Nutrition and Bone Growth*

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Eleven years ago, I presented before the International Orthodontia Society skulls of macacus rhesus monkeys which had been subjected to experimental feeding for a period of from nine months to one and one-half years. Although these feeding experiments have been continued to the present time, only refinements in results have occurred.

The pathological changes have been studied in greater detail and analyses of the bone composition have been carried out with more accuracy, due in part to perfection of methods. Among the changes observed are deformities in the mandible, particularly at the condyles, sigmoid notch, coronoid process and ramus. In some deficiencies, the growth of the supreior maxillae shows arrest with open sutures. The arch is narrowed, the anterior teeth protrude and give the face the appearance of the adenoid, mouth-breathing type. The posterior nasal passages are constricted in the deficiently-fed animal, the degree depending upon the extent of the specific deficiency and the duration of the experiment. In most of these cases, general bone changes are found. The effects shown have come about through protracted mineral and vitamin deficiency feeding. Since it is in this field that most of my investigation has been conducted I will at this time confine myself to this aspect of the subject. The particular mineral deficiency has been calcium, which Sherman has stated to be the greatest deficiency in modern diets. Diets deficient in vitamins A, C, and D were also fed. The pathological consequences, as described by Professor Wolbach, are briefly as follows:

Hemorrhages and bone changes are the outstanding features of scorbutic conditions. The explanation lies in the fact that the cells of supporting tissues are unable to make and maintain normal intercellular substances. The substance of particular interest is the collagen of all fibrous structures, the matrices of bone, dentine, and cartilage, and all non-epithelial cement substances including that of vascular endothelium. Thus, in bone disintegration, the change is in the character of the matrix. Hemorrhages occur through the diminished cohesion of endothelial cells.

Proliferation of osteoblasts of the periosteum is a conspicuous feature

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of scurvy in infants. A layer of cells without intercellular material results in separating the periosteum from bone and lays the ground for change in bone form. The histological changes in scurvy have been more fully described elsewhere. Of all the known vitamins, the anti-scorbutic is the most likely to be destroyed in the treatment which our food receives. Now that its chemical nature has been determined, it has been subjected to renewed study only to verify the earlier principles of its action. In practice, the latent and protracted, or intermittent deficiency, is most frequently encountered. So far as has been determined, saturation seems desirable.

In scurvy, there is no interference with calcium metabolism, so far as one can see. On the other hand, one can hardly think of rickets without taking up the subject of calcium and phosphorus supply and its metabolism. The precise cause of rickets is not as clearly determined as that of scurvy. There are curative effects brought about in two separate ways. One is attained by instituting a proper ratio between calcium and phosphorous in the diet at a proper level. The other is by administering the D vitamin.

The pathology of rickets is briefly described as an interruption in the normal sequences of the replacement of cartilage by bone. Growth is accomplished by continuous proliferation of cartilage cells on the epiphyseal side and simultaneous degeneration of matured cells on the diaphyseal side. The cavities occasioned by the degeneration and disappearance of the cartilage cells are entered by capillaries accompanied by osteoblasts which deposit osteoid on the exposed cartilage matrix; hence the first formation of bone is within spaces previously occupied with cartilage cells.

This is the first osteoid to become calcified in repair. The osteoid of the diaphysis that has accumulated during the deficiency subsequently becomes calcified and is then largely removed by osteoblasts. Then the osteomalacia of adults presents striking deformities of the skeleton and complicated microscopic pictures, all to be explained by loss of calcium salts in bone, secondary resorption of the matrix, and new formation by way of reparative response of cells producing osteoid which does not calcify.

While the effect of vitamin A deficiency relates primarily to epithelia, the secondary effects of interest here are anemia, cessation of growth of bones and degenerative lesions of the skeletal muscles. The introduction of vitamin A into the diet is followed by regeneration of the bone marrow and an outburst of erythroblastic activity.

