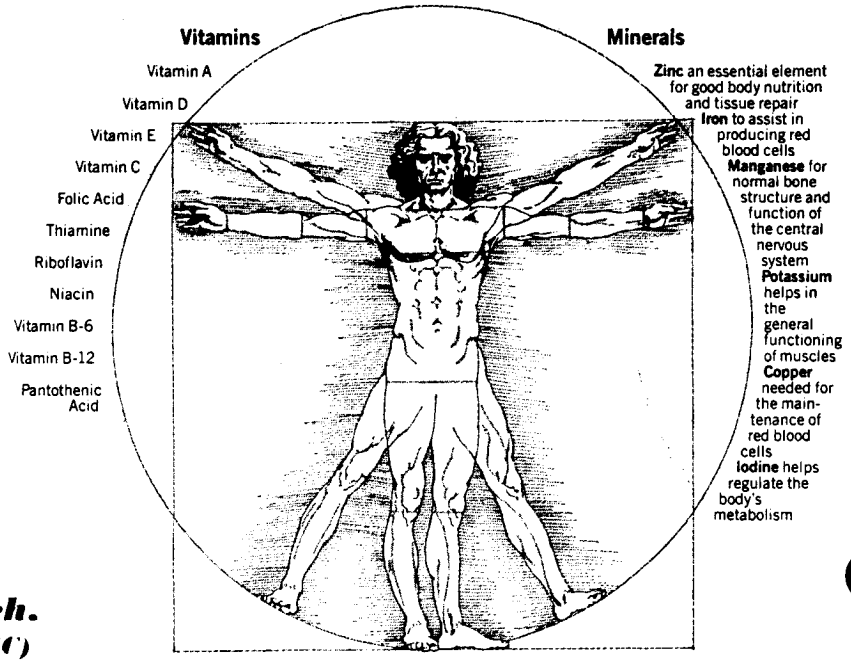


CIRCA 1980

by Carl J. Reich, M.D., F.R.C.P. (C)

megavitamin & mineral treatment of arthritis

Medical science is baffled as to the cause of the two major forms of arthritis: namely, degenerative presenile osteoarthritis and inflammatory rheumatoid arthritis. This mystery still exists today despite the fact that these two diseases affect over 10% of the population. While coronary heart disease kills or disables approximately one-third of the male executive class in our country, arthritis likely causes more time lost from work and causes more suffering. Moreover, because of its high incidence and the added care required in physiotherapy and nursing home care, the national health care costs incurred by this disease is likely greater than for any other disease.

ARGUMENTS AGAINST THE ORTHODOX CONCEPT OF ETIOLOGY

Etiology of osteoarthritis

Many theories have been presented as to the cause of each of these two major forms of arthritis, ranging from simple physical wear and tear to pure psychic factors. In the case of osteoarthritis — despite the fact that it frequently occurs in 50-year-olds as typically as it occurs in the 70's and 80's, it is most commonly attributed to the aging process and is considered a natural degenerative phenomenon. In startling contrast to this concept is the fact that some individuals who have led the most active lives, such as the modern trappers of the North who are still capable of hiking or snowshoeing 10 to 15 miles while in their 70's or 80's, show hardly a trace of this disease! Increased physical activity therefore cannot be considered as a cause of osteoarthritis and to attribute such absence of disease wholely to inherited genetic factors, while ignoring the possible influence of biochemical factors arising for reason of environmental influence, seems unwarranted. Therefore the profession, and especially those of the public who may have begun to develop the painful knees, hips, or fingers of presenile osteoarthritis in their early 50's, have been left to wonder why the joints of their skeletons have begun to degenerate while their neighbors and friends who, to all appearances, apparently live the same or even more vigorous lives, remain entirely free from the least vestige of an ache or pain! The term "apparently" above is the clue that indeed the contrary may be true and that a difference in lifestyle not yet recognized by the profession or the public may be responsible for the development of this disease in relatively early life.

Etiology of rheumatoid arthritis

The most common suggestions made regarding the cause of rheumatoid arthritis are (1) that it is the result of some chronic infection of the joint; (2) that it is the result of psychic stress; i.e., that it is a psychosomatic disease; or (3) that through a derranged immune mechanism the body has become sensitized to some factor within itself. In the instance of the infection theory, the daily press and medical journals almost annually announce the discovery by some research institute of a bacteria or virus isolated from the joint fluid of individuals with rheumatoid arthritis which is responsible for the generation of this disease. All such hopeful announcements are never followed up with an effective treatment for the disease. The best and most recent support for this infection concept is the effectiveness of an anti-protozoal agent, "clotrimazole." Recent clinical trials by the Bayer Drug Company of West Germany unfortunately have revealed considerable toxicity to this drug.

As will be detailed later, I appreciate that obscure infection may play a part in the etiology of some cases of rheumatoid arthritis. But this infection may be made possible only through the effects of long-term dietary deficiency which resulted in a demineralization inflammatory reaction and devitalization of the joint through a process which I define as "maladaption."

