

THESIS:

An investigation of a series of renal cases  
with special reference to some of the  
new renal efficiency tests.

-----oOo-----

*Grace M<sup>c</sup>Lintock M.B.C.B.*

ProQuest Number:27660814

All rights reserved

INFORMATION TO ALL USERS

The quality of this reproduction is dependent upon the quality of the copy submitted.

In the unlikely event that the author did not send a complete manuscript and there are missing pages, these will be noted. Also, if material had to be removed, a note will indicate the deletion.



ProQuest 27660814

Published by ProQuest LLC (2019). Copyright of the Dissertation is held by the Author.

All rights reserved.

This work is protected against unauthorized copying under Title 17, United States Code  
Microform Edition © ProQuest LLC.

ProQuest LLC.  
789 East Eisenhower Parkway  
P.O. Box 1346  
Ann Arbor, MI 48106 – 1346

AN INVESTIGATION OF A SERIES OF RENAL CASES WITH  
SPECIAL REFERENCE TO SOME OF THE NEW RENAL  
EFFICIENCY TESTS.

-----

Nephritis being so common, I had many opportunities of studying this disease in its different forms, while in the Royal Infirmary, Glasgow.

I present in this thesis the results of my study.

In recent years, various methods have been devised for examining the kidney functions. I have carried out some of these methods; but before discussing them, the purpose of them may be briefly summarised as follows:-

- (1) To obtain insight into the existence of any renal disturbance.
- (2) To determine the extent of impairment for
  - (a) Diagnosis.
  - (b) Prognosis.

By examining the urine only as was done before blood examination was known to be of clinical value, albumin was discovered in non-nephritic cases. Whatever the cause, it has been proved by methods to be mentioned later, that there is no pathological condition of the kidney and that the renal efficiency is in no way impaired. Thus there are numerous theories about albuminuria not due to nephritis. This type of albuminuria described by  
(1)  
Tessier in 1899 is called orthostatic, postural or intermittent.

<sup>much</sup> Not globulin is present. Tessier has come to the conclusion that the kidney cannot keep pace with the growing body or there is a faulty development of the glomeruli. Erlanger and Hocker state this is due to a low pulse pressure when patient assumes the upright position. Jehle thinks it is due to a lordosis causing obstruction to the venous outflow from the kidneys. The prognosis in all these cases is good.

After athletic exercises, the urine, when examined, has been found analogous to the urine of nephritic cases - casts and albumin being present. It was found that 6% of soldiers who had finished their training for the war had albuminuria, yet these men were able to go through the war with perfect health. McLean<sup>(2)</sup> also states that over 5% of apparently fit men in the army had albuminuria; 2% had gross albuminuria. Thus the new renal efficiency tests are invaluable when doubt arises. By the new renal efficiency tests, we can not only distinguish between true nephritis and albuminuria of no significance, but can classify the different forms of nephritis.

The classification of nephritis has been the subject of a great deal of discussion. From the symptoms alone, it seems classification would be valueless as often the patient who is more seriously ill appears to be in better condition than the acutely ill nephritic. On the other hand, although cases may have a sudden onset, they do not belong necessarily to the acute type/

type of nephritis. For instance, two females were brought into the wards and each had a history of suddenly taking ill and becoming unconscious. They died a few days after admission and post-mortem examination showed that in

- (1) The kidneys were small and granular
- in (2) Cystic kidneys with very little functioning tissue left.

Since the cause of nephritis is still a debatable point, the etiological classification does not seem to me to be satisfactory. Little light has been thrown on the vexed question of etiology. However, there is one thing clear, and, that is that there are two main groups of nephritic cases.

- (1) Inflammatory
- (2) Degenerative.

I think the best classification described by Widal, Weil and Ambard is from the functional standpoint. De Wesselow<sup>(3)</sup> describes two main groups. In the first, the nitrogenous waste products are retained in the blood. This is called the Azotaemic type. In the second, the Hydraemic type, there is a deficiency in the urine - the threshold body Sod-chloride practically ceases to appear. Sometimes the two groups are correlated and we get a mixed type where urea is retained in the blood and there is a defective elimination of sodium chloride.

The hydraemic type seems mainly inflammatory. The degenerative type belongs to the azotaemic group and is always associated with a high blood pressure. The inflammatory process may supervene/

supervene on a degenerated kidney and then we have a mixed type. As regards prognosis, however important the efficiency tests may be, no investigation is complete without taking into account the cardio-vascular system.

In studying the renal efficiency tests, I have come to the conclusion that a number of them are too intricate for use in an ordinary clinical laboratory. The simplest methods have been found to be the best. These are the Diastatic test, the Urea Concentration test and the estimation of blood urea, together with an examination of the cardio-vascular system.

Renal function can be estimated by finding

- (1) Presence of abnormal urinary constituents.
- (2) Altered physiological balance between blood and urine.
- (3) Eliminating power of the kidney after administering a natural substance like urea.

(1) The abnormal constituents are tested for in the usual way. In nephritis, these consist of albumin, blood and casts. Casts are found in the centrifugalised deposit when examined microscopically either with low or high power. Red blood cells are identified in this way. Albumin may be found by various tests. The heat test is very simply carried out. I shall briefly describe it. The test tube is half-filled with urine. The upper part/

part of the column of the tube is heated and, on boiling, we discover a white clour or haze, which, if it persists after acidifying urine, is due to albumin, knowing it cannot be due to earthy phosphates, nucleo-albumin or mucin.

The best test for blood is the spectroscopic test. When not available, we can test with tinct. Guaiac and ozonic ether. To about 10 cc. urine, a few drops of freshly prepared tinct. Guaiac added. A precipitate of Guaiac resin is thrown down; ozonic ether is poured on the surface. If haemoglobin be present, a blue ring forms, being due to oxidation of the Guaiac resin by the oxygen of the ozonic ether, the haemoglobin acting as carrier between them.

In this test, the fallacies are

(1) due to pus which gives a blue colour with tinct.

Guaiac alone:

(2) Iodides giving a blue colour all through.

(2) In the blood, in certain cases of nephritis, there is an increase in the amount of urea. The estimation of this is one of the simplest methods of blood analysis.

(4)  
In estimating the blood urea, De Wesselow's method was carried out. The urea in the blood is converted into ammonium carbonate by the enzyme urease in the soya bean meal. The ammonia in an alkaline solution is propelled by a current of air/

air into a standard acid solution. The number of c.cs. of standard alkaline solution required to neutralise the acid solution represents the amount of urea in the blood.

Three large strong test tubes are fixed in a stand and connected together by means of glass and rubber tubing, so that air can be drawn through by means of a water suction pump. Let the test tube through which air is drawn first be called A, the middle tube B, and the last one C. Five to ten c.c.s of blood are withdrawn (from a hollow needle inserted into a vein in the antecubital fossa) into a test tube containing powdered potassium oxalate which is thoroughly mixed with the blood to prevent coagulation.

