

The following paper is a summary of the work done in the Laboratory of No. 2 Prisoners of War Hospital, Abbassia, Egypt, during the winter of 1917 - 1918, on the Pellagra cases then in Hospital. The work falls naturally into two halves, (I) The Post-Mortem examinations and their results, (II) Blood counts and urine analysis.

Part I.

It has been my custom to weigh all the large organs at all post mortem examinations, and my attention was drawn to the abnormal wasting in these Pellagra cases as compared with other post-mortems. The organs weighed were the Heart, the Spleen, the Liver and the Kidneys. Fifty Autopsies on Pellagra cases were performed during the above period, and the weights of the organs in these cases were tabulated. As these Autopsies contained many cases complicated by Phthisis Pulmonalis and acute Bacillary Dysentery, such cases were eliminated, and the thirty cases remaining were taken as representative Pellagra cases. Of the fifty cases all had well-marked Pellagra rashes and symptoms, though some did not show much body wasting. Of the thirty cases all were well marked Pellagrins except one who had a well marked rash but was well nourished, and died of an intercurrent malady, in this case ^{ovis} Cancerum ~~ovis~~. Three of these cases had a terminal Bacillary Dysentery, diagnosed post-mortem, and two others had slight active Tubercular Foci in their lungs.

In order to get a standard of comparison for the weights of these organs, a series of forty cases of Pulmonary Tuberculosis and fifty cases of Dysentery^e, all of which showed considerable body wasting, were tabulated. The combined results of these were also tabulated. The normal body weights of Turks and Arabs has not yet been found, and it would take many years to obtain accurate figures, but I have tabulated a series of sixty/

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sixty autopsies on Turks and Arabs in order to get some, even vague, idea of the normal. Abnormal autopsies such as the following were purposely omitted, but those showing weights smaller than normal were included. The following were omitted.

	Heart.	Spleen.	Liver.	Kidney.
(I) Banti's Disease.	12 oz.	54 oz.	112 oz.	13 oz.
(II) Malaria.	10 oz.	33 oz.	60 oz.	10 oz.
(III) Blackwater Fever.	12 oz.	48 oz.	64 oz.	10 oz.
(IV) Lobar Pneumonia, Splenomegale.	17 1/2	41 oz.	77 1/2	13 1/2
(V) B.Coli Septicaemia.	12 oz.	32 oz.	100 oz.	25 oz.

Tabulation of results giving the Mode,
the Median and the Mean. Weights all in ounces.

No. I. PELLAGRA. 50 cases.

	Heart.	Spleen.	Liver.	Kidney.
Mode.	7	8	31-35.	8
Median.	7	7	36	9
Mean.	6.6	6.7	37	9

NO. 2. PELLAGRA. 30 cases.

	Heart.	Spleen.	Liver.	Kidney.
Mode.	6	6	31-35	8
Median.	6	6	33	8
Mean.	6.2	6	33.5	8.6

No. 3. PULMONARY TUBERCULOSIS. 40 cases.

	Heart.	Spleen.	Liver.	Kidney.
Mode.	7	8	41-45	8
Median.	8	8	46	9
Mean.	7.8	8.8	47.6	9.6

No. IV. DYSENTERY. (50).

	Heart.	Spleen.	Liver.	Kidney.
Mode.	6	8	46-50	8
Median.	7	8	48	9
Mean.	7.2	9.2	45.5	9.5

NO. V. PULMONARY TUBERCULOSIS
AND DYSENTERY. 90 cases.

	Heart.	Spleen.	Liver.	Kidney.
Mode.	8	8	45	8
Median.	7.5	8	46	9
Mean.	7.5	9	46.4	9.47

NO. VI. GENERAL TURCO ARABIAN
AUTOPSIES. (60).

