STUDIES OF SERUM ELECTROLYTES: *III. In Infections, Nephritis, and Other Pathological Conditions*

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STUDIES OF SERUM ELECTROLYTES

III. In Infections, Nephritis, and Other Pathological Conditions

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The study of the electrolytes in the serum in health and disease has received an impetus from the introduction of methods for estimating the total base of the serum. This more complete picture of the electrolyte composition as compared with the study of merely the bicarbonate or the chloride of the serum invites a reinvestigation of the blood in various diseases. Attention was directed to the possibilities of such studies by the paper of Van Slyke, Wu and McLean (1923) upon the distribution of electrolytes between serum and red blood cells. Several workers have applied similar methods to the study of the changes in the serum electrolytes in disease. Gamble and his associates (1923, 1925) have studied the picture during fasting and after pyloric obstruction. Peters and his associates (1925, 1926) have made studies of this type in diabetes and nephritis and to a lesser extent in certain other diseases. Atchley and Benedict (1927) have reported the picture following experimental ureteral ligation in dogs. In a previous study (1926) we reported the picture of the serum electrolytes throughout the course of lobar pneumonia. We have continued studies of this type in other infections: tuberculosis, pleural effusions, rheumatic fever and peritonitis. It is the purpose of this paper to record the results of these studies to date. In addition, a few cases of renal disease and a few miscellaneous conditions are reported.

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TABLE 1

Individual case analyses

Case number	Diagnosis	Date (1927)	Temperature	Pulse	Respiration	Total base: Chemically determined	Total base: 1.17 × corrected conductivity	Total base: Average	Cl	CO_2	Protein	Specific gravity	Non-protein nitrogen	Calculated: HCO_3'	Calculated: B Pr (2.03 Pr)	Calculated: Residual anions
			°F.	per minute	per minute	m.Eq. per liter	m.Eq. per liter	m.Eq. per liter	m.Eq. per liter	mM. per liter	grams per 100 cc.	20°C./20°C.	mgm. per 100 cc.	m.Eq. per liter	m.Eq. per liter	m.Eq. per liter
Normal values	Minimum....					150	152	151	98	24	7.6	1.0260	18	23	15	4
	Maximum....					164	162	163	108	33	8.8	1.0300	25	32	18	18
A 10	Tuberculous meningitis	March 15	100.2	98	30	**144**	**146**	**145**	**92**	25.1	8.6	1.0281	**33.3**	24	17	12
A 11	Pleural effusion	March 17	99.4	136	24	**147**	**145**	**146**	100	24.5	**5.1**	**1.0187**	**32.3**	23	**10**	13
A 12	Generalized tuberculosis	March 25	100.4	116	34	154	**144**	**149**	**90**	25.3	**9.5**	**1.0315**	**29.7**	24	**19**	16
		March 31	**99.6**	124	38	**142**	**148**	**145**	**89**	26.5	**9.5**	**1.0317**	**53.7**	25	**19**	12
A 13	Acute proliferative pulmonary tuberculosis	February 15	102.0	108	32	160	**149**	155	**97**	24.8	**8.7**	**1.0320**	**37.7**	24	18	16
		February 22	100.0	112	28	159	156	158	**96**	28.0	**9.3**	**1.0313**		27	**19**	16
A 14	Miliary tuberculosis	April 26	102.8*	112	40	**138**	**148**	**143**	**87**	27.5	**7.5**	**1.0256**		.26	15	15
A 15	Pleural effusion; pulmonary tuberculosis	February 22	102.0	132	32	**149**	**139**	**144**	98	**23.6**	7.7	1.0280		**22**	16	. 8
		March 3	101.4	116	29		**142**		**85**	26.8	8.7		**32.2**	26	18	13
		February 22†				*147*	*136*	*142*	*90*	*21.1*	*6.7*	*1.0250*	*27.1*	*20*	*14*	*18*
A 16	Pleural effusion	March 31	99.0	80	26	**147**	153	**150**	**94**	27.4	7.8	1.0266	23.2	26	16	14
		March 31†				*146*	*145*	*146*	*94*	*10.7*	*5.9*	*1.0229*	*41.9*	9	*12*	*31*
A 17	Acute nephritis	March 7	98.2	102	24	156	157	157	**96**	25.5	**7.2**	**1.0257**	**49.3**	24	15	**22**
A 18	Eclampsia	February 16	101.0	140	20	153	**143**	**148**	**97**	**15.6**	**6.8**	**1.0254**	**46.5**	**14**	**14**	**23**
A 19	Bichloride of mercury poisoning	June 10	98.2	72	22	**147**	**166**		**76**	**23.5**	**9.0**		**282.0**	**22**	18	**41**