The occasional onset of acute rheumatoid arthritis following rapidly on severe psychic stress, such as a bereavement, has repeatedly led to the assumption that the somatic or physical disease of bone could be completely attributed to psychic stress. Similar relationships in the occurrence of the disease following childbirth or major surgery have also led many observers to attribute the disease to the physical and psychic stress inflicted on the individual because of physical demands of pregnancy, physical injury, anesthetic, blood loss, etc.

Contrary to this direct relationship I feel that, while psychic and physical stress may indeed be the final factor which may "trigger" the disease through demineralization, such individuals have been prepared for years or decades; i.e., they have been "pre-loaded" by the effect of chronic dietary deficiency. In these cases this pre-loading has proceeded to the degree that the balance is very easily tipped in the direction of disease, even by stresses that normally are endured almost endlessly or repeatedly by other non-deficient individuals.

By their ample mineral reserves, such non-deficient individuals are able to adapt to the acute demand for minerals.

The auto-immune theory of rheumatoid arthritis is the most recent. Indeed, the work of immunologists, analyzing each antibody protein fraction of blood serum related to body defense in search of a persisting abnormality which then could be related to the cause of this disease, has been prestigious. The fact that the maladaptive syndrome occurs as consistently in the chronic allergic patient as in the rheumatoid arthritic patient incriminates the immune system in arthritis. In my appreciation, however, the immune system so involved is one that has been seriously derranged by the effect of chronic deficiency; and unless the immunological reactions in these patients are compared to the immunological reactions of the non-deficient individual, the study will likely not be profitable.

THE ORTHOMOLECULAR MALADAPTIVE CONCEPT

In the late 1960's — almost fifteen years after my identification in 1954 that chronic asthma was largely a deficiency disease and my very successful treatment of several thousand cases with megadoses of vitamins A and D and minerals present in bone — I discovered that the arthritic and the asthmatic patient had many features in common, despite the differing major diseases they complained of. This similarity was particularly true of the chronic dietary and other deficiencies they experienced and the resulting effects of these chronic deficiencies on the function and physical state of other tissues and organs of the body, giving rise to compaints and physical changes other than those due to asthma or arthritis.

This study of the many and varied clinical associations with arthritis led me to an appreciation of the arthritic that can best be expressed by the quotation: "There, but for the grace of God, walks an asthmatic." With this in mind I treated the arthritic patient the same way I had treated so many chronic asthmatics: with megadoses of the A and D vitamins. The relief from pain, swelling, and disability of the disease experienced in a much larger percentage of these patients than could be expected from conventional therapy with drugs lead to the appreciation that indeed this concept was valid!

MALADAPTION

Some years prior, in 1958, after approximately four years of studying a large number of patients experiencing and not experiencing chronic dietary and other deficiencies and also the results of megavitamin therapy on a large number of chronic asthmatic patients, I arrived at the concept of maladaption. In this concept I considered that the disease of chronic asthma arose largely because of the breakdown of a bronchial constrictive adaptive maneuver, which was attempting physiological biochemical compensation to the chronic deficiency state. I proposed that this adaption was possibly effected through autonomically excited bronchial constriction and exudation which would alter the gaseous exchange of the lung to induce changes in the acid base balance of the body. I moreover proposed that maladaption occurred when physiological mechanisms involved in this process were derranged or became pathological because of chronic deficiency.

As may be expected, attempts were then made to relate other disease to this concept. In the instance of rheumatoid arthritis, I concluded that prior to the onset of the disease an adaptive mechanism involving autonomically excited subsynovial absorption of mineral compounds in the bone had been in effect. I suggested that this process was designed to provide minerals, particularly the calcium reserves in the skeleton, in times of deficient intake or deficient absorption of minerals for the needs of other tissues (such as nerve or muscle) which have a higher priority than bone for these minerals. Unfortunately this adaption to chronic mineral deficiency, through utilization of the reserves of the skeleton, is not an infinite or unlimited process. Instead, once the demineralization process has progressed to the point that the mineral content of subsynovial bone had been depleted to a critical level, an inflammatory process of

synovia results. This inflammation, consisting largely of hyperemia and edema, gives rise to the tissue swelling and production of excessive synovial fluid, etc. which are characteristics of acute rheumatoid arthritis and which so frequently results ultimately in the total destruction of the articular end of the bone.

In the application of this concept of adaption to the creation of osteoarthritis, one must concede that the breakdown of the adaptive process likely does not give rise to such a very active and inflammatory adaptive process produced indirectly by the autonomic nervous system and involving vascularity of synovial, etc. Instead the clinical characteristics of this disease incriminate a far less active direct process, such as the simple demineralization of bone through slow chemical absorbtion. In such a direct adaptive process, calcium salts of bone are dissolved from the matrix by simple excessive osteo clastic over osteo blastic activity of bone cells. Exostoses of bone, which occur at the sites of demineralization, likely are attempts at healing by the deposition of calcium in an inflammatory response of bone. Kinetic forces within the joint, introduced by weight bearing or by the usage of the joints, which have been demineralized and so weakened, may accentuate the irritative response and so be partially responsible for the foundation of exostoses.

