DENTAL CARIES AND A CONSIDERATION OF THE ROLE OF DIET IN PREVENTION

The foundation for dental health is established early in life. The greatest single cause of dental disease is caries which, in turn, is largely a disease of the first two decades of life. The deciduous teeth are no less susceptible than the permanent ones, and disease in them is not without serious consequence for the permanent dentition. Since those physicians caring for children should be informed of current knowledge concerning the relation between diet and caries prevention, representatives of the American Academy of Pediatrics and the American Society of Dentistry for Children met to prepare a joint statement on this problem. The report which follows represents a summary of the position taken by this Committee.

This report has been reviewed by the Committee on Nutrition of the American Academy of Pediatrics; it has endorsed those portions which relate specifically to nutrition. The dental aspects, particularly the pathogenesis of caries, are the responsibility of dentists. Since there is significant difference of opinion on these problems, even among experimental pathologists in dentistry, the burden of responsibility must be borne by those assisting in the preparation of this report.

Anyone interested in a broad survey on the pathogenesis of dental caries may find a comprehensive statement in a publication of the National Research Council, Control of Tooth Decay, from the Committee on Dental Health, Food and Nutrition Board (N. R. C., Washington, D.C., 1953).

Dental caries is a disease of the calcified tissues of the teeth. It is generally believed to be caused by acids resulting from anaerobic glycolysis by microorganisms, is characterized by decalcification of the inorganic portion, and is accompanied or followed by disintegration of the organic substance of the tooth. The lesions tend to occur in particular regions of the teeth, i.e., the occlusal fissures of the molar teeth, the contact areas between adjacent teeth, and, in cases of rampant caries, the cervical areas near the gingiva. These are areas which are not self-cleansing.

Lactic acid, which has been demonstrated in areas of initial caries activity (Fancher et al., Muntz,) and advanced caries (Armstrong et al., Miller,) is the principal acid involved in the caries process. It is derived from bacterial action upon a carbohydrate substrate. Any microorganism, or combination of microorganisms, capable of producing an acidity of about pH 5, which is sufficient to decalcify enamel, can initiate dental decay. The time that the acid must be in contact with the tooth in order to produce decalcification is not precisely known, but from in-vitro studies of adult teeth, may be as short as 10 to 15 minutes.

Whether or not the acid formed will decalcify the enamel of a tooth is dependent on the concentration of the acid, its protection against dilution, and its duration of contact with the tooth.

There are natural factors in the mouth which contribute to the dissipation of acids formed on the tooth surface, such as the amount of saliva and the buffering capacity of the saliva. Specific inhibitory factors may play a part.

Of course, variations in the inherent re-
sistance of the teeth to destruction are important in determining the onset of caries.

FACTORS DETERMINING CARIES FORMATION

Certain conditions are essential for development of dental caries: 1) a caries-susceptible individual or teeth; 2) the presence of acid-producing bacteria which are capable of producing a sufficient concentration of decalcifying acids; 3) the presence of a substrate of orally fermentable carbohydrate; 4) bacterial plaque or accumulations which will concentrate the action of acid at caries-susceptible areas of the teeth.

Most individuals are caries susceptible; less than 5% of the population is immune. Animal studies indicate that caries immunity and susceptibility may be partly a matter of heredity. Klein and associates suggest the same possibilities in humans, but changes in caries activity in a single generation seen in Esquimaux and Maoris following changes in dietary habits indicate that heredity is only a minor factor. Caries activity is greatest during early childhood and adolescence and tends to taper off after maturity is reached. This is believed to be the result of a decreasing susceptibility of individual teeth with increasing exposure in the mouth.

The bacteria necessary for producing acid are always present in the mouth (Miller et al.) and in dental plaques. Many microorganisms have been found capable of producing the pH necessary for decalcification of enamel, including: lactobacilli, aciduric streptococci, diphtheroids, leptotrichia, actinomyces, fusiform bacilli, staphylococci and certain strains of sarcina. The lactobacilli have frequently been shown to have a numerical correlation with caries experience.