A 20	Glomerular nephritis	February 25	98.6	88	14	138	137	138	83	6.5	7.9	1.0294	25.8	5	16	34
A 21	Broncho-pneumonia; subacute nephritis	May 5	103.6	128	28	155	156	156	90	15.7	9.0	1.0303		14	18	34
A 22	Chronic nephritis; cerebrospinal and cardio-vascular syphilis	April 23	100.0	120	24	147	142	145	88	29.2	6.6	1.0262	144.0	28	13	16
		May 5	100.0	132	30	143	142	143	86	21.5	7.5	1.0276		20	15	22
A 23	Rheumatic fever	March 18	100.0	124	24	152	155	154	94	27.7	8.6	1.0293	24.9	26	17	17
A 24	Acute rheumatic fever	April 5	98.6	80	20	165	163	164	101	25.4	8.4	1.0274	24.6	24	17	22
A 25	Rheumatic fever	April 8	100.4	98	30		151		95		7.0	1.0253		(27)	14	(15)
A 26	Recurrent rheumatic fever; chorea	March 7	99.0	92	20	154	156	155	97	27.5	8.6	1.0286	24.5	26	17	15
A 27	Rheumatic fever	April 5	98.4	128	22	163	158	161	92	26.2	8.2	1.0271	28.4	25	17	27
A 28	Lung abscess	February 16	99.8	84	28	147	146	147	98	26.5	7.5	1.0294	26.8	25	15	9
A 29	Carcinomatosis	April 21	101.2	98	26	154	152	153	98	27.3	6.8	1.0249	37.5	26	14	15
A 30	Cardiac decompensation; auricular fibrillation	February 5	97.0	6	54	152	155	154	103	26.2	7.6	1.0288	34.5	25	15	11
A 31	Pernicious anemia	February 9	100.8	98	26	159	155	157	95	30.9	7.6	1.0249	29.4	30	15	17
A 32	Lung abscess	March 7	101.4	104	24	161	153	157	113	27.3	8.9	1.0291	35.2	26	18	0
		March 18	98.4	80	24	152	156	154	98	29.6	9.0	1.0298	18.4	28	18	10
A 33	Peritonitis	March 7	100.4	100	20	161	156	159	96	33.5	8.3	1.0261	44.9	32	17	14
A 34	Peritonitis	March 17	100.0	100	20	160	162	161	101	23.0	8.1	1.0281	57.0	22	16	22
A 35	Subacute infective endocarditis	April 28	101.0	144	40	146	149	148	97	23.0	6.1	1.0255		22	12	17
A 36	Chronic lymphatic leukemia	March 10	98.4	104	19	145	148	147	100	26.8	6.7	1.0243	34.8	.26	14	7
A 37	Malaria	June 8	99.2	92	26	154	157	156	95		6.9			(27)	14	(20)
A 38	Amoebic dysentery	June 3	99.2	80	20	150	154	152	95		7.0			(27)	14	(16)

* Rectal temperatures.

† Analysis of pleural effusion.

MATERIAL AND METHODS

Thirty-three specimens of blood serum and two pleural fluids from twenty-nine patients on the Medical Services at the Pennsylvania Hospital and at the Hospital of the University of Pennsylvania in Philadelphia were examined.

The technical methods used were those employed in our previous study (1926). Chemical determinations of total base by the method of Stadie and Ross have been compared further with conductivity determined with the Christiansen ionometer corrected by the formula of Gram and Cullen. We find on further study the corrected conductivity times 1.17 equals in the average the total base chemically determined and we use this factor at present instead of 1.13 reported in our previous study.

