is only a slight increase in glycogen content and no change in the degree of fatty metamorphosis.

Discussion. In order to evaluate these findings, knowledge of normal values and their fluctuations is essential. The only observations of the glycogen content of the liver in human beings have been obtained at operation, when the metabolism had been deranged by operative trauma, anesthesia, and by previous illness.[5] Autopsy material may not give an accurate picture because of the rapidity of postmortem glycogenolysis, and because even brief agonal states are known to cause drastic changes in the liver glycogen of animals. We are, therefore, accumulating a series of observations in normal individuals in the postabsorptive state, after meals, and after the administration of various drugs and stresses.

A quantitative correlation between histochemical and chemical methods for the determination of liver glycogen would be desirable. The study by Deane, Nesbett and Hastings[6] indicates that a good correlation can be obtained. To this end, we are performing simultaneous quantitative and histochemical determinations of glycogen in livers obtained from rats under various experimental conditions, as well as from human tissue obtained at autopsy. To date, it appears that a roughly quantitative correlation can be observed, and it is hoped that ultimately this can be done with a fair degree of accuracy.

Summary. Liver biopsy specimens obtained during diabetic acidosis and its treatment were examined by the Gomori technic for glycogen. Severe glycogen depletion was found before treatment. Restoration of glycogen content occurred after a few hours of therapy.

[5] MacIntyre, D. S., Pedersen, S., and Maddock, W. G., *Surgery*, 1941, **10**, 716.

[6] Deane, H. W., Nesbett, F. B., and Hastings, A. B., PROC. SOC. EXP. BIOL. AND MED., 1946, **63**, 401.

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Scleroma: Complement Fixation Test.

MILTON GJELHAUG LEVINE AND ROBERT E. HOYT.

From the Institute of Experimental Medicine, College of Medical Evangelists, Los Angeles.

From the time that rhinoscleroma was discovered in 1870 by von Hebra,[1] the disease has been the subject of a good deal of controversy. The name of the disease itself has been considered inadequate, and in 1932, at the Second International Congress Otorhinolaryngology[2] held in Madrid, it was changed to the more inclusive term, scleroma. The lack of definite information on the etiology of the disease has probably been the main source of confusion. Despite the inclusion of the supposed causative organism, *Klebsiella rhinoscleromatis*, in Bergey's Manual,[3] this bacillus is far from acceptable as the etiologic agent. Wilson and Miles[4] say, ". . . there is very little evidence that this organism is primarily responsible for it. There is no means by which it can be distinguished with certainty from other members of the capsulated group; and, since we know that members of this group may be present in the nose of healthy persons, it is difficult to prove

[1] von Hebra, R., *Wien. med. Wchnschr.*, 1870, **20**, 1.

[2] Rapp, Cong, *Madrid Internat. d'Oto-rhenolaryng.*, 1932.

[3] Bergey, D. H., Breed, R. S., Murray, E. G. D., and Hitchens, A. P., *Bergey's Manual of Determinative Bacteriology*, Baltimore, 1939.

[4] Wilson, G. S., and Miles, A. A., *Topley and Wilson's Principles of Bacteriology and Immunity*, 3rd Ed., Baltimore, 1946.

TABLE I.
Complement Fixation of Routine Patients' Sera.

No. of sera	Serum titration 2.5	5	10	20	40	80	160	320	640
5	1+	1+							
3	2+	1+							
1	2+	2+							
1	3+	3+	3+	3+	2+	0			
1	3+	3+	3+	2+	0				
1	3+	3+	0	0					
1	4+	4+							
*1	4+	3+							
*1	4+	4+							

* On repeating with new samples of sera, titration was negative.

TABLE II.
Complement Fixation of Sera from Patients with Scleroma.

No. of case	Serum titration 2.5	5	10	20	40	80	160	320	640	1280
1L	4+	4+	4+	4+	4+	2+	1+	0		
2M	4+	4+	4+	4+	4+	4+	2+	0		
3F	4+	4+	4+	4+	4+	4+	1+	0		
4J	3+	2+	1+	1+	0	0	0	0		
5C	4+	4+	4+	4+	4+	1+	0	0		
*6M	4+	4+	4+	4+	2+	1+	0	0		
7	4+	4+	4+	4+	4+	3+	3+	0		
8	4+	4+	4+	4+	4+	4+	4+	3+		
9	4+	4+	3+	3+	1+	0	0	0		
10	4+	4+	4+	4+	4+	4+	4+	4+	2+	1+
†11	4+	4+	4+	4+	4+	4+	4+	4+		

* Cases 6M, 7, 8, and 9 submitted by Dr. M. Ruiz Castaneda of Mexico City.
† Case 11 submitted by Dr. M. Gerundo, Hilo, Hawaii.

that they play any part in production of rhinoscleromatis."

We have studied many phases of the problem and have reported our conclusions in a publication now in press.[5] We have described an organism (isolated from cases of scleroma) which we feel to be the cause of the disease. It is a Gram-negative, nonmotile rod, forming mucoid colonies on nutrient agar and eosin-methylene-blue agar. It ferments maltose, mannite and dextrose, but never lactose. Occasionally sucrose is fermented. All sugars fermented form acid only.

This paper is intended to describe a complement fixation test for scleroma in which the above bacterium is used as the antigen.

Experimental Procedure. The organism used was isolated from a patient with scleroma. It was grown in nutrient broth containing 5% dextrose for 7 days. At the end of that time it was centrifuged clear of the supernatant which was discarded. The organism was then washed twice in saline, each washing equivalent in amount to the original broth. The organism was exposed to 65°C for 30 minutes and suspended in 1:5000 merthiolate in .85% saline. Organisms from 10 liters of the original broth were resuspended in 100 cc of the merthiolate-saline solution. An anticomplimentary titer was now run. The antigen suspension was found to be anticomplementary in a dilution of 1:48. In preliminary tests, a dilution of 1:60 gave what was considered an excess of mildly positive reactions. A dilution of 1:75 of the antigen was used in the final complement fixation tests.

The actual titration was carried out according to the routine Wassermann procedure. Dilutions of the patient's serum were made in physiological saline. The titration figures

[5] Levine, M. G., Hoyt, R. E., and Peterson, J. E., *J. Clin. Invest.*, 1947, **26**, 281.

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.