presented in Tables I and II are based on serum in saline dilutions. To each tube was added 1 cc of guinea pig complement (2 units), and .5 cc of the 1:75 dilution of antigen. The whole was stored overnight in the refrigerator and then incubated for 10 minutes at 37°C. Following this, .5 cc of rabbit amboceptor (2 units) and .5 cc of a 2% sheep cell suspension were added. The tubes were then incubated at 37°C for 30 minutes and read for hemolysis according to the usual technic of measuring the degree of hemolysis by the symbols, 1+, 2+, 3+ and 4+, the last indicating no hemolysis. Proper controls were run for anticomplementary action of each ingredient of the test.

Results. A total of 534 patients' sera were tested for complement fixation. The individuals were suffering from a variety of diseases such as are to be seen in a consecutive series of applicants for admission to any hospital. Occasional tests showed a doubtful positive, 1+ or less in tube 1. These were not counted. All other positive reactions are listed in Table I. Fifteen patients, or 2.8% gave a positive reaction.

If we now examine the results obtained with sera from patients with clinically diagnosed rhinoscleroma, we find in 11 patients a positive reaction in each case and a relatively high titer of complement-fixing antibody.

From Table II we may conclude that the complement-fixation test is of value in the diagnosis of rhinoscleroma, but in addition it gives further evidence of a relationship of the organism described to the disease itself. Further work is in progress to determine if the sensitivity of the test is such as to permit its use in the diagnosis of early unrecognized scleroma.

Conclusion. A complement-fixation test for scleroma is described which offers evidence for the relationship between an organism described and the disease. The value of the test is indicated in diagnosing scleroma.

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Urobilinogen in Cerebrospinal Fluid.

HANS N. NAUMANN.

From the Department of Pathology, Taunton State Hospital, Taunton, Mass.

With the exception of references[1] to old observations by Mestrezat and by Milian on cerebrospinal urobilin and fluorescence, respectively, only the report by Creyx, Georget and Bonnel[2] on urobilin in spinal fluid in a case of Weil's disease was found in the literature. In the present investigation cerebrospinal fluids obtained at autopsy were tested with Ehrlich's reagent for urobilinogen and the results were compared with the reactions observed in urine and with Schlesinger's test for urobilin in the blood.

Technic. The *cerebrospinal fluid* was obtained by cisternal puncture and a red cell count was made to determine the degree of contamination with blood, after which the fluid was centrifuged. 2.5 cc of clear fluid were mixed in a small tube with 2 drops of 1% *p*-dimethylaminobenzaldehyde in concentrated hydrochloric acid[3] and the color viewed through the column of fluid against a white background. Normally a faint yellow tinge is observed which may become more intensely yellow if the concentration of urea[4] is high. In the presence of urobilinogen a pink

1 Cited in Greenfield, J. G., and Carmichael, E. A., *The Cerebrospinal Fluid in Clinical Diagnosis*, p. 197, Macmillan, 1925.

2 Creyx, M., Georget, F., and Bonnel, H., *J. de Méd. de Bordeaux*, 1935, **112**, 527.

3 Niemann, G., *Z. f. physiol. Chem.*, 1925, **146**, 182.

4 Naumann, H. N., *J. Lab. Clin. Med.*, 1938, **23**, 1127.

TABLE I.
Cerebrospinal Urobilinogen, Urobilin, and Bilirubin Compared to Bile Pigments in Blood and Urine.

Diagnosis, hr post-mortem	Cerebrospinal fluid			Blood		Urine		Other data
	Urobilinogen mg %	Urobilin	Bilirubin mg %	Red cells mm³	Bilirubin mg %	Urobilin	Urobilinogen mg %	Bilirubin	
1. Lung abscess, liver cirrhosis, intestinal hemorrhage. H.P.M. 8½	9.0	3+	0.4	5000	0.3	3+	27.5	±	—
2. Cholelithiasis, obstructive jaundice, pleural exudate, arteriosclerosis. H.P.M. 19	4.4	3+	<0.1	8000	4.5	2+	36.3	+	Fecal urobilinogen 1560 mg %
3. Encephalomalacia, pneumonia, fatty degeneration of heart, liver, kidneys. H.P.M. 14	1.8	2+	<0.1	400	0.4	1+	112.0	±	—
4. General paresis, malaria treatment, pneumonia. H.P.M. 10	1.4	1+	<0.1	1500	0.5	1+	104.0	±	—
5. Cardiac hypertrophy, pneumonia, subdural, subarachnoid hemorrhage. H.P.M. 13	±	±	5.5	1500	3.0	±	0.8	±	—
6. Arteriosclerosis, pulmonary infarct, subdural hemorrhage. H.P.M. 8	±	±	2.3	10800	0.8	±	0.5	±	—

color develops which shades into orange if the yellow color due to urea is also increased. The quantitative urobilinogen determination was performed by a modified Terwen method.[5] Schlesinger's zinc acetate-alcohol reagent was used for urobilin. When bilirubin was indicated by the icterus index, van den Bergh's reaction and Fouchet's test were applied. The quantitative bilirubin determination was performed according to Malloy and Evelyn.[6] *Blood* was obtained from the heart or its large vessels and was oxalated and centrifuged. The clear plasma, which contained some hemoglobin, was used for a qualitative urobilin test by a modification of Blankenhorn's procedure,[7] and for a determination of bilirubin according to van den Bergh. Urine was analyzed for bilirubin by the author's talc adsorption technic[5] and urinary and fecal urobilinogen were determined as indicated above.