An approximate figure for the base bound by protein [B Pr] has been calculated by equation 54 of Hastings, Salvesen, Sendroy and Van Slyke (1927).

$$[\text{B Pr}] = 0.97\,[\text{Pr}]\,(\text{pH} - 5.26)$$

taking for pH, 7.35. Hence

$[\text{B Pr}]_{\text{m.Eq.}}$ is approximately $2.0\,[\text{Pr}]_{\text{gms./100 cc.}}$
$[HCO_3]_{\text{m.Eq.}}$ is taken as equal to $[CO_2]_{\text{mM.}} - 1.3$
Residual anion = $[\text{B}] - ([Cl^-] + [HCO_3] + [\text{B Pr}])$

Protein was determined with the Abbe refractometer as in our previous study (1926). Specific gravity was determined at 20°C. in a 2 cc. pyknometer. In graph 1 we have plotted all measurements of protein against specific gravity. The correlation is fair.

RESULTS

Brief descriptions of each case studied are appended at the close of the paper. The individual analyses are tabulated in table 1. Values outside of the normal range are in bold-faced type. Groups of cases representing lobar pneumonia, tuberculosis and rheumatic fever are tabulated in table 2 showing the maximum, minimum and number of observations outside the normal range for each electrolyte in each group.

The results will be discussed according to disease groups.

Tuberculosis and pleural effusion

(Table 1, cases A 10 to A 16.) All of these patients except A 16 were diagnosed as tuberculous. The range of values and the number outside the normal range are shown for the group in table 2. Chloride concentration is abnormally low in 9 of the 10 observations and low

TABLE 2

Range of values and number of observations ouside the normal range for each disease group

	Number of patients	Number of sera	Base			Cl			HCO_3			B Pr			Residue		
			Range	Number outside normal range		Range	Number outside normal range		Range	Number outside normal range		Range	Number outside normal range		Range	Number outside normal range	
				Below	Above		Below	Above		Below	Above		Below	Above		Below	Above
			m. Eq. per liter	*sera*	*sera*	*m. Eq. per liter*	*sera*	*sera*	*m. Eq. per liter*	*sera*	*sera*	*m. Eq. per liter*	*sera*	*sera*	*m. Eq. per liter*	*sera*	*sera*
Normal.....			163			108			32			18			18		
			151			98			23			15			4		
Lobar pneumonia before crisis*..	14	19	155			102			26			18			20		
			129	18	0	80	16	0	19	2	0	12	10	0	8	0	1
Tuberculosis and pleural effusion†....	7	10	158			100			26			19			16		
			142	8	0	85	9	0	22	1	0	10	1	3	8	0	0
Rheumatic fever‡.....	5	5	164			101			26			17			27		
			151	1	1	92	4	0	24	0	0	14	1	0	15	0	1

* These observations are all of those reported by Sunderman, Austin and Camac (1926) on or before the day of crisis.

† See table 1: A 10, A 11, A $12^{1,2}$, A $13^{1,2}$, A 14, A $15^{1,2}$, A 16.

‡ See table 1: A 23, A 24, A 25^{1}, A 26, A 27.

normal in 1. Of the 10 observations on base, 8 were below the normal range. The change in electrolytes is similar to that observed in pneumonia but less in degree.

In two cases (A 15, A 16) from which pleural fluids were analyzed the electrolyte concentration was approximately the same as in the blood serum taken at the same time with the exception of the decreased protein and CO_2 content in case A 16.

Renal

(Table 1, cases A 17 to A 22.) The electrolyte distribution in two cases of acute nephritis (A 17 and A 21) differed from that in the pneumonia group in the fact that the reduction in chloride was compensated by an increase of one or more of the residual anions without any appreciable change in the total base. This is similar to Atchley's observations with ligation of dogs' ureters. On the other hand in our case of mercurial poisoning (A 19) with anuria for six days, two-thirds of the decrease in chloride and bicarbonate concen-

TABLE 3

Analyses of serum from mercurial poisoning case A 19

	Case A 19	Average normal	Difference
	m.Eq. per liter	*m.Eq. per liter*	*m.Eq. per liter*
Total base (chemically determined)	146.5	154.7	−8.2
BCl	76.2	104.0	−27.8
$BHCO_3$*	22.2	25.8	−3.6
$[B_2HPO_4 + BH_2PO_4]$†	9.4	3.0	+6.4
B_2SO_4	12.0	1.0	+11.0
B Pr‡	18.0	16.0	+2.0
B organic acids§	8.4	4.9	+3.5
pH	7.31		
Non-protein nitrogen mgm. per 100 cc.	282.0		
Degrees depression in freezing point	0.63°C.		