In tube A are the following solutions

15 c.c. 5% Sulphuric acid

(To catch ammonia from the air).

A few drops of amylic alcohol to prevent frothing,  
and a drop or two of methyl red as indicator.

In tube B:

3 c.cs. of blood (whole blood).

5 c.cs. of 6% Acid potass. phosphate.

Few drops of amylic alcohol.

.39 m. Soya Bean meal.

Tube/



Tube B is now shut off so that no air can enter. It is kept in a water bath at 37°C. for fifteen minutes so that the urease from the soya bean converts the urea into ammonia. Tube B is now opened and 4 c.c. saturated solution of Potassium carbonate and 3 gms. solid potassium carbonate added quickly.

The water suction pump is turned on so that the ammonia generated in B is driven into Tube C where already are the following solutions -

25 cc. N/100 Sulphuric acid

Few drops of amylic alcohol to prevent frothing.

Few drops of methyl red as indicator.

Aeration is continued for fully thirty minutes. The contents of Tube C are then removed to an Erlenmeyer flask. To ensure the complete transference of these, both inlet and outlet tubes are washed with distilled water which is then added to the Erlenmeyer flask. The acid is then titrated with N/100 Sod. hydrate using the methyl Red as indicator. .3 c.c.s of N/100 acid is neutralised by the ammonia generated from the soya bean. It is necessary, therefore, to subtract .3 c.cs. from the number of c.cs. required to neutralise the acid solution. With 3 c.cs. blood, each c.c. of acid neutralised is equivalent to 10 mgs. of urea per 100 c.c. blood.

(5)

According to Myers and Fine and American writers, uric acid, since it is the most difficult to excrete, accumulates first, if there is any renal deficiency. Therefore it would seem advisable/

advisable to estimate this substance at an early stage of the disease. Any defect in the first place would lead to accumulation of uric acid.

(6)  
According to Folin, all nitrogenous waste products accumulate in the same proportion in the blood; and therefore if uric acid increased, blood urea increased to the same extent.

Since, it seems, no difference to the prognosis so far has been made by examining such substances as creatinin, uric acid or phosphates, urea alone has been estimated in the nephritic cases I have examined.

(3) . Renal efficiency can be judged by collecting the urine. { ?  
The volume of the urine secreted in the 24 hrs. is measured and the Specific Gravity taken. This is to show that the volume of urine and the low S.G. indicate renal impairment. Rowntree and Fitz argue that the actual volume of urine secreted over a given period of time bears little or no relationship to renal function, although S.G. of urine is markedly decreased in advanced cases of chronic nephritis.

The water test is often carried out to determine the efficiency of the kidneys. This is done in different ways by giving water to the patient to drink. Albarran, Strauss and Mosenthal have almost similar methods.

(7)  
Albarran gives to a starving patient 500 c.cs. water. Half-hourly specimens of urine are collected. Polyuria should appear in the first half-hour and reach its maximum and sink rapidly.

The/

(8) The Strauss-Greenwald method consists in giving one pint of water to a starving patient and <sup>collecting the</sup> urine collected at hourly intervals. Normally the sum of the first three specimens should equal the quantity of fluid taken.

(9) Mosenthal's method is to examine two-hourly specimens of urine during the twenty-four hours, and compare it with the normal, noting Specific Gravity and quantity of each specimen collected. Normally, the S.G. has a wide variation and the quantity of night urine is only the quarter of the day urine. The patient must be given four pints of fluid and a dietary with fairly high Protein content with about 5 grammes salt.

The objection to the tests described is that water given in large amounts might and does fatigue the kidney, and result in a diminished urinary output.

Maclean stated in the British Medical Journal that he has tried the water test to find out the eliminating power of the kidney, but there was no evidence that it gave more information than the urea concentration test.

(10) The objection to the Phenol sulpho<sup>n</sup>ephtalein test is that the reaction of the kidney is tested to a foreign substance; and that normal metabolic products do show a different excretion rate. This test requires a colorimeter which makes it less applicable clinically. Six grammes of the dye <sup>are</sup> injected intravenously and the urine, collected by catheter, is rendered pink by 25% sod. hydrate, and compared with a standard in a colorimeter. Normally 60-85% of the dye is excreted in two hours.

The eliminating power of the kidney is best tested, therefore, by the administration of a natural substance like urea. This test depends on the fact that patients with defective kidneys are incapable of secreting urine with a high concentration of urea.

At 4 a.m. fifteen grammes of urea are given to the patient. A specimen of urine is collected before urea is given and three hourly specimens after. The amount of urea excreted is estimated by a ureometer. McLean's ureometer consists of a burette attached at one end by rubber tubing to a flask and at the other end to a wide open-mouthed cylinder. The burette and U shaped tube are filled with water so that at atmospheric pressure the reading is at 0. Twenty five c.cs. of freshly made sodium hypobromite and 4 c.cs. of the urine are thoroughly shaken in the flask, the cork-screw of the burette being horizontal in order to prevent any air entering the burette. The number of c.cs. of nitrogen evolved is noted, the water in the burette and cylinder being on the same level, at atmospheric pressure. From McLean's table the number of c.cs. of nitrogen represents the percentage of urea. If the percentage is under 2 then it is below normal.

The Diastatic test I describe <sup>last</sup> as it is not so reliable as the former tests. Six tubes of small calibre are required.

No.	Urine in each.	Normal Saline.	Starch.	N/10 Iodine.	Units of Diastase.
1	1 c.c.	0	2 c.c.	One	2
2	.6 "	.4	2 "	drop	3.3
3	.4 "	.6	2 "	added to	5
4	.3 "	.7	2 "	each after	6.6
5	.2 "	.8	2 "	$\frac{1}{2}$ hr. at	10
6	.1 "	.9	2 "	37°C.	20

The six tubes containing their respective quantities of urine, normal saline and starch are kept in a water-bath at 37°C. for <sup>an</sup> a half hour. Immediately afterwards, they are almost filled with ordinary cold tap water to stop the ferment action, and a drop of N/10 Iodine added to each tube beginning with number 6. The appearance of the pink colour gives the index of diastatic activity. For instance if the pink colour first seen is in No. 3 the diastatic activity is 5. If the kidneys are efficient, 6-20 units of diastase <sup>are</sup> found as a rule.

In some cases I have done the Urea Concentration test and the Diastatic test together. In other cases, only one test was carried out, either the Urea Concentration test or the estimation of blood urea. In other cases, all three tests were performed. The blood urea estimation was done in some cases every week where possible/

possible to find out the progress the patient was making.

Later, I shall discuss the value of these tests from my examinations; but I now present my account of the cases tabulated as follows.