The substrate necessary for bacteria to produce acid is an important variable in caries attack rate. Fosdick and Burrill pointed out in 1943 that the only available substrates from which acids can be formed in the mouth are the carbohydrates and that easily fermentable carbohydrates, such as sucrose and glucose, are the ones most likely to be quickly converted to decalcifying acids under conditions existing in the mouth.

The importance of the bacterial plaque in the development of caries was pointed out in the early 1890's. Williams, in a study of 400 subjects, found that caries invariably occurred under a felt-like mass of microorganisms in which he postulated the necessary acid had to be formed. Recent animal studies support the idea that decalcification occurs principally under fixed deposits on the teeth. Rapid acid formation giving a pH as low as 4.5 has been shown to occur in vivo when sugars are placed on plaques on human teeth.

Various oral conditions may modify the activity of the preceding factors in caries. Principal among these would be: the destructibility of enamel in organic acids as it might be influenced by fluorine or other chemicals; the flow, consistency, neutralizing power and antibacterial action of saliva; irregularities of teeth or tooth surfaces, which contribute to bacterial and food deposits; and the presence of phosphates or other buffers in the food or of certain proteolytic bacteria. The absence of certain amino acids and vitamin fractions in the mouth may also play a part.

PREVENTION OF DENTAL CARIES

Since resistance to caries is determined in part by the ability of the teeth to withstand caries attack, it is logical that much attention should have been given to the effects of nutrition on tooth structure and caries resistance. The adequacy of the diet is often considered as being related to the dental caries experience. There is, however, a division of opinion concerning the relationship between dental caries and either specific dietary factors or the general nutritional status. Mellanby has offered evidence that teeth formed on vitamin D deficient diets were defective in surface structure and more susceptible to caries, but
many have questioned her conclusions. Bunting et al. observed 611 children in five public institutions. He concluded that the feeding of an adequate, well-balanced, low-sugar diet definitely decreased the caries activity. Dental caries occurs in well-nourished children, and it is of interest that the condition of the deciduous teeth of children suffering from malignant malnutrition (kwashiorkor) is reported to be good. There are no data indicating that a lack of minerals (Ca, P, Mg) or vitamins in the diet contributes specifically to the development of caries in humans.

Hence, there is insufficient evidence to claim a causal connection between general nutritional status and caries susceptibility. Furthermore, since calcification of all deciduous teeth is completed by 3 years of age, it is difficult to ascribe cavities which develop in deciduous teeth subsequent to this age to lack of minerals or vitamins in the diet. Certainly the improvement in the nutritional status of children in the United States during recent decades has not been associated with a decline in the prevalence of caries. Enamel hypoplasia, which is believed by some observers (though not all) to result from nutritional deficiency, is not associated with caries susceptibility.

Whether or not proper formation of the tooth is dependent on adequacy of the diet, it seems clear that once the enamel is complete it becomes relatively unresponsive to systemic influences of a nutritional nature. This is shown by the fact that there is no evidence of repair of carious lesions and the finding that there is essentially no passage of radioisotopes, such as P, from the tooth pulp to the enamel. The little which does reach the enamel arrives there through the saliva (Sognnaes and Shaw). The nutritional status can influence the integrity of the various periodontal structures and no one questions its importance in maintaining their health.

In practice, the best proven way of increasing the resistance of the teeth and preventing dental caries is by the addition of fluoride to drinking water and the topical application of fluoride. Although other methods of prevention on a mass scale have been attempted in recent years, none has proven effective. The information now available clearly indicates that fluoridation of public drinking water leads to a significant decrease in dental caries. The observed reduction in the incidence rate of decayed, missing and filled teeth (DMF) among children drinking fluoridated water has varied between 30 and 70% in different studies. In general, the magnitude of the reduction is inversely related to the age at which the fluoridated water is first regularly consumed. The caries-preventive effect is comparable to that seen in populations drinking naturally fluoridated water.

