decrease in serum albumin. The total serum globulin fractions, in the malnourished and cancrum oris groups were maintained at levels equal to or slightly higher than those seen in the children considered well-nourished. This maintenance of the globulin levels in the malnourished and cancrum oris groups is no doubt due to the production of immunoglobulins by these patients in a host-response to the infections which afflicted most of them. Thus, these laboratory data on serum proteins supported the three-group classification and strengthened the clinical diagnoses of malnutrition. The two patients with cancrum oris were diagnosed as having noma, based on criteria outlined in previous publications by the authors.[16,17]

Table 2 shows the prevalence of enamel hypoplasia, dental caries, and gingivitis, as determined for each group.

It was possible to follow up on eleven patients from the malnourished group, who were maintained on a high protein and balanced diet for six months. Of these, eight had gingivitis on the initial examination, six months earlier. Five of the eight showed improvement of their gingivitis: three improved from severe to mild, and one each went from mild and moderate to essentially normal gingiva.

Other findings included fourteen children with black stain on the teeth. Thirteen of these were in the malnourished group and one was in the cancrum oris group. Eight patients in the malnourished group and one in the cancrum oris group showed tongue changes consistent with their physical state. Two patients in the malnourished group had angular chelitis. One patient from the malnourished group had a cleft palate and one had candidiasis involving the tongue and buccal mucosa. Two of the children in the malnourished group manifested a marked delay in tooth eruption. Six patients in the malnourished group recently had measles, according to their hospital charts. One child in the mal-

nourished group and one of the two patients with cancrum oris had confirmed cases of pulmonary tuberculosis. The patient with tuberculosis in the malnourished group showed clinical evidence of cervical lymph node involvement. All of the children in the malnourished and cancrum oris groups had fine reddish hair (Figure 3) and the vast majority manifested distended abdomens and swelling of the lower extremities seen in malnutrition.

DISCUSSION

Sweeney and co-workers studied a group of 104 Guatemalan children, two to seven years of age, with third-degree malnutrition, requiring hospitalization; and 150 others, who were less severely malnourished.[6] In those most severely malnourished, 73.1 percent showed linear hypoplastic lesions of the enamel in the primary incisors, while only 42.9 percent of the less severely malnourished had similar lesions. Enwonwu examined 872 Nigerian children, infancy to seven years of age, to ascertain the influence of socioeconomic conditions on enamel hypoplasia.[1] While the prevalence of enamel hypoplasia was nil in his "optimal" (well-nourished) group, 21 percent of the children who came from the village with a high frequency of malnutrition, manifested enamel hypoplastic defects in the incisal third of the primary incisors. In the present study, while none of the seven well-nourished children showed enamel hypoplastic defects in the primary incisors, eight (18.6 percent) of the malnourished group and one of the two (50 percent) children with cancrum oris had enamel hypoplasia (Table 2). Of the eight malnourished children who had enamel hypoplastic defects, seven were among the most severely malnourished, based on clinical and laboratory findings. Likewise, these children showed the most severe involvement. This higher prevalence of hypoplastic defects in the malnourished is to be expected, because the process of amelogenesis is among the most sensitive to adverse body changes.

Enwonwu also studied the relationship of socioeconomic factors on the prevalence of necrotizing ulcerative gingivitis.[13] While none of his "optimal" well-nourished group was affected by the disorder, 15.3 percent of the malnourished village children and 27.2 percent of the children hospitalized with kwashiokor had the typical crater-like depressions covered by a grayish pseudomembranous slough and debris, characteristic of necrotizing ulcerative gingivitis. In the present study, there were an increasing prevalence and a growing severity of gingivitis, by group, beginning with the well-nourished, to the malnourished, to the group with cancrum oris (Table 2). Eleven (25.6 percent) of the forty-three malnourished children were diagnosed to have necrotizing ulcerative gingivitis. Both of the cancrum oris patients had it. In their cases, however, the necrotizing ulcerative lesions had extended beyond the gingiva to involve the buccal mucosa, and the adjacent cheek, as well as the underlying bone. For discussions of the relationship between these lesions and cancrum oris, the authors suggest papers by Enwonwu, Sawyer and Nwoku, Nwoku and Sawyer, and by Tempest.[1,13,16-18] As noted in the results, the authors were able to observe eleven patients from the malnourished group, for six months, during which time they were maintained on a high protein and balanced diet. Five of the eight, who had gingivitis on initial examination, showed improvement over this period. They showed marked improvement in their nutritional status and general health, including weight gains as ascertained

