turned to connect the sampler with a detachable gas storage pump, and the collected gas forced over by revolving the brass collar in the reverse direction to the one in which it was previously turning. For this purpose a long piece of hollow brass rod (*16*) with a crank (*17*) at the free end is slipped over the shaft, and two projections engaged in slots cut in the small portion of the collar (*8*). A bronze plug (*18*), pressed into the rear block (*19*), holds in place the rotating components of the gear-drive mechanism and also serves as a fulcrum against which the force of *8* and *9* can be exerted when the sample is being discharged.

In order to insure a tight, permanent connection between the sampler and the flowing gas, the projecting tip (*20*) is set into a washer in a hole in the pipe carrying the flow, a clamp drawing the pipe and the sampler firmly together. The other tip (*21*) is for connection through a short piece of heavy-walled rubber tubing to a detachable pump for storage and analysis. The detachable pumps have no gears, but have the same type of plunger and double shaft, which ends in a knob concealing the adjusting nut for the inner shaft. The capacity of these, as well as of the sampler, is about 700 cc. Excessive weight is avoided by employing duralumin wherever possible, except in the case of moving parts, for which brass is used as a dissimilar metal.

10004

The Excretion of Porphyrin in Pellagra.

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Beckh, Ellinger and Spies[1] and Spies, Gross and Sasaki[2] have described an increased output of porphyrin in the urine in both endemic and alcoholic pellagra. A normal excretion of porphyrin was observed after the symptoms regressed following suitable treat-

[1] Beckh, W., Ellinger, P., and Spies, T. D., *Quart. J. Med.*, New Series, 1937, **6**, 305.

[2] Spies, T. D., Gross, E. S., and Sasaki, Y., PROC. SOC. EXP. BIOL. AND MED., 1938, **38**, 178.

ment. In these experiments, isolated, small samples of urine were examined, determinations of the porphyrin content of the stools were not made, and the type of porphyrin excreted was not established. Hence it is not possible to conclude whether the porphyrinuria is due to an increase in the total output of porphyrin, or represents a normal total output with an increased proportion excreted by the liver through the urine. Furthermore, the lack of melting point determinations and quantitative studies makes it impossible to conclude whether the porphyria represents a simple increase of a normal process as shown by Dobriner and coworkers for orderly increased hematopoiesis, or a pathological metabolism of pigments marked by the excretion of coproporphyrin Type III.[3, 4]

During a series of studies of the rate of excretion of porphyrin as influenced by the normal and pathological construction and destruction of hemoglobin it has been possible to make detailed observations in a case of pellagra. The rôle of the porphyrins in pellagra is of considerable importance, since the disease presents a certain similarity to porphyria in that both the conditions are characterized by a photosensitivity of the skin. Previous investigators, Massa[5] and Bassi[6] have suggested that the dermatitis of pellagra, like that of porphyria, is consequent to an increased content of porphyrin in the tissues.

Case Report. The patient, a 26-year-old colored woman, was admitted to the Strong Memorial Hospital, Rochester, New York, from May 11, 1936, to June 7, 1936, with a chief complaint of stomatitis, edema of the vulva, dermatitis, and diarrhea. Two months prior to admission the patient, a chronic alcohol addict who had been on a deficient diet for some time, noted mild stomatitis which became progessively worse. Shortly thereafter she developed dermatitis of the hands, feet, and elbows, and 2 weeks before admission the vulva became edematous and was associated with a foul smelling vaginal discharge and severe diarrhea.

Physical examination revealed typical symmetrical pellagrous dermatitis and stomatitis. The vulva and perineum were ulcerated and edematous. The remainder of the physical examination was irrelevant. The temperature was normal. Free hydrochloric acid was present in the gastric juice.

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3 Dobriner, K., Strain, W. H., and Localio, S. A., PROC. SOC. EXP. BIOL. AND MED., 1937, **36**, 752.

