In none of the cases had there been recognizable clinical manifestations of splenic involvement. The spleen was not enlarged. In 12 cases with healed miliary tubercles of the spleen no other tuberculous process was found.

The splenic infection was a hematogenous one arising most commonly from antecedent foci in the lungs or peribronchial glands. Those cases in which no primary tuberculous focus was found probably had a similar mode of origin in which, however, the initial focus was of minor extent unrecognizable at the time of autopsy. We would point out that the fibroses which are observed in anthracotic peribronchial glands are difficult of analysis as indicating a preceding infectious origin of the fibrosis.

The healed splenic tubercles are recognized only by careful search and complete gross sectioning of the tissues of the organ. The presence of the healed miliary tubercles of the spleen indicates the frequency of a tuberculous bacteriemia from which the tissues may entirely recover. The different organs demonstrate a variable resistance to the tuberculous infection. Reinfection may take place in the spleen.

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THE ASSOCIATION OF GASTRIC SYMPTOMS IN NEPHRITIS WITH RETENTION OF NITROGENOUS WASTE PRODUCTS IN THE BLOOD.¹

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SINCE the time of Richard Bright it has been recognized that renal disorders are accompanied by digestive disturbances. The

¹ Read before the American Gastro-enterological Association, Washington, D. C.

pathological basis for this association has been the source of much speculation for the reason that there are no physiological facts which adequately explain it. The symptoms occur in all stages of the disease, from the most incipient to the furthest advanced. Many cases of unrecognized nephritis have only symptoms of dyspepsia. We have been particularly impressed with the number of cases of latent nephritis sent to the hospital with a diagnosis of gastric ulcer or of toxic vomiting. The more common symptoms from which patients of this type complain are nausea, vomiting, loss of appetite, flatulency, abdominal distress, usually without definite relationship to meals, and headaches, frequently of the migrainous type. Owing to the depressed gastric secretion the diagnosis of asthenic gastritis is occasionally made. On account of the toxic character of the vomiting these patients are frequently told that they are suffering from enterogenous intoxication.

In view of the important advances recently made in the estimation of the various nitrogenous waste products which accumulate in the blood (and tissues) in nephritis it appeared of interest to compare the type and extent of this retention with any gastric symptoms which might occur. The end-products of nitrogenous metabolism which deserve consideration are the uric acid, urea, and creatinin. As has already been pointed out in contributions from this institution,² uric acid is normally the most difficult and creatinin the easiest to eliminate. It is not surprising, then, that very early cases of nephritis should show a retention of uric acid without any corresponding retention of urea or creatinin, while cases in the last stages of the disease should be the only ones in which a decided increase in creatinin is encountered. Following this scheme we have divided the cases we propose to discuss into two groups, viz., those showing high creatinins and others in which the high uric acid was the prominent feature. This arrangement has been made for the reason that the retention of creatinin has been found to be of great prognostic value in nephritis³ while the uric acid appears to be an early diagnostic sign. The cases showing marked nitrogen retention, particularly with respect to the creatinin, will first be considered. The similarity in the gastric symptoms in this group of 10 fatal cases (see Table I) is very striking. It is worthy of note that 3 of these cases (II—T. D., VI—E. P., and VII—J. W.) were called to my attention on account of their gastric symptoms. Although examination at once revealed the fact that these patients were suffering from nephritis, the gravity of the situation was not appreciated until a blood examination disclosed a marked retention of creatinin. These cases greatly impressed me and, in a measure, suggested the title of the present paper. Their histories are worthy of brief review:

² Myers, Fine, and Lough: *Arch. Int. Med.*, 1916, xvii, 570.

³ Myers and Lough: *Arch. Int. Med.*, 1918, xxi, 536.

CASE II.—T. D., male, aged thirty-four years, at the time of admission to the hospital, on January 13, 1915, was able to be up and about. His chief complaint was vomiting and pain in the epigastrium of five to six months' duration. A chemical blood examination was made and gave a creatinin of 7.8 mg. to 100 c.c., showing that the patient was in the terminal stages of interstitial nephritis. He died on March 23, 1915.

CASE VI.—E. P., female, aged forty years, was admitted to the hospital with a history of persistent vomiting for the previous two months. Physical examination showed a fairly well-nourished woman with no evidence of edema. The chemical examination of the blood not only established the diagnosis of nephritis, but the high creatinin (8 mg. to 100 c.c. of blood) gave a fatal prognosis. The woman died six weeks after leaving the hospital.

