Vitamin C in Health and Dentition*

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There is an old story of a man who, seeking a means of helping human progress and happiness, was one day taken for a walk in the world by the devil. As they went about, Satan rather proudly as it seemed, called his attention to discoveries, inventions, and innumerable signs of rapid progress in knowledge in various fields. With surprise, the man inquired, "But does not all this progress annoy you, threaten your kingdom?" Smiling and serene, Satan replied, "Not at all—I shall soon tempt them to organize their knowledge." If the story indicates no more than half a truth, that part is none the less insidious. The temptation is ever present to the modern, as to the alchemist, to seek—and to see, as an end of seeking—the philosopher's stone, a panacea, in the organization of new knowledge, always hoping that the newest thing may be the explanation and answer to the perplexing and fascinating puzzle. Then some piece fails to fit and one must go on with the search again. So progress is tangential, but it *is* progress. Such history is well exemplified in the growth of knowledge of the vitamins. Deficiency diseases were long unrecognized as such, while human thought was engaged with toxic, bacterial and infectious theories. The backward look from our peak of recent advance has value in the attempt to understand dental caries. The literature abounds with theories as to etiology and cure, with only an occasionally clear voice of warning that the answer is not complete in any one factor; rather all factors—infections, mouth and systemic reaction, fully adequate diet leading to optimal health—must not be lost sight of. This introduction seems advisable in a discussion of vitamin C and caries, for so much evidence is available as to specific effect that there is a tendency to consider that vitamin C may give the complete answer.

The statement is often made that we no longer see cases of scurvy either in infants (infantile scurvy used to be quite general) or in adults. Such a statement is doubtless true as far as it goes. However the problem, as with the other vitamins, and indeed with requirements in general, is with relative deficiency and this still remains a serious one. A relative deficiency usually does not manifest itself in a clear cut syndrome but in more or less ill-defined poor health. The indefinite picture is further complicated by the fact that in most human subjects there is more than one such deficiency—a diet

*(This is the third of a series of essays dealing with diet in relation to dental health.)

poorly balanced in regard to one requirement is often low in several. There is no true security in the non-occurrence of typical scurvy. Human complications are well exemplified in the early work leading to the discovery of the antiscorbutic vitamin. As mentioned in the previous paper, it was rather early differentiated from the other water-soluble vitamins, due, primarily, to the work of Holst and Fröhlich. After beri-beri was demonstrated to be due to a dietary deficiency, these workers undertook further investigation of so-called "ship beri-beri" to which the Norwegian sailors were subject when on long trips. Using guinea pigs, since shown to be highly susceptible to scurvy, they got results "identical in all essentials" with human scurvy. Further work, with swine particularly, served to establish ship beriberi as a deficiency of two separate but both water-soluble vitamins, now known as B and C. Later work (1,2,3) amply confirmed the postulate that the antineuritic and the antiscorbutic vitamins are separate identities, both essential to good nutrition. Subsequent work has also confirmed the original findings as to occurrence and stability of vitamin C—of which more later.

Typical scurvy symptoms are (2, 4): debility or loss of weight, tenderness of the joints, especially wrists, ankles and knees (animals and babies with scurvy usually cry on being handled) followed by swelling, and sometimes spontaneous fracture. In older animals there is stiffness, loosening of teeth, gum and joint hemorrhages and, later, hemorrhages into the skin. Autopsies show separation of the epiphyses from medulated bone and characteristic bone marrow changes. When treatment is instituted in the early stages, many of the symptoms do not develop or are readily healed; if delayed too long, they may never be completely obliterated—e.g. joint swellings may harden instead of being reduced. Animals vary markedly in susceptibility; man is neither so susceptible as the guinea pig or as immune as the rat. Even in the latter, however, there is evidence to show that a high order of health and well-being demands an adequate supply of antiscorbutic food in the dietary.

As has been mentioned, the human problem is more concerned with relative rather than absolute deficiency—so-called latent, rather than typical scurvy. It is through experiments planned to elucidate this problem that serious injury to teeth induced by low vitamin C has been detected. Zilva and Wells (5) pointed out that when the deficiency was so slight as to be unrecognized by the usual symptoms, there were marked changes in the teeth. They concluded that the teeth are the first parts of the body to be affected by low vitamin C. Howe (6) studying the teeth of scorbutic guinea pigs, found extensive absorption of the alveolar processes (similar to pyor-

rhea(and marked decalcification and loosening of the teeth; the gums bled easily and showed much pus. Alternating normal with deficient diets produced definite markings in the tooth enamel; decalcified areas were noted also in the bones—head, leg, rib, and maxillary bone. Other workers (7) confirmed and extended these results, showing in severe cases degeneration of pulp tissue, beginning with the upper part and extending to the apex, with the pulp entirely replaced by osteodentin or bone.

