UNCINARIAL NEPHRITIS*

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INTRODUCTION

It has long been recognized that anemia and dropsy are common symptoms of the more severe forms of uncinariasis, and edema is usually a prominent feature in fatal cases. Subcutaneous edema, ascites, hydrothorax, and hydropericardium are commonly found, and a general anasarca sometimes occurs. It has been impossible to establish the relation between the presence of hookworms in the intestines and the nephritis suggested by the above symptoms by means of the limited methods of investigation heretofore employed; but albumin, casts, leukocytes and injured epithelial cells have been found in the urine, and typical lesions of chronic parenchymatous nephritis have been found at necropsy in patients who have died in an edematous state.

The present study was undertaken to determine whether patients suffering from uncinariasis, and exhibiting edema, have any constant disturbance of renal excretory function, and whether this function is improved after anthelmintic medication. It had already been observed that, as a rule, the general health of the patient improves as soon as the parasites have been expelled, and that edema and anemia soon disappear. It is known that at least certain of the symptoms may be reproduced in animals by the injection of an emulsion of the heads of hookworms, or of the urine of individuals infested with the parasites. If any derangement of renal excretory function is present, it is probably due to toxic substances generated by the hookworms, and we should expect to find a normal or improved function after the parasites have left the body.

For the purposes of the present study, application has been made of the most recent knowledge concerning renal excretory function. The results of the study contribute support to the hypothesis that there is a form of nephritis due to hookworm infection, and that recovery from the infection is followed by recovery from nephritis.

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The analysis of the following cases supports the findings of Ambard, Widal and Weill concerning the chlorid threshold in nephritis with chlorid retention.

METHOD OF STUDY

1. On the day of entering the hospital, samples of blood and urine of each patient were obtained in the usual manner to determine the Ambard's coefficient and the urea index. The sample of urine was also examined for albumin, casts, leukocytes and sugar. Blood viscosity and systolic and diastolic pressure were also noted. Ophthalmoscopic examination was made at the same time.

2. Salt poor diet (sodium chlorid, 2 gm. daily), rest.[1]

3. When a balance between the chlorid intake (2 gm.) and output was obtained, full doses of digitalin and theobromin were administered in the hope of lowering the chlorid threshold below the point reached on the salt poor diet with rest. At this time, a red cell count was made, and the Ambard's coefficient and urea index were again determined.

4. Sodium chlorid, 10 gm. daily, was administered, and the chlorid excretion was followed. If the added chlorid was excreted with normal rhythm, 20 gm. was administered daily and the excretion was followed. The weight of the patient was observed.

5. Anthelmintic treatment. The indication for discontinuing the treatment was the reduction of the hookworms in the fecal matter to a very small number.

6. Red cell count and the study of the urea coefficients on a salt poor balance.

7. The same as No. 4, plus the determination of the urea coefficients and an ophthalmoscopic examination.

Occasional doses of quinin were given during the time required for the study of each patient, as they had all been suffering from malarial fever before entering the hospital.

In the quantitative examination of the urea, the hypobromite method recommended by Ambard has been employed.

For plasma chlorids, an application of the Volhard principle made by Rappleye [2] was selected; this method has been effective in the hands of Van Slyke.[3]

Blood tests for syphilis were made with the homohemolytic system of Noguchi.[4]

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1. During the stay of each patient urine was collected and examined in twenty-four hour periods in order to estimate the total excretion of sodium chlorid, to study the staircase rhythm and to compare the functional coefficients at the different rates of chlorid excretion.