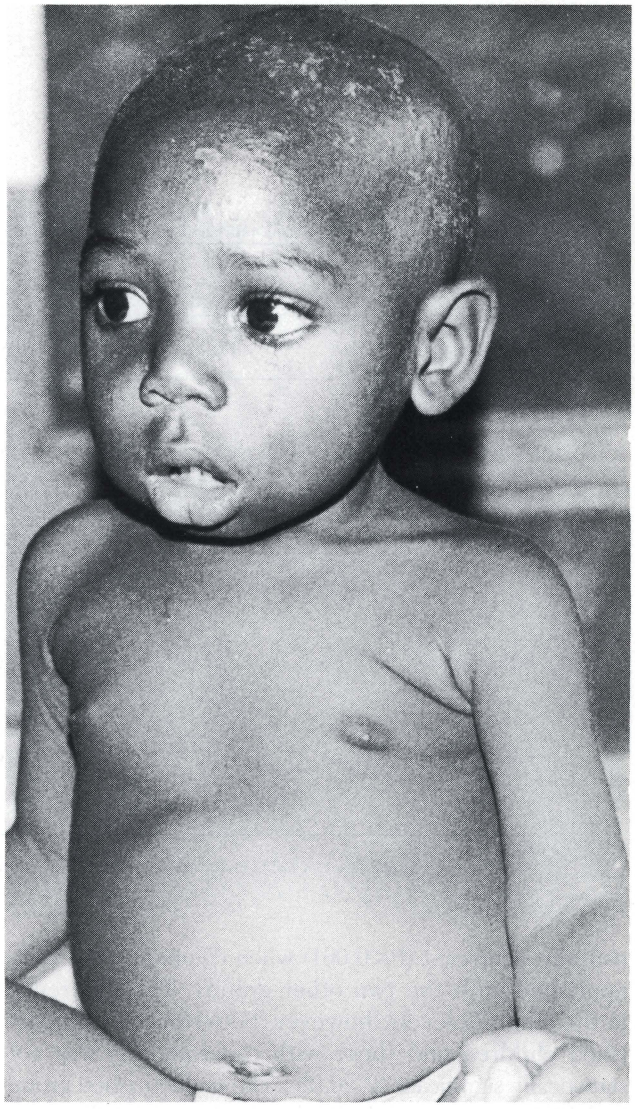

Figure 3. Nigerian child on the road to recovery from severe malnutrition. The child has fine reddish hair and still manifests a distended abdomen.

by the criteria used by the center. It was felt that their improved oral health could be related to the improved nutritional status, but was no doubt also influenced by the improved oral hygiene that followed instruction on oral health care at the time of the initial examinations.

While Enwonwu did not report the prevalence of dental caries in the primary teeth in malnourished Nigerian children, in his studies, the present study did examine the malnourished children for the frequency of this disease. None of the well-nourished children or those with cancrum oris had any caries lesions. Only five (11.63 percent) of the forty-three malnourished children had dental caries. While oral hygiene is poor in most Nigerians, including the children examined in this study (slightly over 50 percent were considered to have poor oral hygiene), the prevalence of caries was low in every group.[7,8] This is not unusual in a Nigerian population, especially for those residing in the rural areas. Sheiham, in a study carried out in Southern Nigeria, found 98 percent of his population to be caries-free; while Henshaw reported that the percentage of caries-free individuals dropped from 91 percent in Port Harcourt (a small community in the East) to 49.1 percent in Lagos, the political and commercial capitol of Nigeria.[9,11] The low prevalence of dental caries in the present rural study and other studies is related more to diet than to other factors, such as fluoride levels in the water. Fluoride levels range from "not detectable" to approximately 0.40 ppm in the region of Oyo State, from where the children comprising the study groups in this report originated. The diet of these rural children is high in fiber (self-cleansing) foods, and low to devoid of the sticky refined carbohydrates that are so easily related to a high prevalence of dental caries.