One of the important contributions to the knowledge of effects of vitamin C is Höjer's (9) extensive study. Realizing the greater importance of latent scurvy in human application, he studied various levels of suboptimal amounts of vitamin C. He found also that tooth changes occurred much earlier than other symptoms and noted progressive changes as follows: gradual change of the odontoblast layer; amorphous calcification of the predentin; widening of Tome's canals; new formation of bone instead of dentin, gradually extending toward the center of the pulp as a spongy, porous mass; dilation of the vessels, hyperemia, occasional hemorrhages; atrophy and resorption of the pulp tissue. He believed the extent of the changes to be determined by the degree of vitamin C deprivation rather than by the length of time of deprival. He considered that the condition of irregular dentin, shown when about one-half the necessary dosage is provided, is the condition most usually seen in human beings. He deducted that the degeneration in the pulp is primarily one of change from odontoblast to osteoblast. Bone changes were similar to the tooth changes but occurred later in the progress of the disease. He states that there is not a primary derangement of calcium metabolism as in rickets, despite the fact that in scurvy, calcium deposits may be found in the liver and muscles. This he explains by the evidences that in early scurvy, bone formation is inferior qualitatively, not quantitatively, resulting in surplus calcium. Essentially he believes that in rickets, osteoid tissue is formed but not calcified, while in scurvy bone is rapidly calcified but is formed to only a limited extent. Two other facts were noted in his studies which have important bearing on tooth and mouth health: there was general tissue atrophy which included the salivary glands; more than half the animals with latent scurvy showed definitely lowered resistance to infection—attributed to weakness of lymphoid tissue. In later papers (10), Höjer developed this early susceptibility of teeth to injury from low vitamin C into a quantitative method for determination of vitamin C. It has the advantage over other quantitative methods, based on the appearance of the more obvious scurvy symptoms, of being shorter, and therefore saving in time and expense, as well as being more delicate. It is criticized however as being possibly less dependable than other

methods, based as it is on a single criterion rather than upon several. Basal diets, standard animals, and the general technique of handling are essentially the same in the types of determinations. In the Höjer method all animals are killed for autopsy on the tenth to fourteenth days. In the other type, the experiments must run on for two to three times as long. In the Höjer method one-half the lower jaw is removed and decalcified, then embedded, sectioned through the foremost molar at right angles to the longitudinal axis of the jaw, and stained. The principal examination is of the cross section of incisor roots. On a fully protective antiscorbutic diet, the stained section shows a broad uniformly stained layer of dentin, white uncalcified predentin, a layer of long, slender, parallel odontoblasts, and a pulp with star shaped cells. With less than the minimum protective dose of C, the uniformity of the dentin-staining no longer appears. In diets lacking in C, the dentin is thin, the predentin, being calcified, is very dark, and the odontoblasts are not in a continuous layer—they have rather become osteoblasts. Results of the Höjer method show, in general, that twice the amount of antiscorbutic is required to protect the teeth as compared with amounts required to protect the individual from other symptoms. Dalldorf and Zall (11) have suggested using the rate of growth of the incisor teeth of guinea pigs, as being more constant and simpler than the Höjer method. Whether or not these methods come to be standard in testing potency of vitamin C, they are worthy of attention as showing how greatly tooth health and development may be influenced by the vitamin.

Seeking an explanation of the mechanism of action of the antiscorbutic vitamin, work of two groups of experimenters should be noted. In 1919, Aschoff and Koch (12), on the basis of extensive study, postulated the theory that the symptoms of scurvy are due to lack of, or failure in production of, "cement substance," intercellular material binding cells together. An essentially similar theory was put forward by Wolbach and Howe (13) who stated that the condition (failure of intercellular material) affects various supporting substances to a different degree, being most marked in those in which the intercellular material is calcified, as in dentin of teeth and in bone matrix. The condition holds for cartilage and connective tissue and (by inference) for others, e.g. blood vessels. Meyer and McCormack (14) also confirm this in general, but they believe that the change is chemical rather than cellular and due to intra rather than inter-cellular changes.

Confirming the animal experiments on scurvy is the work of Hanke (15), who has made careful studies of dietary habits with relation to dental disorders—dental caries and pyorrhea, both alone and associated. He found

that 27% of the patients were deficient in vitamin C only; all patients showed marked improvement when this vitamin was much increased. These and other striking evidences of the marked effect of C in arresting caries and correcting pyorrhea has seemed to support the theory that the vitamin is a specific. A word of warning seems necessary. Hanke himself points out that in his clinical studies he supplied high vitamin C because diets in general were particularly low in this factor, *not* because he believed it to be the only factor implicated; rather he stresses the necessity of diets adequate in every respect as necessary to good dental health. In this connection he points out that, even should mouth infection be eventually shown to be the primary cause of caries, an adequate diet has much to do with reducing susceptibility to such infection.