2. Rappleye, W. C.: J. Biol. Chem **35**:509, 1918.

3. Van Slyke, D. D., and Donleavy, J. J.: J. Biol. Chem. **37**:551, 1919.

4. Noguchi, H.: J. A. M. A. **70**:1157 (April 20) 1918; J. Exper. M. **28**:43, 1918.

The formulas used for calculating the urea coefficients were those given by Ambard and by McLean in their original contributions:

For urea:

$$\frac{Ur}{\sqrt{D \times \frac{70}{P} \times \sqrt{\frac{C}{25}}}} = K \qquad \frac{D \times \sqrt{C} \times 8.96}{Wt \times (Ur)^2} = I$$

Standard normal K=0.07

For chlorids:

$$\frac{\text{Excess over the threshold}}{\sqrt{D \times \frac{70}{Wt} \times \sqrt{\frac{C}{14}}}} = K$$

Standard normal K (NaCl)=0.11

Threshold=Plasma NaCl—Excess over the threshold

$$\text{Excess over the threshold} = K\ (0.11) \times \sqrt{D \times \frac{70}{Wt} \times \sqrt{\frac{C}{14}}}$$

(McLean)

$$\text{Threshold=Plasma NaCl—} \sqrt{\frac{D \times \sqrt{C}}{Wt \times 4.23}}$$

From a tota of nine cases studied in the present work only two are taken for detailed discussion, although several of the others are summarized. As a matter of fact, the same disturbance of the kidneys was noted in every instance, and the slight variations observed were of minor importance and merely confirm the evidence of uncinarial nephritis and its disappearance as a result of the expulsion of the hookworms.

Special attention is demanded by the fact that a higher rate of chlorid excretion is obtained after a few days of treatment in anemic subjects who had previously exhibited a chlorid excretion insufficient to maintain a balance, without abnormal retention, even on the minimal intake of sodium chlorid necessary for a normal alimentation.

REPORT OF CASES

CASE 1 (9).—L. I., man, aged 24, was admitted to the hospital Feb. 18, 1920, with a diagnosis of uncinariasis. He had lived twenty months in an uncinarial zone. During the last six months he complained of dizziness, vomiting of mucus in the morning, cramps in the legs, dyspnea on exertion, nocturnal pollakiuria, disorders of the vision, and ringing in the ears.

Examination.—Noticeable pallor of the skin, edema of the face and feet; radial pulse 108; systolic murmur at base of heart; slight edema of optic papillae. The urine showed a trace of albumin and many finely and coarsely granular, cellular and hyalin casts, leukocytes, no sugar. Table 1 gives the urinary findings.

CASE 2 (8).—C. A. Z., man, aged 39, was admitted to the hospital Feb. 18, 1920, with a diagnosis of uncinariasis. He had lived twenty-four years in an uncinarial zone. Anemia, dizziness and headache, cramps in the legs, nocturnal

pollakiuria, transitory edema of the eyelids and malleolus were the symptoms present some years ago. This man had previously entered the hospital complaining of uncinarial anasarca, but he had recovered under anthelmintic therapy. At the present time, marked anemia and slight edema of the feet and legs were noted; radial pulse 84; papillary edema and congestion in the ocular fundi. The urine showed the same findings as in Case 1.

Since Cases 1 and 2 are similar, they will be discussed together. From the investigations made by Ambard, Widal and Weill,[5] it is known that the same general laws apply to the excretion of urea and of sodium chlorid, but with the important difference that there is a

TABLE 1.—URINARY FINDINGS IN CASE 1*

Date	Urine			NaCl Intake, Gm.	Blood Viscosity	Ambard's Coefficient	Urea Index	Urea per Liter of Plasma, Gm.	Sodium Chlorid		Threshold	
	Quantity per 24 Hrs., C.c.	NaCl per Liter, Gm.	NaCl in 24 Hrs., Gm.						Per Liter of Plasma, Gm.	Rate of Excretion per 24 Hrs., Gm.	Ambard, Gm.	McLean, Gm.
Feb. 18	.....	....	.....	..	2.0	0.059	184	0.271	6.45	13.64	6.12	6.05
Feb. 19	2,080	5.00	10.40									
Feb. 20	1,810	4.00	7.24									
Feb. 21	2,000	3.00	6.00									
Feb. 22	2,060	1.50	3.07									
Feb. 23	2,450	1.00	2.45									
Feb. 24	2,000	1.00	2.00									
Feb. 25	1,340	1.60	2.14	..	2.2	0.069	134	0.242	6.41	4.39	6.22	6.20
Feb. 26	750	5.00	3.75	10								
Feb. 27	1,580	7.00	11.06	10								
Feb. 28	2,240	3.00	6.72	20								
Feb. 29	3,330	5.50	18.31	20								
Mar. 1	3,070	7.00	21.49	20								
Mar. 2	2,580	8.00	20.64	20								
Mar. 3	3,300	8.00	26.40	20								
Mar. 28	1,300	2.00	2.60	..	2.2	0.066	144	0.200	5.76	12.00	5.453	5.43
Mar. 29	2,450	5.00	12.25	15								
Mar. 30	4,700	8.00	37.60	35								
Mar. 31	4,820	14.50	69.89	70	...	0.100	62	0.178	6.37	195.13	3.82	4.54
April 1	6,810	14.60	99.42	100†								
April 2	5,170	14.10	72.89	120								
April 3	1,120	3.50	3.92									