* $[BHCO_3] = [CO_2] - 1.27$.

† $\frac{B_2HPO_4}{BH_2PO_4}$ at pH 7.31 $= \frac{77}{23}[PO_4] = 5.3$ mM/L; $[B_2HPO_4 + BH_2PO_4] = 9.4$ m.Eq.

‡ B Pr = 0.97 (Pr) (pH − 5.26) (Pr) = grams protein per 100 cc.

§ Calculated by difference.

tration in the blood serum was accounted for as is shown in table 3 by increase in serum phosphate, sulphate and organic acid and one-third by decrease in the total base chemically determined.[2] There occurred in this serum a marked discrepancy between total base chemically determined and the serum conductivity so that it would not be permissible to average the value by the two methods. The cause for the discrepancy is not clear. The relative rise in phosphate, sulphate and organic acid in the serum in this case with displacement

[2] This case has been presented from a somewhat different aspect by J. M. Hayman, Jr. and J. T. Priestley, Am. J. Med. Sci., 1928 (in press). The Importance of a Diuresis in the Treatment of Certain Cases of Mercuric Chloride Poisoning.

of chloride is probably to be attributed to the loss of the selective excretion through the kidneys of the former group of anions which are constantly liberated in the metabolism. That such selective excretion normally occurs is indicated by the fact that the ratio of the normal daily excretion of phosphate, sulphate and organic acid in the urine to the daily excretion of chloride is many times greater than the ratio of the normal concentration of the former group in the serum to the concentration of chloride in the serum. In our two cases of advanced chronic glomerulo-nephritis (A 20 and A 22) there was reduction of total electrolyte which accounted for about half of the combined loss of chloride and bicarbonate.

Rheumatic fever

(Table 1, cases A 23 to A 27.) Although five cases of rheumatic fever is scarcely a sufficient number from which to draw any conclusion, it would appear that the total concentration of base was practically unaffected but that the base bound by chloride was somewhat reduced.

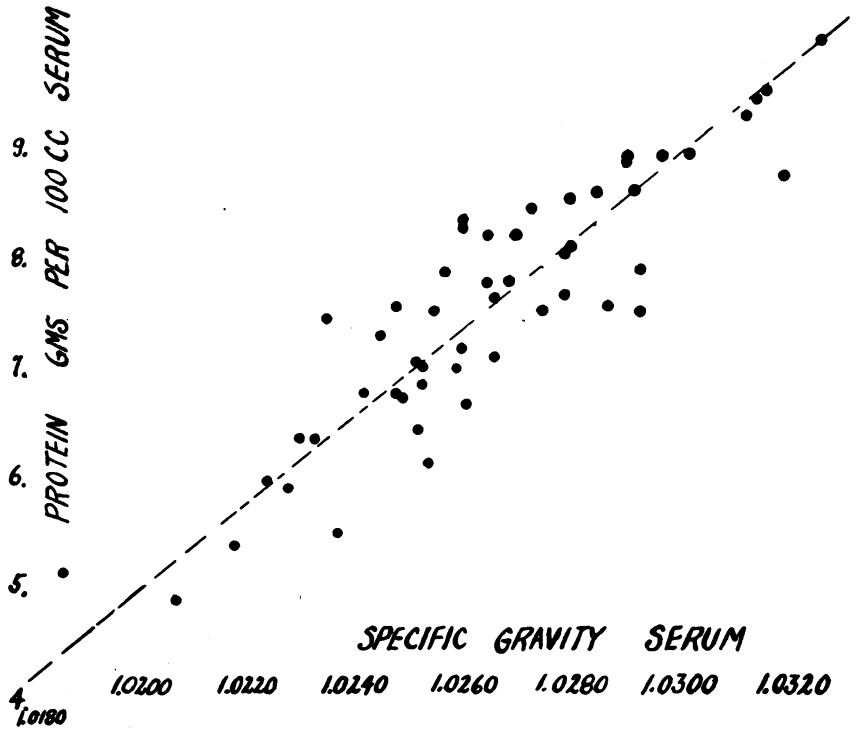
Miscellaneous

(Table 1, cases A 28 to A 38.) We have included here two cases of peritonitis (A 33 and A 34) following appendectomy. These cases received nothing by mouth, but were getting daily hypodermoclysis of saline solution. They gave no evidence of peristalsis, and were very acutely ill, nevertheless, the blood electrolyte equilibrium was little disturbed; serum Cl was normal in A 34 and only slightly low in A 33. This is interesting in view of the hypochloremia reported by Haden in cases of intestinal obstruction following strangulation of which we have none. Whether in our cases the normal or almost normal chloride concentration was maintained by the daily saline hypodermoclysis received requires further investigation.