Dietary effect on susceptibility to infections is something of a moot question. Vitamin A has been called the anti-infective vitamin. While this has been shown to be a misnomer, both because it evidently has no influence on the opsonic index and because other factors than A are involved in increasing immunity, yet there is little doubt that its influence on mucous membranes (as pointed out in the first paper of this series), and hence on one of the first lines of defense of the body against bacterial invasions, adds to general immunity. Much the same thing applies to vitamin C. Findlay (16) showed that scorbutic animals succumbed to smaller doses of pathogenic bacteria than do those on a normal diet; his animals suffered from latent scurvy and manifested no *clinical* symptoms of the disease. Jackson and Moody (17) had previously shown that some symptoms of typical scurvy may be due to intercurrent infections—i.e. here, as in other instances (compare ophthalmia and low vitamin A), certain types of infectious organisms were shown to be present in scorbutic lesions, although both induction and healing response of such lesions leave no doubt that the dietary factor is the primary one. Werkman et al (18) also found decreased resistance to infection in scorbutic animals and suggested lowered body temperature as the cause (they found no change in agglutinin production nor in opsonic activity). Such findings are certainly of possible importance in considering infection theories of caries. Bunting, who is the most important supporter of this theory of primary infection in caries, reported (19) that improved diets markedly aided prophylactic measures.

A unit of vitamin C is defined (20) as that amount which, when fed daily, just suffices to protect from scurvy, as generally recognized, a standard guinea pig prepared and fed by a standard method. If the Höjer me-

thod is used, the minimum protective dose is twice that required when the more manifest symptoms are used as criteria. The international unit is based on lemon juice as the standard antiscorbutic. In contrast with the other vitamins, there are not, at present, commercial concentrates of this vitamin on the market. This is chiefly for two reasons: first, it is the most unstable of the vitamins; secondly, its richest source being the juice of citrous fruits, it is readily available for inclusion in the average dietary. The latter reason might lead one to infer that the average dietary would contain ample amounts of the vitamin, but such an inference is far from warranted. A finding of poor dental health should lead to the question as to the supply of vitamin C. It should be emphasized, moreover, that daily supply is required, since this vitamin is not appreciably stored in the body.

I have said that this is the most unstable of the vitamins and hence difficult to prepare commercially in concentrated form. In the early work of Holst and Fröhlich it was already shown that the vitamin is readily destroyed by heat and by oxidation, with the marked exception that it was found comparatively quite stable in acid foods even when heated in the presence of oxygen. As knowledge has developed with explanations of this behavior, it has been possible also to develop a technique in handling whereby the vitamin is protected. In general however it is necessary to depend upon raw foods as sources. Pasteurized milk, cooked fruits and vegetables, unless strongly acid, have lost large proportions of the vitamin C which they originally contained. This is not an argument against pasteurization nor for faddism for raw foods; as will be shown, it is simple enough to get an adequate supply without resorting to fads.

As in other fields of medicine, knowledge of vitamins grew rapidly during the World War, in response to the necessity of keeping troops well fed and protected from deficiency diseases. In this respect a striking example of the value of food was in Mesopotamia where scurvy was widespread among the Indian troops, not among the British, while the reverse was true of beri-beri. Habitual diet, previous to the siege, had doubtless already brought many of the Indians to the verge of scurvy, while the British, on a varied diet, were well protected. In the emergency, the latter had large amounts of fresh meat from which the Indians refrained on account of religious scruples. While meat is a very poor antiscorbutic, in this case it helped out a very limited dietary. Both groups were greatly improved when herbs and fresh vegetables became available. Incidentally the beri-beri was overcome among the British by including in their bread the Indian "atta", which contains wheat germs. This strikingly demon-

strated need for knowledge was followed by intensive research at Lister Institute and elsewhere, from which we have many practical results regarding the distribution of vitamin C.