4 Dobriner, K., Rhoads, C. P., and Hummel, L. E., *J. Clin. Inv.*, 1938, **17**, 125, and earlier papers.

5 Massa, M., *Riforma Medica*, 1932, **48**, 1669.

6 Bassi, U., *Clin. Med. Ital.*, 1934, **65**, 241.

During the first 6 days of observation the patient was maintained on a fluid diet containing 2,000 calories daily, without clinical improvement. Following this control period, 75 g of a yeast extract (Vegex) were administered daily by mouth and 10.0 cc of liver extract intramuscularly (Eli Lilly and Co.) for 10 consecutive days. Improvement was noted on the second day of treatment and progressed rapidly. On the 8th day the lesions were greatly improved and the stools formed. Liver extract was discontinued on the 10th day and the Vegex was reduced to 50 g daily. During the 30 days of hospitalization the erythrocytes rose from 3.5 to 4.07 million, the hemoglobin from 11.2 to 12.2 g, the mean corpuscular volume fell from 102 to 92, and the reticulocytes reached their peak of 4% on the 16th day. Three months later the patient returned with a slight recurrence which responded rapidly to 4 injections of liver extract.

The methods used for quantitative determinations of the daily total excretion of porphyrin were similar to those previously described.[3, 7] The excretion rate of coproporphyrin is presented graphically in Fig. 1. During the 6-day control period the total coproporphyrin excretion averaged 897 mg per day; of this 254 were excreted in the urine. In the first 9 days of therapy coincidently with clinical improvement the total excretion fell to an average of 458 mg per day, of which 157 mg were in the urine. In the 2 following 9- and 6-day periods of observation the average total daily coproporphyrin excretion fell to 381 and 298 respectively. Besides the increased excretion of coproporphyrin, large amounts of protoporphyrin and deuteroporphyrin were found in the feces, a phenomenon which may have been consequent to bleeding into the intestinal canal.

The excreted coproporphyrin was a mixture of coproporphyrin I and coproporphyrin III[7] but since only small amounts of the Type III were excreted, sufficient material was not available for its final identification. Crystals of coproporphyrin I ester were obtained and were identified by their melting points (234°C). This mixed porphyrin excretion is similar to that reported for certain cases of porphyria, pigment cirrhosis of the liver, and lead poisoning and is different from that of pernicious anemia, hemolytic jaundice, and other diseases in which only increased coproporphyrin I and no coproporphyrin III is produced and excreted.[4, 7, 8–11]

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[7] Dobriner, K., *J. Biol. Chem.*, 1937, **120**, 115.

[8] Vigliani, E. C., and Libowitzky, H., *Klin. Wchnschr.*, 1937, **16**, 1243.

[9] Grotepass, W., *Z. f. physiol. Chem.*, 1932, **205**, 193.

[10] Watson, C. J., *J. Clin. Inv.*, 1937, **16**, 383, and earlier papers.

[11] Mertens, E., *Klin. Wchnschr.*, 1937, **16**, 61.