CASE VII.—J. W., male, aged thirty-four years, walked to the hospital and was admitted on February 22, 1915, complaining of headaches, nausea, vomiting, and constipation. A blood examination gave a creatinin of 8.3 mg. Clinically the patient quickly improved and was continuously up and about the ward. He repeatedly requested to be allowed to go home, but as the creatinin of the blood remained high, he was persuaded to remain. He was up on April 14, but on becoming faint was put to bed, gradually declined, and died on April 16, 1915.

Figures for the CO_2 -combining power of the blood (according to Van Slyke's method) are given for 5 of the 10 cases. Although these are all low it is scarcely believed that the acidosis bore a causal relation to the vomiting. Although we have no definite evidence showing that the nitrogenous waste products have such a relationship it is of some significance that the gastric symptoms seem to vary, to a certain extent, with the amount of retention.

In Table II are tabulated 12 cases with gastric disturbances in which the special feature of the blood analysis is the high uric acid. Many of these cases showed symptoms directly comparable with the preceding group, although none of them was terminal. In general the symptoms were more mild than the preceding, but it seems safe to conclude that they were the early manifestations of the same general condition.

In cases of migraine the estimation of the uric acid of the blood has been of great value in detecting a latent nephritis. Four years ago we were impressed with a case of this character.

D. L., aged forty-five years, had suffered from periodic attacks of typical migraine for the previous five years. These attacks were intense and had resisted all forms of treatment both here and abroad. Examination of his blood proved to be normal with the exception of a uric acid retention of 5 mg. per 100 c.c. When treatment was instituted to reduce the uric acid accumulation in the blood the sick headaches suddenly stopped and have not returned. The only

TABLE I.—GASTRIC SYMPTOMS AND BLOOD FINDINGS IN FATAL CASES OF NEPHRITIS.

Case.	Age.	Sex.	Urine.		Blood-pressure.		Phthalein 2 hrs. Per cent.	CO ₂ combining power. Per cent. Ca. to 100	Blood- sugar. Per cent.	Uric acid.		Creatinine.	Gastric symptoms.	Result.
			Albumin.	Coats.	Systolic.	Diastolic.				Mg. to 100 c.c. of blood.	Mg. to 100 c.c. of blood.			
I—S. H.	37	M	++	++	220	138	0	..	0.14	14.3	202	22.2	Vomiting; gaseous eructations	Died.
II—T. D.	34	M	+++	+	210	105	0	..	0.14	14.3	152	20.5	Nausea; vomiting;	Died.
III—I. D.	17	F	++	++	182	138	0	..	0.10	27.0	200	20.0	epigastric pain	Died.
IV—P. J.	43	M	+++	-	..	..	0	31	0.25	12.5	102	20.0	Vomiting; abdominal pain	Died.
V—E. C.	50	F	++	-	210	100	..	..	0.09	22.4	230	10.7	Vomiting 2 mos.; duration	Died.
VI—E. P.	40	F	+++	+	205	145	0	30	0.10	4.2	40	12.5	Vomiting 2 yrs.; constipation; poor appetite	Died.
VII—J. W.	34	M	++	++	220	100	trace	..	0.14	8.7	144	11.0	Vomiting 4 mos.; epigastric pain; constipation	Died.
VIII—O. G.	22	M	++	-	225	115	23	45	0.13	7.3	05	4.5	Nausea and vomiting; constipation	Died.
IX—P. L.	47	F	+	-	127	85	13	27	0.10	13.3	28	4.3	ing: constipation	Died.
X—M. S.	02	M	++	+++	105	100	18	20	0.08	7.3	30	4.0	Vomiting; no relation to food intake; constipation	Died.

Normal blood findings: CO₂ combining power 54 c.c. per 100, sugar 0.10 per cent., and uric acid 2.3, urea N 12-15, and areatinina 1-2.5 mg. to 100 c.c.

TABLE II.—GASTRIC SYMPTOMS AND BLOOD FINDINGS IN NON-FATAL CASES OF NEPHRITIS.

