TABLE I.
Excretion of 17-Ketosteroids by Patients Suffering from Poliomyelitis.

No. of patients	Sex	Age range	Type of poliomyelitis	Mg 17-Ks. excreted/24 hr range	avg
10	Male	5-13	Bulbar (4)	0.2-1.8	1.0
			Spinal (4)	0.8-4.8	2.5
			Bulbar-Spinal (2)	0.9-2.0	1.5
1	Female	7	Spinal	2.3	
15	Male	14-39	Bulbar (3)	11.4-36.0	19.0
			Spinal (8)	3.4-27.1	10.3
			Bulbar-Spinal (4)	14.9-22.1	19.0
13	Female	14-35	Bulbar (3)	3.1-7.2	5.4
			Spinal (8)	2.0-21.1	8.0
			Bulbar-Spinal (2)	8.6-14.6	11.6

patients entered at the University of Minnesota Hospital during the 1946 epidemic were studied. Of these, 11 were under age 13, 11 were females over age 14, and 15 were males over age 14. In some cases urine specimens were collected from the same patient on more than one occasion and these were subjected to separate analyses. All specimens had been stored in a frozen condition since the time of collection. The 17-ketosteroid fractions were prepared from 200-400 cc aliquots of 24-hour specimens by a modified Callow technic (Callow, *et al.*[2]) and were colorimetrically assayed by the technique of Holtorff and Koch[3]. The results are shown in Table I.

The values for the 17-ketosteroid excretion of the patients suffering from poliomyelitis for the most part fall within the normal ranges reported by others (cf. Talbot and Butler[4]) and as found in our own laboratory. These data thus tend to indicate that there is no relationship between the condition of poliomyelitis and the excretion levels of 17-ketosteroids. No significant differences were observed for the 17-ketosteroid excretion of patients suffering from different types of poliomyelitis.

Summary. The 17-ketosteroids excreted into the urine by patients suffering from different types of poliomyelitis showed no significant variations from normal excretion levels.

[2] Callow, N. H., Callow, R. K., and Emmens, C. W., *Biochem. J.*, 1938, **32**, 1312.

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Growth Promoting Activity of Vitamin B_{12} in Rats Receiving Thyroid Substance.

GLADYS A. EMERSON. (With the technical assistance of Marilyn A. Bingemann.)
From the Merck Institute for Therapeutic Research, Rahway, N. J.

The retarded growth observed in young rats fed toxic doses of thyroid powder is counteracted by liver.[1,2] Extracted Liver Residues (Wilson) had "anti-thyrotoxic" activity when fed at a level of 10% in a diet containing 22% casein.[3] Reticulogen also was effective in reversing the growth retarding action of thyroid substance in chicks fed a diet devoid of animal protein.[4,5]

[1] Ershoff, B. H., PROC. SOC. EXP. BIOL. AND MED., 1947, **64**, 500.

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TABLE I.
Effect of Vitamin B_{12} on Growth of Male Rats Receiving Thyroid Powder.

Groups of 10 male rats each	Supplement	Gain in wt 15 days, g	Avg daily food intake 15 days, g
H_4 (Casein)	None	65	16
282 (Soybean meal)	,,	49	17
286 (Soybean meal + 0.25% thyroid powder)	,,	28	17
,, ,, ,, ,, ,, ,,	5.8 mg cobalt nitrate	27	—
,, ,, ,, ,, ,, ,,	0.5 γ B_{12} p.o.*	79	17
,, ,, ,, ,, ,, ,,	,, ,, ,, s.c.†	74	—
,, ,, ,, ,, ,, ,,	0.25 ,, ,, p.o.	72	—
,, ,, ,, ,, ,, ,,	,, ,, ,, s.c.	73	—
,, ,, ,, ,, ,, ,,	0.125 ,, ,, p.o.	65	—
,, ,, ,, ,, ,, ,,	,, ,, ,, s.c.	71	—
,, ,, ,, ,, ,, ,,	0.0625 ,, ,, p.o.	51	—
,, ,, ,, ,, ,, ,,	,, ,, ,, s.c.	50	—

* *Per os.*
† Subcutaneously.