Most foods contain fluoride at a level of 0.2 to 0.3 parts per million (ppm) as consumed, except for seafoods and tea which contain considerably more. In this country about 3,500,000 people drink naturally fluoridated water. Excessive intake is known to result in mottled dental enamel in children and, when taken in very large amounts over long periods of time, in skeletal fluorosis in both children and adults. No confirmed deleterious effects have been observed in the United States.

The ideal vehicle for dietary fluoride should be such that its consumption is self-limiting, it is easily and cheaply available, and it is readily accessible to regulatory control. The fluoridation of communal water supplies meets these qualifications and is, in principle and in practice, the most effective approach to caries prevention on a large scale. The adjustment of the fluoride content of drinking water to 1 ppm in temperate climates (or about 0.7 ppm in hotter areas) appears to provide an optimal intake. This amount results in

* Recently consideration was given to a plan to include fluoride in milk formulae fed to infants living in areas where fluoridation of community water supplies was not practiced. This plan was rejected as unsafe, since positive control of intoxication under these circumstances was not believed possible.
significant reduction of caries without evidence of toxicity. To achieve maximal caries-preventive effect, fluoride should be ingested during that time when the teeth are in the formative stage and throughout the caries-susceptible years. This ingestion must cover a period from the fourth month in utero (when the first deciduous central incisors begin to calcify) to the age of 18 years.

Studies of children who have drunk artificially-fluoridated water for periods up to 10 years have failed to disclose any evidence of adverse effects on growth, or general health and well-being, or any changes in skeletal density or rate of skeletal maturation. Twenty-six million people in the United States are currently drinking artificially fluoridated water. Fluoridation of communal water supplies is a safe and effective means of caries control and should be extended to as wide a segment of the population as possible.

In areas where fluoridated water is not available, the topical application of a 2% solution of a fluoride to the crowns of the teeth, soon after the teeth are erupted, should be substituted. Many studies indicate a 40% decrease in the dental caries attack rate after such applications. Evidence available suggests that the reduction of caries is related to lowered solubility of fluoridated enamel in acid.

Regulation or restriction of intake of carbohydrate serves not only to foster adequate nutrition but also to withdraw the substrate from which bacteria form decalcifying acids. If strictly enforced, it also reduces the numbers of lactobacilli, which are used by many as an index of caries activity.

There is increasing evidence that between-meal eating and the frequency of eating are related to the dental caries experience of children. Gustafsson et al. conducted a well-controlled study of dental caries and observed that a group of patients who received a diet high in fat and very low in carbohydrate, and practically free from sugar, exhibited low caries activity. When refined sugar was added to the diet in the form of mealtime supplement, there was still little caries activity. In the same study, when caramels were given between meals, there was a significant increase in the numbers of new carious lesions. It was concluded from these studies that dental caries activity could be increased by the consumption of sugar, if the sugar consumed was in a form easily retained on the tooth surface. The more frequent the latter form of sugar was consumed between meals, the greater was the tendency for an increase in dental caries.

Mack studied a group of institutionalized children who were receiving an adequate diet. These children received sugar at mealtime only. She studied the effect of further additions of carbohydrate to the diet in the form of candy. This did not significantly increase dental caries activity, but the children did not receive candy between meals and they were encouraged to brush their teeth after meals.

Potgieter et al. surveyed the dental status in relation to diet as determined from records of weekly food intake of 864 Connecticut school children. Children who consumed more fruits and vegetables and who had better diets had a lower incidence rate of decayed, missing and filled teeth. The frequency of between-meal snacks also showed a slight positive relationship to the dental caries activity.