* Medication: Feb. 24, digitalin, 0.0006 gm.; Feb. 24, theobromin, 2.5 gm.; from March 4 to 21, thymol, 12 gm.

† April 1, fever, with temperature 40.3 C.; it was not possible to complete intake.

threshold for chlorid excretion. If the concentration in the plasma falls to a value below the threshold, the excretion of chlorid practically ceases; therefore, it is the sodium chlorid above the threshold which determines the rate of excretion, in accordance with those laws that hold in the case of urea. A greater excess over the threshold means a correspondingly greater rate of excretion.

5. Widal, F., and Javal, A.: Compt. rend. Soc. de biol. **57**:301, 1904; Ambard, L., and Weill, A.: Semaine méd. **32**:217, 1912; Widal, F., Ambard, L., and Weill, A.: Semaine méd. **32**:361, 1912; Ambard, L., and Chabanier, H.: Arch. urol. clin. de Necker, **1**:248, 1913.

The excess over the threshold can be increased by the following changes: By the lowering of the threshold, by the elevation of the actual plasma sodium chlorid, or by the fall of the threshold and the elevation of the actual plasma sodium chlorid at the same time. In certain cases an increase in the excess above the threshold occurs in spite of a decrease in the actual plasma sodium chlorid as the result of an even greater fall of the threshold. Indeed, the characteristic of normal kidneys is their capacity to move the threshold and actual plasma chlorid values in an effective manner to maintain in equilibrium the intake and output of chlorid without abnormal retention.

TABLE 2.—URINARY FINDINGS IN CASE 2*

Date	Urine			NaCl Intake, Gm.	Blood Viscosity	Ambard's Coefficient	Urea Index	Urea per Liter of Plasma, Gm.	Sodium Chlorid		Threshold	
	Quantity per 24 Hrs., C.c.	NaCl per Liter, Gm.	NaCl in 24 Hrs., Gm.						Rate of Excretion per	Per Liter of Plasma, 24 Hrs., Gm.	Ambard, Gm.	McLean, Gm.
Feb. 18	.....	.....	.....	..	2.5	0.082	93	0.305	6.55	16.32	6.00	6.07
Feb. 19	2,100	5.50	11.55									
Feb. 20	800	4.20	3.36									
Feb. 21	1,300	0.50	0.65									
Feb. 22	1,400	1.00	1.40									
Feb. 23	910	1.75	1.59	..	2.5	0.093	73	0.237	6.18	2.19	6.03	6.07
Feb. 24	1,560	1.50	2.34	10								
Feb. 25	1,400	4.00	5.60	10								
Feb. 26	1,750	5.00	8.75	10								
Feb. 27	1,900	7.00	13.30	10								
Feb. 28	2,180	5.00	10.90	20								
Feb. 29	2,190	8.00	17.52	20								
Mar. 1	2,000	7.00	14.00	20								
Mar. 2	1,860	11.00	20.46	20								
Mar. 3	2,100	10.20	21.42	20								
Mar. 15	2,060	4.00	8.24									
Mar. 16	1,450	5.00	7.25									
Mar. 17	1,000	2.00	2.00									
Mar. 18	1,680	1.00	1.68	..	2.7	0.090	79	0.253	5.63	2.73	5.42	5.47
Mar. 19	770	7.50	5.77	10								
Mar. 20	1,000	10.20	10.20	10								
Mar. 21	1,780	9.00	16.02	10								
Mar. 22	1,900	11.50	21.85	20								
Mar. 23	1,660	14.80	24.56	20	3.1	0.082	93	0.243	5.78	26.64	4.97	5.07

* Medication: Feb. 22, digitalin, 0.0006 gm.; Feb. 22, theobromin, 2 gm.; from March 4 to 14, thymol, 9 gm.

