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Megavitamin Arthritis Treatment, Part 5

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CHAPTER 5

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THE COMMON FORM OF JOINT DYSFUNCTION
by William Kaufman, M.D., Ph.D. (1949)

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Some Inferences Concerning Joint Dysfunction

(Dr. Kaufman, writing in 1949, shows remarkable foresight half a century into the future of orthomolecular (megavitamin) medicine. In this chapter, he describes how the lack of a single nutrient can cause diverse diseases; the need for a new way of looking at arthritis, and reviews his treatment and what level of success to expect with it.)

References cited are posted at this link: http://www.doctoryourself.com/kaufman11.html

It has been shown that the clinical diagnosis of joint dysfunction and the clinical classification of the severity of joint dysfunction are made on the basis of an objective criterion, the Joint Range Index, which is the weighted mean of the numerical values obtained upon measurement of 20 specified joint ranges. Measurement of the Joint Range Indices of an unselected, untreated population of 455 ambulatory male and female patients, 4 to 78 years of age, subsisting on the average American diet of 1945 to 1947, demonstrated that joint dysfunction, as defined by a Joint Range Index of less than 96, was almost universal in occurrence, and was present in individuals with or without complaints referable to muscles and joints, and with or without clinically obvious rheumatoid arthritis or clinically obvious hypertrophic arthritis. The decline of the means of the Joint Range Indices for each succeeding 5-year age group was practically linear in the untreated population, indicating that joint dysfunction tends to increase in severity with increasing age, although within any 5-year age group there are individual variations in the numerical values of the Joint Range Indices which can best be seen in Table 1G. Usually, in the more severe clinical grades of joint dysfunction, clinically obvious rheumatoid and hypertrophic arthritis are commonly found. It has been observed that a person less than 40 years of age with a severe or extremely severe clinical grade of joint dysfunction is more likely to have rheumatoid than hypertrophic arthritis, and that a person more than 50 years of age with a severe or extremely severe clinical grade of joint dysfunction is more likely to have hypertrophic than rheumatoid arthritis. However, a person with mild, moderate or severe joint dysfunction may show no evidence of either hypertrophic or rheumatoid arthritis, or a person with slight joint dysfunction may have local evidences of hypertrophic arthritis (Heberden's nodes) and no other clinical evidence of arthritis.

During the first month of adequate therapy with niacinamide (alone or in combination with other vitamins) a patient with joint dysfunction (with or without rheumatoid or hypertrophic arthritis) will have a rise in the Joint Range Index of at least 6-12 points, and thereafter will have a rise of at least 0.5 to 1 point per month of adequate niacinamide therapy, provided he eats the average American diet containing adequate calories and sufficient protein, and provided he does not mechanically injure his joints excessively. **This improvement in joint mobility occurs regardless of**

the age or sex of the patient, and regardless of whatever other health problems he may have. Subsequently, with continuously adequate niacinamide therapy, the Joint Range Index of 96-100 (no joint dysfunction) is reached, and **maintenance doses of niacinamide are required** to keep the Joint Range Index at this level.

The only observed exception to the attainment in time of a Joint Range Index of 96-100 with prolonged adequate niacinamide therapy occurs in persons who have ankylosed joints which have not regained any mobility in response to adequate niacinamide therapy. In such persons with one or more ankylosed joints, the rise in the Joint Range Index with adequate prolonged niacinamide therapy is the same as the patterns described above, although in time serially rising values of the Joint Range Index may stabilize at a level below 96, which represents the maximal Joint Range Index attainable by the patient when the ankylosed joints do not regain full mobility in response to prolonged adequate niacinamide therapy, even though all the other joints measured for computation of the Joint Range Index can be moved through their full ranges; e.g., if one wrist joint is irreversibly ankylosed, the highest attainable Joint Range Index is 90.9. As has been previously noted, some joints which initially appear to be clinically ankylosed, in time regain their full ranges of movement in response to prolonged adequate niacinamide therapy.

Therapeutically induced improvement in joint mobility, as shown by increasing values of the Joint Range Index, cannot be maintained without continuously adequate niacinamide therapy. When adequate niacinamide therapy is discontinued, there is a regression in the Joint Range Index from the therapeutically improved value to the pre-treatment value. When niacinamide intake is reduced from adequate to inadequate levels, the Joint Range Index decreases and stabilizes at a level above the pre-treatment level and below the maximum level therapeutically achieved.