One other oral finding bears discussion, as it was seen with some degree of frequency and is somewhat unusual to those of us from developed parts of the world. Fourteen children had a black stain of the teeth. Although the authors were familiar with most of the extrinsic stains one sees in a Nigerian population such as a red stain from betel-nut chewing, and most of the intrinsic stains, this one puzzled us. After seeing several children with black stained teeth, we questioned the director, physicians, and Nigerian staff of the center about this stain. It was reported to have been seen by some of these workers for many years and they related the stain was the result of the child ingesting a native "blood tonic". In questioning the relatives of the children in this study, the authors discovered that only those who were given this local "blood tonic" showed a staining of their teeth. "Native medicine" does on occasions have "side-effects", which are even more serious than staining of the teeth. The one child who had cancrum oris, and who died later in Baptist Hospital Ogbomosho, was taken from the center after beginning treatment, by his family. They returned him to the center after a month or two of native treatment. He had been blinded by the native medication and was so seriously ill he died within a day, despite all Western medicine could do for him.

Enamel hypoplasia, dental caries, and gingivitis were all seen with greater frequency in the malnourished child than in the well-nourished, in this study. There was improvement in the gingivitis when these malnourished children were given proper diets, and shown how to perform oral hygiene procedures properly. Thus again, the importance of good oral hygiene and proper nutrition to the development and maintenance of good oral health is reinforced.

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Some thoughts on

Michael J. Album, Esq.

Dr. Marcus has written a thoughtful and provocative article. His premise that the takeover of the health care industry by entrepreneurial "paymasters" (such as HMOs and emergency treatment centers) requires physicians to unionize for their own protection may, however, rest on mistaken assumptions concerning "market conditions" at large.

As an attorney involved in labor matters, I agree with Dr. Marcus that there is a new effort afoot to unionize the white collar and professional workforce, a natural result of the change in our country from a production economy to a service economy. Unions are organizing doctors and lawyers, but more importantly, they are organizing the white collar support staff intimately involved in the delivery of professional services—nurses and secretaries. The sheer size of this workforce of support personnel will prove to be the real focus of union organizing efforts.

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It is precisely the large scale "entrepreneurial paymasters" in the health industry who will have the greatest number of support staff and prove most attractive to union organizing efforts. The "market conditions" referred to by Dr. Marcus may well produce a delightful irony: across-the-board unionization of HMOs and the like, particularly among the support staff, may result in overall labor costs that are too high, and which give a competitive edge to private practitioners with smaller non-union support staff. In his article, Dr. Marcus cited the unionization of pilots with approval, but the current state of the entire airline industry may provide greater support for my thesis. Put more simply, would one rather be a working pilot for a small, aggressive, and non-union carrier such as Peoples Express, or a laid-off pilot for one of the more entrenched and unionized industry giants?

My analysis is not meant to suggest that independent practitioners are immune under the law from unionizing efforts aimed at their support staff. It might be

conditions. For example:

"Yes, in the context that Dr. Marcus exhibits a trade union for professionals. He illustrates how a trade union can aid salaried physicians in maintaining a "standard" wage and improving working conditions (despite his claims of success, 14,000 physicians and dentists is not a large sample of comparison). But today I do not think that a trade union is essential to combat our changing economy. If the government intervention escalates to the point that all health professionals are government employees (as in Great Britain and some other European countries) then I believe we need some vehicle of input into the system."

"Yes, but only in a socialized dental system."

