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Food and the periodontal diseases

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The periodontal diseases, as the name is intended to imply, are a group of diseases which affect the tissues around the teeth, the gingiva or gum, the alveolar bone, the fibrous joint or periodontal membrane, and the cementum. It is important to note that this is a motley collection of tissues, one epidermal, the gingiva, and three mesodermal, two of which, the alveolar bone and the cementum, are calcified. It is only to be expected, therefore, that the effect of particular dietary deficiencies is confused and the subject of controversy.

If we look more closely at the fibrous joint between the tooth and the bone, we find that it is similar to other fibrous joints in the body. On the one side is the alveolar bone which is very cancellous, therefore very labile, and presumably easily affected by changes in the metabolism of the individual, and also by changes in the stresses applied to the tooth. From it, collagenous fibres run to the cementum, which invests the tooth and, as the name implies, cements the fibres to the tooth and is a very stable tissue which once formed is unlikely to be affected by the general bodily metabolism. This joint, however, differs from other fibrous joints in that, to all intents and purposes, the fibrous connective tissue of the periodontal membrane is exposed to the mouth, the ingress of bacteria and food debris being prevented by the physical adaptation of a cuff of epithelium around the neck of the tooth. If this cuff loses its tonicity for any reason, then invasion of the underlying tissues can occur.

Although the loss of teeth is not a lethal disease, yet it presents a serious public health problem. If people have no teeth, they cannot masticate properly and their diet becomes restricted: they must therefore be provided with some at a cost to the country of millions of pounds a year. Unfortunately even this expenditure restores the masticatory efficiency at best to only about half of that of the natural dentition. Unfortunately, the denture wearers are in charge of the selection and preparation of food for the younger generation and their own inclination is to provide food that requires no chewing, which, as we shall see later, predisposes the rising generation to poorly developed periodontal tissues.

Of the two dental-disease groups, dental caries and periodontal diseases, by far the greater number of teeth are lost as a result of the latter. The profession knows all too little about these diseases, so that what I have to say will be to a great extent theory rather than fact, and it will be important to distinguish the one from the other.

Periodontal disease, like most other diseases to which man is prone, can be looked upon as the result of a battle between the forces of destruction on one side and the resistance of the individual on the other. In more particular terms, between the forces

applied to the tooth, the invasiveness of the bacteria and the accumulation of tartar on the destructive side, and the built-in strength of the periodontal tissues and the general bodily resistance on the other.

If we examine large numbers of patients, it is quite obvious that the results of this battle vary considerably. At the one extreme there is the person whose resistance is so high that whatever the abuse to which the dentition is put periodontal disease does not seem to occur. At the other extreme, there are those in whom even the normal exigencies of everyday life seem to produce a breakdown of the supporting tissues of the teeth. There is a considerable amount of knowledge of the factors responsible for destruction, such as the accumulation of tartar and the effects of the application of excessive loads to teeth, but almost nothing is known about the factors involved in the resistance of the patient to these forces.

Into this pattern of a constant battle we must try to fit the part played by the food the patient consumes: it can be considered under two heads, the local effect of the food on the environment of the mouth, together with the effect of the masticatory effort necessary to comminute the food, and the systemic effect caused by the presence or absence of the necessary nutriments.

Local effects of the diet

We should, I think, consider first the local effect of the diet on the dentition, as it may colour to a large extent the experimental results obtained when investigating the systemic effects of various foods. The environment of the mouth is dictated by the saliva, and it is from the saliva in the form of tartar that one of the chief irritants to the soft tissues of the mouth arises. The amount of calculus deposited on the teeth varies of course from patient to patient, but it is influenced by the constituents of the diet, and the frequency of food intake. A high-carbohydrate diet stimulates the secretion of saliva of a high water content and rich in inorganic salts, which latter go to form an increased deposition of calculus, thus rendering the patient more susceptible to periodontal disease. This is further accentuated by the greater refining of the carbohydrate in the diet, so that roughage is eliminated. It has been said that the only way to halt calculus formation is to starve the patient; conversely the best way to speed up formation is to eat continuously. The modern diet, which contains a high proportion of refined carbohydrates, is passed through the stomach very rapidly so that the subject feels hungry shortly afterwards and therefore eats frequently.