EXAMINATION OF THE ARTHRITIC STATE

Clinical

In the majority of arthritis cases the diagnosis of joint disease, which most frequently is either rheumatoid or osteo-arthritis, is apparent. One of the diagnostic problems that may arise, however, is in the relatively rare cases in which both diseases co-exist in the same patient. The more frequent diagnostic problem is that of prodromal or early clinical stages of these diseases. Early stages of osteoarthritis not yet showing definite x-ray changes, which the physician may be reluctant to diagnose as arthritis, are most certainly an inflammatory state of the joints. Similarily, in some instances of rheumatoid arthritis, a long period of migratory pain may precede the inflammatory phase of the disease. Therefore, in both of these types of early arthritis, no gross signs of joint disease may be apparent for years and only pain with some moderate to mild limitation of movement may be present.

Laboratory

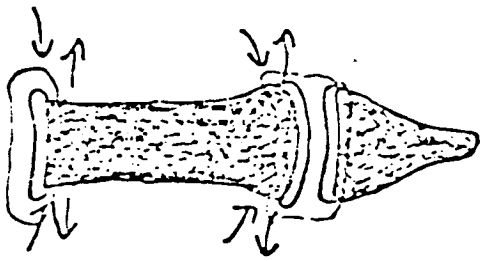
Just as clinical signs of joint swelling, etc. are frequently not present in an early stage of these diseases, so also laboratory tests are occasionally of no help and may even be detrimental to the making of a diagnosis in advanced cases. For example, the rare case of active rheumatoid arthritis may show only slight elevation of the sedimentation rate or, more frequently, may demonstrate a negative latex fixation test, etc.

X-ray

Positive x-ray changes are frequently not present in early cases of osteoarthritis or rheumatoid arthritis. In the latter instance, the radiologist's report often will indicate the presence of a degree of generalized osteoporosis while x-rays of the joints may reveal no pathology except occasional effusions. This type of report indicates that a process of de-mineralization of bone had predated the acute onset of the arthritis, likely by years.

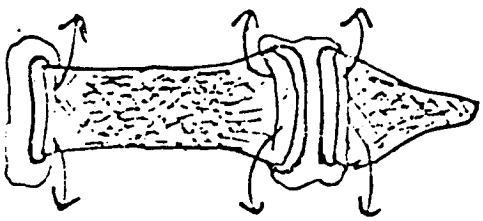
—Continued in next issue

Osteoarthritis



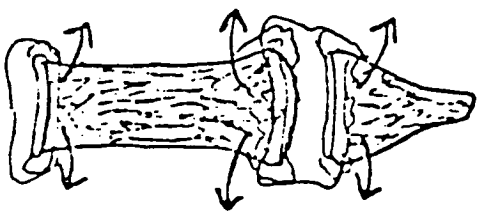
Normal Bone

Balanced Activity: bone formation (osteoblastic) vs. bone absorption (osteoblastic).



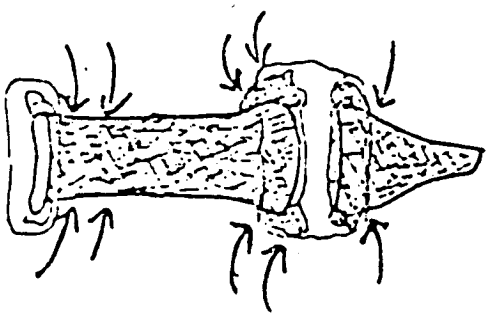
Stage I

Adaptive Demineralization: minimum of 5 to 10 years; slow absorption; mild pains.



Stage II

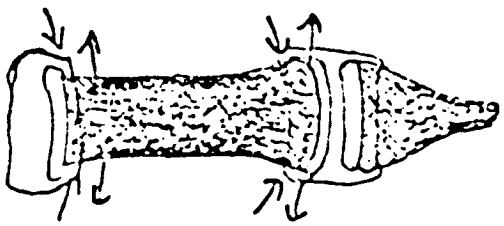
Inflammatory Bone Growth: growth of soft bony "spurs" or exostoses; widening of bone, soft texture plus inflammation; mild or severe pain.



Therapy

Remineralization: mineralization of total bone, including the "spurs;" firm texture; decrease in inflammation and pain.

Rheumatoid Arthritis



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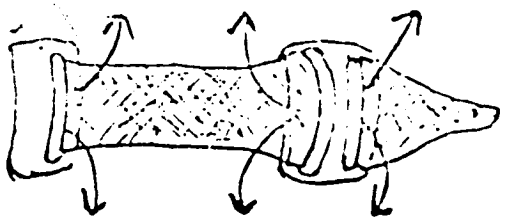
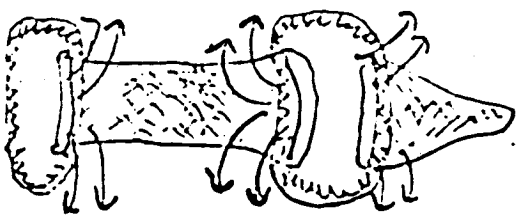
Balanced Activity: bone formation (osteoblastic) vs. bone absorption (osteoblastic).