"I feel that Dr. Marcus is describing the role that organized dentistry should play in speaking for the practitioner. That means that the ADA and its components will have to make drastic changes in their thinking and direction of activities. If they do not make these changes then that role would be appropriate for a trade union. If that should happen I wonder what role will be left for the ADA to play."

"The arguments that Dr. Marcus makes are sound and reasonable. He neglects, however, to examine the consequences of these actions. The mere fact that insurance companies and other groups must deal with us as individuals, rather than as one group is to our advantage at times. For some, the ability to maintain their 'independence' is a significant reason for joining our profession."

"It would be hard for me to support his statements unless someone had a gun at my back."

"In theory, yes, but many of Dr. Marcus' assumptions are based on the prediction that government intervention in the medical profession is inevitable. As long as the AMA and the ADA continue to act responsibly in "socially policing" its members, this should suffice until Federal Control becomes a reality."

"My preference is for dentists, through their associations, to work collectively toward revising the laws that currently seem to work against the dental profession. If this were accomplished, there would be little need for labor unions within the profession."

trade unionism in medicine

helpful to review briefly the law governing just whom can be organized. Under Federal law, employees can unionize, if their employer is engaged in an industry "affecting commerce," and the employees are not considered "supervisory", "managerial", or "confidential" personnel. As the law now stands, every practitioner probably engages in an industry "affecting commerce", just as a result of the supplies and equipment purchased to practice medicine. And as the law now stands, the categories of "supervisor," "manager" and "confidential" employee are narrowly defined; in most cases employees will be found to fall outside these categories and be permitted to unionize. For example, only employees who have direct and independent control over the jobs of other employees, or who establish management policy are considered "supervisory" or "management" personnel, just as only employees who assist in formulating labor policies are considered "confidential" employees. In a hypothetical dental practice consisting of two partners, one newly employed graduate of dental school, an office supervisor, and four hygienists or assistants, one thing is clear: only the partners and office supervisor are exempt from unionization.

The ability then of the independent practitioner to compete in the era of modern health care will not result from some legal immunity of his or her practice from unionization. In the practical world, unions direct their efforts at large targets. From the start, the support staff of the entrepreneurial clinics provides such a target. Moreover, as competition among these clinics grows, pressure will arise to decrease labor costs by routinizing the treatment of patients, expanding the job responsibilities of nurses, technicians and other less costly health care personnel, and using automation to its fullest. In the end, many current members of Dr. Marcus' physicians union may be forced into more supervisory positions, and even find themselves exempt from the protection of the Federal labor laws. At that point they just may decide to enter private practice as independent practitioners.

Abstracts

Mejàre, I. and Brännström, M: Deep bacterial penetration of early proximal caries lesions in young human premolars. J Dent Child, 52:103–107, March-April, 1985.

The aim of the study was to assess the frequency and extent of bacterial invasion in proximal small caries lesions of young human premolars. The teeth were fractured longitudinally through the lesion and examined under a scanning electron microscope. In two out of twelve teeth with incipient caries and without any cavitation, bacteria could be seen in both enamel and dentin; in one, penetrating the dentin to a depth of 1.5 mm from the dentinoenamel junction. In both lesions the chaulkiness extended to about half of the enamel thickness.

Bacteria, Enamel caries lesions

Rubenstein, L. K. and Campbell R. L.: Acquired unilateral condylar hyperplasia and facial asymmetry: report of case. J Dent Child, 52:114–120, March-April, 1985.

A clinical report is presented of a 12-year-old female with right mandibular hyperplasia, facial asymmetry, bitemporal headaches and left temporomandibular joint pain. Past medical history was noncontributory with the exception of a bicycle accident at age eight, which resulted in minor facial trauma. The management and treatment of this case illustrates the importance of long-term follow-up by the pedodontist and the necessity of a team approach involving pedodontics, orthodontics and oral surgery.