In the patient with amebic dysentery (A 38) there was practically no significant alteration in the serum acid-base equilibrium. This patient had more than ten stools per day and the dry skin and mucous membranes suggested an anhydremia at the time when the blood was taken; however, the serum salt distribution was practically normal and the serum protein low, the latter probably as a result of the poor state of nutrition.

Changes in CO_2 content

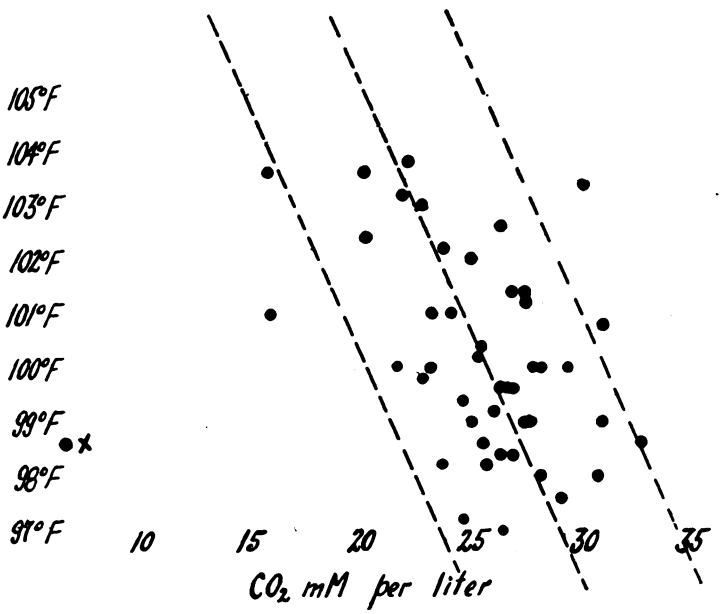
A recognizable though slight correlation between the concentration of CO_2 in the serum and the patients' temperatures is apparent in our cases as shown in graph 2 which includes also our cases of pneumonia. It was found by Stadie, Austin and Robinson (1925) that,



GRAPH 1. PROTEIN REFRACTOMETRICALLY OF ALL SERA, AGAINST SPECIFIC GRAVITY

at either constant CO_2 tension or constant pH, an elevation of the temperature lowers the carbon dioxide capacity about 1 mM. for every 4.6°F. If the body temperature at the time when the blood was withdrawn in our cases be plotted as ordinate, and the CO_2 content be plotted as abscissae, it will be seen in graph 2 that the general trend of the CO_2 content was toward fall of CO_2 content with rise

in temperature but that the magnitude is greater than that found in the study quoted, in vitro at constant pH or constant CO_2 tension, and must be due therefore to other factors such as acidosis or hyperpnea correlated with the increase of temperature. In one case marked x (graph 2) which was entirely outside of the general range, the patient was in uremic coma, with convulsions and a marked acidosis.



GRAPH 2. PATIENTS' TEMPERATURES AT TIME OF BLEEDING, AGAINST CO_2 CONTENT OF SERA

CONCLUSIONS

In tuberculosis, pleural effusion, in chronic glomerulo-nephritis and in mercurial poisoning there was decrease in both base and chloride of the blood serum with the decrease in chloride tending to be relatively greater. The magnitude of the depression of chloride in the mercurial poisoning case far exceeded that in the other cases.

In acute nephritis, eclampsia and rheumatic fever, there was a decrease in chloride without significant change in base.

Reduction in bicarbonate occurred in certain individuals but was not clearly characteristic of any of these groups.

Reduction in refractive index was observed in the chronic glomerulonephritics. A tendency to abnormal variations, sometimes high, sometimes low in refractive index was observed in the other pathologic cases.

Elevation of the temperature was generally associated with a lowered CO_2 content. This was greater than could be accounted for by the change in base bound by protein with change in temperature and must be attributed either to acidosis or to hyperpnea.