	Heart.	Spleen.	Liver.	Kidney.
Mode.	10	12	56-60	11
Median.	10	12	56-60	11
Mean.	9.6	11.8	56.3	12.7

According to Daniells and Newham (1) these organs weigh as follows:

	Heart.	Spleen.	Liver.	Kidney.
European (Quain).	11	6	53	11
British Guiana Negro.	10.8	6.9	47.7	9.9
Indian.	8.7	18.4	48.4	8.7
Chinese.	9	14.8	43.4	8.7

It will be seen that the Turco Arabic weights compare very favourably with an admixture of the European and Indian with the exception of the kidney tissue weight, and as a large number of my autopsies were on cases of surgical kidney, i.e., Abscess formation in the kidney due to B. coli infection with much enlargement/

enlargement of the kidneys, my figures err on the high side.

A comparison of the 50 cases of Pellagra with the other groups gives very interesting results.

- (I) The Heart. The Pellagra Heart weighs at least $1\frac{1}{2}$ ounces less than that of the Pulmonary Tuberculosis group and 3 ounces less than the Turco Arabian Heart.
- (II) The Spleen. The Mode and Median are practically the same for all the wasting diseases, but the Pellagra mean is 2 ounces lower than that of the two other groups. Compared with the Turco Arabian group it is 5 ounces lower.
- (III) The Liver. The Liver shows the most marked wasting. It is 10 ounces less than the other wasting diseases and 20 ounces below the Turco Arabian Liver.
- (IV) The Kidney. The kidney weight compares favourably with the kidney in the wasting diseases and also with the Indian kidney, but it is 2 ounces less than the European and the Turco Arabian group.

The above figures show very considerable wasting in the organs in Pellagra, but if the 30 cases in which death was due solely to Pellagra be compared with the larger group of 50 cases of Pellagra, then the wasting is even more evident. For this purpose I have prepared four charts of the organs in weight groups. (See Appendix I.)

There is no difference in the two kidney curves, but in the other three charts the difference is well marked. This difference is best shown in the Liver chart, where there is a well marked secondary rise in the 50 group which completely vanishes in the 30 groups. There is well marked secondary rise also in the Heart and Spleen charts but it is not shown so distinctly/

distinctly as in the Liver group. This secondary rise is undoubtedly due to the complicating factors of Pulmonary Tuberculosis and Dysentery which have caused death earlier than would have been the case had the Pellagra been left to run its course.

The Post-Mortem appearances found in these cases were as follows. Pellagra rashes were present generally, and if not present the white parchment like appearance of the skin was well marked over the sites where the rash had been. Except in one case emaciation was extreme. They were absolutely without any body fat. Oedema was present to a marked extent in about 60% of the cases, and Ascites in 33%.

The Heart showed Brown Atrophy frequently, slight valvular disease was present in five out of the thirty cases, and a chronic form of Pericarditis in one case.

Perisplāenitis with various degrees of thickening of the capsule was found in 10 cases. Nothing of note about the spleen itself except occasional increased fibrous tissue. Marked pigmentation (malarial) was noted only twice, but undoubtedly would have been remarked more frequently had smears been examined microscopically.

The Liver was usually dark in colour, ^mall and of firm consistency. In three cases it was nutmeg in type. Fatty degenerations, patchy in type, were common, and amyloid degeneration was also noted. An early surgical kidney was found in one case with Bacillus coli abscesses in the cortex. Sub-^{acute} Aente nephritis was present in four cases. Three cases had some fatty degeneration, and three showed cloudy swelling with loss of nuclear staining of the tubules, the glomeruli were normal, and there was no proliferation of the lining cells of Bowman's capsules.

Of the thirty cases three had terminal Bacillary Dysentery diagnosed Post-Mortem. The Y type Bacillus was isolated from two/

two of these cases and B. Shiga from the third. Ulceration of the rectum and an acute inflammatory condition causing a deep crimson discolouration of the ^omuc^aus membrane was present in a few cases. This ulceration often extended well up into the large intestine and was quite unlike Dysenteric ulceration. The rectal ^omuc^aus membrane in these cases was almost polypoid in character.