Fruits and vegetables are far the richest sources of this vitamin. There is wide variation among them however, and much which occurs natively is destroyed by storage, drying, ordinary methods of cooking, etc. Lemons are outstanding. In passing it may be noted that the "limes" which led to the elimination of scurvy from the British navy, were really lemons. True limes are only one-fourth as rich as lemons. Lemon juice has been successfully "treated" (21) and concentrated so that it is possible to give large doses even to infants. Such a concentrate also has value on long voyages (22). Fresh orange juice has a potency about the same as lemons. Both, as explained above, lose much of their potency on standing (breakfast orange juice should *not* be prepared the night before) especially if rendered alkaline. Drying has been done with only a small loss of potency (23), at least if sulphur rather than sun drying is practised (24). Commercial concentrates were studied by (25) Goss at the California Experiment Station. His results showed some destruction of C although the products remained good antiscorbutics. Reports on grapefruit are conflicting,—some class it with lemon and orange juice, others consider it of distinctly less value but still a very good antiscorbutic. Tomato juice is of about the same value as lemons, and due to its high acidity, there is relatively little destruction in canning. Canned juice, weight for weight, is richer than fresh tomatoes (26, 27). The value is much influenced by the degree of ripeness. Thoroughly ripe tomatoes whether induced by ethylene gas, or occurring in the greenhouse or the open field, are richer than if ripeness is incomplete (28). Strawberries, raspberries, currants, gooseberries, are all excellent sources and acid enough to protect this vitamin from deterioration. Raw cherries are good sources but when cooked are poor. Apples are a fair source (one variety has been reported as excellent), and are so often eaten raw as to be of considerable importance. Bananas are fair sources; pears, apricots, peaches, and plums, are low. Among the vegetables, the juice of rutabagas and of raw cabbage are about as good sources as lemon juice; lettuce and watercress are high. Potatoes are only fair but important because of the large amounts consumed. Mature seeds, either cereals or grains, are devoid of C, but develop it on sprouting; young succulent peas and beans are good sources. It must be emphasized that most vegetables lose much or most of their potency when cooked. Heat, especially in the presence of air (oxygen), is very destructive, (see below). Liver is higher

than muscle meat which is poor. Milk varies with the feed of the animal—that is, it is higher when animals have fresh, succulent feed. It is mostly destroyed by pasteurization, but this should by no means be considered as an argument against pasteurization, since milk is too variable to be relied upon as a significant source of this vitamin. To summarize the occurrence of vitamin C in foods: (20) the citrous fruits—lemons, oranges, tomatoes, grapefruit—are the richest sources; berries, young succulent vegetables, and some other fruits are good sources if eaten raw; liver and other organs are fair sources but muscle meat is poor; raw milk is fair to good, depending upon the feed of the producing animal.

From the earliest knowledge of this vitamin and exemplified in its occurrence in foods, it has been shown to be very unstable. In many foods, originally good antiscorbutics, Holst and Fröhlich noted great losses on cooking, drying, and even merely standing. The notable exception was in the citrous sources when the vitamin was—although to a degree only—protected by the high acidity. Gradually, by much research, it has been shown that the vitamin is extremely sensitive to destruction by oxidation, and that the oxidation takes place most readily at nearly, or on the alkaline side of, neutrality. Ordinary cooking, of course, subjects foods to the oxygen of the air; moreover vegetable tissues contain more or less oxygen or show a high oxygen potential. It has been demonstrated that the higher this potential, the greater and more rapid is the destruction of the vitamin. Modern methods of canning have been devised to overcome this effect; vacuum exhaust and sterilization temperatures are used with much reduced exposure to oxygen so that canned foods show relatively little destruction. Indeed, foods canned with such precautions are better sources of C than "fresh" foods which reach our tables only after some days in transit from the fields through the markets. As a result of a great amount of research concerning the occurrence, lability, and general properties, it has recently been impossible to isolate the pure vitamin (29,30). Due primarily to the work of Szent-Györgyi in Hungary and others, vitamin C has been shown to be identical with a hexuronic acid or "ascorbic" acid. This acid occurs in relatively large amounts in the suprarenal glands. In animals which are susceptible to scurvy, such as guinea pigs, it has been shown that the amount of acid in the glands becomes markedly less on a low or free vitamin C diet. It has also been shown that the vitamin value of lemons is accurately accounted for by their content of this acid. Ascorbic acid has been successfully prepared in relatively large amounts from Hungarian paprika (Capsicum annuum).

Summary

1. Low amounts of vitamin C have been shown to have very great effects on teeth and gum structure and consequently upon resistence to caries and to pyorrhea.

2. It is very generally low in the average dietary.

3. The best sources of the vitamin are the citrous fruits and young succulent vegetables—but these latter must be eaten raw.

4. The vitamin is readily destroyed in ordinary cooking. It is to a great extent protected by modern methods of canning and by sulphur drying. Alkaline reaction and presence of oxygen (air) greatly increase its destruction.

5. The vitamin is chemically identical with ascorbic acid, as established by recent research.

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