Case.	Age.	Sex.	Urine.		Blood-pressure.		Phthalein 2 hr.	Blood- sugar.	Uric acid.	Urea nitrogen.	Creatinine.	Gastric symptoms.	Result.
			Albumin.	Cast.	Systolic.	Diastolic.							
I—M. S.	41	F	+	++	140	75	53	0.00	0.7	22	1.5	Vomiting; abdominal pain; diarrhea	Improved.
II—A. R.	57	M	-	-	130	70	40	0.12	0.3	22	2.5	Gaseous eructations; no vomiting; constipation	Unimproved.
III—J. C.	00	M	+++	-	150	100	20	0.11	0.3	40	1.5	Vomiting; diarrhea	Unimproved.
IV—P. P.	28	M	+	+	135	70	01	0.11	0.3	15	2.4	Vomiting; regurgitation of food; appetite poor	Improved.
V—M. S.	40	F	+	+	210	150	51	0.12	0.1	12	2.0	Vomiting; flatulency; constipation	Unimproved.
VI—L. B.	50	M	-	-	100	05	40	0.11	5.4	11	0.0	Vomiting; no relation to food intake	Improved.
VII—N. B.	71	M	+	+	130	75	54	0.11	5.4	17	2.8	Occasional vomiting	Improved.
VIII—A. B.	38	F	-	+	110	80	50	..	5.0	27	3.3	Vomiting; gaseous eructations	Improved.
IX—G. G.	01	M	+	+	210	110	48	0.11	4.8	22	3.4	Nausea and vomiting; constipation	Improved.
X—L. K.	72	M	+++	++	200	100	..	0.12	4.0	37	1.8	Epigastric pain	Improved.
XI—F. S.	07	M	+	+	125	00	10	..	4.2	10	1.5	Occasional vomiting	Improved.
XII—S. F.	58	F	+	+	100	70	44	0.12	3.7	22	3.2	Nausea and vomiting; epigastric pain	Improved.

other evidence of nephritis during the past four years has been an occasional faint trace of albumin in the urine with a systolic blood-pressure of 155 and a diastolic of 95. These conditions have been transitory.

Aside from the stomatitis and ulcerative colitis of uremia the gastro-enteric symptoms of nephritis are probably central. Evidence is lacking that uric acid, urea, and creatinin are toxic in themselves. The amount of their retention is a probable index, however, of the accumulation of unknown substances which may possess some toxic importance. A poor oxidation of creatinin in the absence of its excretion would give methyl guanidin, a substance of known toxic properties. The possible relation of such substances to the gastric symptoms of nephritis is, at present, only problematical.

NOTES ON CASES IN TABLE I.

CASE I.—S. H., male, aged thirty-seven years. Headaches; dizziness; constipation; alcoholic; had stomach trouble five months; vomiting; edema of feet and ankles. Diagnosis: chronic interstitial nephritis; died.

CASE II.—T. D., male, aged thirty-four years. Vomiting; epigastric pain for five to six months; alcoholic; constipation; had scarlet fever; weight in abdomen after food intake. Diagnosis: chronic interstitial nephritis; died.

CASE III.—I. D., female, aged seventeen years. Frontal headaches; epigastric pain; dizziness; vomiting; dyspnea; tachycardia. Diagnosis: chronic interstitial nephritis; died.

CASE IV.—P. J., male, aged forty-three years. Gastritis; diarrhea; headaches; dizziness; vomiting two months; edema of feet and ankles. Diagnosis: chronic interstitial nephritis; died.

CASE V.—E. C., female, aged fifty years. Dizziness; inability to pass urine; vomiting for past two years; no relation to food intake; constipation; nycturia. Diagnosis: chronic interstitial nephritis; died.

CASE VI.—E. P., female, aged forty years. Vomiting four months with headaches; slight epigastric pain; vomiting; bears no relation to food intake; constipation. Diagnosis: chronic interstitial nephritis; died.

CASE VII.—J. P., male, aged thirty-four years. Syphilis sixteen years ago; marked alcoholic; dyspnea; headaches; vomiting; edema of face and lower extremities fifteen months. Diagnosis: chronic interstitial nephritis; died.

CASE VIII.—O. G., male, aged twenty-two years. Fainting spells; impairment of vision; vomiting two weeks; alcoholic. Diagnosis: chronic interstitial nephritis; died.