The cessation in bone growth is due to cessation of proliferative activity of the epiphyseal cartilages. A narrow band of atrophic cartilage results which becomes bound by a thin layer of bone of the diaphyseal side. In recovery, the cartilage regenerates and blood vessels from the diaphyseal

marrow penetrate the limiting bone plate, and normal endochondral bone formation is resumed. Thus, it can be seen how the formation of either intra-cartilaginous or intra-membranous bone may be altered by a few dietary deficiencies. Electrolyte metabolism, acid-base balance or food ash may all enter into the problem.

Nutrition means more than food selection, although this is a first consideration. How the mechanism of the body disposes of the raw building materials is a second consideration. Digestion, absorption and, in the case of bones, mineral fixation enter into the problem. Indeed, Cannon has shown the effect of the emotions on digestion, and in these days of high pressure existence, it deserves thought. Then, too, endocrine action is, in this connection, another factor.

From my experience in experimental work, there are a few matters that I should like to mention to you for what they may be worth. For one thing, I will refer to the changes in bone in which it is the organic substance that is the deciding factor. It is so in scurvy and it is so in rickets. X-ray diffraction studies substantiate this view. One cannot escape noticing that in bone growth and development, in nutritional deficiencies, the fundamental action is on cellular activities. It is seen that as long as uninterrupted growth of the bones in question is allowed to proceed, regularity in tooth arrangement has occurred. It is to be recalled that the results seen are from protracted deficiencies.

Deformity in the region of the condyle, at the sigmoid notch, and in the coronoid process and ramus of the mandible leads one to wonder if changes may not be effected in the temporo-maxillary region in some cases of orthodontic treatment. In the differences observed at the sutures in the superior maxillary bones, one again wonders if, in orthodontic procedures, changes take place here. In passing, I should like to suggest that it would be to the advantage of Orthodontia if there were more definite information as to when and where centers of ossification put in their appearance and also as to the time in life when maxillary sutures close.

From a further examination of these skulls, one notes the difference in the size of the posterior nasal openings in the normal and in the deficiently-fed animals. Here mouth breathing is not the cause, but the result, of constricted nasal architecture brought about by nutritional faults. In a study of the effect of nutrition on bones, one observes that a notable change occurs usually at the point of muscular insertion. This may be of interest because so much of orthodontic therapy consists of intelligently applied stress or tension. In order that this may not occupy the mind of the operator too exclusively, let me quote from Harris as follows:

"We see the manner in which the calcification of the matrix of the senescent cartilage provides the only means whereby the hereditary form of bone can be maintained from the 'slimy' mesenchymal stage through the 'gristly' cartilage stage to the highly vascular 'bony' stage.

"We have a new conception of the manner in which both the hereditary form of the bone and the dispositions of the longitudinal and transverse trabeculae are impressed upon the bone at so early a period in the life of the embryo that strains and stresses and the pull of the as yet undifferentiated muscles cannot be a primary determining factor of the form of bone. This is a fundamental objection to the acceptance of the various mechanical and mathematical theories of bone growth."

Studies of the muscles themselves in the deficiently-fed animals show degenerative changes in the skeletal muscles. The stimulation of muscular tone appears to be a worthwhile consideration in Orthodontia.

Let me also call your attention to the fact that these experimental monkeys have inherited tendencies of no-one-knows-how-many-generations toward normal tooth arrangement. Yet, within a few months, one is able to produce mandibular and maxillary bone deformities with irregularly placed teeth by instituting nutritional faults.

Such carefully controlled studies as the one now being conducted at the clinic on growth and development by Dr. Stuart at the Harvard School of Public Health will eventually be a great help in determining the relation of early nutritional factors to bone growth.

It is one of the responsibilities of the orthodontist to see that conditions are as favorable as possible for bone growth. From the time when man begins his existence as a simple cell of about .2 mm. in diameter until he becomes a unit of highly specialized cells, he is dependent for changes involved in the transition on nutrition. To this rule, bone is no exception.