Vitamin F and Carbamide in Calcium Metabolism

BY ROYAL LEE, D.D.S.

CARBAMIDE, otherwise known as urea, is a physiological constituent of all body fluids which has heretofore been mistakenly considered as a waste product. Its presence in urine in considerable amounts has been responsible for this conclusion. Sodium chloride, too, is a constant constituent of urine, and is no more a waste product than carbamide. Both are necessary blood and lymph salts, but carbamide is made by the cells for use as an osmotic regulator, and either carbamide or sodium chloride must be available to promote water output in kidneys or sweat glands where an aqueous solution of waste products is to be thrown off by osmosis. A deficiency of salt in hot weather is a serious matter for the body, but a deficiency of carbamide has, heretofore, not been recognized as possible or probable. I believe the evidence is clear to show that such may be and often is the case.

Carbamide is made only in the liver, and is a product of the splitting of arginine by arginase, to yield ornithine and carbamide. Arginine, of course, is available from protein digestion; it is one of the commonest amino acids. Arginase is present in male liver in higher amounts than in female, according to Tauber, and in highest concentration after sexual maturity.[1]

Carbamide denatures proteins, according to various investigators. That means that it alters the affinity of the protein molecule for any mineral salts which may be conjugated with it, and we know that the calcium in the blood which is destined for bone or tooth use is carried as a nondiffusible molecule of calcium phosphate attached to a protein carrier.

Vitamin F promotes the release of calcium bicarbonate in the blood, and its consequent diffusion into the tissues. How? Apparently in this way: Vitamin F is known to promote the action of the sex hormones. The extra activity of the male hormone may cause the increased promotion of arginase activity, with consequent increased blood carbamide. The carbamide denatures the protein-calcium complex, releasing into the blood free ions of calcium phosphate, which react with sodium bicarbonate always present, to afford calcium bicarbonate and sodium phosphate. It is probably this same protein-calcium complex which is built up in the blood stream by the action of vitamin D. It is obvious that without the aid of vitamin F this calcium cannot be unloaded after it reaches its destination.

The rise in blood urea also would account for the great relief of sunstroke victims experienced from the use of vtiamin F, for their distress is known to be due to two reactions: One, a stoppage of perspiration which we can now see is a consequence of depletion of both salt and carbamide, without which no sweat can be osmotically secreted; second, a high blood calcium from too much vitamin D which acts as a tissue poison until brought under control. We must recall that the only way excess vitamin D can cause harm is by its boosting of the blood calcium levels.

It also explains why older persons, especially women, can tolerate less sunshine than younger persons.

Chemically, carbamide is carbon dioxide combined with ammonia. As such, it must be considered as a buffer salt, for while neutral in itself, it can release ammonia for neutralizing acids if urease is present, an enzyme that catalyzes this reaction. Now, signficantly, urease is found in the stomach mucosa. As such it should be expected to act as a protection against the tendency of the gastric acid to irritate the stomach wall, by locally providing ammonia to neutralize acid. Suppose the carbamide levels were lowered by reason of sex hormone deficiency? The natural reaction would very possibly be a tendency to gastritis and ulcers, for peptic ulcer is definitely a result of uncontrolled gastric acidity. It is not strange that one important clinical use which has developed for vitamin F is in gastritis, now that we see this connection.

Vitamin F has never been actually isolated and identified. It is an associated factor found in certain unsaturated fatty acids. Its highest potency is found in arachidonic acid of kidney fat, or the fat of other mammalian glandular organs. Some fish oils are 40% arachidonic acid, but they fail to have a vitamin F potency in proportion. It is probably the synergistic association of vitamin F in fish oils that affords the better effect of vitamin D from that source, and it is probably the high sex hormone content of the blood in pregnancy which by its stimulation of carbamide

production prevents vitamin D from raising the blood calcium to toxic levels, it being an established fact that in pregnancy, the female is immune to vitamin D toxicosis.