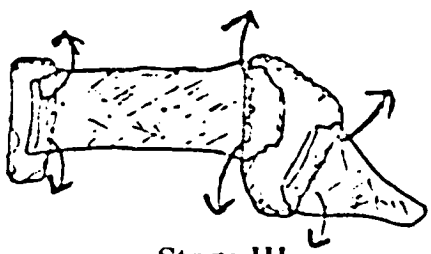
Stage I

Adaptive Demineralization: minimum 5 years; slow demineralization; migratory pains.



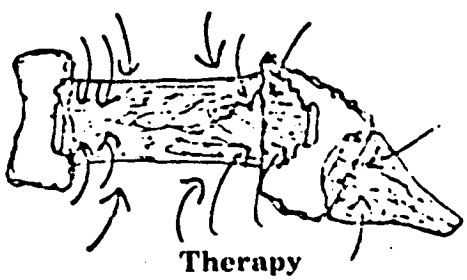
Stage II

Acute Inflammation: hyperaemic synovia; rapid demineralization; severe pain.



Stage III

Chronic: deformity; progressive demineralization; pain.



Therapy

Remineralization: mineralization of bone, decrease in inflammation; decrease in pain.

Megavitamin and mineral treatment of arthritis

by Carl J. Reich, M.D., F.R.C.P.(C)

PART II

OTHER CLINICAL ASSESSMENT of the osteo and rheumatoid arthritis patient is that directed toward discerning the existence of the chronic A and D mineral deficiency state and defining the physical signs and functional complaints indicative of the effect of this deficiency state on other tissues of the body.

The responsible deficiencies

The A and D deficiency state is dependent on a deficient dietary intake of calcium, phosphorus, and vitamin D responsible for the absorbtion and utilization of ionized calcium. Such a diet is usually deficient in milk and butter fat. A daily optimum amount of milk for an adult is 2 to 3 glasses of whole milk; an optimum amount of butter fat is that included in the whole milk and in the additional butter used on bread, vegetables, in cooking, etc.

Since the trend has been to replace butter with margarine, and to replace whole milk with 2% or skim milk, or to replace milk entirely with other beverages such as fruit juices, I have adopted the custom of describing disease arising out of this deficiency as the "2% milk and margarine disease."

Ultra-violet radiation of unfiltered sunshine on untanned skin creates activated seven-dehydrocholesterol, or natural vitamin D-3, in the skin. Deficiency of this possibly very efficient D vitamin frequently is an additional factor for the malabsorbtion of dietary minerals and hence for adaptive demineralization of the skeleton.

The deficiency syndrome

Other clinical signs, which exist for reason of the effect that the same deficiency state has on other tissues, are those which have been described as the "A and D vitamin deficiency maladaptive state."

These clinical findings may also be frequently found existing alone; that is, not associated with overt disease, but only associated with the responsible dietary deficiency state. In these instances, this syndrome is taken or indicative of the disease prone state for the maladaptive disease. When found associated with diseases such as asthma or arthritis, these diseases represent complications of the maladaptive syndrome. The tissues most frequently involved in the vitamin A and D plus mineral deficiency state are the tongue, fingernails, skin, skeletal muscles, and intestinal smooth muscle.

Physical findings. The physical indications of deficiency are:

Tongue. Degrees of coating.

Fingernails. Degrees of layering, longitudinal lining, longitudinal splitting and breaking; loss of the undernail red flesh color by a degree of opaqueness of the nail.

Skeletal muscles. Increased irritability as indicated by (1) increased contraction of the muscle of the thighs, arms, and of the anterior and posterior chest on percussion; (2) the occurrence at the site of percussion of a tonic nodule of contracted muscle known as "myoedema;" (3) increased pain on moderate palpation of the trapezius and soleus muscles.

Tendon reflexes. Increase in the tendon reflex response of the patella and biceps tendons.

Skin. Degrees of dryness of the skin; hyperkeratosis of the upper arm.

Functional complaints. Functional criteria of the deficiency syndrome are:

Fatigue. General and chronic; lack of stamina; intolerance to cold.

Central nervous system. Headaches, dizziness, and anxiety.

Gastrointestinal system. Gas, bloating, constipation, alternating constipation and diarrhea.

Skeletal muscles. Aching, spontaneous cramping, usually of the calves of the leg at night.

Skin. Dryness.
Nose and throat. Dryness or congestion.

The above-mentioned criteria are very rarely encountered in their entirety in a single patient. More frequently only a fraction of the most important yet still diagnostic physical signs of the syndrome may be elicited in the deficient patient. For this reason, a large number of deficient patients should be studied in this fashion until the examiner becomes well aquainted with the complete picture of the deficiency syndrome and with partial or incomplete presentations of the syndrome.

The physical criteria of the syndrome are more constant than the functional criteria; indeed, the latter may be entirely absent in some cases. I believe that the reason for this disparity lies in the adaptive potential of the individual, which can maintain correct function of tissues and of organs in the face of chronic deficiency.