On the contrary, the renal incapacity to excrete chlorid would result from an abnormal and permanent elevation of the threshold and the impossibility of lowering it to within normal limits in order to increase the excess above the threshold and to induce a sufficient rate of excretion to balance chlorid ingestion. For it is not possible for the actual plasma sodium chlorid to rise above certain invariable limits. For instance, an individual in whom the greatest excess over the threshold is capable of generating only a rate of excretion of 10 gm. sodium chlorid, if the ingestion be increased to 20 or 30 gm., will exhibit

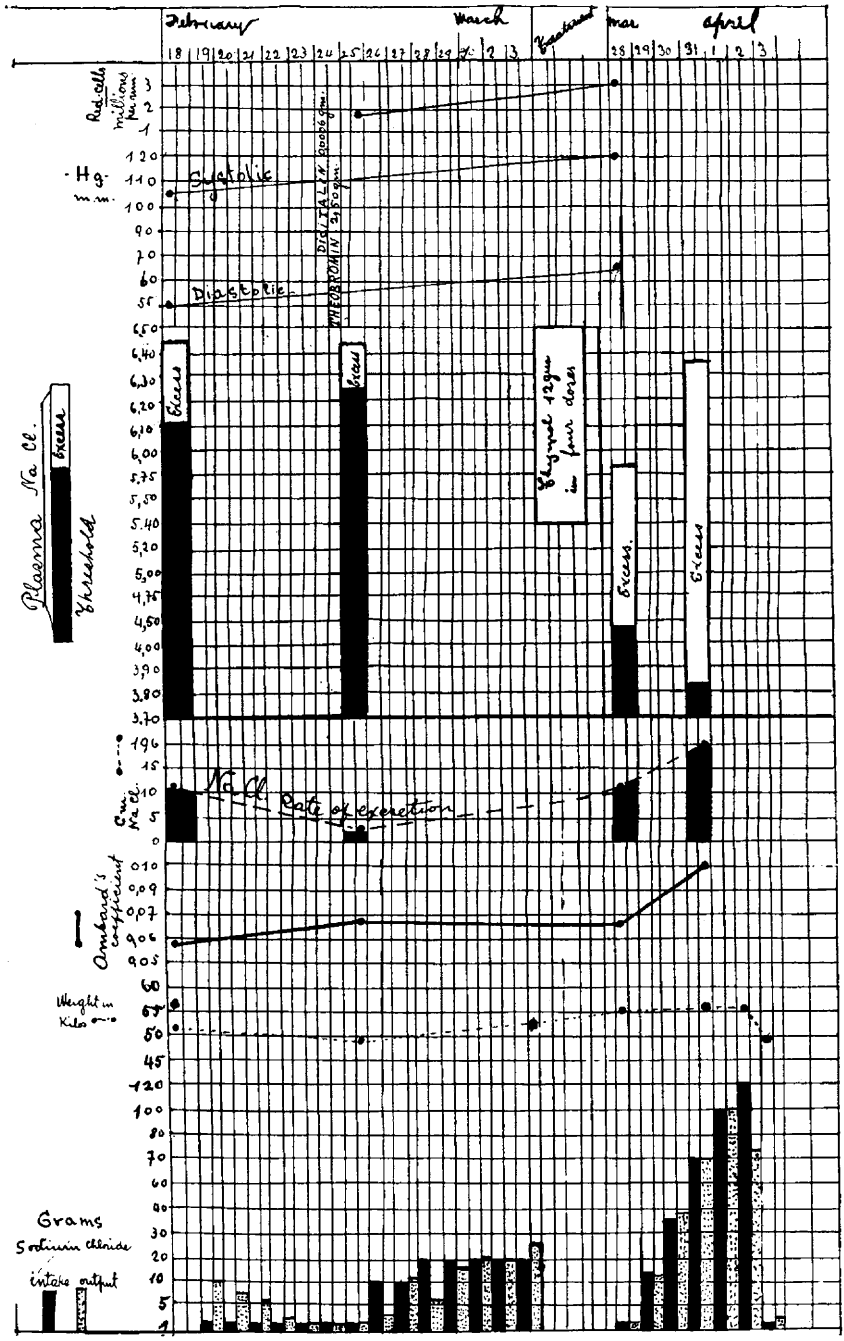

Fig. 1.—Graphic representation of data in Table 1.

a retention of 10 or 20 gm., since the threshold cannot be lowered further. Furthermore, the concentration in the blood and the rate of excretion of urea and of chlorids depends not only on the ingestion of nitrogen or chlorid, but also on the balance between the ingestion and elimination of fluids.

With this basis established, together with the graphic and tabular presentation of two of the nine cases studied in the present work, it is not necessary to enter into a longer discussion. A marked insufficiency of chlorid excretion is demonstrated in these patients, which in certain instances is associated with a disturbance of urea excretion.

In Case 1, when infected by hookworms, there is shown a normal rhythm of retention of sodium chlorid; but the failure of the kidneys when exhibiting a rate of excretion of 13 gm. sodium chlorid, is shown by the high values of the threshold and of the actual plasma sodium chlorid. The same subject, after treatment, excreted at a similar rate, 12 gm. sodium chlorid, stimulated only by a threshold and actual plasma sodium chlorid of 5.453 and 5.760. After treatment a rate of excretion of 195 gm. was obtained by means of a rise to 6.370 gm. of the actual plasma sodium chlorid and the remarkable fall of the threshold to an unusual level, 3.82 gm.