<u>No.</u>	<u>Sex</u>	<u>Age</u>	<u>Admitted</u>	<u>Dismissed</u>	<u>Symptoms</u>	<u>Syst.</u> <u>B.Pr.</u>	<u>U.C.T.</u>	<u>Dias.T.</u>
1	F.	34	20.12.24	17.2.25 Well.	After pleurisy patient noticed swelling of face and feet	170 to 120	1.15	2
2	M.	57	20.11.24	27.1.25 Much Improved	Oedema of sudden onset. Headache.	140	1.8	20
3	M.	28	20.11.24	27.2.25 Improved	Uraemic Convulsions. to Oedema.	130 to 100	(6.1.24) 1.4 1.9 (11.2.25)	6.5
4	M.	25	15.12.24	27.1.25 Recovery	Oedema Headache Shivering	100	2.1	6.5
5	F.	10	20.12.24	10.3.25 Recovery	History of nephritis 1 yr. ago. Oedema and sudden convulsions	140	1.6 to 1.7	2

<u>No.</u>	<u>Sex</u>	<u>Age</u>	<u>Admitted</u>	<u>Dismissed</u>	<u>Symptoms</u>	<u>Syst.B.Pr.</u>	<u>U.C.T.</u>	<u>Dias.T.</u>
6	F.	31	30.12.24	29.1.25	Headache and Recovery Oedema from 27.12.24	130	1.3	5
7	F.	42	30.12.24	24.1.25	Dyspnoea. Pain in Much Improved back. Slight Oedema	145	1.4	5
8	F.	12	30.1.25	17.3.25	Oedema. Improved Uraemic Convul- sions.	130 to 115	1.6	2
9	M.	20	14.1.25	20.2.25	Oedema of back and Recovery face. Vomiting.	120	1.7 to 2.2	5
10	M.	49	3.2.25	27.4.25	General Improved Oedema. Sore Throat.	165	1.3 to 1.7	3.3
11	F.	29	1.2.25	28.2.25	Post Recovery Scarla- tinal. Slight Oedema.	120	1.4 to 1.6	5
12	M.	65	13.2.25	3.4.25	Dyspnoea. Great Improve- ment. Oedema of feet. Bronch- itis.	170	1.1	5

<u>No.</u>	<u>Sex</u>	<u>Age</u>	<u>Admitted</u>	<u>Dismissed</u>	<u>Symptoms</u>	<u>Syst.B.Pr.</u>	<u>U.Conc.T.</u>
13	M.	44	29.1.25	28.4.25 Greatly Improved	General Oedema. Dyspnoea. Haematuria.	190 to 150	1.6
14	M.	13	23.2.25	20.3.25 Recovery	General Oedema. Haematuria.	100	2
15	M.	23	23.2.25	15.3.25 Recovery	Sore throat Oedema, Haematuria.	110	2.2
16	M.	14	14.4.25	15.5.25 Improve- ment.	Swelling of face	165	.75
17	F.	50	5.12.24	10.1.25 Improve- ment.	Severe headache. Swelling of feet.	180	2.3
18	F.	11	20.3.25	12.5.25 Recovery	Nephritis. 1923. Gen- eral Oed- ema.	110	2.2



<u>No.</u>	<u>Sex</u>	<u>Age</u>	<u>Admitted</u>	<u>Dismissed</u>	<u>Symptoms</u>	<u>Syst.B.Pr.</u>	<u>U.Conc.T.</u>
19	M.	24	31.10.24	30.12.24 <i>Recovery</i>	Haematuria. Headache. Shivering.	110	1.1 to 1.6
20	F.	8	5.11.24	15.11.24 Improve- ment.	Transitory Oedema of face.	100 to 90	1.2
21	F.	49	20.11.24	15.1.25 Recovery	Headache. Oedema.	140	3
22	F.	15	14.1.25	2.2.25 Recovery	Shivering. Headache. Oedema.	150	2.1
23	F.	44	10.11.24	5.12.24 Recovery	Anuria. Swelling of feet.	160 to 130	2.8
24	M.	41	11.10.24	30.12.24 Greatly improved.	General Oedema.	160	1.4 to 1.75

No.	Sex	Age	Admitted	Dismissed	Symptoms	Syst. E.Pr.	Mgs.Urea per 1000 cc.Bld.	
							Date.	Result.
25	M.	58	10.3.25	11.5.25 Died.	Weakness, Anaemia. Retinitis Consti- pation.	205		300 <i>Always the same.</i>
26	F.	12	13.3.25	3.4.25 Much Improved	General Oedema with re- currences	90		47
27	M.	65	13.2.25	3,4.25 Greatly Improved	Dyspnoea. Bronch- itis. Oedema of feet.	160		37
28	M.	55	5.11.25	17.11.25 Died.	Sudden coma.	220	6.11.25 7.11.25	700 451
29	F.	16	27.8.25	18.11.25	Patient developed mental symptoms and trans- ferred to observ- ation ward.	115	7.11.25	105
30	F.	19	26.10.25	27.11.25 Recovery	Acute onset Oedema. Headache.	130	16.11.25	40.5

<u>No.</u>	<u>Sex</u>	<u>Age</u>	<u>Admitted</u>	<u>Dismissed</u>	<u>Symptoms</u>	<u>Syst.</u> <u>B.Pr.</u>	<u>Mgs.Urea per 1000</u> <u>cc. Bld.</u>	
							<u>Date.</u>	<u>Result.</u>
31	F.	19	30.12.25	3.1.26 Died. (Mother died of renal disease).	Swelling of face 3 wks. before admission.	160	2.1.26	113
32	F.	48	20.12.25	2.2.26 Recovery	Swelling of face & feet. Bronchitis.	140 to 110	21.12.25 23.12.25 5.1.26	34 23 40
33	M.	19	1.10.25	4.12.25 Recovery	Coma with History of Convulsions & Oedema.	140	14.11.25 20.11.25 25.11.25	69 65 29
34	M.	51	16.10.25	1.12.25 Recovery	Swelling of face & feet.	200	14.11.25 18.11.25 25.11.25	41 50 31
35	M.	38	26.10.25	25.1.26 Recovery	Swelling of face and legs of 4 weeks' duration.	150	10.11.25 27.11.25 11.12.25 4.1.26 20.1.26 25.1.26	55 35 37 46 57 25

<u>No.</u>	<u>Sex</u>	<u>Age</u>	<u>Admitted</u>	<u>Dismissed</u>	<u>Symptoms</u>	<u>Syst.</u> <u>B.Pr.</u>	<u>Mgs.Urea per 1000</u> <u>c.c. Bld.</u>	
							<u>Date.</u>	<u>Result.</u>
36	M.	13	4.11.25	9.12.25 Recovery	Sore Throat Headache Oedema Sickness	115 to 110	20.11.25 25.11.25 2.12.25 9.12.25	52 48 29 11
37	M.	29	30.12.25	5.3.26 Recovery	Headache. General Oedema.	160 to 110	5.1.26 20.1.26 3.2.26	63 40 15
38	F.	34	9.1.26	11.1.26 Died.	Drowsiness Convulsions	150	10.1.26 11.1.26	183 215.5
39	M.	21	10.1.26	17.2.26 Recovery	Oedema - began 10 weeks before admission	120	16.1.26 23.1.26 3.2.26	38 39 19
40	F.	32	24.4.25	1.7.25 Recovery	Uraemic Convulsions	190	25.4.24	205
41	F.	46	23.2.26	1.3.26 Died.	Scarlet fever as a child. Dyspnoea. Epistaxis. Headache.	220 to 190	24.2.26	175