OSTEOARTHRITIS

Contrary to current concepts, which consider this disease to be present only when increasing pain and swelling lead to crippling and to x-ray changes within a relatively brief period of time, it occurs in two stages: a long, relatively quiescent period of demineralization followed by a stage of increasing pain, swelling, etc.

Stage I: quiescent demineralization

Swelling and pain usually are absent but may be transient and diagnosed as "rheumatism." While x-rays of the joints are negative, they frequently reveal that some osteoporosis of the entire bone due to demineralization has taken place.

Stage II: obvious disease

There is increasing pain, swelling, stiffness, increasing deformity, and x-ray changes. Thinning of the cartilage often occurs and, with the opposition of heaped up bone growth, may give rise to rigidity.

The x-ray changes are of irregularity of bone at the edge of cartilage surfaces or definite "spurs" or osteophyte formation in this area. This laying down of additional bone likely occurs because demineralized bone acts either as an irritant to the covering periosteum of bone at the periosteal cartilage junction; or the demineralized, weakened bone is irritated by the normal stress of weight bearing or by pressure caused when the joint is used, such as in the fingers.

Because of the x-ray evidence of demineralization, we may suspect that the total weight of minerals within the bone may be 10% below normal, even though these growths of bone may increase the volume of the bone by 1 to 3%.

Therapy

Therapy is directed toward remineralization of the bone, including the inflammatory exostoses, with A and D vitamins and minerals and toward reduction of the inflammatory raft tissue swelling. Once accomplished, better weight bearing and usage is possible.

While function in early cases may be greatly restored even though x-ray changes are not altered, little more than some decrease in pain can be expected in advanced cases.

RHEUMATOID ARTHRITIS

The evolution of this disease occurs in three stages: Asymptomatic demineralization, followed by acute arthritis, and finally deformity.

Current concepts, however, consider this disease to occur in two stages, at first heralded by the acute onset of arthritis and later characterized by crippling, etc.; demineralization is considered a result of the disease process, which has onset because of stress or infection, etc.

Stage I: quiescent demineralization

During a long, quiescent period of years or decades there is a gradual demineralization of the skeleton, particularly of the more vascular ends of the bones. This is due to an imbalance between the deposition and removal of minerals from the bone required in the mineral adaptive process for use elsewhere.

Later there may be fleeting or migratory mild to moderate pain; transient joint swelling

may occur, but does not deserve the nomination of "arthritis."

Stage II: active arthritis

Acute onset of pain and swelling, often in several joints, is due to rapid acceleration of the adaptive demineralization process. Demineralization results from the added demand for minerals created by some stress such as the healing of major surgery, psychic influences, etc. which result in loss of rest, hyperventilation, etc. This demand on bone minerals may also be enhanced by anorexia leading to diminished intake and by losses incurred through vomiting or diarrhea.

Nervous and muscle tissue have a higher priority than other tissues for bone minerals such as calcium because of increased vascularity and swelling of the synovia, tissue which lines the joint exclusive of the area covered by cartilage.

Because of the acute pain and swelling of joints, the disease has sometimes been referred to as "inflammatory arthritis," in contrast to the more quiescent "degenerative arthritis" of old age or "presenile osteoarthritis."

Current concepts diagnose rheumatoid arthritis only at this stage of acute inflammation. X-rays usually are taken within several days or weeks of the acute onset of pain and swelling. These are invariably negative, except for occasional effusion, but occasionally will show that the bones reveal some degree of osteoporosis (i.e., they have been demineralized). This aspect of the x-ray report has been consistently ignored, and demineralization has not been considered a result of the arthritic process.

Pain is usually excessive in this stage, and the inflammatory reaction in and about the joint usually results in a raised sedimentation rate and rheumatoid factor concentration of the blood.

Stage III: joint destruction

Because of relentless adaptive demineralization, part of the bone structure of the joint has been totally absorbed; deformity, such as the characteristic deviation of the fingers, occurs. Partial or full fixation of the joint may eventually occur because of the proximity of one bone surface to another.

X-rays usually will indicate severe demineralization. This demineralization currently is considered to result from the arthritic process and the inactivity arising from it; it is not considered to be of prime importance in *causing* the disease.

COMBINED RHEUMATOID AND OSTEOARTHRITIS

In some rare instances, the history of chronic and slow progressing osteoarthritis is obviously altered or complicated by the acute onset of typical rheumatoid arthritis, with its resulting additional characteristic deformities.

In these instances the direct adaptive maneuver of progressive slow absorption of minerals of bone has been complicated by the inflammatory reaction of synovia, or joint linings, indirectly excited via the autonomic nervous system to hasten the arthritic process.

RESULTS OF CASES TREATED

To date I have given this A and D megavitamin therapy to 1500 to 2000 osteoarthritic patients and possibly to 300 to 400 rheumatoid arthritic patients. No signs of toxicity to the vitamins has been noted.