In Case 2, when infected by hookworms, there is demonstrated an abnormal rhythm of retention and high values of the threshold and actual plasma sodium chlorid which induce, however, only a rate of excretion of 12 gm. sodium chlorid; while after the expulsion of the hookworms, a higher rate of excretion was obtained with a simultaneous fall in the values of the threshold and actual plasma sodium chlorid.

CASE 3 (7).—J. A. R., man, aged 23, was admitted to the hospital Feb. 18, 1920, with a diagnosis of uncinariasis. He had lived five years in an uncinarial zone. Symptoms which had lasted several months were: cramps in the legs, ringing in the ears, nausea and vomiting of a bitter and salty fluid in the morning, headache.

Examination.—Revealed noticeable pallor of the skin and conjunctiva; edema of the face and feet; radial pulse 120; systolic murmurs at the apex of the heart; enlarged spleen; edema of the optic papillae. In the urine were the same findings as those described in previous cases.

February 18: Ambard's coefficient 0.076; actual plasma sodium chlorid, 6.44 gm., threshold, 5.94 gm.; rate of excretion, 12.14 gm.

February 23: Ambard's coefficient 0.083; actual plasma sodium chlorid, 6.32 gm.; threshold, 6.06 gm.; sodium chlorid per twenty-four hours, 3.36 gm.; red blood cells, per c.mm., 1,500,000; blood pressure: systolic 110, diastolic 45; blood viscosity 2.1.

Medication.—February 22: Digitalin, 0.0004 gm., theobromin, 1.50 gm. From March 5 to 16, 9 gm. thymol in three doses.

March 18: Ambard's coefficient, 0.074; actual plasma sodium chlorid, 6.16 gm.; threshold, 6.01 gm.; sodium chlorid per twenty-four hours, 3.27 gm.; blood pressure: systolic, 106, diastolic, 68; blood viscosity 2.4.