<u>No.</u>	<u>Sex</u>	<u>Age</u>	<u>Admitted</u>	<u>Dismissed</u>	<u>Symptoms</u>	<u>Syst.</u> <u>B.Pr.</u>	<u>Mgs.Urea per 1000</u> <u>c.c. Bld.</u>	
							<u>Date.</u>	<u>Result.</u>
42	M.	50	20.3.26	22.3.26 Died.	Unconsciousness.	160	21.3.26	195
43	F.	46	9.4.26	10.4.26 Died.	Dyspnoea Cardiac Irregularity Cyanosis Oedema.	Not able to be taken		112.5
44	F.	33	28.2.26	6.4.26 Recovery	Headache. General Oedema.	180 to 115	29.2.26 3.3.26 11.3.26	53 47 48
45	F.	15	8.2.26	13.4.26 Recovery	Very marked Oedema.	100	27.2.26	35
46	F.	31	18.2.26	6.4.26 Recovery	Headache Oedema	160		40

No.	Sex	Age	Admitted	Dismissed	Symptoms	Syst. B.Pr.	Blood Urea		U.C.T.	D.Act- ivity.
							Date	Result		
47	M.	36	12.3.26	18.5.26 Improved	Oedema of feet & face. Dyspnoea Headache Shivering	160	14.3.26 22.3.26 3.4.26 9.4.26 19.4.26 27.4.26 18.5.26	53 95 120 60 85 111 70	1.25	3.3
48	M.	54	16.3.26	8.5.26 Improved	Feeling unwell & weak for a month or two. Then noticed swelling of face.	160	16.3.26 19.3.26 29.3.26 3.4.26 13.4.26 17.4.26 28.4.26	210 279 150 75 70 60 77	1.25	2
49	M.	50	30.11.25	20.4.26 Did not clear up.	Weakness and oedema of face & feet	130	2.12.25 9.12.25 4.1.25 19.1.25 5.2.26 3.3.26 13.3.26 2.4.26 17.4.26	25 23 51 30 10 43 65 60 60	1.4	3.3

No.	Sex	Age	Admitted	Dismissed	Symptoms	Syst. B.Pr.	Blood Urea		U.C.T.	D.Act- ivity
							Date	Result		
50	F.	44	2.5.26	10.6.26 Improved	Oedema of feet. Sick-ness. Vomiting. Re-current attacks of nephritis.	120	3.5.26 15.5.26	90 45	1.5	2
51	F.	55	24.4.25	13.5.26 Improved	Headache for two wks. before admission. Oedema face and feet.	240	* 28.4.26 3.5.26	83 80	1.75	20
52	F.	45	5.5.26	22.6.26 Improved	Nephritis 17 years ago. Dyspnoea. Oedema face and feet.	200	7.5.26 10.5.26 17.5.26 21.5.26	189 73 75 75	1.9 1.8	20
53	F.	22	14.5.26	5.6.26 Improved	Dyspnoea. Severe Occipital Headaches. Oedema of feet.	120	17.5.26	90	1.65	20
54	M.	72	13.3.26	4.6.26 Improved	Giddiness Failing eyesight.	240 to 200	15.5.26 18.5.26	37 65	1.85	5.5

No.	Sex	Age	Admitted	Dismissed	Symptoms	Syst. B.Pr.	Blood Urea		J.C.T.	D.Act- ivity.
							Date	Result.		
55	M.	62	14.5.26	25.6.26 Improved	Giddiness in the morning.	240 to 160	18.5.26	95	2.5	6
56	F.	41	8.6.26	22.6.26 Greatly Improved	Headache and Vomiting of 1 mth's duration.	100	9.6.26 22.6.26	86 45	2.7	10
57	F.	39	23.6.26	10.8.26	Dyspnoea. Oedema. During the past yrs. Inter- mittent swelling of feet. Headaches	190	25.6.26 9.7.26 21.7.26	87 138 50	1.3 2.2 to 2.6	20
58	F.	23	18.6.26	9.7.26	General weakness. Polyuria. Dyspnoea. History of Convulsions with first baby.	120	29.6.26 9.7.26	92 90	2.5 2.8	5.5
59	F.	27	25.3.26	15.8.26	Cardiac failure. Dropsy. Oedema.	120	112 (29.5.26).		1.4	6



No.	Sex	Age	Admitted	Dismissed	Symptoms	Syst. B.Pr.	Blood Urea		U.C.T.
							Date	Result	
60	M.	64	19.5.26	2.7.26 Improved	1917-Kidney Disease. Dyspnoea. Diminished output of urine for the last month.	180	24.5.26 25.6.26	180 88	1.6
61	M.	48	28.5.26	9.7.26 Improved greatly.	Cardio Renal Dyspnoea. Oedema.	235 to 190	17.6.26	52	8
62	M.	53	8.6.26	15.8.26 Died.	Oedema Dyspnoea Cardiac Disease	132 to 120	17.6.26	45 Patient too ill to be further examined.	1.15
63	F.	12	3.7.26	13.8.26 Greatly improved	Nephritis twice before. Oedema of face and feet.	100	9.7.26 23.7.26	111 75	1.8 2.4
64	F.	35	8.7.26	4.8.26 Recovery	Oedema of feet a few weeks before admission.	130	23.7.26	50	2.3
65	F.	43	3.8.26	25.8.26 Died.	Ill for past few weeks. Weakness. Failing Eyesight.	240	6.8.26 9.8.26 14.8.26	160 160 210	1.3

The interpretation of these tests and the effects of disease on the kidney function as indicated by the retention of metabolic products.

-----

Considering the normal blood urea, the degree of nitrogenous retention in a normal individual always remains constant, I have done blood urea estimations with no kidney disease and I agree with McLean that there is a fairly constant level of urea in the blood. The normal variations of any found do not come (12) (under the sphere of practical politics. Some American writers have stated that under 20 mgs. urea per 100 c.c. blood is normal and figures over that indicate a defect in the kidney. According to McLean and de Wesselow (13) 20-40 mgs. seem to be about the normal level.

7 With one exception, my figures were all between 17 and 45 mgs. Urea per 100 c.c. blood. The cases I had examined, knowing they had no kidney trouble, all gave a blood urea under 60. The latter figure was from a case of pleurisy and probably indicated a slight toxic damage to the kidneys. Even minor degrees of failure of concentrating power tend to be accompanied by a rise in blood urea. I have found figures above 40 to indicate a lesion of the kidney - however slight.