In 60-70% of early and moderately advanced cases of both forms of arthritis, a gradual diminution of pain and swelling is apparent in the first 4 to 6 weeks of therapy, providing at least moderate reduction of pain and restoration of function. In approximately 50% of these cases, continued improvement ultimately provided an excellent degree of restoration and relief from pain.

In advanced cases of both forms of arthritis, the most frequently experienced result is only mild to moderate relief of pain.

Megavitamin and mineral treatment of arthritis

by Carl J. Reich, M.D., F.R.C.P.(C)

PART III

BIOCHEMICAL MONITORING

Estimations of serum calcium and alkaline phosphatase were performed on most children and young adults. In my experience with many thousands of chronic asthmatics on similar dosages of these vitamins, biochemical changes were not noted in adults. Therefore, biochemical monitoring was not performed on adults but was continued in children. To date ony moderate elevation of alkaline phosphatase has been noted rarely. This was not at high enough levels, and was not associated with an elevation of serum calcium, to warrant the discontinuance of therapy.

In the large groups of chronic asthmatics previously mentioned, some rare unexplained findings of raised alkaline phosphatase were noted prior to therapy. I suspect that these may be due to a hypersensitivity to synthetic vitamin D-2, or "Ergo Calciferol," which currently is used to fortify milk and other foods.

No clinical signs of toxicity to these dosages of the A and D vitamins — such as pseudo tumour-cerebri, papilloedema, chronic nephritis, severe dermatitis, exostosis, or osteoporosis — has been noted. Minor intolerances such as gastritis, headaches, milk transient skin rashes, etc. were noted only occasionally. These reactions were all immediately relieved once therapy was discontinued and resumed at a reduced dosage. For this reason, one can hardly define these reactions as toxicity.

PREPARATIONS USED

The following preparations were used in therapy:

1. *Aquasol A & D.* A solution of semi synthetic vitamin D-3 and natural vitamin A from fish oils, in a water soluble glycol; contains 40,000 IU of vitamin A and 8,000 IU of vitamin D per cc.
2. *Halibut liver oil capsules.* Each capsule contains 5,000 IU of natural vitamin A present in fish oil; approximately 100 IU of natural vitamin D-3 ("Chole Calciferol") present in fish oil; and approximately 300 IU of synthetic vitamin D-2 ("Ergo Calciferol").
3. *Bone meal tablets.* Usually a ½ g. tablet; contains approximately 2/3 calcium and 1/3 phosphorus.

VITAMIN A & D TOXICITY

I think it was Linus Pauling who stated that the minimum daily requirement (MDR) calculated by health authorities for vitamin C — 3 mg in Germany and 50 mg in the United States — arose from their estimates of the vitamin content of the respective national diets. As Pauling and others appreciate, these MDR values reflect severe vitamin deficiency in these national diets.

Vitamin A ("Palmitate") was synthesized in the 1930's and vitamin D-2 ("Calciferol") was synthesized several years later. Subsequently, toxicity was observed when investigators gave 250,000 IU or more to aging and ill patients. Possibly for this reason, the MDR of vitamin D was fixed at 400 IU and of vitamin A at 5,000-10,000 IU. I believe these values represent severe deficiency and instead the daily intake of these two vitamins should be 3 to 6 times these amounts.

Average dosage of A and D vitamins in the therapy of chronic asthma and arthritis.

	Aquasol A & D	Halibut Liver Oil	Total	Bone Meal
Child, 3 years	*2-3 drops, 2-3 times/day*			*½ tablet, 2 times*
Vitamin A dosage	5,000-12,000 IU		5,000-12,000	½ g
Vitamin D dosage	1,000-2,400 IU		1,000-2,400	
Child, 15 years	*5 drops, 3 times/day*	*1 capsule, 2 times/day*		*1 tablet, 2 times*
Vitamin A dosage	20,000 IU	10,000 IU	30,000 IU	1 g
Vitamin D dosage	4,000 IU	800 IU	4,800 IU	
Adult, 150-175 lb.	*6 drops, 3 times/day*	*2 capsules, 3 times/day*		*1 tablet, 3 times*
Vitamin A dosage	24,000 IU	30,000 IU	54,000 IU	1½ g
Vitamin D dosage	4,800 IU	2,400 IU	7,200 IU	

Depending on response to therapy, these dosages are maintained for several weeks or months; however, in most cases, they will be reduced to a half or a third of these amounts within that period.

I have given 10 times the MDR of these vitamins — up to 6,000 IU of vitamin D and 60,000 IU of vitamin A — to many thousands of patients for periods of 3 to 6 months without the development of toxicity and phosphatase, raised phosphorus, or raised calcium. Toxicity to vitamin D is calcinosis of the kidneys, producing changes equivalent to a nephritis. Vitamin A toxicity may cause exostoses of bone or may be neurotoxic, causing papilloedema and ataxia. But the spectacular feature about toxicity to these vitamins is that the pathological features induced are not irreversible; they begin to resolve within one week after discontinuance of the overdosage.