Results. Of a total of 41 postmortem cerebrospinal fluids examined 4 gave a distinct pink reaction with Ehrlich's reagent, 11 gave a faint pink or orange color, and 26 were negative. The first 4 mentioned and 2 xanthochromic fluids were studied in greater detail.

As noted in Table I, urobilinogen ranged from traces to 9 mg % and the urobilin gave doubtful to strong fluorescence. There was a rough parallelism between cerebrospinal and urinary urobilinogen and plasma urobilin with increases in the first 4 cases while the increase in the last 2 cases was uniformly very slight or doubtful. The direct van den Bergh reaction in Case 1 was delayed positive, and negative in Cases 5 and 6. The Fouchet test was positive in Cases 1, 5 and 6 and doubtful in Cases 2, 3 and 4.

An interesting observation was the gradual appearance of a green fluorescence in the untreated cerebrospinal fluids of Cases 1 to 4

[5] Naumann, H. N., *Bioch. J.*, 1936, **30**, 762 and 1021.

[6] Malloy, H. T., and Evelyn, K. A., *J. Biol. Chem.*, 1937, **119**, 480; Lepehne, G., *J. Lab. Clin. Med.*, 1942, **28**, 229.

[7] Blankenhorn, M. C., *J. Biol. Chem.*, 1928, **80**, 477.

after being kept in the refrigerator for several days. This fluorescence was proportional in strength in the previously determined urobilinogen content, indicating the slow oxidation of the latter into urobilin which under the conditions of its solution in the cerebrospinal fluid exhibited a fluorescence greater than usual. This observation corresponds to that of Creyx, Georget and Bonnel[2] who found a fluorescence in the fresh spinal fluid of their case of Weil's disease, prompting them to establish the occurrence of urobilinorachia.

Comment. The origin of urobilinogenorachia may be explained by the diffusion of urobilinogen from the blood into the cerebrospinal fluid whenever the blood urobilinogen is sufficiently increased and the blood-cerebrospinal fluid barrier is disturbed. The conditions under which urobilinogen and bilirubin may pass the blood-cerebrospinal fluid barrier do not necessarily appear to coincide, as illustrated by Case 2 in which the cerebrospinal fluid contained 4.4 mg % urobilinogen and practically no bilirubin while the plasma bilirubin amounted to 4.5 mg %. On the other hand, Case 1 seems to indicate that diffusion of both pigments took place at a similar rate. As xanthochromic spinal fluids in jaundice are rare[8] factors other than liver damage can be assumed to be responsible for the increased barrier permeability, the most likely being infection.[9] The present cases showed severe pulmonary infection and in one instance malaria in addition.

Summary. Six cases of urobilinogenorachia have been presented in which the postmortem cerebrospinal fluid urobilinogen ranged from traces to 9 mg %.

8 Klein, N., and Szentmihalyi, S., *Dtsch. Arch. f. klin. Med.*, 1932, **173**, 234; Lickint, F., *Z. f. d. ges. Neurol. u. Psych.*, 1931, **136**, 291.

9 Malamud, W., and Rothschild, D., *Arch. Neurol. Psych.*, 1931, **24**, 348.

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Effect of Thiols on Mercurial Diuresis.

CARROLL A. HANDLEY AND MARGUERITE LAFORGE.

From the Department of Physiology and Pharmacology, Baylor University College of Medicine, Houston, Texas.

The formation of stable complexes between mercury and thiols[1,2] led us to believe that the use of the latter compounds might yield further evidence of the mechanism of action of mercurial diuretics as well as the mechanism of the normal transport of water across the renal tubule. Peters[3] and his associates have presented ample evidence that many of the physiologic actions of the trivalent arsenicals may be explained by the reaction of the arsenical with essential sulfhydryl groups of certain enzymes, thereby inactivating the enzyme. Barron and Singer[4] have demonstrated that organic mercurials are also powerful inhibitors of some enzymes containing sulfhydryl groups.

This report concerns the effect of several compounds that contain sulfhydryl groups on mercurial diuresis.

Methods. The basal urine output of female dogs with inlying catheters was measured; the mercurial diuretic was then given intravenously, and the diuresis curves (Fig. 1) were obtained by recording 20-minute urine volumes over a period of about 3 hours.

1 Gilman, A., *Fed. Proc.*, 1946, **5**, 285.

2 Braun, H. A., Lusky, L. M., and Calvary, H. O., *J. Pharmacol.*, Suppl., 1946, **87**, 119.

3 Peters, R. A., Stocken, L. A., and Thompson, H. S., *Nature*, 1945, **156**, 616.

4 Barron, E. S. G., and Singer, T. P., *J. Biol. Chem.*, 1945, **157**, 221.