Report on investigations on the marasmus syndrome in the Indian soldier from the General Headquarters (India) Medical Research Organization, July, 1945-March 1946.

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ARMY PATHOLOGY ADVISORY COMMITTEE
GENERAL HEADQUARTERS (INDIA)

REPORT

ON

INVESTIGATIONS ON THE MARASMUS SYNDROME IN THE INDIAN SOLDIER

FROM

THE GENERAL HEADQUARTERS (INDIA)
MEDICAL RESEARCH ORGANIZATION

July, 1945—March, 1946.

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REPORT ON INVESTIGATIONS ON THE MARASMUS SYNDROME IN THE INDIAN SOLDIER FROM THE GENERAL HEADQUARTERS (INDIA) MEDICAL RESEARCH ORGANIZATION, BASED ON A STUDY OF 2,000 CASES, CHIEFLY INDIAN PRISONERS OF WAR REPATRIATED FROM JAPANESE PRISONERS CAMPS

July, 1945—*March*, 1946.

By

Lieut.-Col. J. H. Walters, I.M.S./I.A.M.C.

Major R. J. Rossiter, R.A.M.C.

Major H. Lehmann, R.A.M.C.

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SECTION I

INTRODUCTION

1. *Historical.*—Marasmus means wasting and has been applied in a broad sense to syndromes of malnutrition arising in Indian Army Units on active Service.

This syndrome does not appear to have been differentiated, either from the dysenteries or starvation oedema during the war of 1914-19, though scurvy was recognised during the siege of Kut el Amara and oedema is said to have occurred among the troops captured there during their subsequent imprisonment. However, cases similar to those studied in the present investigation were treated by one of us (J. H. W.) in Iraq in 1941 and 1942, and again in Egypt in 1943, where from one mixed Battalion of Punjabi Mussulman and Jats, 16 severe cases of marasmus occurred among the vegetarian Jats while the meat-eating Mussulman companies were found to be healthy.

The number of marasmus cases evacuated from the Burma-Assam Theatre during the difficult defensive fighting of 1942-44 is not accurately known, but was sufficiently great to make it second to malaria as a cause of manwastage in the Indian Army. At the time this investigation was planned (early 1945) the views of a number of medical officers had been gathered both from official and unofficial reports, but no detailed study, with laboratory facilities, had been made. The reports on the starvation conditions in European prison camps, which have recently come to hand, were of course not then available.

2. *G. H. Q. Medical Research Organisation, Marasmus Research.*—On June 4th and 5th 1945, a conference was called at G.H.Q. (India) to discuss research into the problem of marasmus in Indian Troops. In consultation with Dr. A. Neuberger of the Medical Research Council the formation of the Marasmus Research Team as part of the G.H.Q. Medical Research Organisation was recommended and a programme of research suggested. This was confirmed by the D.M.S., India, in June 1945.

Clinical material for the investigation was to be drawn from units engaged in the anticipated Malayan Campaign and the Research Team was consequently organised in two components; the forward or field section, consisting of a clinician and a nutrition officer, was to select suitable cases and to investigate in detail the actual dietetic conditions existing in the units in which these cases arose. The patients selected were then to be evacuated without delay to the Base Team working at 145 I.B.G.H. (I.T.), Hospital Town, near Bangalore, where a formal investigation with laboratory facilities would be undertaken.

The constitution of the Base Team was to be (letter 3607/12/D.M.S. 5(c) of 23rd July 1945 from D.M.S., India, to D.D.M.S. Southern Army):—

(1) Clinician
(2) Biochemist (*a*)
(3) Biochemist (*b*)
(4) 3 Trained Laboratory Assistants (Sergeants R.A.M.C.)
(5) 1 Sergeant Cook R.A.M.C.
(6) 2 Ward Servants I.A.M.C.
(7) 2 Sweepers I.A.M.C.

The capitulation of Japan, following her defeat in Burma, necessitated a modification of the original plan, and the Base Research Team only was raised, consisting of:—

Lt.-Col. J. H. Walters, I.M.S./I.A.M.C.	.. Clinician
Major R. J. Rossiter, R.A.M.C. ..	.. Biochemist.
Major H. Lehmann, R.A.M.C. ..	.. Biochemist.

Lt.-Col. Walters was in charge of the clinical care of the patients. Major Rossiter carried out the investigation of blood and plasma volume and fat and carbohydrate tolerance tests, while Major Lehmann was responsible for haematology, blood chemistry and gastric function tests. Capt. Dhurjaty, I.A.M.C. worked throughout as Medical Officer in charge of the ward in which the cases under investigation were concentrated. Sgt. F. Kayser, R.A.M.C., and Sergeant Housy, R.A.M.C., assisted ably in the laboratory investigations, while Sgt. Cook G. Pigram organised the marasmus kitchen from which special diets were supplied.

3. *Material Presented.*—The plan for the repatriation of Allied prisoners provided for the accommodation of those requiring hospital treatment in the new hospitals grouped at Jalahalli, near Bangalore, and Indian patients selected from these provided the greater part of the material studied. This line of evacuation from the Burma—Malayan theatre of occupation has been maintained and latterly many cases drawn from the occupying forces have been received. This series of cases has provided a very useful contrast to the repatriated prisoners.

It is convenient at this stage to sketch the aetiological back-ground of the various syndromes found in the successive groups of returning prisoners.

The standard daily ration issued by the Japanese to Indian prisoners consisted of 12 oz. rice and about 8 oz. green vegetables, usually the leaf and stem of sweet potatoes, a little tea and a small supply of sugar. Following admission to hospital, this ration was automatically reduced to half. Medical supplies were issued in totally inadequate quantities, anti-malarial measures were little practised, while alimentary infections and infestations were widespread, and, in the absence of even the crudest drugs for their treatment, imposed a heavy mortality on the prisoners. Minor cruelties and torture added their quota to the heavy burden of suffering which these men had sustained.

The first batch of repatriated prisoners received were evacuated from Bangkok via Rangoon ; the men had been held for 3½ years under comparatively healthy conditions on an aerodrome and, being on friendly terms with the Siamese, had been able to supplement their rations by the secret purchase of extra food. They were all thin but in ' hard ' physical condition and provided no cases suitable for study.

The following convoys were received from Singapore and most of the patients had been treated, often for many months, in a large camp hospital at Neesoon, staffed by Indian Medical Officers working under Lt.-Col. M. Chaudry and Major Elahi Bux, Medical Specialist. In this hospital most commendable efforts had been made to keep poultry and to grow vegetables, while success had been achieved in the production of small amounts of liver extract, for parenteral as well as oral use. Crude vitamin B group extracts were prepared, first from lentils (Moong Dal) and later from rice polishings, and were effective both orally and parenterally, but the supply could never meet the great demand, and many of the malnutrition syndromes were present in this group of patients.

Two succeeding convoys came from Hong Kong and Canton; among these patients grave malnutrition and beri-beri were rare. The men stated that they had received fairly regular supplies of stale fish from the Japanese in addition to their basic ration and had been able to make small additional purchases from the Chinese.

The last convoys brought men repatriated from the Japanese Pacific Island bases of New Guinea and New Britain. These men, in addition to severe malnutrition had suffered severly from malaria, tuberculosis, yaws and tropical ulcers of the legs, and had been taken over in extremely poor condition by a number of Australian Field Medical Units, at whose hands they had received most excellent treatment for 3 months prior to their return to India.

Cases studied at the latter end of the series were drawn from units who had fought in the Burma Campaign and had subsequently been included in the forces of occupation. Their usual statement was that they were vegetarians or, if meat-eaters, had received only rare meat rations. Dried meat appears to have been regarded with much suspicion on religious grounds, and was rarely accepted by the type of patient received.

Some 2000 patients in all were examined and, from a study of these, our clinical impressions were formed. In addition a small group of 50 patients were selected, on clinical grounds only, for a more intensive investigation. This group was comprised of cases illustrative of the different types of deficiency syndrome encountered and is described in more detail in Section II. The patients of this group also formed the subjects of the laboratory investigation described in the succeeding sections. Table I gives a complete list of the names of the patients studied, together with the relevant particulars of regiment, age and religion.

4. *Control Group.*—A control group of 9 Indians was also studied at the I.M.H. Jalahalli, near Bangalore. These were patients, otherwise healthy, who were resting after a short course of penicillin thereapy for venereal disease. They were kept in bed during the investigations. The relevent particulars of this series are given in Table 2.

5. *Objects of the Research.*—Because of the cessation of hostilities, the objects of the research, as outlined in the G.H.Q. Conference on the Marasmus Syndrome in Indian Troops, had to be modified considerably. The investigation had now to be centered for the most part round the repatriated prisoners and because of the excellent work done immediately following upon their release and during their subsequent evacuation to India, much of the planned work, especially that on vitamin deficiency tests, had to be abandoned. The objects of the research at the time the team commenced to function could now be defined as follows :—

(1) To examine clinically as many patients as possible with a view to obtaining a satisfactory classification of the various clinical states which may be associated with marasmus and also with a view to gaining as much information as possible concerning the aetiology, treatment and prevention of the condition (Section II).