CASE IX.—P. L., female, aged forty-seven years. Cough for three years; headaches; dizziness; palpitation of heart; constipa-

tion; vomiting; no relation to food intake; arthritis of right elbow. Diagnosis: carcinoma of liver; died.

CASE X.—M. S., male, aged sixty-two years. Headaches; occasional vomiting spells; dyspnea; increased frequency of urination; edema of feet and ankles; arthritis for ten years (?); enlargement of heart to right. Diagnosis: chronic interstitial nephritis, endocarditis; died.

NOTES ON CASES IN TABLE II.

CASE I.—M. S., female, aged forty-one years. Scarlet fever; diphtheria as child; abdominal pain; frequency of urination; diarrhea; vomiting; dyspnea. Diagnosis: neurasthenia; improved.

CASE II.—A. R., male, aged fifty-seven years. Constipation; gaseous eructations; nycturia; painful urination; no vomiting. Diagnosis: carcinoma of stomach and liver; unimproved.

CASE III.—J. C., male, aged sixty-nine years. Vomiting; diarrhea; edema of feet and ankles. Diagnosis: chronic interstitial nephritis; endocarditis; transferred; unimproved.

CASE IV.—P. P., male, aged twenty-eight years. Regurgitation of food; moderate alcoholic; occasional vomiting spell. Diagnosis: gastric ulcer (?); improved.

CASE V.—M. S., female, aged forty-six years. Weakness; anemia; headache; vertigo; cardiac pain; diarrhea; flatulence; epigastric pain; vomiting. Diagnosis: chronic interstitial nephritis; unimproved.

CASE IV.—P. P., male, aged twenty-eight years. Regurgitation of food; moderate alcoholic; occasional vomiting spell. Diagnosis: gastric ulcer (?); improved.

CASE V.—M. S., female, aged forty-six years. Weakness; anemia; headache; vertigo; cardiac pain; diarrhea; flatulence; epigastric pain; vomiting. Diagnosis: chronic interstitial nephritis; unimproved.

CASE VI.—L. B., male, aged fifty years. Headache; pneumonia eight years ago; vomiting spells every ten to fourteen days; no relation to food intake. Diagnosis: chronic interstitial nephritis; cecal stasis; improved.

CASE VII.—N. B., male, aged seventy-one years. Scarlet fever; constipation; pain in chest; difficulty in voiding. Diagnosis: chronic interstitial nephritis; improved.

CASE VIII.—A. B., female, aged thirty-eight years. Headache; stomach trouble seven months' duration; gaseous eructations; dyspnea; vomiting; accentuated P₂. Diagnosis: mucous colitis; improved.

CASE IX.—G. G., male, aged sixty-one years. Constipation; dizziness; vomiting three years; dyspnea; accentuated A₂. Diagnosis: chronic interstitial nephritis; hemiplegia; improved.

CASE X.—L. K., male, aged seventy-two years. Pneumonia; diphtheria; epigastric pain; relieved after food intake. Accentuated A². Diagnosis: chronic pancreatitis; improved.

CASE XI.—F. S., male, aged sixty-one years. Dyspnea; occasional vomiting spell; regardless of food intake; headache; weakness. Diagnosis: chronic interstitial nephritis; improved.

CASE XII.—S. F., female, aged fifty-eight years. Arthritis in all joints (?); headache; dizziness; constipation; pains in shoulder; epigastric pain; nausea and vomiting; jaundice. Diagnosis: chronic interstitial nephritis; improved.

CONCLUSIONS. Gastric symptoms are among the most common early symptoms of nephritis. In cases with obscure gastric disturbances the chemical examination of the blood has been found very valuable. Several cases are reported in which the estimation of the blood creatinin not only showed that the patients were suffering from severe nephritis, but gave a fatal prognosis. In some of the earlier cases the blood uric acid was of value as an early diagnostic sign.

STUDIES IN THE PHYSIOLOGY AND PATHOLOGY OF THE STOMACH AFTER GASTRO-ENTEROSTOMY.¹

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THE operative treatment of ulcer of the stomach or duodenum is in the largest percentage of cases a curative one. Experiences, however, have demonstrated that the continuous repetition of instances demonstrating the splendid results obtained by operative interference is constantly and not infrequently being disturbed by a group of cases in which some of the preoperative symptoms persist after the surgical interference or in which new symptoms of an apparently different nature supervene during or after the period of recovery.

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