Although it has been found <sup>that</sup> urea in itself is not toxic, yet <sup>the</sup> level of blood urea is an index of the extent to which renal function is impaired.

It/

It is stated, <sup>but</sup> nitrogenous retention, as indicated by a high blood urea, is increased in certain diseases, e.g., gastrointestinal disorders, and cardiac disease. In every cardiac condition I have examined, the renal tissue was found to be damaged also as in a nephritic; and those cases which reached the post-mortem room always showed degenerative changes in the kidneys and had a blood urea of over 100 mgs. per 100 c.c. blood.

The gastric cases were never very high and never above 50 mgs. per 100 c.c. blood.

Any condition with a blood urea of over 100 mgs. urea seems to me to indicate a severe nephritis. I have never found any other disease giving as high a blood urea. Before giving a definite prognosis, the blood urea must be estimated several times. If this precaution ~~is~~ not taken, one might think, at first, a patient had a very bad prognosis; but on further examination, the blood urea figure falls and the patient quickly recovers.

(14)

De Wesselow considers that if the blood urea is found to be in the neighbourhood of 100, life is rarely prolonged for more than two years; while patients in whom blood urea is over 200 rarely live for more than six months.

In my examination of nephritic cases, I have come to the conclusion, that a figure of 100 mgs. is on the border line between recovery and a fatal issue. In studying all cases - acute/

acute and chronic - I have found that even if the symptoms suggest an acute onset, if the blood urea remains over 100, the prognosis is bad, and always indicates some gross change in the kidneys which will never recover their normal standard.

In the preceding pages, I have stated the reasons why I prefer to divide nephritic cases into two groups - Azotaemic and Hydraemic. By examining the blood urea, I have found that all acute cases do not come under the category of the Hydraemic type. In one of the charts, there is a graph showing the variations of the blood urea from cases which were divided into two groups "acute" and "chronic" according to the symptoms and history of the patient. Five cases of the acute type had a blood urea of over 100 mgs. - one developed mental symptoms, and had to be transferred to an observation ward; the other two had a slow recovery. One of them whose blood urea was over 100, developed uraemic symptoms the following day but recovered eventually after several weeks. The remaining two died.

These hydraemic cases are therefore of two kinds. The acute nephritis of probably infective origin subsequent on cold, shivering, sore throat, or fever, and the inflammatory type super-imposed on an already degenerated kidney. Thus although most acute cases recover remarkably well, it is advisable to do the blood urea estimations.

All the more chronic cases had, as a rule, a relatively high blood urea. (15) I have never found a low blood urea associated with/

with uraemia.

The question that arises with all hospital patients is "What is the immediate prognosis?" The blood urea is a very good guide as to how the patient is progressing.

As regards diagnosis, when the blood urea is constantly over 60, there is no hesitation in stating that the renal efficiency is impaired. Cases of hydraemic nephritis give a blood urea figure below this, and thus the blood urea estimation is not a good test to do alone for diagnostic purposes.

The results of the urea concentration test performed on patients other than nephritics vary between 1.8 and 3.1%.

When a patient is examined in the early stages of acute nephritis, the urea concentration test of the urine is low and the results are similar to what is found in the more chronic affections of the kidney. One cannot at this stage, differentiate with certainty a grave from a milder acute attack, when the urea concentration test is the only one used for diagnostic purposes. Nevertheless if done at intervals, the U.C.T. gives us a fair idea of the condition of the kidney function and how far the damage done to the kidney is beyond repair.

When doing the Urea Concentration test alone, as has been done in 24 cases, the results in most nephritic cases <sup>is</sup> below 2. When this is above 2, the damage, if any, done to the kidney seems to be quickly removed for the cases have a short convalescence as indicated by Nos. 4, 9, 14, 15, 17, 18, 21, 22, 23. Although/

Although No. 18 had U.C.T. of 2.2, one would have thought from the history it would have been less. The child, nevertheless, had no symptoms except oedema and the recovery was uninterrupted. The damage could not have been marked.

I have made a graph showing the relationship between the Urea Concentration test and the Blood Urea. According to some observers, the blood urea rises as the Urea Concentration of the urine falls. Thus if we have a very low figure for the U.C.T., we would have a high blood urea. I have found this to be so in the majority of cases. I have observed as low a figure as .8% for the U.C.Test and 55 mgs. for the blood urea estimation. In the majority of cases, a urea concentration between 1 and 2 indicates a blood urea of over 60.

Lastly, the Diastatic test is to be considered. It does not seem to me to give a very reliable interpretation of the excretory function of the kidney. Thus the normal between 6 and 20 has been found in 12 cases out of 24 nephritics I have examined. This test done alone is not sufficiently accurate to be the sole test for diagnostic purposes. It can only be used as a confirmatory one.

The best test to do alone is the Urea Concentration Test.

When the blood urea is the only test and is found low at the beginning of a mild type of nephritis, one cannot take this to be of diagnostic value. In this case, the Urea Concentration test/

test is a better one for diagnosis. When the blood urea is relatively low and the urea concentration test also low the effect of disease on the kidney function is very slight. But in case No. 49 where the blood urea and the U.C.T. were both low at the beginning the patient did not recover quickly, as the blood urea, instead of remaining the same, rose steadily. The blood urea thus gives one a very good idea of how the case is progressing.