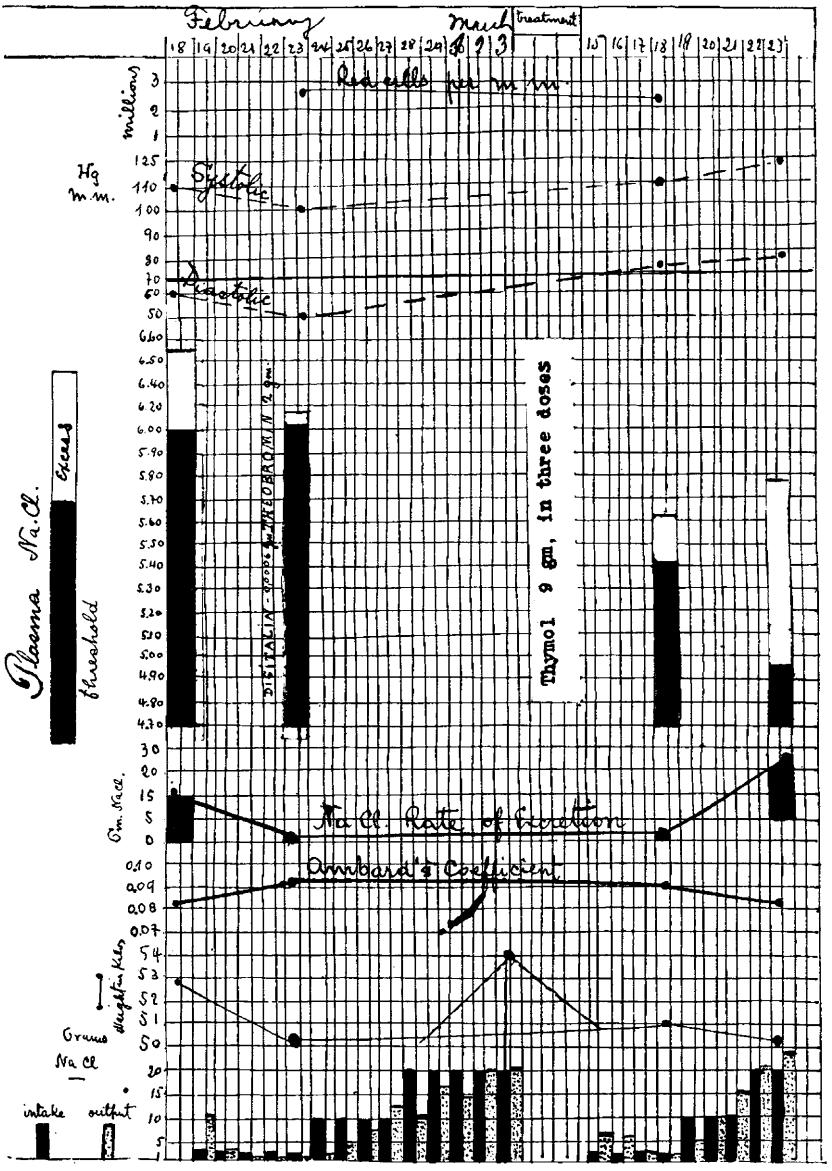

Fig. 2.—Graphic representation of data in Table 2.

March 28: Ambard's coefficient, 0.075; actual plasma sodium chlorid, 6.01 gm.; threshold, 5.44 gm.; rate of excretion, 17.95 gm.; red blood cells per c.mm., 1,500,000; blood pressure: systolic, 104, diastolic, 58; abnormal rhythm of retention on 10 gm. sodium chlorid before treatment; normal rhythm of retention on 10 and 20 gm. sodium chlorid after treatment.

CASE 4 (6).—S. H., boy, farmer, aged 17, was admitted to the hospital February 16, with a diagnosis of uncinariasis. He had been resident three years in an uncinarial zone. Chilliness, headache, nausea and vomiting, dizziness, ringing in the ears, edema of the face and lower extremities were the symptoms.

Examination.—Severe anemia; radial pulse, 108; systolic murmurs at base of heart; pollakiuria; enlarged spleen; trace of edema in optic papillae. In the urine were albumin and casts as described in previous cases.

February 16 (day of entering hospital): Ambard's coefficient, 0.066; actual plasma sodium chlorid, 6.55 gm.; threshold, 5.94; sodium chlorid per twenty-four hours, 21.19 gm.; blood pressure: systolic, 115, diastolic, 55; blood viscosity, 1.9.