Mandibular condyle, Condylar hyperplasia, Facial asymmetry, Temporomandibular joint, Mandibular trauma

Humerfelt, D.; Hurlen, B.; Humerfelt, S.: Hyperdontia in children below four years of age: a radiographic study. J Dent Child, 52:121–124, March-April, 1985.

Sixty-six children (forty-one boys and twenty-five girls) were evaluated for differences in characteristics of premaxillary hyperdontia between the primary and permanent dentitions. The study population had forty-five supernumerary teeth in their primary dentitions and fifty supernumerary teeth in their permanent dentitions. Eighty percent of the primary supernumerary teeth were located in the lateral incisor regions, in proper alignment within the dental arch. Sixty-two percent of the supernumerary teeth in the permanent dentitions were situated in the central incisor regions; 90 percent were outside the dental arch, mostly palatal; and 42 percent were inverted. Seventy-three percent of the supernumerary teeth in the primary dentitions had erupted. Twin tooth formation occurred in twenty-eight patients, the majority in the primary dentition; but in eleven children, both dentitions were involved. Interference with the normal dentition occurred most often in the permanent dentition. Children with primary supernumeraries exhibit a greater chance of hyperdontia in the permanent dentition than do others.

Hyperdontia, primary and permanent dentitions; Twinning

Ranta, R.: Hereditary agenesis of ten maxillary posterior teeth: a family history. J Dent Child, 52:125–127, March-April, 1985.

A family is described, where the mother and her two sons had similar bilateral congenital absences of the following maxillary teeth: the second premolars, the first, second and possibly the third molars, as well as, in the sons, the second primary molars. The size, shape and formation of the remaining teeth were within normal limits.

The near relatives of the parents had no history of hypodontia. The family members were otherwise healthy except that the mother and one of the sons had a slight nasality of speech. Moreover, the mother and both sons had adhesive otitis.

Agenesis, primary and permanent teeth; Familial traits

Hainline-Raez, A. G. and Richardson, D. S.: Abnormal odontogenesis: report of case. J Dent Child, 52:130–133, March-April, 1985.
This report describes a case of abnormal odontogenesis. No definitive diagnosis was achieved, but a differential diagnosis between ameloblastic fibro-odontoma, complex or compound odontoma, odontodysplasia and displaced maxillary first premolar were explored. The treatment rendered and the most probable prognosis were presented.

Odontogenesis

Sawyer, D. R. and Nwoku, A. L.: Malnutrition and the oral health of children in Ogbomosho, Nigeria. J Dent Child, 52:141–145, March-April, 1985.
Fifty-two Nigerian children, forty-five of whom were severely malnourished were examined to ascertain their level of oral health. Enamel hypoplasia, dental caries, gingivitis and periodontitis were given special attention in this study, and each of these disorders was found to be more frequently seen in the malnourished child. In a followup study of the malnourished children treated at the Baptist nutritional center there was improvement in the children's oral health, manifested by improvement of their gingivitis and periodontitis as the child's nutritional level and general health improved. Other oral findings were noted and recorded, including two patients with cancrum oris (noma) and others with angular chelitis, cleft palate, fungal infections, and a peculiar intrinsic black stain of the teeth, among others.

Nigerian children, Dental caries, Hypoplasia, Gingivitis, Periodontitis.

Friis-Hasche, E. *et al:* Dental health status and attitudes to dental care in families participating in a Danish fluoride tablet program. Community Dent Oral Epidemiol, 12:303–307, October 1984.
The caries experience and dental fluorosis of 84 Danish children, who had used fluoride tablets for 1-4 years in the period 1976-1980, were compared with those of a group matching in sex, age, place of living, and socioeconomic status. The average age of the children at the time of examination was 5.8 years. A recording of mothers' attitudes to dental care, knowledge about tooth brushing, attitudes to candy, and number of teeth in the maxilla showed no difference between the fluoride tablet group and the nonusers' group. Moreover, there was no significant difference between the two groups with respect to dental caries. The findings are discussed in relation to recent reports on the decline of dental caries resulting from widespread use of local administration of fluorides.

