# Editorial

## *Human Pantothenic Acid Deficiency*

Pantothenic acid deficiency has been experimentally induced in man by Bean and Hodges.[1] They administered a diet deficient in this factor together with a metabolic antagonist (omega-methyl pantothenic acid) to four volunteers.

A number of interesting clinical and laboratory changes were noted. Among these were postural hypotension, fatigability, and, later, epigastric distress, numbness and paresthesias of the hands and feet, hyperreflexia, and emotional changes. Despite a dietary intake of more than 1000 calories and 100 Gm. protein daily, weight loss was progressive. An increased susceptibility to respiratory infections was noted. Impaired acetylation and impaired adrenal function, gastric hypochlorhydria, and a fall in blood cholesterol levels were also found.

Pantothenic acid alone was then added and although the paresthesias improved promptly, the urinary 17-ketosteroid excretion, already depressed, decreased further. It became clearly evident that the clinical manifestations of the deficiency were progressing; in fact, rather alarming symptoms developed which were suggestive of adrenal insufficiency, and cortisone treatment was necessary. A rapid and complete recovery in clinical and laboratory spheres followed administration of a *general diet* supplemented with oral and parenteral vitamins.

As the authors note, spontaneous development of such a deficiency syndrome is highly unlikely because of the wide distribution of the vitamin in foods and, unlike pyridoxine, because of its resistance to destruction by heat or chemicals. Nevertheless, this is an important subject because pantothenic acid is a portion of that remarkable and crucial substance, coenzyme A. Some of the signs and symptoms and biochemical alterations in this syndrome are undoubtedly due to impaired acetylation —a metabolic process dependent on an intact coenzyme A mechanism.

It is possible that the metabolic antagonist may have had some toxic properties of its own,[2] because a favorable response was not induced by pantothenic acid alone. It is equally possible that the deficiency was present for so long that progressive bodily changes were developing which could not be simply reversed by pantothenic acid supplementation. It is perhaps more likely that the diet plus antagonist produced a deficiency of more than the single vitamin (perhaps an as yet unknown factor), so that it took a general mixed diet in addition to vitamin B complex supplementation to bring about a cure.

These observations are of considerable interest and suggest that investigators and clinicians will be hearing much more about pantothenic acid.

—S. O. Waife, M.D.

REFERENCES


1. Bean, W. B., and Hodges, R. E.: Pantothenic acid deficiency induced in human subjects. *Proc. Soc. Exper. Biol. & Med.* 86: 693, 1954.
2. Induced pantothenic acid deficiency in man. *Nutrition Rev.* 13: 36, 1955.