(2) To make a detailed clinical study of selected patients representing each of the main groups in the clinical classification, and to observe their progress and response to treatment (Section II).

(3) To carry out a haematological investigation on these representative patients (Section III).

(4) To make a study of the blood chemistry of the representative patients, including a study of the serum proteins, serum calcium, serum inorganic phosphorus, and serum phosphatase (Section IV.)

(5) To determine the blood volume and plasma volume of a smaller selected group of patients and, from these data, to determine the total circulating haemoglobin, red blood cells and plasma protein ; to observe the rate of recovery in terms of the rate of production of haemoglobin, red blood cells and plasma protein and so to determine the pattern of recovery in marasmus patients (Section V).

(6) To study the effect of transfusion of both plasma and whole blood to patients suffering from malnutrition (Section VI).

(7) To examine gastric function as represented by the fractional test meal findings and to investigate the effect of different treatments on those tests. (Section VII).

[(8) See at the bottom of page 5.**]

6. *Technical Methods.*—The following were the technical methods used :—

Haemoglobin was at first estimated by the alkaline haematin method recommended by King et al (1944), using a Klett colourimeter. Trouble was, however, experienced in the maintaining of a suitable standard. Most of the determinations were therefore done by the acid haematin method using an Adams diluting type haemoglobinometer with a Sahli—Adams square—sectioned diluting tube. The instrument, including the one pipette used throughout, was calibrated by the alkaline haematin method of King et al (1944) from two separate specimens of haemin, one

obtained from Professor E. J. King, British Post-Graduate Medical School, Hammersmith and one from Major M. Hynes R.A.M.C. of the G.H.Q. (India) Anaemia Research Team. The two specimens gave identical results.

Red Blood Cells were enumerated in a Spencer "Bright Line" counting Chamber with an Improved Neubauer ruling. The dilution was made with Hayem's fluid in each of two calibrated pipettes, and two counts were done on each dilution. Thus four counts in all were performed on every specimen of blood. The recorded result was the average of these four.

The Haematocrit was measured in a Wintrobe tube using the Wintrobe Oxalate mixture as an anticoagulant. The tubes were spun at 2500 r.p.m. for 1 hour.

Haematological Indices.—The Mean Corpuscular Volume (M.C.V.), Mean Corpuscular Haemoglobin (M.C.H.) and Mean Corpuscular Haemoglobin Concentration (M.C.H.C.) were calculated as described by Wintrobe (1932).

The Serum Protein was measured by the copper sulphate specific gravity method of Phillips et al. (1945). There is now ample evidence (see for example Hoch and Marrack (1945)) to show that this method is reliable, and repeated checks with the micro-Kjeldahl and the biuret method have confirmed this. When unusually low figures were obtained, the results were checked chemically. All determinations were made on serum, and the relation :

$$Pr—343\ (Gp—1.0070)$$

was used throughout, where Pr=Serum Protein Concentration in gm./100 cc. and Gp—the serum specific gravity. This relation was found to be correct for the low serum protein concentration found in patients suffering from malnutrition, but gave results up to 5% too low in normal and recovered patients. For convenience, however all results have been expressed in terms of this relation.

The Albumin/Globulin Ratio was measured by the biuret method using half-saturated ammonium sulphate as a precipitating reagent, and checked by the micro-Kjeldahl method using 22% sodium sulphate.

Serum Calcium was measured by the method of Kramer and Tisdall (1923).

Serum Inorganic Phosphorus was measured by the method of Briggs (1922).

Serum Phosphatase was determined by the method of King and Armstrong (1934).

Surface Area was calculated from body weight and height by the Du Bois formula.

Plasma Volume was measured by the Evan's blue technique. At first the method of Crooke and Morris (1942) was used but when the pocket photometer of King (1943) became available, the colour matching was done directly on diluted plasma without previous removal of proteins. The instrument, using an Ilford's Spectrum Orange light filter, is well suited for plasma volume determinations and it is felt that such errors as occurred were physiological rather than technical. Samples were withdrawn 10, 20 and 50 minutes after the dye injection and the plasma volume at zero time determined graphically.

Blood Volume was calculated from the plasma volume and the haematocrit.

Blood Sugar was estimated by the micro-method of Folin and Wu as described by Harrison (1944).

Serum Fat.—Serum total fat (ether soluble matter) was estimated gravimetrically after extraction in an all-glass Sexhlet apparatus, and the cholesterol was determined in the dried extract by the method of Myers and Wardell (1918).

Faecal Fat was determined as described by Harrison (1944).

7. *Acknowledgments.* We wish to acknowledge our indebtedness to the following :—

The many individuals of G.H.Q. (India) Medical Directorate, including the Director of Hygiene and Pathology and the Assistant Director of Pathology, who have assisted in the planning, organisation, and the reporting of this work ; the G.H.Q. Research Statistical Officer (Med. Dte.) who has assisted with the statistical evaluation of many of the results ; G.H.Q. Directorate of Services Kinematography for the photographic reproduction of figures ; D.D.M.S. Southern Army for his interest ; A.D.M.S. 109 L of C Area and Administrative Commandant, Hospital Town, for their help in overcoming many of the local difficulties ; Garrison Engineer, Hospital Town, for his many speedy installations in the laboratory and for his help in the preparation of graphs for this report ; O.C. 145 I.B.G.H. (I.T.) for making us so welcome in his hospital and for his continued help; O.C., I.M.H. Jalahalli, for providing us with the facilities for studying the 'control series'.; O.C., 146 I.B.G.H. (I.T.) for permitting us to study 4 cases in his hospital; the many medical officers of 145 I.B.G.H. who helped us greatly ; the nursing, ward, and laboratory staff ; and above all, the patients themselves, who had endured much, but were always co-operative, sometimes to the degree of embarrassment.

**(8) To investigate gastric function as represented by carbohydrate and fat tolerance tests and to judge the effect of different treatment on these tests (Section VIII).

Table 1.

Particulars of Patients of the Special Investigation.

Serial No.	Name.	Regiment.	Age.	Religion.
1	Venkataswamy.	13 I. P. C.	21	Hindu.
2	Abdul Hakim.	R.I. A. S. C.	25	Mohammedan.
3	Rikhi Ram.	2/17 Dogra.	25	Hindu.
4	Amar Singh.	R. I. A. S. C.	33	Hindu.
5	Dundu Mali.	13 I. P. C.	35	Hindu.
6	Rustam Ali.	R. I. A. S. C.	47	Mohammedan.
7	Mohan Lal.	I. A. O. C.	33	Hindu.
8	Abdul Rehman.	2/16 Punjab.	25	Mohammedan.
9	Khandoji Rao.	1st Mysore Inf.	25	Hindu.
10	Mohd. Araf.	2/10 Baluch.	25	Mohammedan.
11	Allah Din.	2/10 Baluch.	37	Mohammedan.
12	Ramaswamy Naidu.	1st Mysore Inf.	27	Hindu.
13	Lal Hussain.	5/2 Punjab.	30	Mohammedan.
14	Sher Gul.	2/16 Punjab.	22	Mohammedan.
15	Ghaus Mohd.	I. A. M. C.	27	Mohammedan.
16	Sher Din.	I. A.	25	Mohammedan.
17	Ghulam Haidar.	I. A.	26	Mohammedan.
18	Lal Khan.	13 I. P. C.	30	Mohammedan.
19	Hazir Khan.	2/10 Baluch.	25	Mohammedan.
20	Ganda Singh.	2/17 Dogra.	40	Hindu.
21	Moula Baksh.	7/8 Punjab.	30	Mohammedan.
22	Babu More.	13 I. P. C.	33	Hindu.
23	Ahmed Khan.	R. I. A. S. C.	32	Mohammedan.
24	Ahmed Din.	1/14 Punjab.	26	Mohammedan.
25	Manik Thakore.	13 I. P. C.	30	Hindu.
26	Surup Singh.	2/9 Jat.	24	Hindu.
27	Ashik Ali.	R. I. A. S. C.	25	Mohammedan.
28	Dalip Singh.	HKS. Heavy AA.RA.	36	Sikh.
29	Bachuttar Singh.	I. G. S. C.	24	Sikh.
30	Syed Ahmed Shah.	HKS. Heavy AA. RA.	30	Mohammedan.
31	*Nihal Singh.	1/19 Hyderabad.	31	Hindu.
32	*Sarjit.	R. I. A. S. C.	28	Hindu.
33	Binda.	I. A. M. C.	45	Hindu.
34	Lal Singh.	I. E.	27	Hindu.
35	Akbar Khan.	1st Burma Regt.	33	Mohammedan.
36	Sohan Sigh.	Singapore Police.	40	Sikh.
46	*Godhu Ram.	R. I. A. S. C.	29	Hindu.
47	*Gayadin.	I. S. C.	30	Hindu.
48	*Nizam Khan.			Mohammedan.
49	*Ram Singh.	7 Rajput Regt.	29	Hindu.
50	Noor Khan.	HKS. Heavy AA. RA.	23	Mohammedan.

*Not repatriated prisoners of war.

Table 2.

Particulars of Patients of the Control Group.