In almost every case there was desquamation of superficial epithelium and thinning of the mucus membrane, and, especially in those cases which showed marked thinning and ballooning, small whitish ^omuc^aus tubercules were seen. On microscopic examination no evidence of T.B. could be found in these small mucocoeles.

No abnormalities were found in the Pancreas or in the Thyroid though occasionally sections showed increase of connective tissue in the latter gland.

In view of the close resemblance between Pellagra and Addison's Disease, a study of the Supra-renal glands is interesting. They show a wasting of about 16% as compared with those of other wasting diseases. I am indebted to Captain H. E. Roaf, R.A.M.C., (T) for the figures of the Suprarenal glands.

Pellagra has always been recognised as a wasting disease, but the extent of the wasting has probably not been sufficiently ~~recognised~~ emphasised. Drewry (2) commenting on twenty one cases of Pellagra on four of which autopsies were held, states only that the Heart and Liver were atrophic, the Spleen congested, marked thinning of the intestines, and in places entire disappearance of the mucosa. The kidney showed irregular congestion. It is worthy of note that all these cases were complicated by some form of tubercular disease. Other literature is equally vague on the subject of wasting.

While serving in the Turkish Army these prisoners probably had/

had a diet poor in Protein and Fat, but that is merely conjecture. Whilst they have been prisoners their diet has been as follows at the various camps.

I. Prisoners of War Camp. Weight in grammes.

	Carbohydrates.	Fats.	Proteins.	Proteins corrected.
	541	32.59	91	52.50
Heat value per gramme.	4.1	9.3	4.1	
Heat value	2220	302	373	
Total Calories 2,895				

II. No. 2. Prisoners of War Hospital Turks and Arabs.

	Carbohydrates.	Fats.	Proteins.	Proteins corrected.
	609.3	34.16	102.7	64.21
Heat value per gramme.	4.1	9.3	4.1	
Heat Value.	2500	317	420	
Total Calories 3,237.				

III. Hospital Diet of European Prisoners.

	Carbohydrates.	Fats.	Proteins.	Proteins corrected.
Heat value per gramme	463.44	59.65	118.6	79.49
Heat value per gramme.	4.1	9.3	4.1	
	1900	555	486	
Total Calories. 2,941.				

The camp diet is ample to preserve the metabolic level, though too much dependence is placed on Carbohydrates. The Protein in the hospital diet is quite sufficient, though the fat figure is low, and on account of the shortage of olives it might be accounted a little lower than is shown on the table.

In all wasting diseases, and especially in Pellagra the wasting occurs as follows. (I) Fats. The superficial, omental, mesenteric and perirenal etc: (II) The stored up lipoids and glycogen of the liver: (III) The muscles. The accompanying curves of the Liver and Heart show this wasting very markedly. (See Appendix I).

It/

It is presumed that these Pellagra Prisoners had a less generous diet of Protein and Fat when they were in the enemy army than they received at our hands. It is thus conceivable that they have been living on their own bodily fat and Protein plus such Carbohydrates as their allies have allowed them to retain. Allow this to proceed further, then, when he has used up his available lipoids, he is practically dependant on Carbohydrate diet. Starling (3) points out that when Carbohydrate goes into circulation it is changed into sugar, and as such needs twenty times its weight of water to carry it. He contends that, as a diet of carbohydrates increases the water contents of the body, this factor, when associated with inanition and fat shortage, may produce dropsy. Excessive fermentation due to the carbohydrate diet might to some extent account for the diarrhoea which is a symptom of Pellagra, and also might account for the thinning of the large intestine and the desquamation of the superficial epithelium.

There appears to be some periodicity about the disease. The most acute cases appeared in February and March, i.e. the rashes were most marked then. On the other hand it does not appear to be infectious. No cases have occurred in hospital amongst those workers intimately associated with the Pellagra cases, and who have subsisted on the same diet.