The series as a whole and the case of mercurial poisoning in particular suggest the readiness with which chloride is reduced in the serum to make way for other anions.

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CASE HISTORIES

Case A 10 (1502). Age 17, white male. Admitted March 13, 1927. Tuberculous meningitis. Symptoms began 10 days before admission. At time of blood examination he was irrational with hyperactive reflexes, cervical rigidity and characteristic changes in spinal fluid. No tubercle bacilli were demonstrated.

Case A 11 (1442). Age 23, white, female. Admitted March 10, 1927. Left-sided pleural effusion; symptoms dating from two days before admission; 1750 cc. aspirated. X-ray evidence of left apical cavity. Considered tuberculous but organisms not demonstrated.

Case A 12 (1532). Age 27, colored, male. Admitted March 15, 1927. Died April 3, 1927. Diagnosis at autopsy: Advanced tuberculous mesenteric, retroperitoneal, retropelvic and mediastinal lymphadenitis; tuberculous peritonitis and suppurative pneumococcic peritonitis; moderate proliferative tuberculosis of lungs and liver; bilateral broncho-pneumonia; bilateral pleural effusion; suppurative pneumococcic pericarditis; ulcerative tuberculous proctitis; tuberculous femoral adenitis. During hospitalization 1500 cc. of fluid were removed from peritoneal cavity and 720 cc. from left pleural cavity.

Case A 13 (192). Age 17, colored, female. Admitted December 29, 1926. Acute proliferative pulmonary tuberculosis rapidly extending in both lungs at time of blood examination.

Case A 14 (955). Age 55, colored, male. Admitted February 11, 1927. Died April 28, 1927. Diagnosis at autopsy: Generalized miliary tuberculosis, fibrous adhesive peritonitis; tuberculous pleurisy; syphilitic aortitis; myocarditis and cardiac hypertrophy; generalized arteriosclerosis. The Wassermann reaction had been strongly positive. During hospitalization 1000 cc. of fluid were removed from peritoneal cavity and in two aspirations 2750 cc. from right pleural cavity.

Case A 15 (1091). Age 21, colored, female. Admitted February 21, 1927. Pulmonary tuberculosis with left pleural effusion; tubercle bacilli in sputum. During hospitalization 900 cc. of pleural fluid were removed from the left pleural cavity.

Case A 16 (1728). Age 19, white male. Admitted March 25, 1927. Left pleural effusion following a cold three weeks before admission. Fluid aspirated four times in first week of hospitalization, a total of 2600 cc. A pure culture of *streptococcus hemolyticus* was obtained from the fluid; no tubercle bacilli were found. Temperature 98.2 to 102.3; leucocyte count 10,000 to 34,000.

Case A 17 (1297). Age 17, white, male. Admitted March 2, 1927. Acute. nephritis following a cold one month before admission. Temperature 97 to 101. Leucocytes 12,000 to 24,000. Phthalein excretion, 5 per cent in 40 minutes, 25 per cent in 6 hours. Blood urea nitrogen, 30.4 mgm. per 100 cc. falling to 9.2.

Case A 18 (4951). Age 28, white, female. Admitted February 16, 1927. Eclampsia developing at term. Blood pressure 140 systolic, 100 diastolic. Blood urea nitrogen 33 mgm. per 100 cc. Blood taken for analysis following the eighth convulsion and while in coma.

Case A 19 (S-100). Age 38, white, male. Admitted to hospital May 22, 1927, and discharged July 4, 1927. On May 22nd the patient took five large bichloride of mercury tablets. Twenty minutes later he was given milk and in one hour he had emesis after taking six raw eggs. On admission to the hospital two hours after taking the poison, he was given gastric lavage. At this time he had developed abdominal cramps and diarrhea. The patient was completely anuric from the time of admission until May 28th during which time he received from 3 to 6 liters of fluid daily. From May 28th to June 6th the urine output varied from 400 to 750 cc. daily and contained a trace of albumin, occasional casts, and red blood cells. From June 8th until the day he was discharged the urine output varied from 400 to 3750 cc. averaging approximately 1200 cc. and contained no casts, a trace of albumin and, after June 13th, sugar in traces. Phthalein test on June 30th showed excretion of 1 per cent in two hours. Edema of the face was observed on day of admission. It became more marked and generalized on May 26th and had disappeared by June 2nd. On May 24th the patient had convulsions and was irrational, becoming rational two days later. The eye grounds showed no hemorrhages, exudates, nor choking of discs. Daily blood urea nitrogen determinations increased steadily from 43 mgm. per 100 cc. on May 23rd to 247 mgm. on June 11th and then decreased gradually to 85 mgm. on July 2nd.