Attitude; Dental caries; Dental enamel; Dental health services; Dental plaque; Dental prophylaxis; Dentifrices; Family; Fluorides; Gingivitis

Eijkman, M. A. J.; Riel, C. B. M. van; Dijk, R.J. van: 873 questions of Dutch dental patients: A challenge to dental health education. Community Dent Oral Epidemiol, 12:308–314, 1984.
Two thousand persons from a select sample of the Dutch population were sent a questionnaire in which the most important question was: "Did you have any questions on matters related to dentistry during the past year?" The question was by nature an open question, and the respondents were entitled to several answers. Six hundred and forty questionnaires were sent back, of which 398 actually provided an answer to the request. Although the request was for questions only, there were also clear answers, in which 751 were identified as questions and 122 as complaints. The problems are highly varied as to their contents. Most questions bear on the dentist's activities (35%). Most striking is that these questions can be answered by any dentist. The category on dental minded behavior accounts for 22% of the questions, that on various aspects of the dentist-patient communication and on fear accounts for 17%, and the category on financial problems in dentistry accounts for 16%. Bearing in mind that the composition of the group of 640 respondents differs from that on the entire Dutch population, the results should be interpreted with due care. The discussion includes a plea for more DHE-activities on an individual basis. Finally, it is shown what the use can be of a coding system of questions and complaints of patients in dental health care.

Dental health education; Evaluation of care; Social dentistry

Poulsen, S. *et al:* Caries clinical trial of fluoride rinses in a Danish Public Child Dental Service. Community Dent Oral Epidemiol, 12:283–287, October 1984.
Three hundred and sixty-five 2nd through 4th graders completed a three-year clinical trial on the caries-preventive effect of rinsings every second week during the school year with 10 ml of an 0.2% neutral solution of sodium fluoride. All children received regular dental examinations and treatment in clinics established by the municipality in which the study took place. The trial was performed under double-blind conditions. The caries increment on teeth erupted at baseline was 1.75 DMFS in the fluoride group and 1.83 DMFS in the placebo group ($P > 0.05$; 95% confidence limits for percentage caries reduction: -20.7% and 29.5%). The caries increment on teeth erupting during the trial was 0.73 DMFS in the fluoride group and 0.99 DMFS in the placebo group ($P > 0.05\%$; 95% confidence limits for percentage caries reduction: 1.0% and 51.6%).

Caries increment; Fluoride rinses; Prevention

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ABSTRACTS *continued from page 95*

Wefel, J. S. and Harless, J. D.: Topical fluoride application and lesion progression *in vitro*. J Dent Res, 63:1276–1278, November, 1984.
The objectives of this investigation were to study the effects of topical fluoride application on white spot enamel and the effect that this application would have on a second acid exposure (lesion progression).

Artificial white spot lesions of approximately 200 um were created with an acidified gel technique, central control sections were removed, and the remaining tooth halves were randomly paired, with one half serving as an untreated control while the other received a single four-minute application of 1.23% (w/v) fluoride solution of either NH_4F, Na_2SnF_6, APF, or TiF_4. Both tooth halves were sectioned following progression and examined with polarized light microscopy. Lesion depth, internal pore volume, and presence, size and shape of dark zones were determined. The considerable variation among progressed lesions revealed no significant benefit from any topical fluoride treatment.

Enamel white spots, Topical fluoride, caries progress

Rasmussen, S. T.: Fracture properties of human teeth in proximity to the dentinoenamel junction. J Dent Res, 63:1279–1983, November, 1984.
Under conditions of controlled failure, the fracture properties of dentin at and in proximity to the dentinoenamel junction (DEJ) were studied with a work-of-fracture (W_f) technique and by scanning electron microscropy (SEM). In 37°C distilled water, fracture of normal human teeth at the DEJ was a difficult failure mode to investigate. Even with high stress concentration at the DEJ, only a few specimens showed apparent DEJ failure at localized regions. SEM fractography of these localized regions revealed surfaces similar in some respects to surfaces found in other studies of the DEJ of diseased teeth and decalcified or desiccated teeth. SEM fractographs varied depending upon the distance of the fractured surface from the DEJ. The results of this research suggest that the work of fracture of dentin increases for failure in the near vicinity of the DEJ, probably reaching a maximum at the DEJ.