One of the predisposing causes would appear to be the nature of severe shock especially to the sympathetic system, as the majority of cases have developed the disease shortly after capture. Some of the cases gave a history of having had the disease every spring for several years, and as the Turkish armies have now been fighting practically continuously for eleven years, the nervous system of the soldiers must be to some extent strained and liable to shock. The similarity of Pellagra and Addison's disease immediately draws attention to the Adrenal. The medulla is normally rich in Chromaffin bodies as are the sympathetic/

sympathetic ganglia, and it is conceivable that this chromaffin is deficient in Pellagra. With this deficiency we would find an associated deficiency of Adrenalin especially as there would be, on account of the excessive fermentation, a deficiency of the mono amine acids including Tryptophane from which the Adrenalin is formed.

Myers and Fine (4) point out a deficiency in hydrochloric acid. As this is found in other wasting diseases, it would appear to be merely a further sign of wasting, and call for the addition of an acid tonic including H.Cl. to the treatment. The excessive carbohydrate diet plus the deficient H.Cl. should account for the excessive fermenting stools passed by these Pellagra cases. In summing up the first part of this paper, the following conclusions are arrived at:

- (I) More wasting occurs in Pellagra than in the other recognised wasting diseases.
- (II) The ^aAtiology is obscure, but diet appears to play a great part. Goldberg (5) has caused the ~~convicts~~ disease in convicts in America by giving Carbohydrate diet. Fat deficiency appears to be a more probable cause of the severe form of the disease found here than Protein deficiency. Vitamines may be discounted, as those Turkish prisoners in Palestine had a sufficient supply of fresh fruit to provide them with the required amount of vitamines. The dropsical condition as prevalent in Pellagra is quite in accordance with Starling's (3) views on fat deficiency.
- (III) The disease is not infectious.
- (IV) Whether the adrenal wasting is just in accordance with the rest of the body wasting, or whether there is a true decrease of the medulla and chromaffin system generally with a failure to produce adrenalin is, at present, being worked out by my colleague, Captain Roaf.

PART II. (See Appendix II for tables.)

Blood counts and Urine Analyses were done on 50 cases selected as being uncomplicated Pellagra cases. A table of these counts and analyses is given below.

The average blood count is as follows:

R.B.C's.	4,115,800 per
W.B.C's.	5,050 per
Hb.	70.7 per cent.
Colour Index	86.
Polymorphonuclear leucocytes	62.9
Lymphocytes.	28.4
Mono-nuclears.	8.18
Eosinophiles.	.52

The medium agrees with the average but the mode is found to differ in two particulars:

R.B.C's.	3.5 to 4 millions.
W.B.C's.	4,500.

No abnormal red cells were noticed, though generally the erythrocytes were smaller and appeared more shrunken than those of other patients examined. Poikilocytosis and basophilic degeneration were rarely noticed. Hillman (6) shows the following result from 12 complete counts and 32 differential counts:-
R.Bc's 4,758,000 average with variation from 3,920,000 to 5,440,000 Leucocytes 10,403. Haemoglobin 83%, Colour index 8. No change in size, shape or colouring of red cells. He found an occasional leucocytosis which may point to an infection of obscure origin. He found a lymphocytosis of 33 % and accounts it to be ⁱⁿ accordance with the general cachectic condition of Pellagrins who are often subject to Gastro-Intestinal disorders. Lavinder (7) reports on 24 examinations, of which three examinations were made from one case and two others were cured cases, as follows - chlorotic anaemica; great variation in the number of leucocytes/

leucocytes with occasional leucocytosis not clinically explicable.

- His conclusions are (I) Constant secondary anaemia;
(II) Leucocytosis rare (not a phenomenon of uncomplicated Pellagra).
(III) Relative increase in large mononuclears probable but all results very discordant.