Dental caries does not often occur when the daily food intake contains no refined sugar and only minimal carbohydrate. When caries-susceptible individuals are given a low-carbohydrate diet, lactobacilli rapidly disappear from the oral cavity, and in many individuals it has been found that, after reducing the salivary lactobacillus counts by the use of a restricted diet, the carbohydrate intake can be gradually increased without a return of the previously high lactobacillus count. It is not necessary to restrict carbohydrate intake in highly susceptible patients as long as the lactobacillus count remains low. Counts of 10,000 lactobacilli per milliliter of saliva,
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or higher, are an indication that these organisms are sufficiently active to present a threat of development of caries.31

Although a low-carbohydrate diet may provide sufficient nutrients for the average individual, it is not consistent with contemporary eating habits in children, may cause ketosis, and is extremely difficult to maintain. Furthermore, the necessity of a diet which restricts not only simple sugars but also complex carbohydrate is not clear, because, in the joint report of the Council on Dental Health and the Council on Dental Therapeutics of the American Dental Association, it was concluded that starchy carbohydrates are of minor importance in the development of caries. In any case, this is therapy and not prophylaxis.32 Furthermore, pediatricians have questioned the advisability of restricting some of the starches as well as sugar, fearing that such a diet might not only be calorically inadequate but also cause emotional strain in some children.

The recommended diets3 can provide the daily allowances of nutrients recommended by the Food and Nutrition Board of the National Research Council. The daily intake of carbohydrate is restricted to 100 gm for 2 weeks. After this 2-week period starch is reintroduced. This procedure produces a rapid change in the oral flora, characterized by a marked reduction in the number of acidogenic bacteria. It is not meant to be a permanent regimen, but one to be followed for a short period of time and for a special purpose.

It is worth remembering that special dietary programs have other implications in childhood. If a child is compelled to eat a diet that is different from that of the other children, even in his own home, and, if the diet is different from the school meal, other children will make life miserable for the child in question. The result may be damaging to the sense of security. This factor should be carefully considered in relation to whatever advantages may be obtained by special diets. It is difficult, because of ready availability, to completely withhold candy from children. It would seem better to provide some candy in the home, to be eaten at the end of a meal, rather than to deny it completely. Furthermore, highly concentrated sources of refined sugar, such as candy, given after meals are apparently less apt to produce caries than if given between meals.

A number of investigators have reported on the decalcifying effect of acid beverages (made effervescent by addition of carbon dioxide or acid by addition of phosphoric or citric acid). McClelland,33 in 1926, reported that the presence of a pH of 3.5 and below, even if existing for only a few minutes, is a potential source of damage to teeth. West and Judy,34 in 1938, stated that "when an individual places a piece of ordinary acidified candy in his mouth and allows it to dissolve slowly against his teeth, the concentration of the solution at the surface of the candy will be very high, with a pH in the region of 3.4."

Restarski et al.,35 in 1945, reported: "In an initial experiment some extracted human teeth were immersed in a common . . . beverage. When first inspected after 2 days immersion, the enamel surfaces were found to be grossly decalcified. Severe destruction of the enamel on the molars of 200 white rats was produced by allowing the animals to drink the popular soft beverage for periods of 5 days or more." However, none of this relates directly to caries in human subjects, and the weight of evidence indicates that carbohydrates taken in liquid form are less destructive than those used in a viscous or solid form.

The role of simple dental hygiene, such as toothbrushing, in prevention of dental caries, while generally accepted, has not been exempt from the type of questioning directed at many other wide-spread hygienic measures. Nevertheless, few dentists or physicians fail to support the practice of proper brushing of the teeth.

Whether or not prepared dentrifices are more effective than simple brushing with water is, at present, the subject of controversy. Nevertheless, as both the lay public and physicians are targets for consid-
erable advertising by manufacturers of
dentifrices, it seems worth including a sum-
mary of the report on this subject* made
by the Council on Dental Therapeutics of
the American Dental Association:

A dentifrice is a substance used with a
toothbrush for the purpose of cleansing the
accessible surfaces of the teeth. Commercial
dentifrices are available in the form of paste,
powder and liquid.