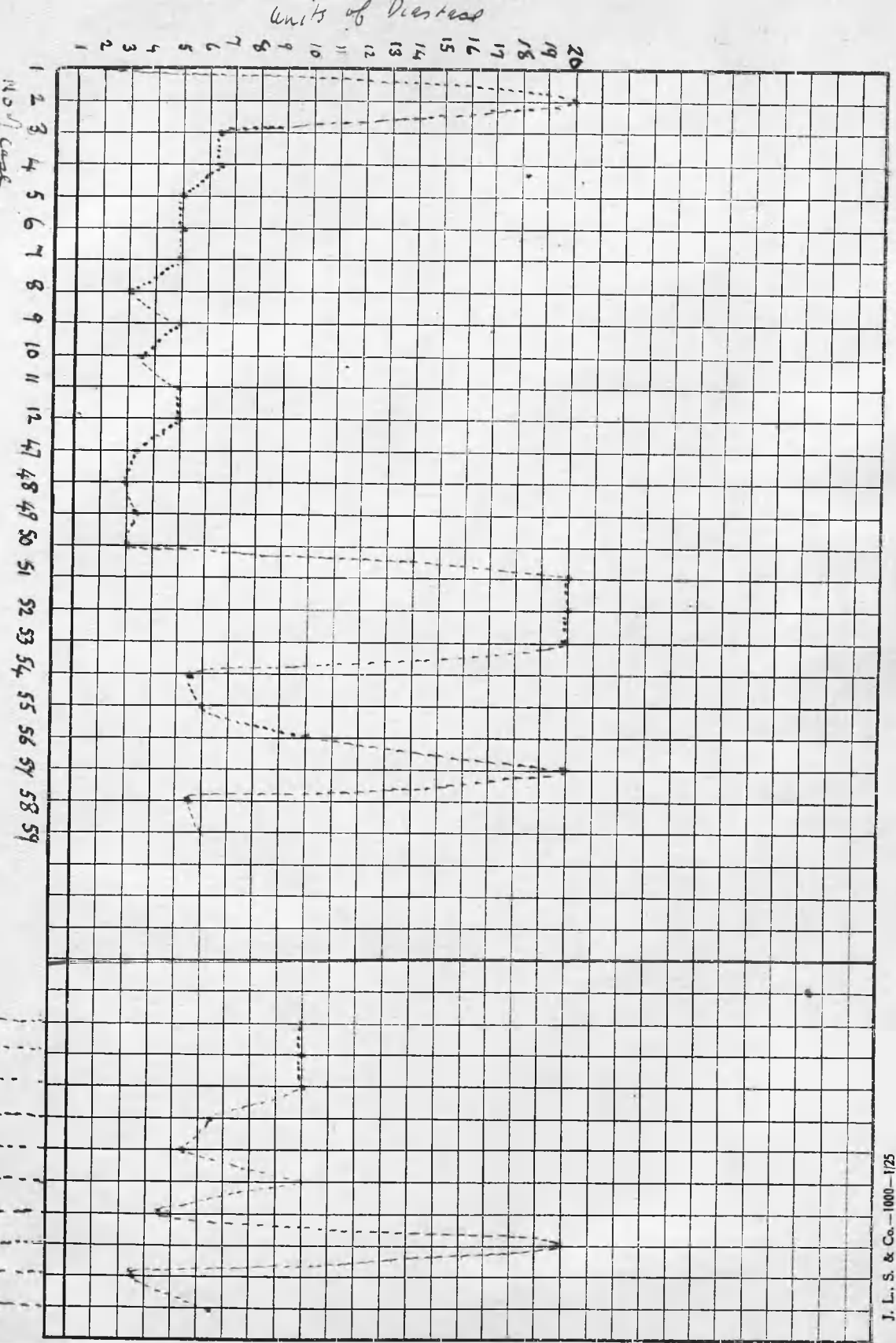
The Urea Concentration test is best for Diagnosis.

The Diastatic test is only a confirmatory test.

-----oOo-----

Diabetic Test - The fluctuations in nephritic cases are similar to those of the non-nephritic cases.

Cases other than Nephritis



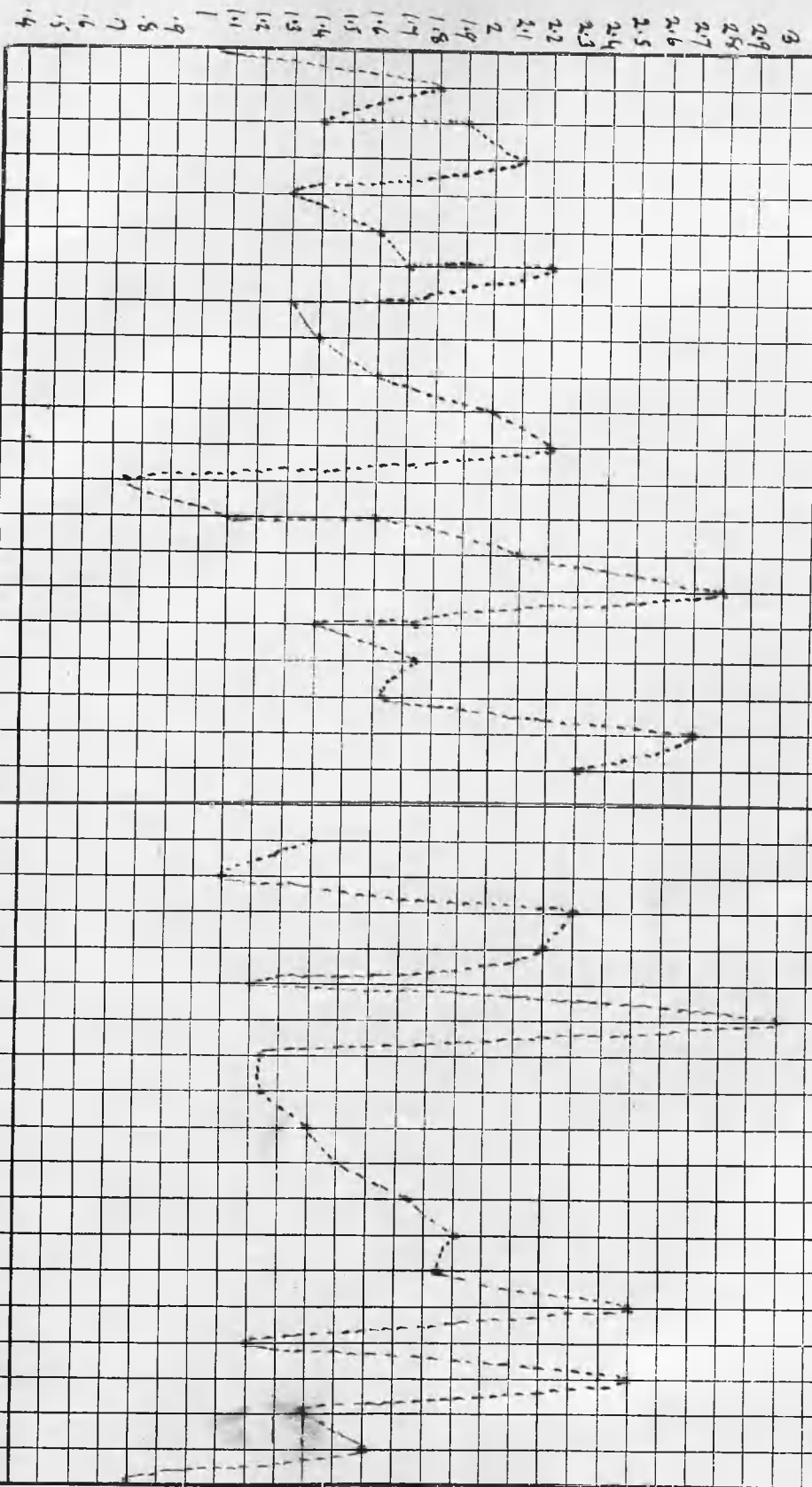


To show that the results of the urea concentration test in Acute and chronic nephritis are similar