In a discussion following this paper Egan stated that he had found a leucocytosis and no increase of large mononuclears. Drury (2) commenting on 21 cases states that slight anaemia is present and the leucocyte count is normal. The consensus of opinion at the National Conference on Pellagra U.S.A. 1909 was that leucocytosis did not exist in uncomplicated Pellagra. In 14 cases, i.e. 28% there was a mononuclear count of 10 or over. This increase of mononuclears was due to recent malarial infection.

Four cases show an abnormal lymphocytosis 40 to 58%. No clinical factors are available which might in any way explain this marked increase. There was no history of recent Variola I believe, but ~~medical~~ ^L history was difficult to obtain and somewhat unreliable. No Eosinophilia has been observed in these cases.

Conclusions from Blood Counts.

- (I) There is an anaemia, chlorotic in type.
- (II) The White Blood Count has never been above the Physiological normal and there is a frequent Leucopenia.
- (III) There is no increase of Polymorpho nuclear leucocytes in uncomplicated cases.
- (IV) There is no lymphocytosis in uncomplicated cases.
- (V) There is no increase of large mono-nuclear leucocytes in uncomplicated cases.
- (VI) There is no eosinophilia.

In/

In summing up, these conclusions point to Pellagra being neither a bacterial nor a protozoon infection.

The urine examination showed albumen present to a varying degree in thirty-one out of the fifty cases. Casts epithelial, hyaline or granular, were present in ten cases, and in seven other cases there was evidence of destruction of renal tissue. It is pointed out that these examinations were carried out in the first quarter of the year when the Pellagra was acute; in the more chronic stage of the disease Albumin may not be present in such a large percentage of cases. Myers and Fine (4) found Albumin in two cases and hyaline casts in six cases out of a total of fourteen cases examined. They lay some stress on the presence of ethereal sulphates and indican in the urine, as being bodies derived from intestinal putrefaction. Indican is derived from Indol which is a tryptophane derivation. It is from the tryptophane group that the chromaffin substance develops its adrenalin, and if there is deficiency of tryptophane there may well be deficiency of adrenalin. As indican is eliminated high up in the small intestine, it might be possible that B imidazolylethylamine, a tonic derivative of hystidin which, as Adam and McCrae (8) point out, can be easily isolated from the duodenum and Jejunum, could be absorbed in small quantities and give rise to severe Auto-intoxication and Death, as frequently these patients are walking about one day, and next morning collapse and die. Normally imidazolyl^eethylamin^e~~e~~ is not absorbed.

The only conclusion to be drawn from the urine examinations as carried out at this Hospital is the large proportion of cases which develop a form of sub-acute nephritis, as shown by the presence of Albumin and casts in the urine.

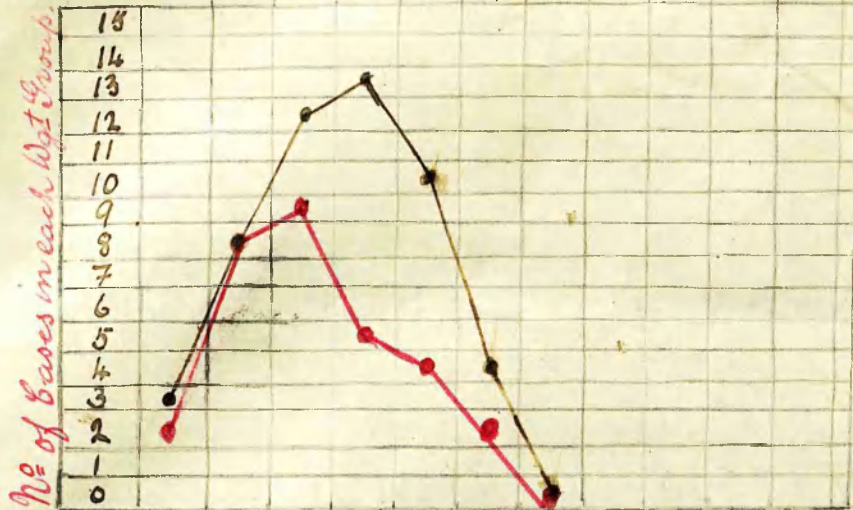
Sept. 1918.