Serial No.	Name.	Regiment.	Age.	Religion.
N37	Kheta Singh.	I. A.	23	Sikh.
N38	Syed Jagaria.	I. E.	21	Mohammedan.
N39	Sonin Samuel.	I. A. M. C.	22	Christian.
N40	Hanu Mathur.	I. E.	22	Hindu.
N41	Chinappa.	I. A. M. C.	20	Hindu.
N42	Annamalay.	I. S. C.	26	Hindu.
N43	Mohd. Khan.	I. A. M. C.	29	Mohammedan.
N44	Rachappapatil.	I. E.	23	Hindu.
N45	Munshi Ram.	I. E.	23	Hindu.

SECTION II

CLINICAL FEATURES.

The cases studied fall into 5 main groups, *viz.*, (1) those of pure marasmus showing marked wasting but little evidence of specific vitamin deficiency sydromes, (2) those of marasmus with gross hypoproteinaemia, (3) those of marasmus with marked alimentary dysfunction due to vitamin B_2 group deficiencies, (4) those showing the various neurological syndromes, and (5) those in whom anaemia was the predominant feature.

1. *Pure Marasmus.* The following cases are taken as representative of the first group :—

(*a*) *Serial No.* 26. Sep. Surup Singh 2/9 Jat Regiment, age 24 captured in Singapore 15-2-42 he remained fairly fit for 2 years when he had malaria with fever lasting for 15 days, which was not treated. Early in 1945 he developed dysentery lasting for 6 days, became oedematous, developed ascites and was admitted to Neesoon Hospital where he remained until released. His diet there consisted of 6 oz. rice, some greens, and occasional fish. He received injections of rice-polishing extract in hospital with some relief.

On examination he appeared alert and intelligent, but his memory was poor. Tongue : large, sore and red and showing hypertrophy of the papillae. Skin : dry and atrophic, generalised hyperkeratosis follicularis with a 'crazy pavement' type of eruption over the extensor surfaces. B.P. 96/54. Abdomen, which had been tapped 4 times at Neesoon Hospital, showed slight ascites on arrival. The C.N.S. showed no change except sluggish knee and ankle jerks.

Investigations showed mild macrocytic anaemia, R.B.C. 3.01×10^6 cells./cmm., Hb 11.0 gm./100 cc., M.C.V. 111 cμ, M.C.H.C. 32.8%, low serum albumin—A, 1.98 gm./100 cc. G 3.12 gm./100 cc., total serum protein 5.10 gm./100 cc., A/G ratio 0.66/1. Gastric acid production was normal and the glucose tolerance curve only slightly flat.

He received two blood transfusions each of 1 pint and 500 cc. double-strength reconstituted plasma, short courses of parenteral nicotinic acid and riboflavin and parenteral liver extract 4 cc. twice weekly. His appetite was good and he immediately began to improve. After 2 months treatment his weight had increaded from 83 lbs. to 123 lbs., his blood volume from 2930 cc. to 4760 cc., his blood pressure to 110/64 and his appearance was that of extreme well being. Blood examination showed R.B.C. 4.39×10^6 cells/cmm., Hb 14 g. % M.C.V. 95 cμ. M.C.H.C. 33.7%, and serum protein 6.44 gm.% (A/G ratio, 1.09/1).

(*b*) *Serial No.* 25.—Pioneer Manik Thakore, 13 I.P.C.; age 30, captured in Singapore 15-2-42, he remained fairly healthy for 2 years when, following two attacks of malaria and dysentery, he developed generalised neuritic pains. Later he became oedematous, but never developed ascites. He was bedridden in Neesoon Hospital for nearly a year before his release.

On examination on 29-9-45 he was listless but rational. His tongue showed central atrophy and marginal redness with scanty swollen papillae. These changes were only of minor degree and rapidly disappeared. There was slight oedema of the ankles. The heart sounds were of poor quality and the pulse soft. B.P., 104/54. There was a complete lesion of the right common peroneal nerve considered to be traumatic, but no other neurological abnormality save sluggish knee and ankle jerks. His height was 67 in. and weight 95 lbs. on admission. There was a mild degree of macrocytic anaemia (R.B.C. 3.08×10^6 cells/cmm. Hb. 9.3 gm. % M.C V. 106 cμ.

M.C.H.C. 28.7%). The serum albumin was somewhat low, the total protein being 5.17 gm. % (A/G ratio, 0.92/1). Fractional test meal showed hypochlorhydria with a good response to histamine ; after 7 days treatment with parenteral riboflavin, 3 mgm. daily, he showed a normal acid curve. The glucose tolerance curve was flat initially but had returned to normal within 4 weeks of admission, blood and plasma volumes were just over 2/3 normal.

Following treatment with plasma transfusion, 2 pints, riboflavin and liver extract, 4 cc. every second day, for 2 weeks, he made steady progress. His weight, which was 75 lbs. initially, increased by 49 lbs. in 8 weeks. He was discharged with a normal blood picture and chemistry after 10 weeks treatment.

Comment.—These cases, on first examination, showed marked wasting without clinical evidence of serious hypoproteinaemia or anaemia, and with only slight signs of ariboflavinosis. Since their digestive functions were but little impaired, they made a rapid recovery and only needed specific vitamin therapy for a short period initially, with blood and plasma transfusion to give them a flying start.

2. *Marasmus with Gross Hypoproteinaemia.*—The following cases are cited as examples of marasmus accompanied by gross hypoproteinaemia.

(*a*) *Serial No.* 6.—Sep. Rustum Ali, R.I.A.S.C., age 47, captured in Singapore in February, 1942, he remained a prisoner on the island. In 1943 polyneuritis began, but he continued to work until in May 1945; following a sharp attack of dysentery, he became oedematous and was treated in hospital with injections (? vitamin B_1) for 2 months, when he was able to leave. In August 1945 he was re-admitted owing to extreme weekness and much oedema. During his evacuation to India by hospital ship, he received a blood transfusion of 2 pints but, despite this, on arrival he showed gross anasarca with anaemia.

His heart sounds were feeble, B.P. 104/60, his tongue showed atrophy of the epithelium, the stools were loose, frothy and fatty and the skin was dry and atrophic.

Investigation showed R.B.C. 2.05×10^6 cells/cmm., Hb 7.5 gm./100 cc. M.C.V. 122 c.μ. M.C.H.C. 30.0%, serum protein 4.20 gm./100 cc. (albumin 1.46 gm. %, globulin 2.74 gm. %, A/G ratio, 0.53/1). Gastric analysis showed a histamine refractory achlorhydria ; the stools contained 32% total fat of which 71% was split. The glucose tolerance curve was flat, with a maximum rise of 33 mgm./100 cc. after one hour.

He received a transfusion of 2 pints whole blood, parenteral liver extract, 4 cc. daily, and parenteral nicotinic acid, 200 mgm. daily. The oedema and ascites cleared within 2 weeks with a resulting fall in body weight of 12 lbs. A second gastric analysis after 2 weeks showed 7 ccs. N/10 free acid per 100 cc. after 45 minutes. with a good response to histamine. Two months after admission his weight had increased from 78 lbs. to 119 lbs. and he appeared well nourished . His R.B.C. was now 3.8×10^6 cells/cmm., Hb 11.5 gm./100 cc., M.C.V. 96 cμ., M.C.H.C. 32%, and serum protein 7.25 gm./100 cc. (albumin, 3.62 gm. %, globulin, 3.62 gm. %, A/G ratio, 1/1.). The gastric acid secretion had returned to normal while the faecal fat (on a diet containing 180 gm. fat) was 27.6 %, of which 70 %, was split, while the fat tolerance curve was normal. This remarkable improveme nt in the blood count and chemistry is reflected in the changes in his appearance during recovery (see photograph p. 10a).

(*b*) *Serial No.* 2.—Sep. Abdul Hakim, R.I.A.S.C. age 25, held prisoner in Singapore since February 1942. Symptoms of polyneuritis began after 3 attacks of dysentery in November 1942. In June 1943 there was oedema of the whole body which was relieved by 22 injections (? vitamin B preparation). By December 1943 his lower limbs had lost all sensation and in March 1944 oedema and ascites again developed ; he became bedridden and so remained until his evacuation.

On admission he showed oedema and gross ascites. His skin was covered with the lesions of infected scabies, his hair was dry and thin and his tongue slightly atrophic. The heart sounds were weak and the B.P. 92/62. There was loss of sensation over the feet and tendon jerks in the lower limb were obtained only with difficulty.

Routine investigation showed R.B.C. 2.2×10^6 cells/cmm., Hb 5.6 gm./100 cc. M.C.V. 136 cμ. M.C.H.C. 29 %, serum protein 4.0 gm./100 cc. (Albumin 1 gm. %, globulin 3 gm. %, A/G ratio 0.3/1), serum calcium 5.9 mg./100 cc. with no overt or latent tetany. The abdomen was tapped and 7 pints of peritioneal transudate drawn off, after which the liver was felt to be enlarged to 4 fingers breadth and hard.