DISCUSSION

Innumerable clinical observations have been made over the past 25 years on the presence of chronic dietary and other deficiencies, on clinical findings resulting from and associated with chronic deficiency, and on the response to therapy of overt disease such as chronic asthma. These observations force the conclusion that the onset of "inflammatory rheumatism" arises from adaptive demineralization of the skeleton, in compensation for dietary mineral deficiency, effected by autonomic stimulation of synovia.

The immune mechanism may be involved in such reactions within the synovia in rheumatoid arthritis. I suspect that these mechanisms have been derranged by the same set of biochemical disturbances which first excited the bynovia. Moreover, like the autonomic nervous system, the immune system may be only secondarily involved in the genesis of this disease; chronic dietary deficiency likely is the prime causative factor. Therefore, if the immune mechanism is to be considered in this disease, it should be studied in reference to its occurrence in (1) the non-deficient individual, (2) the deficient non-diseased individual, and (3) the deficient diseased individual. If not evaluated in this context, its importance in the causation of rheumatoid arthritis will continue to be misinterpreted.

CONCLUSION

Bone is not a static mineralized tissue like the biting edge of a tooth; it is subject to constant absorption and laying down of calcium salts, particularly in the joint areas. This ebb and flow of minerals is part of the repairative process; but it is also required in the mineral adaptive function of the skeleton. If these adaptive processes are balanced, the density of bone will be preserved. If, however, after many periods of demand, the redeposition process is less than the absorption process, then gradual demineralization will result and give rise to one of the major forms of arthritis. Rarely, these diseases will occur in combination.

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ORTHOMOLECULAR* ADAPTIVE MEDICINE

Carl J. Reich, M.D.
Fellow, Royal College of Physicians (Canada)

*treating disease with natural substances; preventing disease with optimum diet.

Some years ago Linus Pauling suggested in an article entitled "The New Medicine" that the application of orthomolecular medicine was so radically different from orthodox medicine as to constitute a new approach to health care.

Dr. Reich agrees wholeheartedly with this view. His more than 20 years in the field has given him insight into the adaptive function of the body in health; the derangement of these adaptive functions by chronic deficiency to create disease; and the correction of this maladaption and disease by megavitamin therapy. Dr. Reich suggest that this "new medicine" is actually "orthomolecular-adaptive medicine."

About 20 years ago Hans Selye popularized the concept of stress and adaption to stress as a disease-causing agent. But he did not believe that chronic dietary deficiency could create one of the most influential biochemical stresses of the body. Nor did he correctly integrate chronic dietary deficiency with the adaptive processes, which suggests that vital adaptive functions of the body can be severly altered by chronic dietary deficiency to the degree that they break down and become ineffective.

Selye proposed that an animal adequately stressed by physical, chemical, or emotional factors (whether alone or in combination) will develop diseases such as intestinal inflammation, spasm, ulceration, arthritis, hypertension, heart disease, etc. as its ability to cope or adapt to this persistent stress is exhausted. I believe that the optimally nourished and rested animal may endure these stresses almost indefinitely until old age; if adaptive diseases occur, they indicate not so much the exhaustion of normal adaptive mechanisms as the breakdown of compensatory defense mechanisms which were deranged because of chronic deficiency.

MALADAPTION

The A & D vitamin and mineral therapy was first given successfully to a chronic asthmatic in 1954. Following a five-year study of the treatment of many chronic asthmatics with this therapy, I arrived at some new definitions: "maladaption" refers to altered adaptive mechanisms of the body; "maladaptive diseases" are those caused by derangement of these mechanisms.

Clinical observations have convinced me that vitamins A and D and minerals, particularly calcium, are intimately concerned with the

STIMULATION OF
function of the autonomic nervous system to excite or retard physiological processes of separate organs or tissues. These alterations in function are designed to compensate for the biochemical changes inflicted by exertion, temperature changes, food deprivation, chronic diet deficiencies, etc.

Ionic or dissolved calcium is one of the most important requirements for the proper function of the nervous and muscle elements of the autonomic system, and hence of the adaptive functions of many organs of the body.

Taken in the form of chemical compounds such as calcium carbonate or calcium phosphate, calcium is ionized through digestion and the action of vitamin D. In this free ionic state it is absorbed from the intestine and made available for the complex cellular reactions of the body.

When the intake of calcium (highest concentrations in milk) or vitamin D (highest concentrations in butterfat and fish oils) is inadequate, nerve and muscle function as well as the secretory potential of almost every internal and external gland, may be seriously altered. These calcium dependent functions are VERY important to the body because calcium, in its free ionic form within the cell, normalizes cellular function. LESS IMPORTANTLY, it gives rigidity to the skeleton as part of a solid compound such as calcium phosphate.

This ionic calcium deficiency of milk and butterfats, and the skin's lack of exposure to the sun, is responsible for the derangement of adaptive functions of the body. An example is the quiescent physiological bronchial contriction which alters the proportion of exhaled gases and also the concentrations of these gases in the blood. Cellular ionic calcium deficiency creates excessive contraction (or spasm) of the billions of contractile molecules of smooth muscle within the bronchial tree. This deficiency produces pathological spasm, creating asthma.

