A Guide to
PRACTICAL
NUTRITION

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Nutritional Aspect of Dental Disease

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THE etiology and prevention of dental caries and periodontal disturbances (gingivitis and pyorrhea) are of major interest to the medical and dental professions. The physician is concerned because these lesions form an important link in the chain of more serious systemic diseases. The dentist, on the other hand, has a paramount interest in maintaining the teeth as a functional unit to maintain optimum mechanical and chemical digestion. It is therefore apparent that dental practice is intimately associated with the science of nutrition. The physician and the dentist should therefore be acquainted with the relation of nutrition to dental disease.

Dental caries and periodontal disorders are among the common human ailments. They present serious economic problems for the average household; they are of great economic importance to the state, and to national defense. Optimum nutrition cannot be adequately maintained without an efficient complement of teeth. The efficiency of dental restorations are in proportion to the number and disposition of natural teeth remaining in the jaws.

The integrity and efficiency of an adequately functioning masticatory apparatus in middle life and in old age depend largely upon the status of nutrition during early periods of life.

The agents leading to susceptibility to dental disease are multiple and complex. The initial lesion of dental caries is due to organic acids formed "in situ" by the action of bacteria on carbohydrate food residues. The rate and concentration of this acid production depends, however, upon many factors such as the degree of refinement of the food eaten, the character of the oral bacterial flora and the presence of certain enzymes, vitamins (83), etc.

The role of nutrition in periodontal disease in man, however is more difficult to establish. It is generally known that inadequate nutrition predisposes to lowered resistance to bacterial invasion. The difficulty of obtaining definite evidence of the influence of subclinical nutritional deficiencies on the dental structures is obvious.

It may be stated from our present knowledge that diseases of the teeth and their investing structures are of a degenerative nature (84); they are related primarily to deficient food factors which predispose the dental tissues to invasion by microorganisms. The diet of primitive peoples who were relatively free from dental caries and periodontal disease, included the essential protective foodstuffs and a wide variation in the proportion of protein, carbohydrate and fat.

"Dental caries and civilization walk hand in hand" (83). The poor class among the early Egyptians had better teeth than the upper classes. The replacement of crudely ground cereals with modern white flour and the increased use of refined sugar has decreased the consumption of vitamin B_1 by approximately one-third. Roller milling and bleaching have removed from wheat and corn half of the riboflavin and calcium, two-thirds of the iron, and practically all of the caro-

tene. These are only two of many examples of how the foods of civilized man have deteriorated in biologic value in comparison with the foods of his primitive ancestors. Robert Harris (85) believed that the increase in the incidence of caries coincided with the decrease in intake of calcium, phosphorus, vitamins A, C, and D, each of which is essential for tooth formation.

Vitamin A deficiency during the period of tooth development leads to the formation of an imperfect enamel of chalky consistency with pits and fissures on the surface, and to the exposure of the dentine with subsequent discoloration of the teeth. Atrophy of the enamel organ may occur in severe vitamin A deficiency leading to "stunted" appearance of erupted teeth (86).

Ascorbic acid deficiency involves changes in the dentine-forming element of the tooth. The rate of tooth growth in experimental guinea-pigs can be controlled by the amount of vitamin C in the diet. A guinea-pig on a daily intake of 0.5 to 1 mg. ascorbic acid will show no clinical signs of scurvy but microscopic study of its tooth structures will reveal degeneration of the odontoblasts (87). The dentine forming cells have a definite reparative function after the tooth is fully formed; this is evidenced by the formation of secondary dentine in carious teeth of children fed on a controlled diet (88). An optimal vitamin C supply would therefore seem beneficial in retarding and walling-off tooth decay. Vitamin C influences the metabolism of calcium and phosphorus. An insufficiency of vitamin C in the presence of mineral deficiencies in the diet may lead to imperfectly formed teeth with fissures in the enamel.

Ultraviolet light and vitamin D appear to influence the body to resist dental caries. McCollum (89) reports that the largest number of cavities are observed in children in New York during the late winter and spring while the smallest number occurs during the summer and fall.

The systemic factor leading to tooth decay is believed by some to be a "disordered carbohydrate metabolism" resulting from an increased intake of refined carbohydrates and a vitamin B deficiency. Vitamin B_1 is necessary for adequate carbohydrate metabolism. Thiamin avitaminosis leads to intermediate breakdown products in carbohydrates which are acid in nature. It is generally accepted that the primary lesion of dental caries is brought about by acid formation in the mouth and there is clinical evidence to indicate the presence of this acid about the necks of teeth in caries susceptible individuals. Hearman (83) is of the opinion that an increased intake of carbohydrates may give rise to this acid production in the mouth.