He was given the dried plasma from 750 cc. blood reconstituted to 400 cc. by intravenous drip. The haematocrit fell from 30% to 23% with no appreciable rise in the serum protein concentration. He was then transfused with 1 pint of whole blood, after which the oedema and the ascites were absorbed and the patient began to gain weight rapidly. He was discharged after 3 months treatment in hospital in good health, but with a liver which was still enlarged and hard ; he had gained 29 lbs. in weight and had altered remarkably in appearance (see photograph p. 10b). An examination on discharge showed R.B.C. 3.75×10^6 cells/cmm., Hb. 11.5 gm./100 cc., M.C.V. 98 cμ., M.C.H. C. 31 %, serump rotein 7.36 mgm/100 cc. Albumin 3.51 gm. %, globulin 3·85 gm. %. A/G ratio, 0.9/1), serum calcium 10.7 mgm./100cc. serum inorganic phosphorus 4.4 mgm. %. He appeared to have developed a symptomless cirrhosis of the liver.

C. Serial No. 24.—Sep. Ahmed Din 1/14 Punjab Regt., age 26. This patient had little memory for events over the past two years ; he was captured in February 1942 and held in Singapore. He became weak following an attack of dysentery in 1943 ; anasarca developed and he remained bedridden.

On arrival, he showed extreme emaciation, many bedsores, and oedema of the dependant parts with much ascites (see photograph p. 10c). He was restless and disorientated but had a voracious appetite. His lips showed angular stomatitis and cheilosis while his tongue, though not atrophic, was bright red and showed swelling of individual fungiform papillae (" red mushrooms ") ; these changes were considered typical of ariboflavinosis (Jones et al. (1944)) and responded within 7 days to parenteral riboflavin 4 mg daily. The skin was dry and showed " crazy paving " eruption over the whole body. The heart sounds were feeble. B.P. 100/74.

The following were the initial findings : R.B.C. 2.85×10^6 cells/cmm., Hb. 8.8 g./100cc. M.C.V.104 cμ. M.C.H.C. 29.5 %, serum protein 2.88 g./100 cc. (albumin 0.75 gm%, globulin 2.13 gm. %, A.Gratio, 0.3/1). Initial plasma volume, 2480cc ; blood volume 3500cc.

He was given transfusions of dried plasma from 1000 cc. blood reconstituted to double strength on 3 occasions over 10 days ; 24 hours after the last of these all the oedema had cleared (see photograph p. 10c). Each transfusion added approximately 70 gm. protein to his circulation. Total circulating plasma protein estimations 24 hours after each infusion showed that only 9 gm. were retained in the circulation from the first, 26 gm. from the second, and the whole amount from the third. (see Section VI). This man thereafter continued to eat enormously and was discharged in good condition after 11 weeks, treatment, during which he had gained 47½ lbs. in weight (see photograph p. 10c). His confusional state had improved leaving him with a somewhat childish mentality. Normal faecal fat and normal fat and glucose tolerance curves were found on discharge. The final plasma volume was 3360 cc. and blood volume 5600 cc.

D. Serial No. 5.—Pioneer Dundumali, I.P.C., age 35. Held prisoner in Singapore since February 1942, he was admitted to hospital in June 1943 on account of generalised oedema, followed a year later by ascites, which had been tapped 5 times before his release. He had a sore tongue and loose motions for many months prior to release.

On admission his face was puffy ; there was moderate ascites but only slight oedema. The skin along the naso-labial folds showed a greasy seborrhoea, the lips showed perleche and the tongue was large and red and was covered by swollen, eroded papillae. The skin was scaly and atrophic but there were no gross neurological signs except loss of ankle jerks.

Investigation showed R.B.C. 2.48×10^6 cells/cmm., Hb. 10.75gm./100 cc. M.C.V. 133 c.μ., M.C.H.C. 32.5%, serum protein 4.18 gm./100 cc. (albumin 2.0 gm.%, globulin 2.18 gm. %, A.G ration B.0.9/1), serum calcium 6.6 mgm. % without signs of latent tetany.

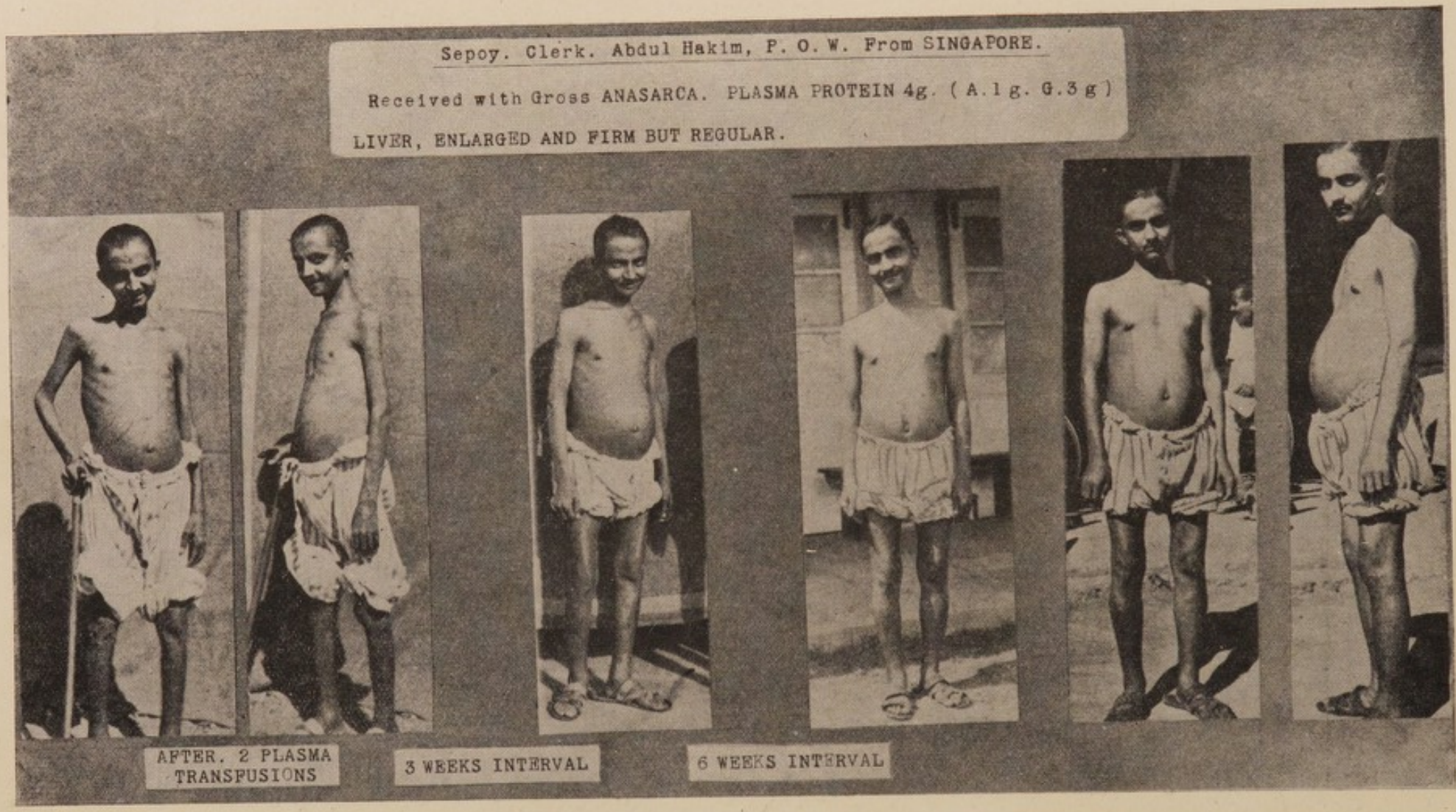
SEPOY. BUTCHER RUSTOM ALI P.O.W. FROM SINGAPORE.