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- References (I) Danielis & NewHam. Laboratory Etudes in Tropical Medicine.
- (II) Drewry. Comments on 21 cases of Pellagra Transactions of National Conference on Pellagra, November 1909. Colombia S.C. U.S.A.
- (III) Starling. British Medical Journal, August 3rd 1918. Page 105.
- (IV) Myers and Fine. American Journal of Medicinal Sciences, May 1913. No 5 Vol. CXIV p.705.
- (V) Goldberg. U.S.A. Public Health Reports Vol.33 pp 481, 488. 1918 and other dates.
- (VI) Hillman. American Journal of Medical Sciences April 1913. No4 Vol CXIV. p.507.
- (VII) Lavinder. Notes on the Haematology of Pellagra Vide supra No II.
- (VIII) Adam & McCrae Text Book of Pathology, 2nd edition Page 106.

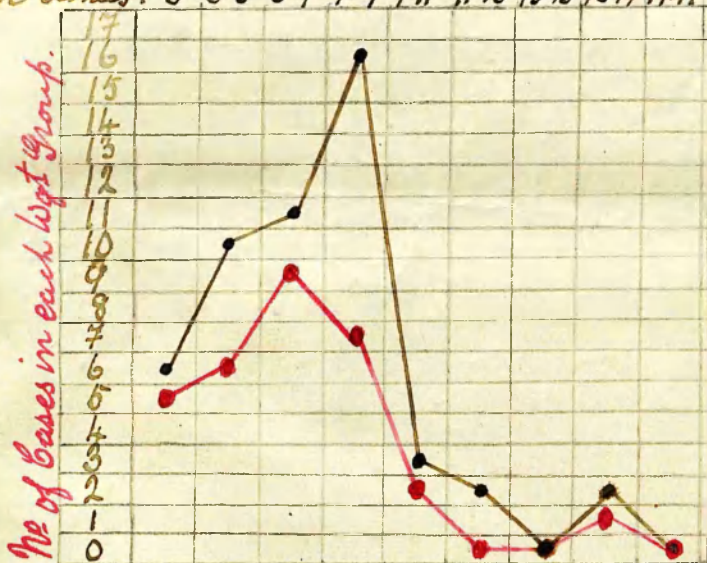
HEART CURVE. 50 Cases "Black." 30 Cases "Red."

Weight in Ounces 4 5 6 7 8 9 10 11 12 13 14



SPLEEN CURVE. 50 Cases "Black." 30 Cases "Red."

Weight in Ounces 1-3 3-5 5-7 7-9 9-11 11-13 13-15 15-17 17-19



PELLAGRA. Amongst the Turkish Prisoners of War

A THESIS for the M.D. Degree of
The University of Glasgow.

