tivity of one of these gastric juices decreased the binding of one of these but did not change the results with the liver slice. 4. The enhanced uptake on the rat liver slice produced by these gastric juice fractions did not seem to be related to proteolytic activity.

1. Miller, O. N., Hunter, F. M., PROC. SOC. EXP. BIOL. AND MED., 1957, v96, 39.

2. Johnson, P. C., Driscol, T. B., *ibid.*, 1958, v98, 731.

3. Herbert, V., *ibid.*, 1958, v97, 668.

4. Latner, A. L., Merrils, R. J., Vitamin B-12 and Intrinsic Factor, 1957, pg. 201, ed. by Heinrich, H. C., Enke, Stuttgart.

5. Richmond, V., Caputto, R., Wolf, S., *Arch. Biochem.*, 1957, v66, 155.

6. Latner, A. L., Merrils, R. J., Raine, L., *Biochem. J.*, 1956, v63, 501.

7. Richmond, V., Caputto, R., Wolf, S., *Gastroenterology*, 1955, v29, 1017.

8. Johnson, P. C., Bird, R. M., Smith, C. W., *South. Med. J.*, 1958, v51, 417.

9. Anson, M. L., Mirsky, A. E., *J. Gen. Physiol.*, 1932, v16, 59.

10. Grasbeck, R., *Acta Med. Scandinav.*, 1956, Supp. No. 313.

Received March 31, 1959. P.S.E.B.M., 1959, v101.

Serum Protein-Bound Carbohydrates and Lipids in Cholera. (24931)

SACHCHIDANANDA BANERJEE AND JAGADINDRA NATH BHADURI
Dept. of Physiology, Presidency College, Calcutta, India

Electrophoretic studies on plasma proteins in cholera showed(1) a diminution in albumin and α-globulin, increase in β-globulin which was further differentiated into β_1 and β_2 fractions and no change in γ-globulin. Plasma proteins are conjugated with carbohydrates and lipids. As distribution of these protein bound hexoses and lipids is likely to be disturbed in pathological conditions(2-8) it was of interest to study glycoproteins, mucoproteins and lipo-proteins in serum of cholera patients. Total lipid, phospholipid and cholesterol of serum were also determined. The above studies were also carried out in normal subjects for comparison.

Methods. Patients admitted into Nilratan Sircar Medical College Hospitals, Calcutta with typical symptoms of cholera were selected. As routine procedure, stool was cultured and cases whose stool showed presence of cholera vibrio were included in the report. Normal subjects were persons admitted in surgical ward of hospital for vaginal hydrocele or hernia. Examinations of blood, urine and stool were carried out to ascertain if subjects were otherwise normal. Blood was collected from anticubital vein in early morning, before breakfast and before any treatment, in centrifuge tubes and clear serum used for different estimations. Glycoprotein and mucoprotein were precipitated from aliquots of serum, dissolved in alkali, treated with orcinol-sulfuric acid and color compared with standard solution of galactose-mannose(9). For estimation of lipo-proteins(10) an aliquot of serum was treated with saturated solution of Sudan Black B in 95% ethanol, alcohol evaporated, centrifuged and supernatant stained serum used for electrophoresis. .04 cc of stained serum was applied to Whatman No. 1 strips, and electrophoretic separation carried out with barbiturate-barbituric acid buffer of pH 8.6 and ionic strength .05 for 8 hours, by using L.K.B. paper electrophoresis apparatus with current strength of 5 mA and 250 volts. Whatman strips were dried at 70°C for half hour, optical density curves of colored zones plotted using Photovolt Photoelectric Densitometer Model 425 on graph paper and areas of component section of graph were measured by planimetry. α,β lipo-proteins and "O" fraction were expressed as percentage of total lipids. Cholesterol was estimated by method of Sobel and Mayer(11), phospholipid by determining P of ether extract of serum(12) and multiplying value by 25(13) and total serum lipids by the method of Bragdon(14). Results are given in Table I.