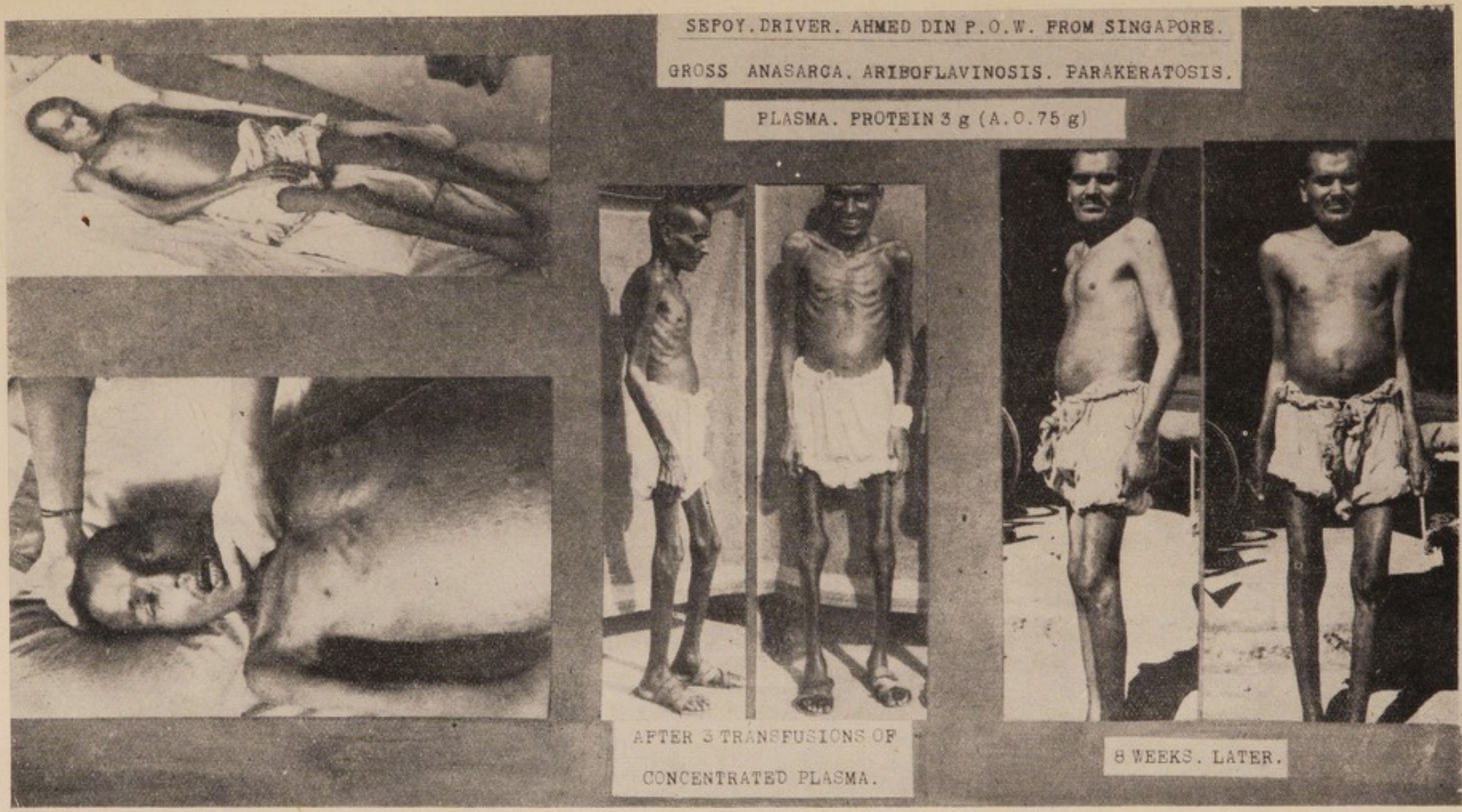
RECEIVED WITH GRAVE MACROCYTIC ANAEMIA & ANASARCA.

AFTER 2 CONCENTRATED PLASMA & 2 BLOOD TRANSFUSIONS

3 WEEKS LATER.

6 WEEKS LATER.


Sepoy. Clerk. Abdul Hakim, P. O. W. From SINGAPORE.
Received with Gross ANASARCA. PLASMA PROTEIN 4g. (A.1 g. G.3 g)
LIVER, ENLARGED AND FIRM BUT REGULAR.
AFTER. 2 PLASMA TRANSPUSIONS
3 WEEKS INTERVAL
6 WEEKS INTERVAL


SEPOY. DRIVER. AHMED DIN P.O.W. FROM SINGAPORE.
GROSS ANASARCA. ARIBOFLAVINOSIS. PARAKERATOSIS.
PLASMA. PROTEIN 3 g (A.O.75 g)
AFTER 3 TRANSFUSIONS OF
CONCENTRATED PLASMA.
8 WEEKS. LATER.

He was treated with riboflavin and, because of the moderate steatorrhoea, with nicotinic acid. He made steady progress and on discharge the findings were : gain in weight, 99 lbs. to 133½ lbs. in 9 weeks, R.B.C. 4.28×10^6 cells/cmm., Hb. 13.0 gm. /100 cc., M.C.V. 93.5 cμ. M.C.H.C. 32.5%, serum protein 1.17 gm./100 cc. (albumin 2.86 gm. %, globulin 4.31 gm. %, A/G ratio 0.66/1), serum calcium 10.5 mgm./100 cc. His liver remained palpable to 2 fingers' breadth below the costal margin and was abnormally hard.

E. Serial No. 22.—Pioneer Babu More, I.P.C., age 33. Held captive in Singapore since February 1942, he suffered from several attacks of malaria but remained at work until May 1945. Then after further attacks of malaria he lost his appetite and his legs began to swell. This oedema was said to have been relieved by injections and was not present on his arrival in India.

On examination he appeared extremely emaciated ; his skin was covered by the lesions of infected scabies and his hands and feet showed extensive epidermophytosis. Although his abdomen was distended, there was no free fluid and oedema of the legs was absent. His tongue showed gross ariboflavinosis, being swollen, raw and bright red ; there was marked excoriation at the angles of the mouth. He had little appetite, vomited periodically and was passing loose watery motions which contained no exudate.

After 10 days stay in a general ward where he received a course of sulphaguanidine with only slight benefit, ascites and oedema began to reappear and full investigation was undertaken, which showed :—R.B.C., 3.01×10^6 cells/cmm., Hb. 10.6 gm./190 cc. M.C.V. 103 cμ. M.C.H.C. 32. 7%, serum protein 5.23 gm./100 cc. (albumin 1.67 gm. %, globulin 3.56 gm. %, A/G ratio 0.47/1), faecal fat 19.8% of which 92% was split. Glucose and fat tolerance curves were within normal limits.

He received a short course of parenteral riboflavin, 4 mgm. daily, for 2 weeks with oral nicotinic acid 300 mgms. daily and, despite a relapse of B.T. Malaria, showed marked improvement with loss of oedema after 2 weeks. He was discharged after 3 months treatment during which his weight had increased from 89 lbs. to 112 lbs. Blood examination showed R.B.C. 5.16×10^6 cells/cmm., Hb. 15 gm./100 cc. M.C.V 89 cμ., M.C.H.C. 32.6%, serum protein 7.12 gm./100 cc. (albumin 3.39 gm., %, globulin 3.73 gm. %, A/G ratio, 0.91/1).

This case illustrates a previously observed phenomenon of 'delayed oedema'. It would appear that just below the critical level of plasma protein concentration (or rather, albumin concentration) the development of oedema may be held in abeyance by salt and fluid deficiency, only to appear later when the salt and water depletion is made good. This man had received no mercurial diuretics.

Comment.—These cases bear out reports of the therapeutic efficiency of transfusion of concentrated plasma for patients with a severe degree of hypoproteinaemia. The cases were comparable in severity with those treated at Belsen (Mollison (1946)) and the response appeared to be just as good (Janet Vaughan (1945)). No dangerous reactions, such as those mentioned by Lipscomb (1945), followed any of our transfusions.

3. *Marasmus Associated with Gross Vitamin* B_2 *Group Deficiencies.*—

(*a*) *Serial No.* 15.—Ambulance Sep. Ghaus Mohd., I.A.M.C., age 27. A captive in Singapore since February 1942, he was admitted to hospital in May 1944 with dysentery and anaemia. He had a malarial relapse in November 1944 and remained bedridden until repatriated.

On admission he looked desparately ill ; he was cachectic, vomited after every feed, and passed semi-solid stools. Signs of chronic bronchitis were present ; the heart sounds were feeble and the pulse weak, B.P. 92/62. There was oedema of the feet and moderate ascites. The lower border of the liver was palpable but not tender. A tentative diagnosis of carcinoma ventriculi was made and full investigation undertaken.

The R.B.C. was 1.66×10^6 cells/cmm., Hb. 9.1 gm./100cc. M.C.V. 154 cμ. M.C.H.C. 35.5%, serum protein 4.78 gm./100 cc. (albumin 1.2 gm./100 cc. globulin 3.6 gm. % A/G ratio, 0.3/1). Fractional test meal showed only traces of free acid even after histamine injection and was not improved 10 days later. He had a flat glucose tolerance curve.

He was treated with nicotinic acid, 200 mgm. parenterally, 300 mgm. orally and liver extract 4 cc. intramuscularly daily. Four weeks later his condition was much improved, vomiting had ceased and his stools were normal and, despite the loss of all oedema, he had gained 8 lbs. in weight. Parenteral nicotinic acid was then discontinued. Although he successively developed a B.T. malaria relapse, cellulitis of the feet secondary to epidermophytosis, and an exacerbation of chronic otitis media, he continued to gain weight rapidly, and was discharged after 4½ months treatment. His weight had increased from 94½ lbs. to 144 lbs. and his blood picture had returned to normal. The liver remained palpable to 1 finger's breadth, somewhat hard, and slightly tender. On discharge his R.B.C. was 5.25×10^6 cells/cmm., Hb. 15.5 gm./100 cc, M.C.V. 84 c.μ., M.C.H.C. 35%, serum protein 7.35 gm. 100 cc. (albumin 4.3 gm. %, globulin 3.05gm.%, A/G ratio 1.4/1). Despite the marked improvement on nicotinic acid, gastric achlorhydria persisted, but gastric acidity ultimately returned to normal after heavy parenteral administration of riboflavin (see Section VII).