Vitamin B_{12}, a crystalline compound which has been isolated from both liver[6] and fermentation sources,[7] neutralizes the growth inhibiting effect observed in immature rats following the feeding of a diet containing soybean meal and thyroid powder. The basal diet (282) employed in these studies had the following composition: soybean meal, 60; salts, 4; dextrose, 24; crisco, 10; cod liver oil, 2 g/100 g and micronutrients: thiamine, 1; riboflavin, 2; pyridoxine, 1; Ca pantothenate, 10; nicotinamide, 10; inositol, 5; p-aminobenzoic acid, 30; choline, 100; biotin, 0.05; pteroylglutamic acid, 0.20; α-tocopherol, 14.2; 2 Me 1:4 naphthoquinone, 14.2 mg/100 g. Desiccated thyroid U.S.P. (Armour) replaced dextrose at a level of 0.25% in diet 286. A purified ration (H_4),* containing 24% Labco casein, served as a standard for comparison.

Male rats, averaging 40 g in weight, were placed at weaning on each of the 3 dietary regimens. After 28 days on experiment the weight increments in g for the several groups were as follows: Diet H_4 (24% casein), 130; diet 282 (60% soybean meal), 120; and diet 286 (60% soybean meal and 0.25% thyroid powder), 80. Following this depletion period the rats receiving diet 286 were segregated into groups as indicated in Table I.

The animals receiving 0.125 γ vitamin B_{12} daily in addition to diet 286 grew over a 15-day test period at a rate which was more than twice that of the controls. Their weight gain, over this period, approximated that of animals receiving the diet containing casein and exceeded that made by the rats fed the ration containing soybean meal without thyroid substance. This level appeared to approach the optimal as indicated by the fact that the feeding of higher levels of B_{12} resulted in only slightly increased weight increments. Vitamin B_{12} appeared to be utilized equally well when administered by either the oral or subcutaneous routes. Rats given cobalt nitrate by the oral route made

* Composition of Diet H_4	g/100 g
Casein (Labco)	24
Dextrose	60
Salts (U.S.P. No. 1)	4
Hydrogenated veg. oil	10
Cod liver oil	2

Micronutrients, mg/100 g: thiamine, 1; riboflavin, 2; pyridoxine, 1; Ca pantothenate, 10; nicotinamide, 10; inositol, 5, and choline chloride, 100.

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weight gains comparable with those of the controls indicating that cobalt could not be employed under these conditions in the biosynthesis of vitamin B_{12}.

It is of interest to note that the food consumption over the 15-day test period for the rats receiving 0.5 γ of vitamin B_{12} daily and those fed the unsupplemented diet but without thyroid powder were the same. The red and white blood cell counts in both the control group and in the groups receiving vitamin B_{12} fell within the normal limits.

Summary. Vitamin B_{12} counteracts the growth retarding effect of thyroid powder when fed in conjunction with a diet devoid of animal protein. The vitamin is equally effective when administered by either the oral or the subcutaneous route.

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Entrance of Intravenously Injected Sucrose into the Cerebrospinal Fluid.*

ROGER S. HUBBARD AND JOHN ZOLL.

From the Department of Pharmacology, the Medical School, University of Buffalo, and the Millard Fillmore Hospital, Buffalo, N. Y.

Sucrose injected intravenously into human subjects can be recovered almost quantitatively from the urine.[1] It has been used as an osmotic diuretic drug[2] although its toxic actions greatly limit its usefulness.[3] The excretion of the sugar by the kidneys appears to result from quantitative filtration through the glomeruli, and, since the compound is very little affected by tubular reabsorption, its rate of excretion has been used to determine glomerular filtration[4] although sucrose is not apparently as satisfactory as inulin for this purpose.

Besides its action as a diuretic, it has been used in the study of body fluids. It appears not to penetrate cells readily[1] and to be contained almost solely in the extra-cellular fluid. Lavietes and his coworkers[5] have suggested that it be used to determine total extracellular fluid when large accumulations of abnormal fluid are absent. On the other hand sucrose appears to penetrate poorly into the spinal canal[6] and has been injected intravenously to reduce intracranial pressure by osmotic action.[7]

Hubbard and Anderson[8] developed a specific method for determining sucrose, injected large amounts of the sugar into human subjects who showed unusual accumulations of extra-cellular fluid, and compared the concentrations of sucrose in blood plasma and extracellular fluid samples withdrawn after various time intervals had elapsed. They found that sucrose diffused readily into extracellular

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