11:12—corneal reflex disappeared and respiration suddenly ceased. The animal was at once injected intravenously $NaNO_2$ 22.5 mg. per kg., followed by $Na_2S_4O_6$ 500 mg. per kg.
11:15—p. 68, r. 12; corneal reflex returned.
11:16—p. 108, r. 92.
11:25—p. 166, r. 132 (panting).
11:31—raised its head.
12:14 p. m.—injected intravenously $NaNO_2$ 10 mg. per kg. and $Na_2S_4O_6$ 125 mg. per kg.
12:23—up on its feet.
12:25—p. 156, r. 80.
1:18—p. 160, r. 20.
2:05—drank water.
4:45—ate food, urinated.
8:00 a. m. next day—no obvious untoward effects.

We have also studied the combination of methylene blue and sodium tetrathionate or thiosulphate in cyanide poisoning, and observed a synergistic action. The results are, however, much less striking.

7081 C

Glycogen Formation after Oral Administration of Mannitol to White Rats.

ALFRED K. SILBERMAN AND HOWARD B. LEWIS.

From the Department of Physiological Chemistry, Medical School, University of Michigan.

Glycogen formation is stated to occur after the oral administration of mannose to animals, but the conversion of mannitol, the hexahydric alcohol which may be obtained by reduction of the aldehyde group of mannose, to glucose or glycogen has not been definitely proven. It has been maintained that sorbitol,[1] the hexahydric alcohol derived from glucose, may alleviate the hypoglycemic symptoms in rabbits which have received insulin and when administered to diabetic patients may increase the respiratory quotient. Rosenfeld[2] was unable to observe any significant increases in liver glycogen after feeding mannitol to dogs. Pflüger[3] in a crit-

[1] Reinwein, H., *Deut. Arch. klin. Med.*, 1929, **164**, 61.

[2] Rosenfeld, G., *Centr. inner Med.*, 1900, **21**, 177.

[3] Pflüger, E. F. W., *Das Glykogen und seine Beziehungen zur Zuckerkrankheit*, Bonn, 1905, 2nd Ed., 215.

ical evaluation of the data of Külz concluded that the increased glycogen deposition obtained by this investigator was within the limits of experimental error.

We have studied the glycogen content of the liver after the oral administration of *d*-mannitol by the method of Cori. Young white rats were fasted 24 hours and fed either 2 or 4 cc. of a 15% solution of mannitol by stomach tube. The amount of mannitol fed was determined by passing the mannitol solution through the stomach tube into an evaporating dish, which was then placed in an oven and dried to constant weight. After absorption periods of 2, 3, 4, and 6 hours, the rats were killed and the glycogen content of the liver was determined as described previously.[4]

TABLE I.

Glycogen content of liver of white rats after oral administration of Mannitol.

Wt. after fast gm.	Absorption period hr.	Mannitol fed mg.	Liver Glycogen %
143	2	214	0.07
160	2½	214	0.05
137	3	214	0.08
129	3	214	0.12
130	4	250	0.16
135	4	250	0.08
110	6	505	0.19
114	6	505	0.05
115	6	505	0.07
120	6	505	0.08
123	6	505	0.06
132	6	505	0.09
136	6	505	0.07
145	6	505	0.07
Average value for 9 control animals			0.05 (range 0.02-0.06).

The results are presented in Table I. The glycogen values for the livers of a control series of 9 rats which received 2 cc. of water after 24 hour fasting periods varied from 0.02 to 0.06% with an average of 0.05%. Inspection of the data of the table fails to reveal any significant increases in liver glycogen after oral administration of mannitol as compared with the values obtained for the control series.

After these experiments were completed, Carr and his co-workers[5] reported a similar study of the glycogen content of the liver of white rats, previously fasted and then fed a mixture of cacao-butter and mannitol (33%) over a period of 80 hours. The glycogen content

[4] Catron, L. F., and Lewis, H. B., *J. Biol. Chem.*, 1929, **84**, 553.

[5] Carr, C. J., Musser, R., Schmidt, J. E., and Krantz, J. C., Jr., *J. Biol. Chem.*, 1933, **102**, 721.

of the liver of the rats fed mannitol and cacao-butter ranged from 0.62 to 1.80% with an average value of 1.23%, as compared with the average glycogen content of the livers of control rats fed cacao-butter alone of 0.14%.

It is impossible to compare the results of this series with our own experiments since the treatment of the animals was quite different in the 2 series. Under the experimental conditions used by us, it is evident that mannitol did not serve as a ready available source of glycogen. A similar lack of ready utilization of mannitol is shown by the reported failure of mannitol to relieve insulin shock in white rats.[6]

7082 C

Concentration of Poliomyelitis Virus by Ultrafiltration.*

PAUL F. CLARK, R. C. AINSWORTH AND L. G. KINDSCHI.

From the Department of Pathology and Medical Bacteriology, University of Wisconsin.

The obvious desirability of finding a means for the more adequate concentration of any of the filterable viruses, led us to attempt to adapt the ultrafiltration method, used by Seibert[1] in the study of tuberculin, to the concentration of poliomyelitis virus. That several of the well studied filterable viruses can be retained by filters or ultrafilters has been known for some time. But the work has been done largely for the purpose of determining the size of the viruses. Krueger and Schultz[2] and Clifton, Schultz and Gebhardt[3,4] for example, have made careful studies of the filterability of poliomyelitis virus through graded collodion membranes. Their results indicate that the size of this virus is certainly less than 50 millimi-

[6] Voegtlin, C., Dunn, E. R., and Thompson, J. W., *J. Pharmacol. and Exp. Therap.*, 1925, **25**, 168.

* Aided by a grant from the Milbank International Committee for the study of Infantile Paralysis and later by a grant from the National Research Council.

[1] Seibert, F. B., *J. Biol. Chem.*, 1928, **78**, 345.

[2] Krueger, A. P., and Schultz, E. W., PROC. SOC. EXP. BIOL. AND MED., 1929, **26**, 600.

[3] Clifton, C. E., Schultz, E. W., and Gebhardt, L. R., *J. Bact.*, 1931, **22**, 7.

[4] Poliomyelitis. 1932, p. 49. Milbank International Committee for the Study of Infantile Paralysis.