(*b*) *Serial No.* 18. Hav. Lal Khan, I.P.C., age 30. This man after his capture in February 1942 remained fairly healthy until August 1945, when he contracted dysentery; his stools thereafter never became formed; he developed complete anorexia and sore tongue and rapidly became weaker.

On admission at the end of September, he was extremely emaciated; his tongue showed both marked atrophy and marginal redness and he was passing 8-10 loose, pale frothy motions daily. There was moderate peripheral neuritis of the legs; vision was 6/18 in each eye and the ocular fundi showed temporal pallor of the disc. Perimetry showed gross concentric contraction of the visual fields. Laboratory findings were, R.B.C. 3.55×10^6 cells/cmm., Hb. 11.5 gm./100 cc., M.C.V. 101 c.μ., M.C.H.C. 32%, serum protein 5.34 gm./100 cc. (albumin 2.1 gm. %, globulin 3.24 gm. %, A/G ratio 0.65/1). The glucose and fat tolerance curves were flat. Gastric analysis showed achlorhydria with slight response to histamine.

He was treated with oral nicotinic acid, 300, mgm. daily; with slight interruptions while the effects of short courses of riboflavin and liver extract on the gastric function were tested. After 5 weeks' treatment his general nutrition was much improved, the tongue epithelium had regenerated and his glucose tolerance curve was nearly normal.

On his discharge, after 3 months' treatment, his weight had increased from 88½ lbs. to 130 lbs. and no evidence of beri-beri remained. There was no increase in the visual fields. Blood, examination showed R.B.C. 4.64×10^6 cells/cmm., Hb 13.8 gm./100 cc. M.C.V. 90 c.μ., M.C.H.C. 32%, serum protein 6.84 gm./100 cc. (albumin 3.80 gm.% globulin 3.04 gm.%, A/G ratio, 1.25/1). Fractional test meal and fat and glucose tolerance curves were all normal.

(*c*) *Serial No.* 28.—Nk. Dalip Singh, Hong-Kong, Singapore Regt., R.A., age 36. A captive from Singapore who had had burning of the tongue and abdominal distension with loose motions for over 2 years prior to his release.

He was very thin with a tongue with central atrophy and marginal redness. He had a gurgling distended colon and his motions, passed 4 times daily, were loose, fatty and offensive. Investigation showed R.B.C. 3.5×10^6 cells/cmm., Hb. 11.2 gm./100 cc. M.C.V. 109 c.μ., M.C.H.C. 33%, serum protein 5.48 gm./100 cc. (albumin 2.8 gm.%, globulin 2.68 gm. %, A/G ratio, 1.04/1). Gastric analysis showed a delayed production of a normal amount of acid and fat and glucose tolerance tests showed flat curves. Faecal fat estimation gave a total fat of 58.4 gm. % of which 68% was split.

The marginal redness of the tongue responded to 3 injections of 3 mgm. riboflavin, but relapsed on withdrawal of the vitamin; steady regeneration of the central epithelium occurred on nicotinic acid 300 mgm. orally. The stools became formed within 3 weeks and a rapid gain in weight took place. He was discharged after 4months' treatment during which his weight had increased from 112 lbs. to 150½ lbs. A final investigation showed R.B.C. 3.75×10^6 cells/cmm., Hb. 14 gm./100 c.c. M.C.V. 106 c. μ., M.C.H.C. 35%, serum protein 6.66 gm./100 c. c. (albumin 4.19 gm. %, globulin 2.47 gm. %, A/G ratio 1.7/1); gastric analysis, normal curve; glucose and fat tolerance curves, normal; faecal fats, 22.9 gm.% of which 35% was split.

(*d*) *Serial No.* 36.—Police Sep. Sohan Singh, Malayan Police, age 40. Following capture in Singapore in February 1942 he remained in fair health, despite attacks of malaria, until March 1945 when diarrhoea (without blood) and vomiting began and continued until his admission. His mouth became sore soon after and oedema of the whole body developed. Prior to his arrival in India he had received a transfusion of plasma, 1 pint, and saline, 3 pints, and 5 intravenous injections of 200 mgm. nicotinic acid.

He was very wasted with a distended tympanitic abdomen, but was free from oedema and ascites. His skin showed hyperkeratosis as well as a 'crazy paving' type of parakeratosis. Investigations showed, R.B.C. 2.21×10^6 cells/cmm., Hb. 7.8 gm./100 c.c. M.C.V. 117.6 c.μ., M.C.H.C. 30%, serum protein 5.13 gm./100 c.c. (albumin 2.56 gm. %, globulin 2.56 gm.%, A/G ratio, 1/1); faecal fat, 82.3 gm. % of which 64% was split; glucose and fat tolerance tests, flat curves; gastric analysis, achlorhydria responding to histamine.

Initial treatment was by riboflavin only, 12 mgm. orally and 4 mgm. subcutaneously; on this the marginal redness of the tongue disappeared, but no general improvement, or alteration of the stool was evident after 3 weeks, and he had gained only 1 lb. in weight. At this stage nicotinic acid therapy was substituted for riboflavin the daily dosage being 500 mgm. orally, 150 mgm. parenterally, with liver extract 8 cc. parenterally. After 7 days his appetite had begun to return, his weight increased by 6 lbs., and his stools became loosely formed. Thereafter rapid improvement continued and he was discharged after 3 months' treatment during which his weight had risen from 109 lbs. to 155 lbs. Final investigation showed, R.B.C. 3.64×10^6 cells/cmm., Hb. 13.1 gm./100 c.c. (albumin 3.87 gm. %, globulin 2.39 gm. %, A/G ratio, 1.6/1); faecal fat 17.1 gm. %, all of which was split; gastric analysis, hypochlorhydria; glucose and fat tolerance tests, normal.

(*e*) *Serial No.* 11.—Sep. Allah Din, 2/10 Baluch. Regt., age 37. Following his capture in Singapore, he remained fairly well until May 1944 when, after a short attack of dysentery, he lost his appetite, his tongue became sore and he began to pass loose bubbly motions with much flatulence. This condition persisted until he reached India.

He was a tall, emaciated man, with an atrophic, glazed, mauve-coloured tongue, and with a much distended, tympanitic abdomen. His stools were pale and frothy. His blood pressure was 90/68. His skin was dry and atrophic and his hair thin. Laboratory findings were :—R.B.C. 2.36×10^6 cells/cmm., Hb. 11.1 gm./100 c.c. M.C.V. 134 c.μ., M.C.H.C. 35%, serum protein 5.4 gm./100 c.c. (albumin 3.22 gm.% globulin 2.2 gm. %, A/G ratio, 1.46/1); gastric analysis, delayed acid production; fat and glucose tolerance tests, slightly flat curves; faecal fat, 46 gm. % of which 68% was split.

Treatment was begun with nicotinic acid, 200 mgm. orally and 150 mgm. subcutaneously, to which was added a short course of riboflavin when he developed, in addition, a marginal redness of the tongue. His appetite soon returned, the tympanites and steatorrhoea subsided, the epithelium of the tongue regenerated and he rapidly gained weight. However after 7 weeks' treatment his blood still showed a macrocytosis (R.B.C. 3.50×10^6 cells/cmm., Hb 12.6 gm./100 c.c. M C.V. 108.5 c.μ., M.C.H.C. 33%) and this still persisted on his discharge after 9 weeks' treatment, despite 5 injections of 4 cc. liver extract. His weight increased by 30 lbs. and his B. P. rose to 110/70. Final laboratory results were :—R.B.C. 3.63×10^6 cells/cmm.,

Hb. 13.1 gm./100 c.c. M.C.V. 108 c.μ., M.C.H.C. 33.5 %, serum protein 6.5 gm./100 c.c. (albumin 3.76 gm. %, globulin 2.74 gm. %, A/G ratio, 1.4/1), total faecal fats 32.1% of which 69% was split; fat and glucose tolerance curves, normal.

(*f*) *Serial No.* 32.—Barber Sarjit, R.I.A.S.C., age 28. This man was not a P.O.W. By habit a strict vegetarian, he served for 6 months in the Middle East in 1941, remaining healthy and, after returning to India was drafted to Iraq in 1942. There he developed 'dyspepsia' and severe macrocytic anaemia in December 1943, and was evacuated as 'seriously ill' to India in January 1944. After 2 months' treatment in hospital, he was sent on active service to Burma in January 1945. He remained on duty until July 1945, when he lost his appetite and became weak and wasted. He was admitted to hospital in August with loose stools and a sore tongue and was evacuated to India in November. During this period he received 4 transfusions, each of 1 pint of whole blood, 14 cc. liver extract by injection and nicotinic acid 300 mgm. daily; however, the lack of symptoms of vasodilation suggested that he was not able to absorb the latter drug.

