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Riboflavin Absorption in Pernicious Anemia.

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Current opinion concerning the cause of the subacute combined cord degeneration in pernicious anemia favors the concept that the neural changes arise from a deficiency of some unidentified factor other than that required for hematopoiesis. Since achlorhydria invariably is present in pernicious anemia, the possibility must be considered that this condition might result in a decreased absorption of certain vitamins. That an avitaminosis might account for the neurological changes has been suggested by a number of observers among them being Field and his collaborators,[1, 2] Gildea, Kattwinkel and Castle,[3] Suh and Merritt,[4] and others.

Street, Cowgill and Zimmerman[5] observed myelin degeneration of the peripheral nerves and posterior columns of the spinal cord in dogs fed a diet adequate except for riboflavin. Their observations suggested the possibility that this component of the B complex may play a rôle in the etiology of subacute combined degeneration of the spinal column. In order to determine if pernicious anemia patients have a diminished ability to absorb this vitamin, its daily urinary excretion by such subjects was estimated.

Experimental. The experiment was conducted in a manner similar to that recently reported for pantothenic acid.[6] Twenty-four hour urine specimens from 12 patients and 12 healthy students and laboratory workers, who served as control subjects, were collected and preserved with benzene and 10 ml of acetic acid. The patients obtained hospital meals, while the latter ate unrestricted diets likewise considered to be adequate. The method of Snell and Strong[7] was used for the determination of the urinary riboflavin.

The values observed are recorded in Table I. Due to the unre-

1 Field, H., Jr., Robinson, W. D., and Melnick, D., *Ann. Int. Med.*, 1940, **14**, 588.

2 Robinson, W. D., Melnick, D., and Field, H., Jr., *J. Clin. Invest.*, 1940, **19**, 399.

3 Gildea, E. F., Kattwinkel, E. E., and Castle, W. B., *New England J. Med.*, 1930, **202**, 523.

4 Suh, T., and Merritt, H. H., *Am. J. M. Sc.*, 1938, **196**, 57.

5 Street, H. R., Cowgill, G. R., and Zimmerman, H. M., *J. Nutrition*, 1941, **22**, 7.

6 Meyer, C. E., Burton, I. F., and Sturgis, C. C., PROC. SOC. EXP. BIOL. AND MED., 1942, **49**, 363.

7 Snell, E. E., and Strong, F. M., *Ind. and Eng. Chem., Anal. Ed.*, 1939, **11**, 346.

TABLE I.
Riboflavin Excretion.*

Pernicious anemia patients			Controls		
No.	Daily excretion, μg	After 5 mg dose,† μg	No.	Daily excretion, μg	After 5 mg dose,† μg
1	1070	3830	1	483	3360
2	15	2760	2	770	4750
3	250	1070‡	3	1200	2770
4	980	—	4	743	3080
5	825	2546	5	810	2180
6	980	4580	6	846	3180
7	—	4890	7	244	—
8	—	4410	8	312	4590
9	448	—	9	525	—
10	650	3530	10	615	2790
11	905	1600	11	1000	1890
12	716	1444	12	305	1700

*μg per 24 hours.
†5 mg riboflavin, Merck, given orally at start of 24-hour period.
‡An outpatient not available for further study.

stricted nature of the diets, of which the riboflavin contents were not known, the results cannot be interpreted in the nature of a comparative balance study. Nevertheless, they do serve to indicate that achlorhydria apparently does not impair the absorption of riboflavin.

More comparable data for the 2 groups of subjects were obtained by administering 5 mg of riboflavin in addition to that contained in the diets. Following this, the individual quantities excreted, listed in Table I, do not reveal any tendency toward a lesser output by the patients.

These results resemble those observed in a similar study of pantothenic acid excretion.[6] They may be interpreted only as indicating that a lack of hydrochloric acid apparently does not result in an impairment of absorption of the accessory food factors studied. That the degeneration of the central nervous system may result from a conditioned avitaminosis arising from a lessened food consumption over a prolonged period remains a possibility.

Conclusions. In patients with pernicious anemia, the excretion of riboflavin, both before and after oral administration of 5 mg of the vitamin, was of the same order as that eliminated by healthy individuals. There is, thus, no indication of impaired absorption of this compound by such subjects as a result of their lack of hydrochloric acid.