"Acute" according to symptoms

"Chronic" according to symptoms

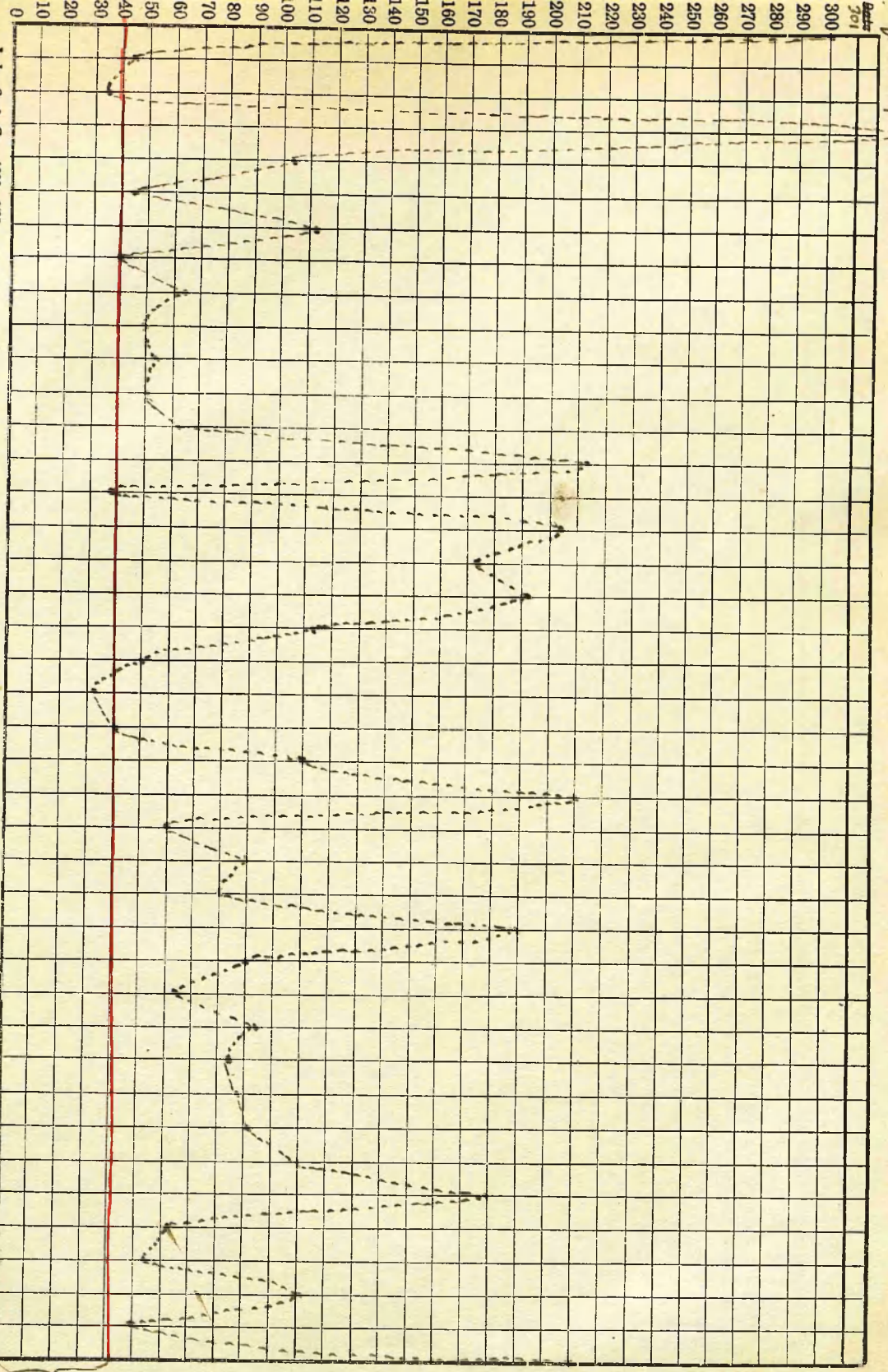
Urea  
in Urine



No. in List

The Variations of the results of blood urea estimations  
in all cases of nephritis (Auer & Christie).

mg. urea per 100 cc. Blood.



