fact. The warning is perhaps especially needed now, inasmuch as hydrolysis by N HCl is generally employed in the study of various phosphate fractions in blood or muscle.

On neutralizing this extra acid before carrying out the colorimetric test a rather high salt concentration is produced, which in our experiments, on account of the final dilution used, amounted to 0.2 M. Not all salts interfere with the colorimetric reaction, as will be shown presently, but the chlorides and nitrates do interfere seriously. We determined in a series of special analyses the various concentrations of sulfates and chlorides at which the colorimetric determination of P is no longer accurate. These results are here summarized:

Salt	Interfering Concentrations
NaCl	0.1 M
NH_4Cl	0.2 M
$(NH_4)_2SO_4$	0.5 M
Na_2SO_4	About 3.0 M

Rimington found the NaF in a concentration of 0.01 M interfered with the phosphate determination. We also find that NaCl interferes seriously, 1.0 M concentration causing a loss of 50% in the P recovered colorimetrically. A 0.1 M $NaNO_3$ gives results that are 20% too low. The fact that the sulfate salts, especially the Na_2SO_4, interfered very much less than the chlorides, accounts for our success in using N H_2SO_4 for hydrolysis. The maximum Na_2SO_4 concentration upon neutralization of the acid and upon dilution of the solution is only 0.1 M, whereas our analyses show that this salt does not interfere even in very large concentrations and at 3 M it actually causes an increase in color by about 7%.

6100

Plasma Protein and Blood Volume.

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Plasma protein deficiency results in a decrease of the osmotic pressure of the blood and has been held to be part of the explanation of edema in nephrosis and certain cases of undernutrition. If this postulation be correct the capillary pressure would be in excess of the osmotic pressure of the blood and as a result of the increased

transudation a state of oligemia should be expected to prevail under such conditions. That this is actually the case in nephrosis has been shown by Darrow[1] and Waterfield.[2] The behavior of blood volume in nutritional edema has, however, not received the attention it deserves. The present report deals with the blood volume changes in both of these diseases, special attention being directed towards correlating such volume changes with the fluctuation of the plasma protein.

The plasma protein was determined by the micro-kjeldahl method and the blood volume by inhalation of carbon monoxide according to the technic reported previously.[3] Hematocrit readings were made by the method of Osgood.[4] In the cases of nephrosis the 24-hour urine output for the day of examination was also ascertained. In all, 5 cases of nephrotic kidney condition and 7 cases of nutritional edema were studied. The findings are given in Tables I and II.

On examining the data two points deserve special emphasis. First, in both diseases, when not complicated by severe anemia, the

TABLE I.
Blood Volume and Plasma Protein Findings in 5 Cases of Nephrosis.

Case No.	Date	Wt. kilo	B. M. R. %	Urine output in 24 hr. cc.	Plasma protein %	Blood Vol. Total cc.	Blood Vol. Per sq.m. cc.	Red cell volume %	Remarks
1	March 9, 1928	91.4	— 5.0	580	4.70	3690	1775		
	March 30, 1928	90.3	+ 1.4	1380	5.20	4215	1970		
	May 1, 1928	52.8	+ 0.5	1950	6.90	5025	3030		
	October 4, 1929		+ 8.8		6.60	5500	2895		Recovered
	October 17, 1930	67.6	+ 6.7	900	6.44	5430	2920	39.5	
2	January 16, 1928	61.8	—20.0	230	4.40	2918	1757		
	March 3, 1928	53.7	+13.4	1220	5.60	3835	2300		Improved
	May 2, 1928	60.9	—26.4	1140	5.50	3700	2200		
3	January 16, 1930	65.5	—41.0	1415	3.46	3155	1793	38.8	
	February 19, 1930	55.4	—27.8	1350	4.00	3540	2172	32.3	
	April 8, 1930	49.2	—16.7	1130	4.38	3670	2370	32.5	
4	October 17, 1929	45.2	—15.4	1630	4.68	4255	2820	39.6	
	December 5, 1929	43.0	— 2.7	765	3.99	3202	2180	35.0	Became
	December 23, 1929	44.8	—16.2	1100	3.81	3580	2370	37.5	worse
5	May 5, 1931	26.7	— 9.0	2500	4.57	1564	1580	37.5	
	May 19, 1931	27.9	— 9.8	790	4.88	1683	1700	37.0	
	June 3, 1931	26.0	—16.6	560	4.89	1638	1706	39.5	
	January 23, 1932	30.8	— 0.3	1450	5.83	1962	1886	37.5	

[1] Darrow, D. C., PROC. SOC. EXP. BIOL. AND MED., 1926, **23**, 740.

[2] Waterfield, R. L., *J. Clin. Invest.*, 1931, **9**, 589.

[3] Chang, H. C., and Harrop, G. A., Jr., *J. Clin. Invest.*, 1928, **5**, 393.