SPASTIC CONDUIT DISEASES

The distortion of a purposeful physiological constriction providing adaption to pathological
INTO
spasm and generating disease are operative in 15
various sections of the intestinal tract; these are WHICH MAY
directed into different physiological maneuvers BE
to compensate for the biochemical errors of the body created by decades of chronic dietary deficiency of minerals and the A and D vitamins. Gastric spasm, hypersecretion, and ulceration, as well as similar changes of other sections of the intestinal tract, MAY represent the breakdown of functions that originally were adaptive or protective. Therefore, just as chronic asthma is largely the end product of a "defense mechanism gone wrong," duodenal ulcerations, ulcerative colitis, and constipation MAY
are BE different forms of "asthma of the intestinal tract." These diseases of muscular conduits are created by deficiency of the contractile macromolecules of muscle and autonomic nervous system stimulation on the same muscle responsible for the initial adaptive constriction.

Diseases of the respiratory and intestinal conduits have not been interrelated, and related with other diseases, in this fashion by mere wishful thinking! For 4 years before I treated the first chronic asthmatic with A and D vitamins, I made many clinical observations on autonomic dysfunction in hundreds of patients that lead me to devise this therapy, and since then I have treated over 10,000 chronic asthmatics. I have also made clinical observations on chronic dietary deficiency and the functional and physical alterations resulting from it in an equal number of non-asthmatic patients. These clinical findings are absent in a very large number of non-deficient patients.

One result of this study was to describe as "spastic conduit diseases" those which are caused by muscle spasm in organs such as the intestinal tract or the arterial system (essentially muscle-lined tubes or conduits) and the lung or heart (which contain highly responsible muscle-lined conduits).

BIOCHEMICAL MECHANISMS

I cannot define the physiological and biochemical mechanisms by which chronic asthma, colitis, hypertension, or coronary spasm, for instance, may compensate for the influences

which chronic dietary deficiency may have on the body. Consider, however, that the restriction of pulmonary ventilation, or the restriction or speeding up of the movement of intestinal contents through the intestinal tract, or the continued kinetic energy exerted on the billions of particles which constitute the blood may induce alterations of the biochemistry of the blood. Then also these organs may be used in defense mechanisms against the imposition of biochemical alterations of the diet, etc.

ADAPTIVE ARTHRITIS

The many clinical interrelations in the different patients can be explained only by the mechanism of adaption and maladaption.

The same clinical data found in patients with one of the "spastic conduit" diseases have been observed in the pre-senile osteoarthritic and rheumatoid arthritic patients. A large percentage of these patients displayed dietary mineral deficiency, spontaneous painful leg cramps at night, layering and splitting fingernails, increased tendon reflexes, pain in various muscles on pressure, etc. The adaptive maneuver to chronic dietary mineral deficiency was concluded to be demineralization of the skeleton which gave rise to these arthritic diseases!

Hence, I believe that a process of gradual asymptomatic demineralization of the skeleton precedes the first pain or joint swelling of arthritis for many years, or even decades! The x-ray report of bone demineralization associated with even the earliest signs of joint changes frequently constitutes irrefutable evidence that such a process has preceded the onset of arthritis for some period of time.

X-ray evidence of pre-senile osteoarthritis, therefore, is the laying down of soft decalcified bone in areas of the joint covered not by cartilage but by joint lining, or synovia. These areas of bone are irritated to lay down the soft bone of "spurs," or exostoses, because such demineralization constitutes an irritant. This synovial covered bone is irritated to such osteoblastic activity either because the demineralized bone it covers cannot tolerate the normal stresses of weight bearing or other joint pressure, or because demineralized bone alone serves as an irritant.

In osteoarthritis I suspect the bone has become demineralized by a gradual leaching out process, a "direct adaptive mechanism." In rheumatoid arthritis, however, I feel that the demineralization process is so greatly heightened — either through increased vascularity, providing increased circulation of the synovia, or through nervous or hormonal autonomic activity — that far greater and more rapid absorption of the bone may occur. Such an absorption may frequently cause total collapse of the joint, providing the characteristic destructive joint changes in this disease.

The relationship of chronic dietary deficiency to an array of seemingly unrelated physical and functional findings, to particular diseases of muscle-lined conduits, and to the arthritic diseases of bone — and the interrelationship of these diseases — have me convinced that calcium has higher priority when present in free form in the body cells than when present in the stable compounds of the skeleton. This ionic calcium is particularly important in cellular activity of nerve, muscle, and secretory function responsible for the normal progress of adaptive functions of the body, particularly in organs containing smooth or involuntary muscle.

Deficiency of such ionic-free calcium, largely created by chronic vitamin D deficiency, may therefore give rise to a host of diseases currently attributed to other factors such as allergy, infection, psychological stress, or just plain "bad luck."

The application of concepts embodied in orthomolecular medicine — or rather, "orthomolecular adaptive medicine" — offers much more in the prevention and alleviation of disease than is currently afforded by orthodox concepts of therapy with drugs which pay little or no attention to nutrition.

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