However, the evidence to date indicates
that, when such dentifrices are employed as
adjuncts to supervised toothbrushing in con-
trolled clinical investigations, their superiority
over conventional dentifrices has not been
clearly established.

Controversial evidence concerning the possi-
ble usefulness of dentifrices containing urea
and dibasic ammonium phosphate is reviewed
in numerous publications.

Some control of dental caries has been re-
ported in controlled and supervised studies of
the use of a penicillin dentifrice. Other studies
have failed to reveal the same amount of use-
fulness from this dentifrice. It has not been
shown that the unsupervised use of a penicillin
dentifrice by the general public will result in
a reduction of the incidence of dental caries.*

There is a slight increase in the number of
penicillin-resistant organisms in the mouths of
the users of penicillin dentifrices.

Dentifrices containing chlorophyll derivatives
have also been placed on the market. There is
some evidence that the use of a chlorophyll
derivative in a dentifrice increased the
rate of improvement of gingivitis in a special
group of children under observation, but this
effect was transitory. Other investigators have
not been able to observe significant beneficial
effect from the use of a "chlorophyll" dentifrice.

Certain new foaming agents have recently
been incorporated into tooth pastes, and some
of these dentifrices have been promoted with
greatly exaggerated claims for "anti-enzyme"
and "antibacterial" activity. Evidence in sup-
port of these claims is controversial, and the
usefulness of these dentifrices in caries control
has not been adequately established.

A paste dentifrice containing stannous fluo-
ride has appeared on the market in some parts
of the country. The inclusion of other fluoride
salts in dentifrices has not been demonstrated
to be beneficial. The published evidence con-
cerning stannous fluoride in a dentifrice is still
too limited to form the basis of a reliable
evaluation.

Adequate dental supervision by a den-
tist seems to be an accepted health prac-
tice in most American communities, and
there is little question that dental supervi-
sion can play a part in caries prevention.
While there are now a limited number of
specialists in pediatric dentistry (pedodon-
tics), supervision must usually be obtained
from dentists not limiting practice to chil-
dren. The pediatrician can recommend that
toothbrushing start at about 24 months of
age and also that dental visits begin at
between 24 and 30 months of age. Then
the dentist will have the opportunity to
give counsel in general hygiene and also
to search for remediable oral pathology.

The pediatrician not only sees children
before the dentist but also is able to care
for their total health needs. Therefore, it
would seem wise to encourage pedia-
tricians to learn more about the dental
care of children, and, at the same time, to
urge dentists to learn more about the gen-
eral health problems of children. There
seems to be need for co-operation between
dentists and pediatricians; this should be-

 begin in the medical and dental schools. The
dental faculty should have an opportunity
to teach the etiology and treatment of
dental pathology to medical students, and
conversely, dental schools should have a
place in their curriculum for the pediatri-
cian to teach those aspects of pediatrics
which relate to dental problems. Pediatric
hospitals and children's services should
have dentists in attendance, and attempts
are now being made to have dental interns
in pediatric hospitals.

SUMMARY

As dental caries is primarily a disease of
childhood and appears to be at least in
part preventable, the pediatrician is obliged to be interested in this problem and can play an important part in prophylaxis. Present knowledge indicates that the most effective prevention available is the consumption of fluoridated drinking water containing a concentration of fluoride appropriate to the environmental temperature. Reduction of the intake of refined sugar both in amount and frequency has a beneficial effect on caries control. The prescription of diets essentially devoid of all sugars should be used to stem the progression of rampant caries. That this regimen would be as effective when complex carbohydrates are permitted and only refined sugars prohibited has been indicated by some studies. However, any highly restricted program must be considered therapeutic and not preventive and should be under pediatric supervision.

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REFERENCES
20. Bibby, B. G.: Neglected factors in the


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