Do not forget that both sexes make use of both the female and male hormones, the difference is in the proportions. One of the best commercial sources of estrogen is stallion urine. It is probable that an increased intake of vitamin F promotes a normal balance between these two hormones. It is a common clinical result of vitamin F administration for a woman patient to report a falling off of a masculine hair growth. The effect seems to be a disintegration of the hair shaft at the skin surface. It seems to indicate that the female grows hair, but the shaft cells are friable. Otherwise, how explain this sudden dropping off of hair? Sometimes children and even adults report handfuls of hair dropping off on the pillow when arising. That is a disorder of the endocrine system that usually responds to a multiple vitamin treatment. Often it is ascribed to a spirochetal infection, but many cases are seen where this explanation is not probable, and which promptly responds to vitamins.

One of the first indications of vitamin F deficiency is nephritis. If carbamide is necessary to osmotic discharge of toxic substances in solution (along with sodium chloride), and its production is dependent upon vitamin F, it is apparent that serious embarrassment of the kidney will inevitably result from F deficiency. It is significant that in sunstroke, not only the sweat glands but also the kidney function is paralyzed more or less. This shows, I believe, that there is a severe deficiency of carbamide, and a vicious circle of symptoms is present which is broken by the vitamin F. The use of carbamide along with salt as a hot weather aid to the osmosis of perspiration would seem desirable.

A mixture of salt and carbamide, equal parts, added to drinking water in small proportions seems to add thirst-quenching properties which the plain water lacks. About a quarter level teaspoonful is ample, to the glass of water.

In allergic conditions a deficiency of calcium is usually considered a contributory factor, and the tissue fluid content of diffusible calcium is usually demonstrably low. Clinically, the use of the vitamin G complex is the most successful of the schedules we have seen tested. A clue is seen here, in that pyridoxine deficiency is known to cause tetanic convulsions, and pyridoxine, plus an unknown B-factor, has been found interchangeable with vitamin F in the treatment of controlled F deficiency in animals.[2]

I consider allergies a result of a combination of circumstances which includes a deficiency of vitamin G complex, a lowered level of blood carbamide (common after middle age) and reduced pancreatic activity in which proteins are incompletely digested and antigenic fractions can get into the blood stream and produce some specific sensitization. I think carbamide is a vital link in this chain, as where it is deficient there is a great enhancement of the tendency to sensitization. That is because carbamide is a mild denaturant in itself of proteins, and probably acts as a first line of defense against sensitizing agents by destroying their antigenic power. Since the carbamide level is secondary to the sex hormone level, the beginning of allergy trouble at middle age is obviously natural. But do not forget that nutritional deficiencies of vitamins A, E, and F, hasten this lowered hormone level, and also, the deficiency of the G complex and calcium greatly enhance the development of symptoms.

Most patients who need calcium do not respond to its administration in the usual forms. That is no doubt because their condition is due more to their inability to assimilate the element than to its actual absence in the food. It is known that an alkaline intestine will prevent the assimilation of calcium.[3]

A deficiency of hydrochloric acid in the gastric secretions will definitely inhibit calcium assimilation. Probably one of the benefits of lactobacillus acidophilus, or lactic acid yeast, is the reduction of intestinal alkalinity and the promotion thereby of calcium absorption.

High phosphorus diets, which may be created by an excess of cereal foods, are stated by the same authority to exert an unfavorable effect on calcium absorption. And calcium phosphate, commonly sold for nutritional purposes, is totally useless nutritionally, according to investigators of the Ohio Experiment Station, reported in Bulletin 347, at least for promoting bone growth in pigs and cows.

The calcium of plant cells and cereal germ is in the form of phytate, an organic combination which carries inositol as well, a factor of the vitamin B complex. This calcium is soluble in acids, and is no doubt normally dissolved by the gastric juice. It can be also rendered soluble with citric acid, so that its assimilation is not dependent upon the presence of gastric juice, and then can be taken without other foods. For calcium is best assimilated on an empty stomach, it tends to combine with foods, especially fatty foods, and remain in the intestinal tract if foods are present. So it appears that we have a complicated situation with respect to the assimilation of calcium, and it is easy to see how a patient can develop a condition of acute deficiency in spite of the universal presence in foods of at least some form of calcium.

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