Case A 20 (815). Age 39, colored male. Admitted February 4, 1927. Died February 26, 1927 during a convulsion. Diagnosis: Chronic glomerulo-nephritis with failing circulation. Blood pressure—220 systolic, 110 diastolic. Fixation of specific gravity. Phthalein excretion 5 per cent in first hour, less than 5 per cent in second hour. Blood urea nitrogen 120 to 300 mgm. per 100 cc. Blood creatinin 13 to 16 mgm. per 100 cc. Hemoglobin 55 per cent.

Case A 21 (1912). Age 42, white, male. Admitted April 5, 1927. Bronchopneumonia with subacute nephritis. Patient was admitted slightly delirious, complaining chiefly of pain in the left knee. The left patella had been fractured three days previously. The patient gave a history of nocturia four or five times a night during the past month. His blood pressure was 124/72. Heart was slightly

enlarged to the left and the sounds were scarcely audible. The patient's face was pale and puffy and there was moderate generalized subcutaneous edema. He became progressively stuporous, developed broncho-pneumonia and died May 7, 1927. Blood for chemical analysis was taken two days before death. The urine showed a constant heavy cloud of albumin with granular casts and many red blood cells. Phthalein excretion was 5 per cent in the first hour and 10 per cent in the second hour. The blood urea nitrogen determinations ranged from 57 to 132 mgm. per 100 cc.; the creatinine ranged from 3.2 to 8 mgm. per 100 cc. Blood Wassermann was moderately positive. The hemoglobin was 40 per cent, red blood cells 1,500,000; white blood cells 15,000. Histological examination of kidney: The tubules are very prominent and distended, with cells thin and nuclei often not visible; in the lumen cellular debris and disintegrating red blood cells are seen. There was no evidence grossly of hydronephrosis. In the capsules of Bowman a few cells and precipitated albumin are seen. A few of the glomeruli are hyaline. There is widespread increase of interstitial fibrous tissue with a few small areas of mononuclear infiltration and irregularly distributed areas in which the blood vessels are distended with blood. Anatomical diagnosis: Subacute nephritis; acute cystitis; fractured left patella; pericardial effusion (40 cc.); broncho-pneumonia.

Case A 22 (2127). Age 51, white, male. Admitted April 18, 1927. Patient had had tabes dorsalis for the past 7 years. Blood and spinal fluid Wassermann were very strongly positive. On admission patient had complete paralysis below the umbilicus. His urine showed a constant cloud of albumin with many pus cells and a fixed specific gravity. The blood pressure was 120/60. The blood urea nitrogen steadily increased from 63.5 to 277 mgm. per 100 cc. and the creatinine from 2.6 to 11.8 mgm. per 100 cc. Patient had increasing congestion of both lungs and died suddenly six weeks after admission. Histological examination of kidney: Generalized increase of fibrous tissue with focal collections of round cells and leucocytes. Glomeruli more widely separated than normal and in some instances hyaline. Adjacent areas show slight compensatory hypertrophy of tubules and glomeruli. Vessel walls thickened. Anatomical diagnosis: Cerebrospinal syphilis; multiple abscesses of the kidneys; syphilitic aortitis with fusiform aneurysmal dilatation of ascending aorta; pulmonary edema; passive congestion and fatty degeneration of liver.

Case A 23 (1566). Age 22, colored, male. Admitted March 16, 1927. Rheumatic fever with onset two days before admission. Leucocytes 6000 to 12,000. Wassermann strongly positive. Received salicylate.

Case A 24 (1598). Age 29, white, female. Admitted March 18, 1927. Rheumatic fever with onset two months before admission. Leucocytes 6600 to 13,000. Had received sodium salicylate and sodium bicarbonate for two weeks before blood was taken for analysis.