The proportion of this calculus that accumulates on the teeth varies with the amount of chewing done by the patient and with the fibrousness of the diet, as the frequent contact of the fibres in the food, the tongue, and the cheeks, with the teeth and gums brought about by chewing helps to remove the deposit as fast as it forms. The more fibrous the diet, the more chewing is required, which acts directly upon the supporting structures of the teeth. A certain amount of use is generally necessary for the proper growth of the various parts of the body and for their continued health once they are fully formed. The masticatory apparatus is no exception.

Vigorous sucking from birth to weaning helps to lay the foundation for well-formed arches. Much energetic chewing during childhood ensures the formation of strong and healthy alveolar bone around the teeth. Adequate use during adult life ensures a first-class blood supply to the tissues. In between the fibre bundles of the periodontal membrane are channels containing the blood vessels and the lymphatics. During chewing, the teeth move in their sockets, alternately compressing and expanding these spaces, so helping to promote the flow of venous blood and lymph away from the area. Lack of use causes a degeneration of the alveolar bone and periodontal membrane and also reduces the resistance to infection of these tissues in the immediate vicinity of the non-functional tooth.

From the point of view of the environment of the mouth, then, the optimum diet is one low in refined carbohydrate, high in fibrous content and with a long stay in the stomach so that eating is less frequent.

Systemic effects of the diet

In passing from the local effects of the diet on the periodontal tissues to its systemic effect, we pass from the known into the unknown. So it would be best to consider on purely theoretical grounds what dietary deficiencies might be expected to influence the supporting tissues, and to pass from that to consider the evidence. In this way it is possible to narrow the field of deficiencies down to: those of calcium, phosphorus and vitamin D, which might be expected to affect the calcified tissues, vitamins A, B and C, which might affect the epithelial and collagenous tissues, and protein (including individual amino acids), which would affect the growth and stability of all tissues. As it would make for boredom to take each of these factors individually, it will be better to discuss only those about which we think something is known.

Here, unfortunately, I must digress to mention briefly the clinical entities which I have grouped together as periodontal diseases.

(1) There are those which are merely an inflammation of the surface epithelial tissues, known as gingivitis. (2) There is a disease known as periodontitis simplex which, for our purposes, may best be defined as a destruction of the supporting tissues caused by the local destructive forces mentioned earlier. (3) A disease known as periodontitis complex which, again purely for our present purposes, we can consider to be a destruction of the periodontal tissues for which no local cause can be found, i.e. indicating a systemic or general metabolic cause.

It is known that gingivitis occurs in the late stages of certain vitamin deficiencies, those of folic acid, nicotinic acid, riboflavin, vitamin A and vitamin C (Dreizen, 1956), but it is not thought to be produced primarily by the deficiency but rather by an invasion of weakened tissues by the normal bacterial flora of the mouth. It has come to be generally accepted therefore that in both gingivitis and periodontitis simplex, dietary deficiencies are rarely of any major importance in the aetiology of the disease, though in their treatment it is advisable to ensure that the patient is consuming a balanced diet.

It is when considering the problem of periodontitis complex that it is important to consider the effects of diet. The first observation to influence this search into the diet was that of destruction of the supporting tissues of the teeth by an absolute deficiency of vitamin C. For a long time it has been assumed that this destruction was similar to that seen in periodontitis complex (Boyle, Bessey & Wolbach, 1937). More recently, however, Waerhaug (1958) has shown one marked difference between the two conditions. Until the late stages of scorbutic periodontitis, there is no loss of tissue around the necks of the teeth, so that the administration of ascorbic acid produces a complete cure and the mouth returns to normal. In periodontitis complex, however, there is an early loss of this cervical tissue which once lost can never be replaced, so that even if the disease is halted the mouth cannot return to normal. It is interesting to note that almost the same remarks apply to deficiencies of calcium and vitamin D. This is not to say that partial or complete deficiencies of these factors will have no effect upon the progress of a lesion, but rather that they may be looked upon as contributary rather than causative.