TABLE I. Serum Protein Bound Carbohydrates and Lipids in Cholera and in Normal Subjects.

	Normal (10)*	Cholera (10)*	"t"
Glycoprotein (mg/100 cc)	194 ± 5 †	272 ± 7	9.2
Mucoprotein (mg/100 cc)	45 ± .7	80 ± 2	16.8
Total lipid (mg/100 cc)	619 ± 3	808 ± 19	9.9
α-lipoprotein (%)	32.7 ± 1.0	21.6 ± 1.0	7.9
β-lipoprotein (%)	59.3 ± 1.0	65.7 ± 1.0	4.6
"O" fraction (%)	8.0 ± .7	12.6 ± 1.0	3.8
Phospholipid (mg/100 cc)	250 ± 6	389 ± 16	8.1
Cholesterol (mg/100 cc)	160 ± 3	137 ± 2	6.4

* No. of subjects. † Mean ± stand. error.

Results. In cholera serum phospholipid, total serum lipid as well as β-lipoprotein were significantly increased with decrease in α-lipoprotein and total cholesterol. Serum glycoprotein and mucoprotein fractions also increased significantly as seen in Table I.

Discussion. β-globulins are increased while α-globulins are diminished(1). The increases in cholera of β-lipoproteins and decrease in α-lipoproteins are commensurate with changes in α and β-globulins observed and are similar to changes reported in the nephrotic syndrome (15,16). Phospholipids and total lipids are greatly increased in cholera. This may be associated with mobilization of fat from depots, or to liver damage. The α-protein elevations may be associated with inflammation and tissue destruction(2-5).

Summary. Serum glycoproteins, mucoproteins, phospholipid, total lipid, total cholesterol and lipoproteins were determined in patients suffering from cholera and in normal subjects. In cholera serum glycoproteins, mucoproteins, phospholipids, total lipids and β-lipoproteins were significantly increased and α-lipoproteins and total cholesterol were diminished.

Dr. A. K. Datta Gupta of N. R. Sircar Medical College and Hospitals kindly rendered hospital facilities.

1. Banerjee, S., Chatterjee, K. P., PROC. SOC. EXP. BIOL. AND MED., 1957, v94, 474.

2. Greenspan, E. M., Dreiling, D. A., *Arch. Int. Med.,* 1953, v91, 474.

3. Greenspan, E. M., Tepper, B., Terry, L. L., Schoenbach, E. B., *J. Lab. Clin. Med.,* 1952, v39, 44.

4. Winzler, R. J., Devor, A. W., Mehl, J. W., Smyth, I. M., *J. Clin. Invest.,* 1948, v27, 609.

5. Winzler, R. J., Smyth, I. M., *ibid.,* 1948, v27, 617.

6. Shedlovsky, T., Scudder, J., *J. Exp. Med.,* 1942, v75, 119.

7. Gjessing, E. C., Chanutin, A., *J. Biol. Chem.,* 1947, v169, 657.

8. Perlmann, G. E., Glenn, W. W. L., Kaufman, D., *J. Clin. Invest.,* 1942, v22, 627.

9. Weimer, H. E., Moshin, J. R., *Ann. Rev. Tuberc.,* 1953, v68, 474.

10. McDonald, H. J., Bermes, E. W., *Biochim. biophys. Acta,* 1955, v17, 290.

11. Sobel, A. E., Mayer, A. M., *J. Biol. Chem.,* 1954, v157, 255.

12. Fiske, C. H., Subbarow, Y., *ibid.,* 1925, v66, 375.

13. Youngburg, G. E., Youngburg, M. V., *J. Lab. Clin. Med.,* 1930, v16, 158.

14. Bragdon, J. H., *J. Biol. Chem.,* 1951, v90, 513.

15. Barr, D. P., Russ, E. M., Eder, H. A., *Am. J. Med.,* 1951, v11, 480.

16. Russ, E. M., Eder, H. A., Barr, D. P., *ibid.,* 1951, v11, 468.

Received April 3, 1959. P.S.E.B.M., 1959, v101.