Case A 25 (1920). Age 26, white, female. Admitted April 5, 1927. Recurrent rheumatic fever; rheumatic endocarditis; pericarditis with effusion; cardiac

decompensation. The patient was orthopneic. There were petechiae over the trunk and right arm. Blood pressure 96/48. Hemoglobin 65 per cent. Leucocytes 12,000. Had received sodium salicylate and sodium bicarbonate for two days before blood was taken for analysis.

Case A 26 (1277). Age 14, white, female. Admitted March 1, 1927. Patient had had rheumatic fever each winter for the last four years and chorea during the past two years. She had a marked mitral valvular lesion and coarse twitchings of all extremities. At the time the blood was taken the patient was afebrile.

Case A 27 (1781). Age 14, white, female. Admitted March 28, 1927. Died April 18, 1927. Recurrent rheumatic fever with cardiac involvement for seven years. Orthopneic, pallid, with large tender liver and spleen. Blood pressure 124 systolic, 40 diastolic. Hemoglobin 75 per cent. Leucocytes 9,000. Developed pericardial friction, petechiae and increasing heart failure. Diagnosis at autopsy: Rheumatic pancarditis with aortic, mitral and tricuspid endocarditis; cardiac dilatation; chronic passive congestion of lungs, liver and spleen.

Case A 28 (703). Age 24, colored, male. Admitted January 28, 1927. Pulmonary abscess in right lower lobe with onset of symptoms one week before admission. Culture of sputum showed a *Streptococcus viridans* predominating. Leucocytes 13,000 to 20,000 Sputum 17 to 37 ounces daily.

Case A 29 (1859). Age 60, white, female. Admitted April 1, 1927. Carcinoma of head of pancreas; secondary metastases to liver and lungs: intense jaundice. Onset of symptoms with jaundice three months before admission.

Case A 30 (821). Age 38, white, female. Admitted February 4, 1927. Rheumatic pancarditis since adolescence; auricular fibrillation since 1915; marked heart failure, ascites and subcutaneous edema.

Case A 31 (1373). Age 53, white, female. Admitted February 7, 1927. Pernicious anemia of four years duration. Hemoglobin 65 per cent; red blood cells 1.9 million; leucocytes 4,400. No free HCl in gastric contents. Blood taken for analysis before transfusion.

Case A 32 (1337 . Age 16, white, male. Admitted March 4, 1927. Pulmonary abscess in right upper lobe following tonsillectomy two weeks previously. Through bronchoscope obtained a pure culture of *Micrococcus catarrhalis*. Sputum 10 to 15 ounces daily.

Case A 33 (1346). Age 28, colored, male. Admitted March 5, 1927. Acute gangrenous perforating appendicitis with generalized peritonitis; onset March 1; operation March 5. Wassermann strongly positive. From March 5 to 7 when blood was taken for analysis the patient received 180 cc. of 5 per cent glucose and 2 per cent $NaHCO_3$ every three hours by rectum.

Case A 34 (1530). Age 30 white, male. Admitted March 15, 1927. Acute gangrenous perforating appendicitis with generalized peritonitis; operation on admission. During following two days until blood was taken for analysis patient had received 100 cc. physiological saline by hypodermoclysis and continuous enteroclysis with tap water.

Case A 35 (2241). Age 20, white, male. Admitted April 25, 1927. *Streptococcus viridans* endocarditis; rheumatic pancarditis. Blood 100 colonies per cubic centimeter. Hemoglobin 70 per cent. Leucocytes 12,700. Pericardial frictions. Liver and spleen large and tender.

Case A 36 (S-102). Age 52, colored, male. Admitted February 23, 1927. Chronic lymphatic leukemia. Symptoms noted for one year. Generalized adenopathy. Enlarged liver and spleen. Red blood cells 3.3 millions. Leucocytes 600,000; 98 per cent lymphocytes.

Case A 37 (3055). Age 40, white, male. Admitted June 8, 1927. Tertian malaria. Onset three days before admission on arrival from West Indies. Paroxysm each evening. Blood taken 8 hours following a chill.

Case A 38 (2907). Age 41, colored, male. Admitted June 1, 1927. Amebic dysentery. History of amebic dysentery in Argentina 19 years before. Syphilis 7 years before. Progressive loss of weight and appetite for 14 months. Reappearance of dysentery 3½ months before admission. Wassermann strongly positive. Skin very dry. Hemoglobin 75 per cent. Leuoocytes 17,000; polymorphonuclears 70 per cent.