February 24: Ambard's coefficient, 0.066; actual plasma sodium chlorid, 6.35 gm.; threshold, 5.98 gm.; sodium chlorid per twenty-four hours, 9.97 gm.; blood pressure: systolic, 108, diastolic, 50; red blood cells per c.mm., 1,020,000.

Medication.—February 23: Digitalin, 0.0004 gm.; theobromin, 1.50 gm.; thymol, 9 gm. in three doses.

March 24: Ambard's coefficient 0.070; actual plasma sodium chlorid, 5.66 gm.; threshold, 5.24 gm.; sodium chlorid per twenty-four hours, 8.91 gm.; blood pressure: systolic 96, diastolic 54; red blood cells per c.mm., 1,490,000; blood viscosity, 3.5.

March 27: Ambard's coefficient, 0.061; actual plasma sodium chlorid, 6.01 gm.; threshold, 5.31 gm.; rate of excretion, 31.51 gm.; blood pressure: systolic, 100, diastolic, 76; sodium chlorid excretion obtained on salt poor diet with rest, 22.50 gm.; normal rhythm of retention on 10 gm. sodium chlorid, abnormal rhythm of retention on 20 gm. sodium chlorid.

CASE 5 (5).—R. Z., man, farmer, aged 25 years, was admitted to the hospital February 6. He had been resident twelve years in an uncinarial zone. He complained of dizziness, general weakness, ringing in the ears, and dyspnea on exertion during the last year, severe anemia, nausea and vomiting.

Examination.—Systolic murmur at base of heart, radial pulse 108 and oliguria. In the urine were the same findings as in the foregoing cases. (The study of this case was incomplete owing to the departure of the patient.)

February 6: Ambard's coefficient, 0.140; acutal plasma sodium chlorid, 6.40 gm.; threshold, 5.95; sodium chlorid per twenty-four hours, 3.52 gm.; blood pressure: systolic, 110, diastolic, 60; red blood cells per c.mm., 1,980,000; blood viscosity, 2.6.

Medication.—Thymol, 9 gm. in three doses.

March 3: Ambard's coefficient, 0.090; actual plasma sodium chlorid, 6.19 gm.; threshold, 5.71 gm.; sodium chlorid per twenty-four hours, 8.21 gm.; blood pressure: systolic, 108, diastolic, 66; blood viscosity, 3.1; red blood cells per c.mm., 2,260,000. This patient showed an abnormal rhythm of retention on 10 gm. sodium chlorid.

CASE 6 (4).—A. V., man, farmer, aged 21, was admitted to the hospital January 29. Resident seven years in uncinarial zone. For one year previous he had complained of headache, ringing in the ears, anemia. Edema appeared in the last four months which increased to anasarca with marked anemia, chilliness, nausea and vomiting, dizziness; mentality was subnormal, he was insensitive to slight stimuli. Papillary edema of both optic nerves. In the urine were the same findings as previously described.

Salt poor diet and rest caused an excretion of 221.93 gm. sodium chlorid and a loss of weight of 10.88 kg. After anthelmintic treatment he was able to excrete enormous amounts of salt without discomfort.

January 29: Ambard's coefficient, 0.130; actual plasma sodium chlorid, 6.20 gm.; threshold, 5.55 gm.; rate of excretion, 8.85 gm.; blood pressure: systolic, 110, diastolic, 50; blood viscosity, 2.3.

Medication.—February 14: Digitalin, 0.0004 gm.; theobromin 1.50 gm.

February 15, digitalin, 0.0004 gm.

February 15: Ambard's coefficient, 0.086; actual plasma sodium chlorid, 6.22 gm.; threshold, 5.91 gm.; rate of excretion, 4.89; blood pressure: systolic, 100, diastolic, 55; blood viscosity, 2.5; red blood cells per cmm. 1,400,000.

Medication.—Thymol, 9 gm. in three doses.