Dentin, fracture properties; Dentinoenamel junction

Prasad, N. and Burdi, A. R.: Crown area as an indicator of changing crown size in human pre-natal teeth. J Dent Res, 63:1302–1305, November, 1984.
This study deals with correlations of tooth size in human primary teeth. Traditionally, mesiodistal or buccolingual measurements have been used to express crown size. However, in this study, crown area was used as a reference parameter.

Graphic reconstructions of 38 histologically prepared human fetuses with Crown-Rump Length (CRL) between 54 and 280 mm were used to obtain linear and area measurements. Based on clinical records and physical examinations, these fetuses were considered typical-for-age, or "normal". Correlation matrices indicated high levels of concordance among all developing primary tooth crowns and arch measurements. It was concluded that crown area is a statistically better trait to use in comparison of teeth rather than the traditional mesiodistal measurement.

Primary teeth, measurement of

Morimoto, T. *et al:* Frequency-dependent modulation of rhythmic human jaw movements. J Dent Res, 63:1310–1314, November, 1984.
Modulation of jaw movement patterns by alteration of the chewing rhythm was analyzed in nine subjects. Jaw movements were monitored by an electronic transducer system. The subjects were asked to chew gum either at voluntarily determined rates or at rates guided by a lightflash that varied from 2 to 7 Hz at 1-Hz intervals. The results can be summarized as follows: (1) Chewing was generally conducted at rates below 3 Hz when the rhythm was voluntarily determined. When the rhythm was guided, however, the maximum chewing rate could occasionally be raised up to 6 Hz; (2) the duration of the chewing cycle was more closely related to changes in the duration of the opening and occlusion phases than to that of the closing phase; (3) at chewing rates below 3 Hz; and (4) at chewing rates below 3 Hz, the jaw opening velocity increased with speed of chewing, whereas it decreased or sometimes fluctuated over a wide range at rates above 3 Hz. It is concluded that, during normal mastication at rates below 3 Hz, chewing rhythm is altered by changing both the velocity of jaw opening and the duration of the occlusion phase.

Jaw movement patterns, Rhythm modulation

Söderholm, K. -J. M.: Influence of silane treatment and filler fraction on thermal expansion of composite resins. J Dent Res, 63:1321–1326, November, 1984.
The coefficient of thermal expansion of experimental composite materials containing either silane-treated or untreated fillers in a triethylene glycol dimethacrylate (TEGDMA) matrix was investigated. The results show that an inverse linear relationship existed between volume fraction filler and coefficient of thermal expansion. No differences were seen between silane-treated and untreated composites, while it was found that repeated heating (aging) caused the thermal expansion to decrease for all material combinations.

Reduction in the coefficient of thermal expansion with increased filler fraction of unbonded filler indicates that the polymerization shrinkage of the matrix induces hoop stresses around the fillers.

By use of a simplified theoretical model (Appendix), these induced stresses were remarkably high, and that increased filler fraction increased

the tensile stress level surrounding the filler particles. Since these tensile stresses could facilitate crazing and crack growth in the matrix, these estimates may explain why filled resins containing low fractions of microfilled particles seem to possess remarkably good clinical wear resistance when compared with composites containing higher filler concentrations, at least during the first years in service.

Composite resins; Fillers, silane-treated; Fillers, untreated; Thermal expansion

Topitsoglou, V. *et al*: Effect of chewing gums containing xylitol, sorbitol or a mixture of xylitol and sorbitol on plaque formation, pH changes and acid production in human dental plaque. Caries Res, 17:369–378, 1983.