[4] Osgood, E. E., *Arch. Int. Med.*, 1926, **37**, 685.

TABLE II.
Blood Volume and Plasma Protein Findings in 7 Cases of Nutritional Edema.

Case No.	Date	Wt. kilo	B. M. R. %	Plasma protein %	Blood Vol. Total cc.	Blood Vol. Per sq.m. cc.	Red cell volume %	Remarks
1	September 25, 1929	41.7	— 5.9	5.09	2560	1821	33.5	
	November 22, 1929	38.8	+13.4	6.56	3715	2925	32.5	
	December 6, 1929	41.4	+13.5	5.90	3550	2670	30.8	
2	April 2, 1930	49.0	—10.8	3.77	2975	1893	41.0	
	May 20, 1930	46.2	+13.7	6.87	3570	2410	40.4	
3	March 31, 1931	46.7	—11.5	5.77	3590	2163	36.8	
	June 9, 1931	50.5	+ 3.7	6.12	4240	2632	38.8	
4	February 17, 1930	41.4		4.07	3335	2315	30.6	Secondary
	March 3, 1930	43.1	— 1.3	5.81	3835	2610	30.8	anemia
5	January 21, 1930	28.4	— 5.5	5.34	2650	2430	26.2	Secondary
	February 21, 1930	30.8	+22.6	5.92	2910	2620	29.4	anemia
	March 7, 1930	32.0			2906	2570	37.8	
6	November 5, 1930	25.2	—11.7	5.37	1950	1930	19.5	Anemia
	May 6, 1931	29.1	— 3.9	6.20	2085	1905	35.2	
	June 26, 1931	34.0	+12.6	6.44	2600	2260	32.8	
	July 29, 1931	37.9	+19.9	6.57	2720	2266	34.8	
7	September 24, 1930	27.1	—22.8	3.46	1510	1510	35.4	
	March 24, 1931	24.9	—15.6	5.89	1612	1662	35.3	
	April 10, 1931	27.4	+ 0.1	6.08	1850	1832	31.4	
	May 1, 1931	28.5	—17.6	6.38	2048	1968	33.5	
	July 2, 1931	31.4	+ 5.0	6.79	2175	2015	37.7	
	July 17, 1931	32.0	+ 0.3	6.86	2355	2160	36.7	

blood volume expressed in cc. per square meter of the surface area (calculated from patient's height and weight) was very much lower than the normal standard determined by the same method.[3] This is in agreement with the previous work on nephrosis[1, 2] and further corroborates the belief that the edema of these 2 diseases may be placed under the same category. Secondly, any change in the plasma protein concentration was reflected in nearly every case by a corresponding variation of the circulatory volume and when an increase of the plasma protein took place, usually resulting in an improvement or disappearance of the edema, then the blood volume almost invariably returned towards the normal range. As the cell volume suffered only very slight changes the volume variation seemed to be chiefly confined to the plasma.

No definite correlation between the volume fluctuation and the extent of diuresis was evident in any of these patients with nephrotic kidney disease.

In conclusion one may assume that the plasma protein concen-

tration among many others is one of the regulating factors of the circulatory volume. Deviation from normal on the part of the former may be attended by a disturbance of the latter. This is well exemplified in such pathological conditions as nephrosis and nutritional edema.

6101

Experimental Production and Cure of Jejunal Ulcers.

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Twenty-six animals were operated upon for the production of jejunal ulcers using a slight modification of the technic of Mann and Williamson[1] for depriving the jejunum of its normal alkalinity.

In each case the duodenum was transected between the pylorus and the ampulla of Vater and both ends inverted with silk. The jejunum was then divided a few inches beyond Treitz's ligament and again both ends inverted. Following this a gastro-enterostomy was performed between the distal end of the jejunum and the stomach and an enteroenterostomy performed between the closed duodenojejunal loop and the upper ileum about 24 inches below the gastroenterostomy, allowing the bile, pancreatic juice and duodenal secretions to be emptied back into the small bowel at this point. These changes were made in the original procedure because it was felt that the nutrition of the animals would be better preserved if the drainage were done higher in the small intestine, thereby eliminating this factor from the ultimate results. Also since a lateral anastomosis has a better blood supply than one made end-to-end, there would be less chance of criticism on this score as a possible source of ulcer formation.

Ten of these animals formed typical chronic ulcers varying in size from ½ to 2½ cm. in diameter. The period necessary for the formation of the ulcers varied from 42 to as long as 428 days with the average of 118 days. The ulcers were multiple in 3 cases. They always formed on the wall of the jejunum opposite the gastroenterostomy stoma and never involved the suture material which was of silk throughout, except in one instance, where the edge of a very large perforating ulcer touched it in one spot. The animals were

[1] Mann, F. C., and Williamson, C. S., *Ann. Surg.*, 1923, **77**, 409.