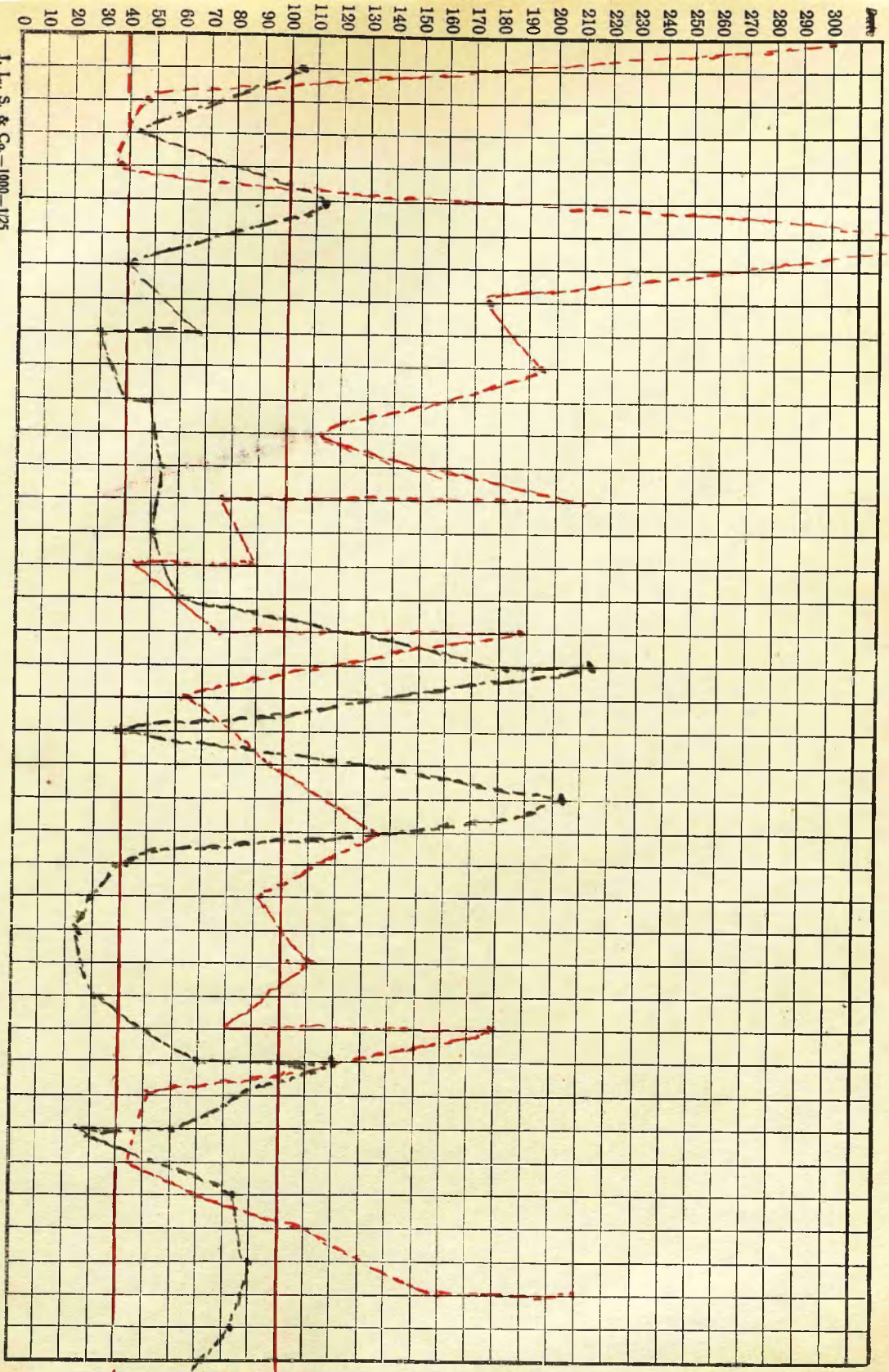
NORMAL  
AMOUNT

J. L. S. & Co. - 1000 - 175  
No. of case  
in list

The Blood Urea in Acute & Chronic Cases.

My Urea  
per 100 cc Blood.

400  
450

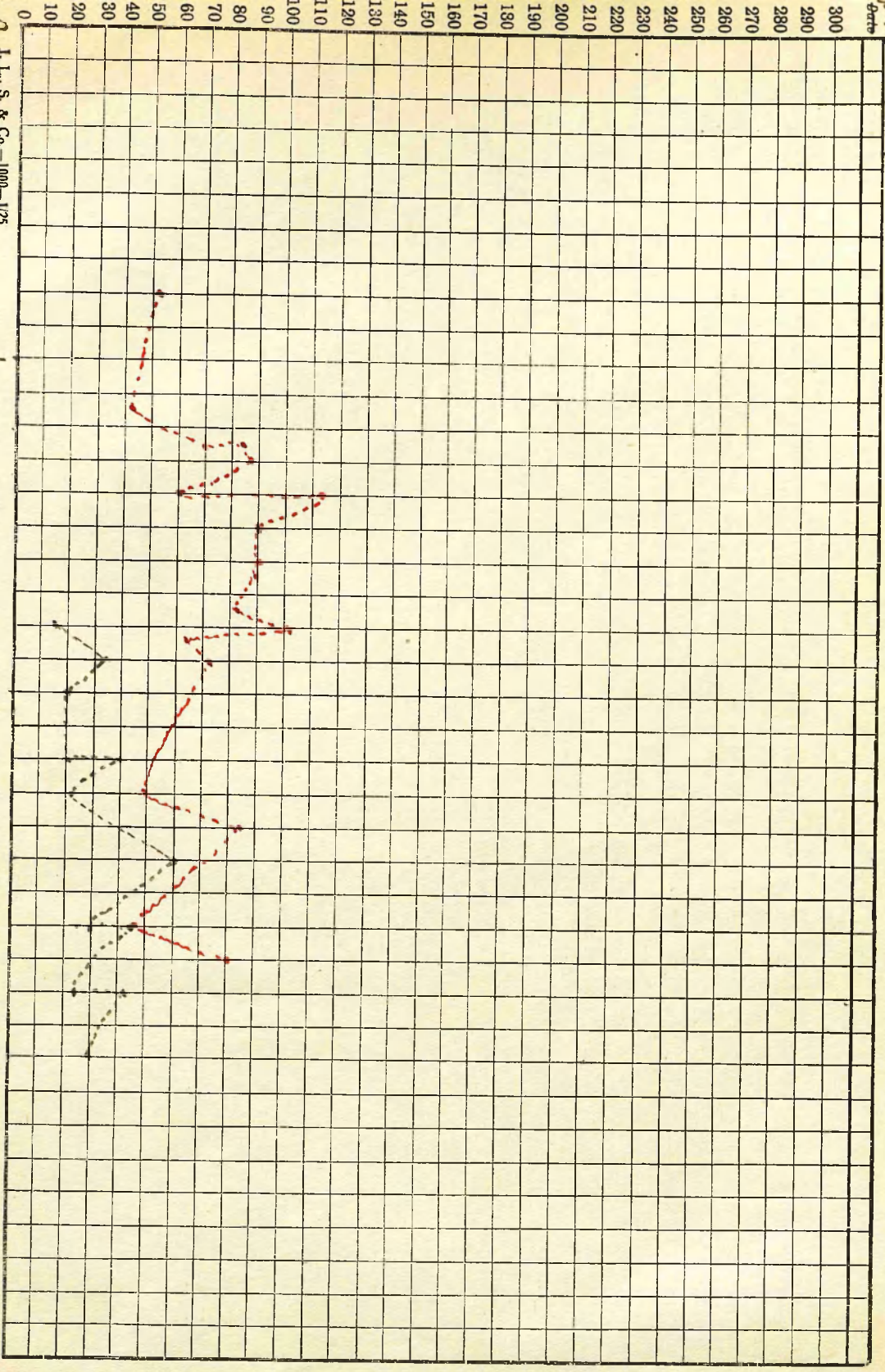


J. L. S. & Co. - 1000 - 125

No of Cases in Order  
25 26 27 28 29 30 31 32 33 34 35 36 37 38 39 40 41 42 43 44 45 46 47 48 49 50 51 52 53 54 55 56 57 58 59 60 61 62 63 64 65

# Relation Between Blood Urea and Urea Concentration Test.

My Urea  
per 100cc blood



J. L. S. & Co. - 1000 - 1725

Urea Concentration Test

--- Nephritic Cases  
- - - Non-Nephritic Cases

A low blood urea does not always indicate a high urea concentration test or vice versa

## B I B L I O G R A P H Y

1. American Journal Medic Science 1918  
Nov. P.643  
Dec. P.830,64
2. British Medical Journal, May 1st. 1926, P.806  
Price's Medicine 1922  
  
The Chemistry of the Blood in Clinic Medicine  
by De Wesselow
3. P.113. 4.P.221. 6. P.122. 13. P.116. 14. P.129.
5. Archives Internal Medicine 1916, P.17,570.
7. ( Beaumont and Dodds Recent Advances in Medicine
8. ( Taken from John Hoppins Hosp Bull 1915, XXVI,292
9. Archiv l n.t. Medic 1915, XVI,733
10. Taken from John Pham and Exp. Therap 1910.579
11. Modern Methods in Diagnosing Renal Disease (Maclean)  
Q.J. Medic 1919 July P.34  
J. Biology Chem. 45, 617, 622
12. Arch. l n.t. Med 1918 Nov. 587  
The New Physiology in Surgic and Gen. Pract. P.33  
(A. Rendle Short.)
15. Beaumont and Dodds Recent Advances in Medicine P.48.