Presented by

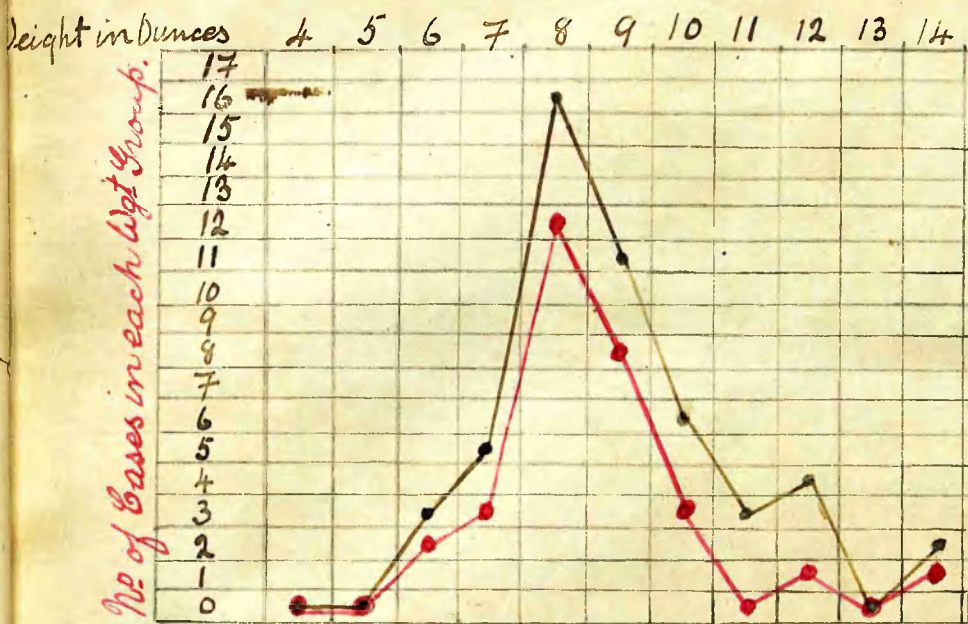
Richard Reid Kirkwood Paton
M.B. Ch.B. Glasgow 1910.

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Cairo.

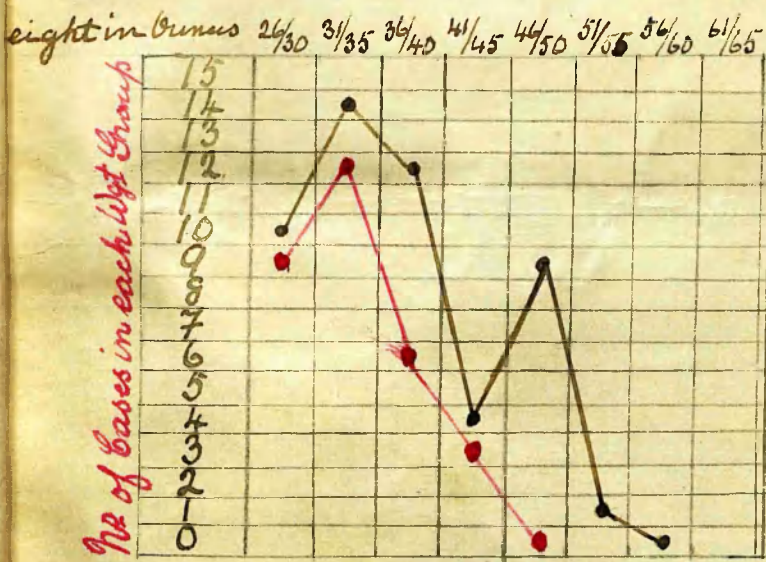
September 1918.

Home Address
Campbell House
Glasgow S.S.

KIDNEY CURVE. 50 Cases "Black." 30 Cases "Red."



LIVER CURVE. 50 Cases "Black." 30 Cases "Red."