On examination in November 1945, he was extremely wasted, with a tongue which was slightly sore, glazed, pale-mauve in colour, and markedly atrophic. His skin was dry, with a Branny desquamation and showed extensive hyperkeratosis follicularis. His resting pulse rate was 104 per minute. There was a loud haemic murmur over the base of the heart and the B.P. was 104/66. Oedema was present up to the knees. The abdomen was sunken with a palpable spleen and liver; both organs were firm and not tender and suggested a chronic malarial infection. The stools were very bulky, pale, frothy and very offensive. Laboratory investigation showed:—R.B.C. 1.96×10^6 cells/cmm., Hb. 5.5 gm./100 c.c. M.C.V. 97 c.μ., M.C.H.C. 29%, serum protein 6.33 gm. 100 c.c. (albumin 3.44 gm. %, globulin 2.89 gm. %, A/G ratio, 1.2/1); plasma volume 2400 cc., blood volume 2960 cc; gastric analysis, histamine refractory achlorhydria; glucose tolerance test, an extremely flat curve; faecal fat, 27.1 gm. % of which 74% was split.

He thus presented a picture typical of gross nicotinic acid deficiency and treatment was begun with this vitamin 300 mgm. orally and 100 mgm. parenterally. After 10 days his appetite had returned, his tongue was no longer sore, its epithelium showed regeneration, and the stools were reduced to 2 daily, while the glucose tolerance curve was normal. Since achlorhydria persisted, riboflavin was substituted for nicotinic acid and he received 12 mgm. daily by mouth and 4 mgm. by injection for the next 6 weeks.

After 3 months' treatment his state of nutrition was excellent, the tongue was normal and the B.P. 122/70, while his weight had increased from 76 lbs. to 110 lbs. Final investigations showed:—R.B.C. 5.19×10^6 cells/cmm., Hb. 15.4 gm./100 c.c. M.C.V. 83 c.μ., M.C.H.C. 36 %, serum protein, 7.46 gm. 100 c.c. (albumin 4.96 gm. %, globulin 2.5 gm. %, A/G ratio, 2.0/1); plasma volume 2420 cc., blood volume 4240 c.c; gastric analysis, achlorhydria responding to histamine.

This vegetarian had therefore, developed a deficiency syndrome for the second time on active service; the presenting features were those of nicotinic acid deficiency and he showed a prompt response to the administration of this vitamin, which was then discontinued in order to observe the effect of riboflavin on gastric acid production; the administration of this vitamin was unnecessarily prolonged owing to the inexperience of the medical officer in charge of the case.

(*g*) *Serial No.* 31.—L/Nk. Nihal Singh, 1/19 Hyderabad Regt., age 31. This man was not a P.O.W. Previously a vegetarian, he had learned to eat mutton with his unit 4 years previously, but suffered the usual privations of the Burma Campaign from October 1944 where the arrival of rations, especially meat, was uncertain and irregular. In July 1945 he was admitted to hospital, following an attack of dysentery, for anaemia, sore tongue, and wasting. He was treated with one blood transfusion, and irregular small doses of liver extract and nicotinic acid, but had not improved by the time he was evacuated to India in November.

He was a big, very emaciated, anaemic man, with a dry skin showing hyperkeratosis follicularis. His lips showed marked angular stomatitis and the scars of

cheilosis, while the tongue was large, folded, very sore and bright purplish-red in colour. In addition to swollen, eroded papillae round the margins, there was marked atrophy of the medial zones of epithelium. His B.P. was 92/60; there was no oedema and only moderate anaemia. Peripheral neuritis of the legs was present. Investigation showed :—R.B.C. 2.10×10^6 cells/cmm., Hb. 9.5 gm. /100 c.c.M.C.V. 137 c.μ., M.C.H.C. 33%, serum protein 5.3 gm. /100 c. c. (albumin 2.75 gm. %, globulin 2.55 gm. %, A/G ratio, 1.08/1) ; plasma volume 2570 cc. and blood volume 3600 cc ; glucose and fat tolerance curves, very flat ; faecal fat, 49 gm. % of which 77 % was split ; gastric analysis, normal acidity, but delayed emptying-time.

Treatment was begun with riboflavin 9 mgm. parenterally for a week, then 12 mgms. orally for 10 days, after which, the lip lesions had healed and the inflammation and soreness of the tongue had been relieved, leaving a pale glazed atrophic epithelium, but there was complete absence of general improvement. Appetite had not increased and weight had decreased by 4 lbs., while the stools and the absorption tests showed no improvement. Treatment was then changed to nicotinic acid 300 mgm. by mouth. There was no evidence that the drug was being absorbed and after a week the weight had fallen. Finally parenteral nicotinic acid therapy, 150-200 mgm. was instituted with immediate improvement, the weight increasing for the first time since admission. After 7 days of this treatment he had gained 11 lbs. and he continued to increase in weight until his discharge 11 weeks later.

During the period of 11 weeks observation in hospital his weight increased from 98 lbs. to 151 lbs. On discharge he was in excellent state of nutrition. His B.P. was 112/60 while the blood picture, serum protein, blood volume, absorption tests and faecal fat estimations were all normal.

This man, therefore, showed a most striking response to parenteral nicotinic acid therapy at a time when he appeared unable to absorb the vitamin from his alimentary tract. He also demonstrated the failure of riboflavin to influence the general alimentary dysfunction, even although it might eliminate specific mouth lesions and improve gastric acid production and mobility.

(*h*) *Serial No.* 20—Sep. Ganda Singh 2/17 Dogra Regt., age 40. This man also was a prisoner in Singapore from February 1942. He remained fairly healthy until April 1945 when he developed recurrent attacks of malaria over 3 months for which he received inadequate treatment. During this illness he developed peripheral neuritis which was treated with 15 injections, and bleeding from the gums, which was still found on his first examination in India.

He appeared wasted and very anaemic ; his tongue showed a mild ariboflavinosis and the abdomen, in which both spleen and liver were much enlarged, was tympanitic. Purpuric patches were found on the limbs, the gums bled easily and sub-conjunctival haemorrhages were seen. A diagnosis of avitaminosis B_2 group with scurvy was made and investigation begun which showed :—R.B.C. 2.26×10^6 cells/cmm., Hb. 8.5 gm./100 c.c. M.C.V. 112 c.μ., M.C.H.C. 34%, serum protein 6.5 gm./100 c.c. (albumin 2.1 gm. %, globulin 4.4 gm. %, A/G ratio, 0.48/1); blood platelets 175,000/cmm. ; coagulation time 5½ minutes (normal for method 5-8 mins.) ; bleeding time 4 mins. (normal for method, less than 2 mins.) ; prothrombin, 80% normal ; serum inorganic phosphorus, 4.0 mgm. %. Following a test dose of 100 mgm. ascorbic acid only 33 mgm. were recovered from urine passed in the next 24 hours, whereas after 3 weeks' treatment on 100 mgm. ascorbic acid daily, followed by a rest period of 3 days, 95 mgm. of a test dose of 100 mgm. was excreted in 24 hours, suggesting that the deficiency of vitamin C in the body had been made good. This patient, the only one among the 2,000 prisoners studied to show signs of scurvy, made a good recovery and was discharged after 8 weeks' treatment with a normal blood picture and chemistry.

Discussion.—Study of thisgroup of patients, confirmss previous impressions as to the syndromes of riboflavin and nicotinic acid deficiency. In our opinion, the former deficiency is characterised by specific lesions of the lips, tongue and mouth and by changes in the gastric acid production and gastric mobility. The commonest

lip lesions are seen as cracks radiating from the corners of the mouth ; rarely a sodden membrane forms (perlèche). Greyish areas of necrotic epithelium may be seen on the inner aspect of the lips, while red, pinhead-sized papules often occur on the buccal aspect of the lower lip. Indurated red plaques occasionally form on the palate while aphthous ulcers are common. The tongue is characteristically swollen and indented by the teeth and assumes a fiery red colour ; when in this state soreness and burning are extreme. Atrophy of the fungiform and filiform papillae does not occur, in contradistinction to the picture seen in nicotinic acid deficiency ; indeed individual fungiform papillae appear swollen and mushroom-shaped, while their denuded summits allow the central capillary loops to appear as prominent red spots ("Red Mushrooms"). See Jones et al. (1944). Gastric acid production and stomach mobility, if defective, appear to be influenced favourably by riboflavin therapy, yet general nutrition and glucose and fat absorption tests show absolutely no improvement on prolonged treatment with this vitamin, even although the specific lesions on the lips and in the mouth can always be banished within a week, provided adequate dosage (about 9 mgm. daily) is employed.

Nicotinic acid deficiency, on the other hand, leads to atrophy of the epithelium of the tongue. The whole organ appears small and pointed ; its surface is denuded of papillae and presents a pale mauve glazed appearance. There is little complaint of soreness and only slight burning follows the ingestion of spiced or hot foods. Anorexia and tympanites are constant symptoms, as is also diarrhoea ; the total fat content of the stools is abnormally high, but in the cases studied, the proportion of split fats was not much increased.

Riboflavin and nicotinic acid deficiencies naturally often occur concurrently, when the tongue shows signs of atrophy of the central areas, usually of a strip to either side of the median fissure with redness along the margins and at the tip, where the characteristically swollen fungiform papillae are seen.