What may turn out to be one of the more fruitful lines of investigation is that of the effects of protein deficiencies on the supporting tissues. There are many areas of the world where periodontitis complex may be said to be endemic, affecting a high proportion of the population. This position may be due to a racial predisposition to the disease, but it could also be due to the type of diet eaten which may be dictated by availability, poverty or religious custom. These areas are all situated in the tropical or subtropical belt, and the people affected are those leading a static pastoral life, depending for their food supplies upon cereal grain and pulses. The nomadic tribes in the same areas are not affected, and the presumption is that this immunity is due to the latter's consumption of animal protein. This observation has naturally led to the investigation of the effects of protein deficiency on the periodontal tissues of experimental animals. As might be expected, an absolute protein deficiency in the diets of young weaned animals halts the remodelling of the alveolar bone by halting the osteoblastic phase, and slowly reduces the collagenous fibrous content of the periodontal membrane (Stahl, Miller & Goldsmith, 1958). Absolute protein deficiency does not, however, occur in these human populations. The children in these areas show a normal deciduous dentition with little or no evidence of periodontal destruction. It is only after the permanent teeth have begun to erupt that the disease manifests itself, and it reaches an incurable state by the time the subject reaches maturity. If protein deficiency is the underlying cause, therefore, the condition is more likely to be due to a shortage over a long period of one or more of the so-called essential amino acids, some of which are not provided in adequate quantity in the diet. It is questionable whether animal experimentation will provide us with the answers to this problem.

I hope that in this short space I have managed to give an indication, if nothing more, of the importance of the food we eat in the control of periodontal diseases and to show how little really precise information is available of the effects on the supporting tissues of partial deficiencies of various essential nutrients.

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Dental caries in relation to nutrition: structural effects

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In discussing the relationship between nutrition and dental caries it is important to distinguish between the systemic effect of the absorbed food upon the developing tooth and the environmental effects of dietary habits. In this paper we are concerned with the former. There is no evidence to suggest that any physiological changes occur in the intact fully formed enamel. It is unlikely therefore that any change can take place in the composition of the fully formed enamel by active cellular processes. If nutritional factors are to influence subsequent caries then it is likely that they will do so during the formative period.

Dental caries is the result of bacterial attack on the surface of the tooth. Some teeth seem better equipped than others to combat this attack. It is pertinent to inquire how far this resistance can be attributed to nutritional factors present during the tooth's development.

Earlier work on the relation between nutrition, tooth structure and dental caries

Various attempts have been made to correlate imperfection of surface enamel structure with susceptibility to dental caries. Mellanby (1918, 1923) was one of the first to suggest such an association. After examining many hundreds of children's teeth she concluded that those teeth with comparatively mild surface hypoplasia were more susceptible to decay than those without such irregularities (Mellanby, 1934). Support for this hypothesis was provided by the observations of Davies (1939) on the first permanent molars in British schoolchildren, by Mellanby (1940) in Finnish children and by Bibby (1943) in a study of New England children. In each study there was some positive correlation between irregularity of enamel surface and dental caries. Contrary to these findings, Day & Sedwick (1934) reported no difference in the caries incidence in children with and without grossly hypoplastic teeth and Staz (1943) in a study of Johannesburg children concluded that hypoplastic teeth were less liable to decay than normal teeth.

There is therefore some evidence that poor surface structure may increase susceptibility to caries. It is in the interpretation of these results that controversy has arisen. There is evidence to show that deficiencies of vitamin D during tooth