	Diff. %				Urine				Deposits	
	Reds.	Whites	Hb. %	P	L	M	E	Sp. Gr.	Alb.	
25787	4,680,000	6,000	75	71	20	9	-	1022	+	Epithelial Cells.
25788	3,960,000	4,600	80	67	27	6	-	1024	-	Urates.
30828	3,970,000	6,200	75	55	35	9	W	1014	+	Hyaline Casts.
26213	3,000,000	6,000	75	67	27	6	-	1018	F.T.	Epithelial Debris.
29717	2,540,000	5,600	60	69	26	5	-	1020	-	-----
30099	3,780,000	3,400	70	63	30	6	1	1022	-	Phosphates.
29286	3,830,000	3,400	75	61	30	7	2	1028	F.T.	Phosphates. ^d
31092	3,360,000	5,600	65	76	19	5	-	1024	-	Urates Oxalates.
32938	4,400,000	5,600	70	61	32	6	1	1012	-	-----
28967	3,740,000	4,600	70	61	29	9	1	1016	-	Uric Acid Crys.
17066	3,830,000	7,800	75	25	38	17	-	1014	-	-----
1918	4,910,000	4,200	70	64	26	10	-	1024	Trace	Urates.
33556	4,930,000	6,000	70	65	24	10	1	1014	+	Epithelial Cells.
26163	3,750,000	5,600	70	70	18	7	5	1018	+	Epithelial Casts.
9386	4,100,000	4,000	75	63	27	10	-	1016	T.	Epithelial Cells.
3650	3,600,000	6,000	70	52	34	14	-	1002	X. +	-----
9453	3,730,000	4,200	65	57	31	12	-	1024	F.T.	Fus Cells.
27710	3,500,000	4,000	60	36	48	16	-	1016	+	Epithelial Debris.
39589	3,850,000	6,400	70	65	23	7	1	1016	+	Epithelial Debris.
29071	3,840,000	4,200	65	68	25	7	-	1016	+	R.B.C's.
32358	3,480,000	5,400	75	61	34	5	-	1026	+	Granular Epith.
25530	5,380,000	5,000	70	53	36	9	1	1024	T.	Oxalates.
29949	4,300,000	5,000	80	68	27	5	-	1022	-	Urates.
15926	4,200,000	4,300	60	58	36	6	-	1022	+	-----

Bloods and Urines. Pellegra.

	Reds	Whites	Hb. %	P. : L.	Diff %	E.	Sp. GR.	Alb.	Deposit
31176.	4,750,000	7,400	80	67:23	9	I	IOIO	-	Phosphates.
26619.	4,340,000	5,200	75	67:26	6	I	IO20	-	-----
26227.	3,700,000	7,000	65	77:16	7	-	IOI8	+	Urates.
30967.	4,190,000	6,800	60	63:25	9	3	IO20	F.T.	Urates Oxalates.
Mustafa Ismail.	4,580,000	5,400	60	62:27	II	-	IOI8	-	-----
Mahmoud Silem.	4,750,000	5,200	75	58:31	II	-	IOI2	-	Phosphates.
Ismail Mahmed.	4,620,000	4,200	80	59:32	8	I	IO22	-	-----
Mehmed Memish.	4,400,000	7,000	70	72:22	6	-	IO20	-	Phosphates.
32399.	3,870,000	6,400	60	77:15	7	I	IO26	+++	Granular Casts.
27322.	4,960,000	5,600	70	73:21	6	-	IO20	++	Granular Casts.
26603.	5,220,000	4,400	80	63:26	II	-	IOI2	+	-----
31415.	4,420,000	6,200	70	50:40	IO	-	IOI2	+	Granular Casts.
684.	3,170,000	3,000	75	73:19	8	-	IO22	F.T.	-----
24839.	6,000,000	4,800	75	66:25	9	-	IO04	+	Epithelial Cells.
27486.	3,520,000	3,600	70	47:42	IO	I	IO20	-	Prostate Cells.
30355.	4,400,000	3,800	70	69:24	6	I	IO20	-	-----
27374.	3,780,000	7,800	80	71:21	8	-	IOI4	+	Epithelial Cells.
23494.	3,910,000	3,400	70	55:39	6	-	IO08	+	Blood, Pus, Epith.
8838.	3,700,000	4,600	60	79:15	5	I	IOI2	-	-----
35118.	4,200,000	6,600	75	58:32	IO	-	IOI8	+	Hyaline Casts.
25881.	3,460,000	3,300	65	59:36	5	-	IOI4	-	Urates.
2336.	5,370,000	6,200	70	68:21	9	2	IOI6	F.T.	-----
33400.	3,000,000	7,200	70	68:26	6	-	IO20	F.T.	-----
35649.	4,520,000	3,800	55	60:30	IO	-	IO20	+	Oxalates. Phosphates.
27375.	2,900,000	4,200	75	68:25	7	-	IOIO	-	Urates.
31914.	4,400,000	3,500	70	60:35	5	-	IOI2	+	Urates.