In this study, in the various case histories it has been shown that joint mobility of patients with either hypertrophic arthritis or rheumatoid arthritis improves in response to the administration of adequate niacinamide therapy. In addition, certain other benefits have been observed from this therapy, most of which are not susceptible of objective measurement. In both hypertrophic arthritis and rheumatoid arthritis, these benefits include a feeling of being more alert, more vigorous, tiring less easily, an increased sense of well-being. The patient may lose certain minor digestive complaints such as constipation and abdominal bloating. Aches, pains and stiffness in muscles and joints gradually disappear, and his joints seem to be injured less easily by mechanical trauma. Crepitus becomes less noticeable, and eventually disappears. The physician may note that the patient appears younger, that his color is improved, his skin is more elastic, and that his tongue shows improvement in mucous membrane morphology. Liver tenderness and enlargement may disappear. Hemoglobin levels tend to improve. Joint mobility improves, joint deformities occasionally resolve, and impaired muscle strength tends to improve (54) (141).

Not all patients with rheumatoid arthritis recover at the same rate from the signs and symptoms of rheumatoid arthritis in response to prolonged adequate niacinamide therapy. In the early stage of rheumatoid arthritis (prodromal period), there is prompt resolution of the patient's symptoms and signs. In the intermediate stage of rheumatoid arthritis (active acute rheumatoid arthritis) there is a slower resolution of the symptoms and clinical signs of this disorder, not unlike that described in Case E, page 46. In the late stages of rheumatoid arthritis (advanced chronic rheumatoid arthritis), there may be so much retrogressive tissue alteration in nonarticular as well as articular tissues, that complete functional and structural recovery may not be possible, even with prolonged niacinamide therapy. Resolution of the clinical signs and symptoms of late rheumatoid arthritis is exceedingly slow, and not unlike that described in Case V, page 39.

Especially noticeable in patients with rheumatoid arthritis who are receiving adequate niacinamide therapy is improvement in appetite, with concomitant gain in weight, recovery from many nervous and mental symptoms, gradual disappearance of muscle atrophy, and improvement of muscular strength; usually, also, the anemia is corrected, the sedimentation rate index decreases to the normal range, and subcutaneous nodules tend to disappear.

At the present time, no explanation can be given to account for the biodynamic mechanism of niacinamide4nduced improvements in persons with joint dysfunction. However, in both hypertrophic arthritis and rheumatoid arthritis, articular and non-articular improvements continue for as long as the patient's niacinamide intake remains adequate, and they tend to fade away when inadequate niacinamide therapy is substituted for adequate niacinamide therapy, or when niacinamide therapy is discontinued.

Most writers have considered hypertrophic arthritis and rheumatoid arthritis to be two distinct clinical entities (147) (33) (121) (195) (196) (197) (198) (161) (79) (12). Hypertrophic arthritis has been considered to be a degenerative joint disease, and rheumatoid arthritis has been considered to be a generalized disease of the entire body, of unknown etiology (57) (58) (132) (134) (145) (227) (228) (229) (230) (231) (28) (77) (140). From this work, no proof can be given for or against the theory that niacinamide tissue deficiency disease is a prime mover in the evolution of both hypertrophic arthritis and rheumatoid arthritis. It should be realized, however, that **merely because hypertrophic arthritis and rheumatoid arthritis are different clinical entities, one cannot exclude the possibility that they may be caused by the same etiologic agent, acting in different ways.** For example, in experimental animals, it has been shown that **the lack of a single essential nutriment can produce a variety of dissimilar clinical disorders in different individuals** of the same species. However, without knowledge of the animals previous nutritional history, one might not suspect that the same etiologic factor, lack of a specific essential nutrient, was responsible for each of the various clinical syndromes of the same tissue deficiency disease which is permitted to develop at different rates in different individuals of the same species. For example, "two distinct syndromes of ascorbic acid deficiency have been observed in the Rhesus monkey depending on whether the deficiency was acute or chronic. An acute deficiency was characterized by a precipitous weight loss and tenderness in the joints of the legs but no gingival lesions. A chronic deficiency was characterized by severe gingival lesions and skeletal changes but no rapid decrease in weight" (192). Similarly, marked differences exist between the clinical manifestations of acute and subacute athiaminosis.

When monkeys were made completely deficient in thiamin, an acute athiaminosis was produced, and "death occurred before any outstanding clinical symptoms or marked histological degeneration of nerves set in." When monkeys were given about one-half of the needed thiamin, subacute athiaminosis ensued, and there were "clinical signs of polyneuritis and cardiac failure, autopsy findings of peripheral nerve degeneration and
hydropericardium" (115).

