material was found to be different from the blue-fluorescent alkaloid harman produced by the mild oxidation of the acetaldehyde-tryptophane complex. Harman when injected intraperitoneally into rats caused paralysis of the hind legs of the animals and slowing of heart action whereas injection of neutralized protein hydrolysate or the adsorbed material from the hydrolysate had no effect.

Lactoflavin, thiochrome, quinine bisulfate and the fluorescent material from protein hydrolysates give broad bands of fluorescent light. The fluorescent spectrum of the material concentrated from casein hydrolysate is in the violet-green region of the spectrum and is very similar to the fluorescent spectrum of quinine bisulfate. The fluorescent spectra of lactoflavin and thiochrome are different from the fluorescent spectrum of protein hydrolysate. The fluorescent spectrum of the material concentrated from casein hydrolysate is in the violet-green region having wavelengths 4100-5300 Å. The fluorescence of protein hydrolysates is excited only by light of wavelengths 3400-3600 Å.

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Production of Pernicious Anemia-like Syndrome in Rats with Bile Acids.

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Because of the property of bile acids to produce hemolysis of red blood cells, cytolysis of leukocytes, destruction of nerve cells and myelin of nerve fibers, as well as to produce gastric lesions, a group of symptoms present in pernicious anemia, 12 rats were injected subcutaneously from one to 6 times a week with a suspension of glycocholic acid from ox bile The effective daily dose after trials with weaker suspensions, was 10 mg. Of 12 rats, 3 developed a mild macrocytic, oval red blood cell anemia during the course of 2 months. During this time, one developed dragging of the hind legs, with a clumsy gait, and in 3 months, 2 showed skin ulcerations in regions which had not been used for injections. These symptoms did not appear in a control group, nor in a group injected with liver extract at the same time as the glycocholic acid.

Table I shows the nature of the blood changes.

The enlargement of the red blood cells appeared before any marked

TABLE I.

Day	R.B.C. millions per mm³	Mean corpuscular vol., μ^3	R.B.C., size†
0	8.4	52.0	25-50-25
1*	8.5	52.0	
7	7.9	51.5	
14	8.8	53.0	
21	7.5	53.5	
28	7.8	55.2	
35	7.0	58.8	11-34-55
42	6.2	55.5	
49	4.5	61.0	
56	4.8	60.2	8-18-74
63‡	4.5	59.7	
70	6.5	60.5	

*Suspension of 10 mg of glycocholic acid in water, 6 times a week.
†Measurements less than 6.0 microns; 6.0 microns; larger than 6.0 microns.
‡Liver extract injections begun.

change in the red blood cell count was noted. An increase in the red blood cell count was noted 2 weeks after the subcutaneous injection of liver extract (7 units, once a week, for 2 weeks). The mean corpuscular volume was still elevated at the end of 2 weeks of therapy (reticulocyte increase).

The fact that but 3 of the 12 rats developed changes, suggests the continuation of the experiments with attention to variations in age of the animals, diet, size of dose, solubility of the preparation used, type of bile acid, frequency and amount of injections, state of the blood and bone marrow before injection, and histological condition of the stomach after the appearance of macrocytes (*cf.* ulcer formation in guinea pigs).

The observations suggest the hypothesis that an increase or defect in the metabolism of bile acids in pernicious anemia causes a defect in the secretion of the "intrinsic factor", resulting in the development of a macrocytic anemia, and permitting an increase in hemolysis. The bile acids, acting on the central nervous system, would cause the degenerative changes. Leukopenia would be another result.

From preliminary observations, it has been established that the bile acids are quite definitely increased in the blood in patients with pernicious anemia in relapse, confirming the report of Blankenhorn.[1]

Summary. A pernicious anemia-like syndrome has been produced in individual rats, characterized by oval macrocytosis and neurological changes following the subcutaneous injection of glycocholic acid.

[1] Blankenhorn, M. A., *Arch. Int. Med.*, 1917, **19**, 344.