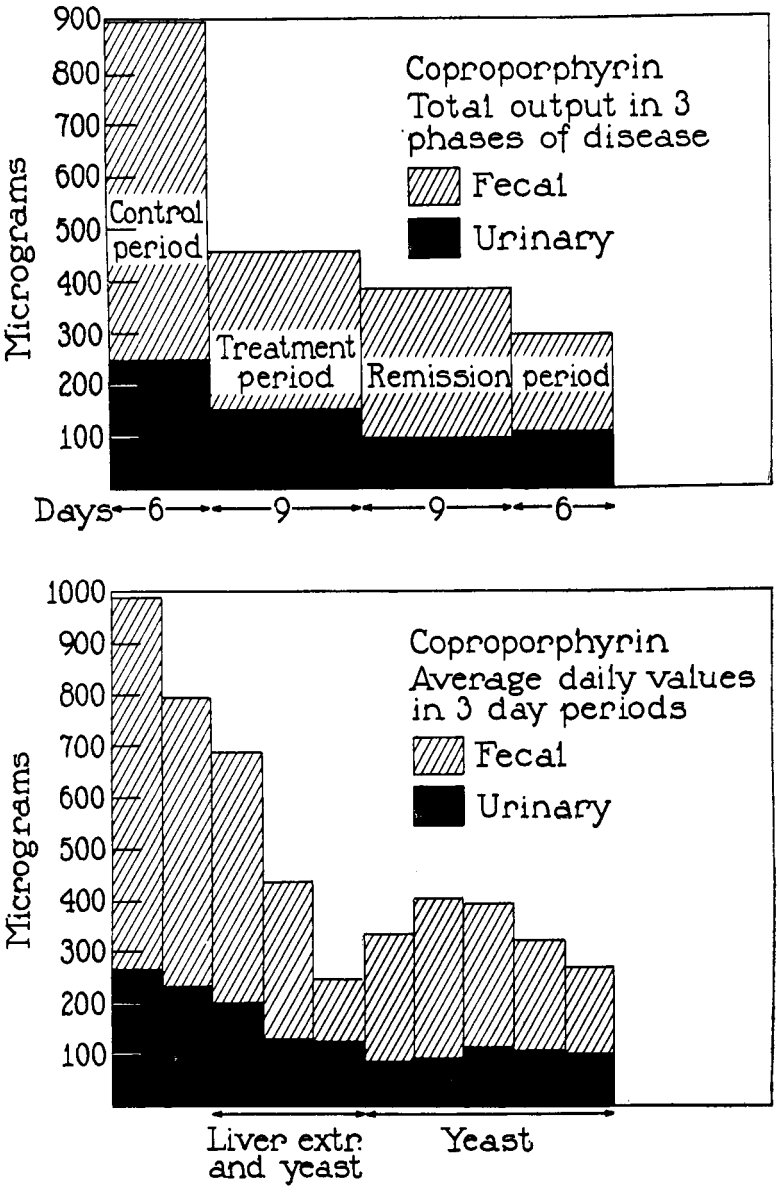

FIG. 1.

The fact that a pathologic construction as well as an abnormally great excretion of porphyrin has been demonstrated in a single case of alcoholic pellagra should be interpreted with care. Unpublished observations of material, furnished by Dr. Sydenstricker, from other cases of pellagra, do not favor the view that increased porphyrinuria

is a constant feature of the disease. Furthermore, since porphyrinuria may indicate simply an hepatic insufficiency similar to that causing urobilinuria, the factor of liver dysfunction in pellagra warrants further study.

Summary. 1. Quantitative determinations of the excretion of porphyrin in a case of alcoholic pellagra are presented. 2. Both quantitatively and qualitatively abnormal excretion of porphyrin featured relapse and disappeared during the induced remission.

10005

Natural Meningococcal Agglutinins and Lysins in Human Serum.

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The study of the susceptibility-problem in cerebrospinal fever, a disease characterized by a highly selective incidence, has divided itself naturally into an analysis of the rôle played by the causative agent and the factors concerned with host-resistance. Investigations dealing with the organisms have centered chiefly around the carrier-condition as a source of infection and the significance of the serological types of meningococci; those dealing with the host have been mostly concerned with attempts to evaluate the humoral defences of the body against meningitic infection, it being unknown what causes the selective localization of the invading organisms in the meninges.

Together with phagocytosis, the bacteriolysin and agglutinin of human blood are regarded by some authors as the primary agents concerned with the removal of the meningococcus from the circulation following its initial invasion.[1, 2] It is generally accepted that not only the normal serum of man but also that of a number of animals (guinea pig, rabbit and monkey) are strongly meningococcidal *in vitro*.[3, 4] The normal bacteriolysin resembles other natural antibodies in that its titer is said to increase with age in man and animals, regardless of previously known exposure to the meningococcus; Silverthorne and Fraser,[5] however, stress the strong menin-

1 Murray, E. G. D., Medical Res. Council, spec. Rep't Series No. 124, 1929.

2 Heist, S., Solis-Cohen, S., Solis-Cohen, M., *J. Immunol.*, 1922, **7**, 1.

3 Flexner, S., *J. Exp. Med.*, 1907, **9**, 105.

4 Matsunami, T., and Kolmer, J., *J. Immunol.*, 1918, **3**, 177.

5 Silverthorne, N., and Fraser, D. T., *J. Immunol.*, 1935, **29**, 523.