Although it may appear from this study that niacinamide has a high degree of specificity in the reversal of the metabolic processes which permit joint dysfunction to evolve, it may well be that a number of other therapeutic agents may influence these metabolic processes in the same direction and to the same extent that niacinamide does. Future clinical studies may indicate that there exist substances other than niacinamide which can produce therapeutic effects which equal or surpass those obtained in response to prolonged adequate niacinamide therapy in the treatment of persons with joint dysfunction. (Editor’s note: Vitamin C is one such substance.)

In the treatment of joint dysfunction in the future there may be clinical applicability of the findings of recent studies concerning the metabolism of niacin under a variety of experimental conditions and in different species of experimental animals (238) (178) (176) (177) (90) (69) (70) (44) (45) (112) (120). (In most of these laboratory studies, niacin rather than niacinamide was administered to experimental animals. While there are certain differences in the biologic utilization of niacin and niacinamide (71) (82) (69) (70), there are sufficient similarities to suggest that biologic utilization of niacin and niacinamide for many metabolic purposes might not be dissimilar, and thus many of the findings that hold true for niacin may also hold true for niacinamide.)

In these studies, certain metabolic interrelationships between niacin, tryptophane, protein, amino acids, pyridoxine and 3-hydroxyanthranilic acid have been discovered (201) (202) (188) (186) (179) (173) (162) (129) (101) (102) (103) (72) (92) (76) (42) (24)

(1). It has been found that when a niacin-deficient animal is fed a sufficient amount of tryptophane, the animal is cured of its niacin deficiency, and increased amounts of niacin-containing molecules appear in the animal's urine. For this reason, naturally occurring tryptophane is thought to be a biological precursor of niacin. In the rat, 50 mg. of tryptophane is the equivalent of 1.0 to 1.5 mg of niacin or niacinamide. In the biotransformation of tryptophane to niacin, 3-hydroxyanthranilic acid is an intermediary compound which is capable of replacing tryptophane in the biosynthesis of niacin and niacinamide (129). It has been found that the amount and type of protein the animal is fed determine the amount of niacin or tryptophane needed by the animal to prevent a niacin deficiency disease, and that a naturally occurring protein (gelatin) (20) and certain naturally occurring amino acids (especially threonine) can inhibit or nullify the effect of niacin or tryptophane in the diet; similarly, other naturally occurring materials (especially corn) (247) (205) and certain synthetic molecules (244) (245) (246) may effectively block the

metabolic action of niacin or tryptophane. The level of pyridoxine nutrition also influences the metabolic pathways by which niacin and tryptophane are utilized (204) (185) (187) (163) (146) (116) (119) (7) (11).

Many diverse types of investigation in humans and in suitable experimental animals remain to be carried out in order to determine the exact nature of the biochemical, metabolic and morphologic changes occurring as a result of the ingestion of different amounts of niacinamide for short or long periods of time. Such studies could evaluate (a) any differences which might exist in the biochemistry of persons with different grades of joint dysfunction, (b) the effect of adequate niacinamide ingestion on the biochemistry of persons with joint dysfunction who are eating a standard diet and are performing a standard daily amount of physical exercise; (c) any sex differences which might exist in biochemical reactivity to niacinamide therapy; (d) the maximal safe doses of niacinamide therapy which would cause optimal improvement in joint dysfunction; (e) various therapeutic agents which have been shown to be capable of replacing niacinamide in animal studies; (f) various conditions which have been found to enhance the effectiveness of niacinamide in animals; and (g) various nutritional conditions which have been found to inhibit the action of niacinamide in animals.

In addition, it would be desirable (a) to study the effects of continuous niacinamide therapy on the biochemical, metabolic and morphologic properties of articular structures in various animal species including bovine animals which are subject to spontaneously occurring hypertrophic arthritis with increasing age (225); (b) to study the distribution of niacinamide in articular structures of experimental animals through the use of tagged molecules (isotopic and radioactive) (85) (17) (212) (164); and (c) to produce graded sub-acute and chronic aniacinamidosis in experimental animals and to study articular structures at various time intervals (244).

While such animal studies and specialized chemical and metabolic studies in humans may prove fruitful in elucidating the mechanisms which permit joint dysfunction to evolve in an untreated population, and to be corrected by niacinamide therapy, from a practical point of view it has been demonstrated that the progressive impairment in joint mobility with advancing age, which has been accepted in the past as an inevitable concomitant of the normal aging process, can be corrected or ameliorated by prolonged adequate niacinamide therapy. In addition, it seems not unreasonable to suppose that the evolution of the common form of joint dysfunction can be prevented by adequate niacinamide supplementation of an adequate diet throughout the lifetime of an individual.

(End of Chapter 5, and of the book. 248 references follow in the Bibliography, which is posted at this link: http://www.doctoryourself.com/kaufman11.html)