The generally accepted views concerning the relation of nutrition to susceptibility and resistance to dental caries are as follows (83, 87, 89, 90):

1. Tooth decay occurs almost exclusively among animals and human-beings subsisting largely on carbohydrate food.
2. Carnivorous men and animals are free from dental caries.
3. Refined carbohydrates in the form of sugar and candy lead to tooth decay.
4. Diabetic patients subsisting on a restricted carbohydrate diet develop arrest of dental caries and show deposition of lime salts within the tubules of their carious dentine.
5. Certain systemic disorders, particularly those involving increased metabolic activity, as fevers, pregnancy and hyperthyroidism, predispose to tooth decay.
6. Dental caries appears symmetrically distributed about the teeth.

Diseases of the attachment apparatus of the tooth have in the past been regarded as the result of local causes. Recent observers seriously doubt the conception of local causes as the primary etiologic factor. Local factors are now considered environmental elements influencing the disease, thus making

them part of the syndrome complex. Percy Howe (83) stated in 1927 that pyorrhea is merely the oral expression of the results of long continued nutritional errors. Many factors besides diet are operative in diseases of the gingivae. The evidence is however that an increased consumption of refined carbohydrates and a deficiency of minerals, vitamins, etc., influence diseases of the gums.

A lack of calcium, phosphorus, and vitamin D weakens the alveolar bone of the jaws. There is evidence to indicate that diets low in calcium produce rarefaction of the bones in animals, more extensive changes appearing in the jaw bones than in the other bones (91). Optimum calcification depends largely on the amount of vitamin D available in relation to other constituents of the diet. The maintenance of favorable conditions in the intestinal tract for adequate calcium absorption and the selection of foods to maintain a normal acid-base relationship are equally important. Inadequate absorption occurs in hypochlorhydria, achlorhydria, during ingestion of alkalis, in liver and gall bladder disease, in hyperthyroidism, and during excessive catharsis and diarrhea.

The utilization of calcium depends upon other factors, among them protein. Insufficient protein in the diet leads to calcium excretion (92).

Ascorbic acid is believed to maintain the integrity of connective tissues. Vitamin C deficiency leads to the inability of certain connective tissue cells to form collagen fibers, osteoblasts and odontoblasts. A diet lacking in vitamin C has long been known to produce scurvy with its characteristic red, hemorrhagic, swollen gums and loose teeth. Boyle, Bessey, and Wolbach (93) have produced in guinea pigs characteristic ascorbic acid deficiency with the diffuse alveolar bone atrophy of pyorrhea. The jaw bones of these guinea-pigs revealed the gross x-ray and histologic changes characteristic of human diffuse atrophy pyorrhea.

Campbell and Cook (94) have recently reported acute gingivitis successfully treated with large doses of ascorbic acid. No other signs of the scorbutic state were evident and local treatment of the gums was not instituted. This corresponds partly to my own experience with this disease; but it seems more practical to consider that, as in pellagra, the majority of mouth lesions are primarily the result of multiple deficiency states. Treatment is always outlined with this plan in mind.

The relationship of vitamin B to the integrity of the soft tissues of the mouth seems firmly established. Topping and Fraser (95) were able to produce gingivitis, stomatitis, periodontitis, and noma in monkeys by feeding them a diet deficient in Vitamin B. Nicotinic acid has been recommended for the treatment of Vincent's disease of the gums. Weston Price (84) and others found a high level of the vitamin B group on a critical analysis of the native food of primitive tribes whose members had healthy periodontal tissues.

It is evident that deficiency in nutrition plays a prominent role in periodontal disease. However, a word of warning is in order against the indiscriminate use of vitamins and minerals in the treatment of these conditions without a general consideration of the patient's nutritional status.

Lesions of the periodontal structures and oral mucosa are so commonly associated with gastrointestinal symptoms, that I rarely see any lasting improvement in the involved oral structures until problems of the digestive tract are adequately corrected; particularly is this true of patients with hypochlorhydria, achlorhydria, gall bladder disease, and motor disturbances of the gastrointestinal tract.

In conclusion, I wish to emphasize that dental lesions can more adequately be managed by closer cooperation between the physician and the dentist.