The aim of the present investigation was to study if xylitol added to a sorbitol-containing chewing gum influenced the pH changes and the acid production activity from sorbitol in plaque. Using a crossover design, a total of 71 persons were given, 10 times per day for 4 days, three types of chewing gum containing: (1) xylitol; (2) sorbitol, or (3) a mixture of xylitol and sorbitol (called xylitol/sorbitol). After the 4-day periods, the plaque pH changes were measured at various time intervals up to 40 min, either after 1 min of chewing on two pieces of the sorbitol or the xylitol/sorbitol gum in 24 of the subjects, or after a 30-second mouth rinse with 10 ml of a solution containing either 25% sorbitol or 25% sorbitol plus 25% xylitol in 23 of the subjects. Moreover, the amount of plaque (wet weight) and the acid production activity of plaque suspensions using glucose, sorbitol or a mixture of xylitol and sorbitol as substrates was also determined in 24 of the subjects. The 4-day periods with the xylitol/sorbitol gum and especially the periods with the xylitol gum resulted in less amount of plaque, higher plaque pH values, and lower acid production activities in the plaque suspensions than the periods with the sorbitol gum. Even though the observed differences were small, the results indicated that the presence of xylitol in a chewing gum, either alone or in combination with sorbitol, is preferable to sorbitol alone as far as the plaque formation, plaque pH and acid production activity in plaque are concerned.

Acid production, Chewing gum, Plaque, Sorbitol, Sugar substitutes, Xylitol

Glass, R. L.: A two-year clinical trial of sorbitol chewing gum. Caries Res, 17:365–368, 1983.

A clinical trial of sorbitol chewing gum was carried out in 540 children aged 7-11 years in a nonfluoride area. Subjects were assigned at random to one of two groups, a no-chewing group and one which chewed gum twice daily. Mean caries increments over the 2-year study period were 4.6 new DF surfaces (SD = 4.8) for the sorbitol gum group and 4.7 new DF surfaces (SD = 5.8) for the no gum group. Differences between groups were nonsignificant. As daily chewing of as much as 2 sticks of gum is unusually high, these findings demonstrate that sorbitol gum is noncariogenic.

Caries; Clinical trial; Epidemiology; Preventive, dietary; Sorbitol

Hoffman, D. *et al*: Reported cigarette smoke values: a closer look. Am J Public Health, 73:1050–1053, 1983.

Effects of mechanical compression of the filter tips and of blocking the air channels of a special filter design on the smoke yields of seven brands of commercial filter cigarettes were investigated. In addition, the influence of these variables on actual uptake of smoke constituents by smokers was studied with four subjects. Compression of filter tips produced major increases in smoke yields for the cigarette which features a filter tip with four longitudinal air channels at its periphery. Blocking of these air channels increased tar yields by 51 percent, and carbon monoxide by 147 percent. Subjects who smoked the cigarette with this special filter design tended to smoke fewer cigarettes per day than when they smoked cigarettes with perforated filter tips, yet their plasma cotinine levels were significantly higher. Blood pressure and pulse rate were markedly elevated after first exposure to smoke from the special filter cigarette, as were plasma nicotine levels. These results point out that individuals inhale different quantities of smoke constituents from cigarettes with reportedly similar smoke yields according to Federal Trade Commission methods. A redefinition of "average" smoking parameters and readjustment of standard laboratory methodology are suggested.

Filter cigarettes, Smoke constituents

West, G.A. *et al*: Autonomic responses to dental procedures in pedodontic patients during a standard restoration session. J Dent Res, 62 (6):728–732, June 1983.

Autonomic and behavioral responses to seven dental procedures were studied in 21 pedodontic patients aged six to 12. When mean pulse rate, pulse amplitude, absolute skin conductance, and skin-conductance responses were compared over all subjects, the injection of the local anesthetic consistently elicited the greatest response. In five patients where anesthesia was inadequate, painful high-speed drilling also elicited large responses. When autonomic responses were compared between high-anxious and low anxious children, both groups showed similar transient responsiveness. High-anxious children showed a significantly higher basal level of sympathetic activity.

Behavioral responses