March 14: Ambard's coefficient, 0.083; actual plasma sodium chlorid, 6.10 gm.; threshold, 5.22 gm.; rate of excretion, 25.33 gm.; blood pressure: systolic, 96, diastolic, 56; blood viscosity, 3.1; red blood cells per cmm. 1,480,000.

March 20: Ambard's coefficient, 0.071 (25 gm. of urea added to diet); actual plasma sodium chlorid, 6.02 gm.; threshold, 5.26 gm.; rate of excretion, 26.07 gm.; blood pressure: systolic, 104, diastolic, 52; blood viscosity, 4; red blood cells per cmm., 1,940,000.

March 21: Ambard's coefficient, 0.081; actual plasma sodium chlorid, 6.12 gm.; threshold, 4.79 gm.; rate of excretion, 60.91 gm.; blood pressure: systolic, 104, diastolic, 52; blood viscosity, 3.9.

Retention of 221.93 gm. sodium chlorid. Normal rhythm of retention on 10 gm. sodium chlorid before treatment. Abnormal rhythm of retention on 20 gm. sodium chlorid before treatment. After anthelmintic therapy it was possible to obtain a normal rhythm of retention even on 57 gm. sodium chlorid.

In the other three cases, the same results were obtained. Case 7 (3) was a syphilitic man to whom mercuric cyanid was administered during the time required for observation, after which there was still a weakly positive Noguchi test in the blood. No patient showed edema in the optic papillae after treatment.

That thymol and sodium sulphate are not the direct cause of the fall in the values of plasma sodium chlorid and of the threshold is demonstrated in the following observations made in a normal subject:

1. March 9, 1920; Ambard's coefficient, 0.066; actual plasma sodium chlorid, 5.89 gm.; threshold, 5.50 gm.; rate of excretion, 24.85 gm.; sodium chlorid per liter of urine (concentration), 3.50 gm.; blood pressure: systolic, 118, diastolic, 64; blood viscosity, 4.8.

Medication.—March 10, 1920: Thymol, 4 gm.; sodium sulphate, 120 gm.

2. March 11, 1920: Ambard's coefficient, 0.060 (urea ingestion 25 gm.); actual plasma sodium chlorid, 5.92 gm.; threshold, 5.44 gm.; rate of excretion, sodium chlorid, 25.49 gm.; (concentration sodium chlorid, 12.10 gm.); blood pressure: systolic, 116, diastolic, 70; blood viscosity, 5.2.

Threshold 2 is lower than Threshold 1 owing to the higher concentration of sodium chlorid in the urine, since the rate of excretion is approximately the same. Rate of excretion Threshold 2 calculated with concentration Threshold 1 gives 0.38 as the excess, which is the same as the excess for Threshold 1; so calculated the Threshold for Threshold 2 would be 5.54 gm.

CONCLUSIONS

In the nine subjects studied, hookworm infection caused nephritis with chlorid retention. The index of urea excretion was low in three cases.

The return to normal took place as soon as anthelmintic therapy expelled the hookworms; although the red cell count was still low.

The etiology of this nephritis is the infection by hookworms; its pathogenesis remains unknown.[6]

6. Additional bibliography not specifically mentioned in text: Chabanier, H., and Lobo-Onell, C.: Arch. urol. clin. de Necker, **1**:235, 1913; Legueu, F., Ambard, L., and Chabanier, H.: Arch urol. clin de Necker, **1**:275, 1913; Peabody, F. W.: J. Exper. M. **17**:71, 1913; McLean, F. C., and Selling, L.: J. Biol. Chem. **19**:31, 1914; McLean, F. C.: J. Exper. M. **22**:212, 366, 1915; J. Exper. M. **26**:181, 1917; J. A. M. A. **66**:415 (Feb. 5) 1916; J. A. M. A. **69**:437 (Aug. 11) 1917; Ambard, L.: Physiologie normale et pathologique des reins, Paris, 1914, Ed. 2, 1920; Castaigne, J.: J. méd. franc, **8**:441, 1919; Ashford, Bailey K., and Gutierrez Igaravidez, P.: Uncinariasis in Porto Rico, Washington, 1911, Gov't Printg. Off., Senate Document, No. 808.