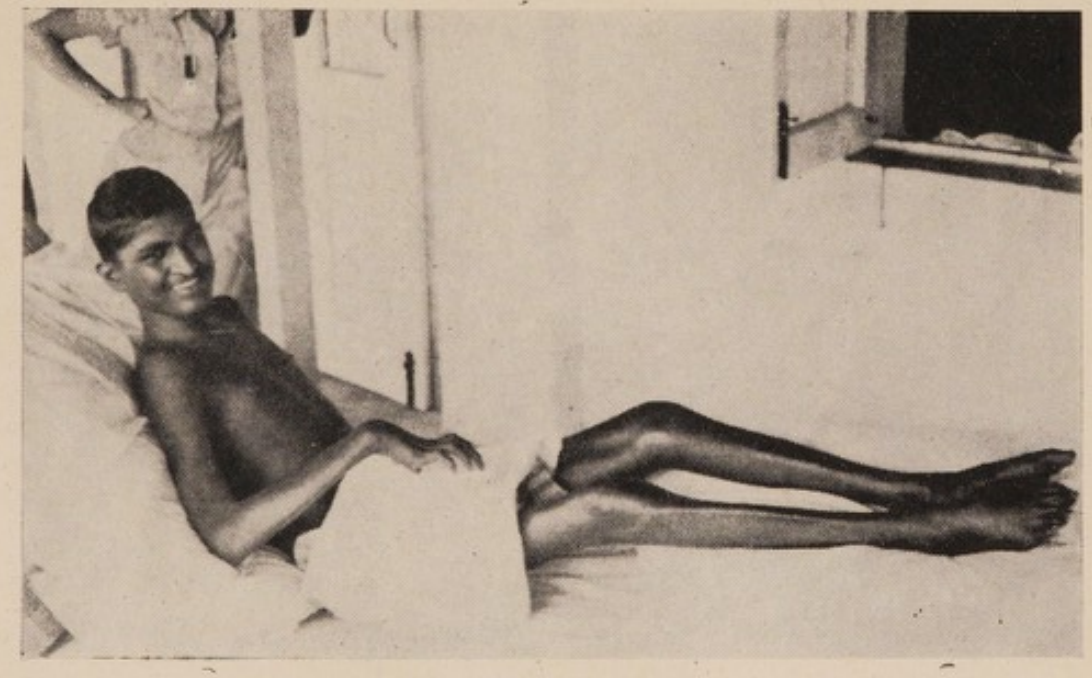
The P.O.W. cases here reported were the only outstanding examples of these syndromes from among the 2,000 prisoners examined, whereas similar cases occur with much greater frequency among combatant troops. For example, out of one convoy of 280 general medical cases drawn from occupational troops in the Burma-Malaya area in February 1946, 50 cases were of this type. The inference to be drawn is that, in the case of the prisoners, the intake of the vitamins of the B_2 group had fallen proportionately to their total food intake, whereas among combatant vegetarian troops, whose caloric intake remains high, there are very many men whose intake of animal protein, the richest source of vitamins of the B_2 in a typical Indian dietary, falls short of their minimum requirements. Analysis of typical diets, providing 3,600 calories in each case, shows that a vegetarian atta-eater should obtain 1 mgm. riboflavin and 32 mgm. nicotinic acid and a vegetarian rice-eater only 0.6 mgm. riboflavin and 14 mgm. nicotinic acid. Since daily requirements are generally agreed to be 2 mgm. riboflavin and 20 mgm. nicotinic acid, the supply obtainable from the diet is insufficient, and is likely to become markedly so when, as often occurred in the extremely difficult terrain over which the Burma Campaign was fought, full rations failed to reach the forward troops. Attacks of dysentery, causing periods of malabsorption from the gut, were also found to have precipitated the deficiency state in many cases.

4. *Marasmus with Neurological Syndromes*

4. (*i*) *Marasmus with Peripheral Neuritis* (*Beri-Beri*).

(*a*) *Serial No.* 23.—Sep. Ahmed Khan, R.I.A.S.C., age 32. Captured in Singapore February 1942, he remained fairly healthy until August 1944 when, following a severe beating, he was bed-ridden and had fever. One month later generalised neuritic pains began and were followed by oedema. He remained in bed until released.

On examination he was thin and showed slight oedema of the ankles. The blood count was normal but the serum albumin was only 2.82 gm./100 c.c. with an



Bilateral Foot and Wrist Drop.

Claw Contracture.

SEPOY VENKATASWAMY, P.O.W. FROM SINGAPORE. PERIPHERAL NEURITIS (BERI-BERI).

A/G ratio of 1·07/1. There were marked signs of polyneuritis; light touch and pain sensation were lost below the lower ⅓ of the forearms and below the lower ⅓ of the thighs. There was an impairment of vibration and joint sense over a similar distribution. Tendon reflexes were sluggish in the arms and lost in the legs and the plantar responses were flexor. There was slight flexor contracture of the knees.

He was given vitamin B_1 15 mgm. orally and 10 mgm. parenterally daily, and soon began to improve. Three weeks later full sensation had returned, but wasting of the peripheral muscle groups in the legs persisted and knee and ankle jerks were still absent. He had gained 16 lbs. in weight and his serum albumin had risen to 4·26 gm./100 c.c. and his A/G ratio to 2·2/1.

(*b*) *Serial No.* 1.—Sep. Venkataswamy, I.P.C., age 21. Captured in Singapore in February 1942, he remained fairly fit until May 1944 when, following an attack of malaria, tingling and numbness of the extremities began. He became bedridden in January 1945 when he had lost all sensation below Poupart's ligament and over the hands. He then lost his voice for several weeks and was unable to swallow fluids without some nasal regurgitation. He had had no illness suggestive of diphtheria.

When received, he was thin but had no oedema or obvious anaemia ; there was a bilateral foot and wrist drop (see photograph p. 16a), and muscular contracture had immobilized the knee-joints in extension. The optic discs showed marked temporal pallor with a crescentic zone of pigmentary disturbance round the temporal margin ; vision was poor and perimetry revealed gross concentric contraction of the visual fields. The arms were anaesthetic below the elbows and the intrinsic muscles of the hands showed much atrophy, giving a claw contracture (see photograph p. 16a). Tendon reflexes were absent. The muscles of the back were weak, but there was no sensory loss over the trunk. The legs showed just appreciable voluntary contraction of the thigh muscles, but none in the peripheral groups. All forms of sensory appreciation were severely impaired below the inguinal ligament and all tendon reflexes were lost. There was a resting tachycardia and the B.P. was 92/58. Investigation showed R.B.C. $3·90 \times 10^6$ cell/cmm., Hb. 12·6 gm./100 c.c. M.C.V. 99 c.μ., M.C.H.C. 33%, serum protein 6·53 gm./100 c.c. (albumin 4·21 gm.%, globulin 2·32 gm.%, A/G ratio, 1·8/1). Serum calcium was very low, 5·1 mgm./100 cc., yet tetanic spasm could not be induced.

After 11 weeks' treatment with vitamin B_1 the patient's nutrition was good, the blood pressure 104/64, the blood picture normal while the serum calcium level had risen to 11 mgm./100 cc. His vision had improved slightly, but there was no alteration in the visual fields. Sensation had returned completely in the upper limbs, but appreciation of light touch was still lost below the knees, while joint sensation was absent over the feet. Tendon reflexes were still unobtainable in both upper and lower limbs. Muscular power had improved in the upper limbs but the intrinsic muscles of the hands showed little recovery. Active flexion of the knees up to 45° had returned, with about 10° of flexion at the ankle joints, but he was still unable to make any movement of the toes. At this stage he was transferred to a hospital near his home, since little prospect of any further recovery was entertained.

(*c*) *Serial No.* 3.—Sep. Rikhi Ram, 2/17 Dogra Regt., age 25. Captured at the fall of Singapore, he remained fairly healthy until July 1944, when symptoms of polyneuritis began and caused his admission to hospital. In November 1944 he developed dysentery which, in the absence of treatment, lasted for 3 months. During this period he developed generalised oedema and a flexor contracture of the knees, which persisted until his return to India. The oedema was relieved by a few ir- injections of a vitamin B preparation and of a liver extract.

On arrival, he was thin with very wasted limbs, and was dull and inattentive. Fundus examination showed temporal pallor of the discs and retinal atrophy. There was impaired appreciation of all sensation below the middle of the forearms, but tendon reflexes in the arms were maintained. The knees were fully flexed and passive extension to 90° only was obtained. There was loss of light touch and vibration

sense below the inguinal ligament, but appreciation of pain was retained down to the knees. Tendon reflexes were not elicited and the plantar response was flexor.

After 14 weeks, treatment with vitamin B_1 and weight extension to the legs, recovery of the range of movement of these joints to within 10° of full extension had been achieved while full sensory function had returned, and all tendon reflexes were obtainable.

This man is notable in that he nearly doubled his body weight during treatment; it increased from 78½ lbs. to 144½ lbs. in exactly 3 months (see photograph p. 18a).

4. (*ii*) *Marasmus with "Captivity Cord Syndrome"*.—Patients showing signs of degeneration of the long tracts of the spinal cord fell into two groups; these showing a sensory ataxia due to loss of function of the posterior columns (common) and those who presented a spastic paraplegia due to pyramdal tract degeneration (rare).

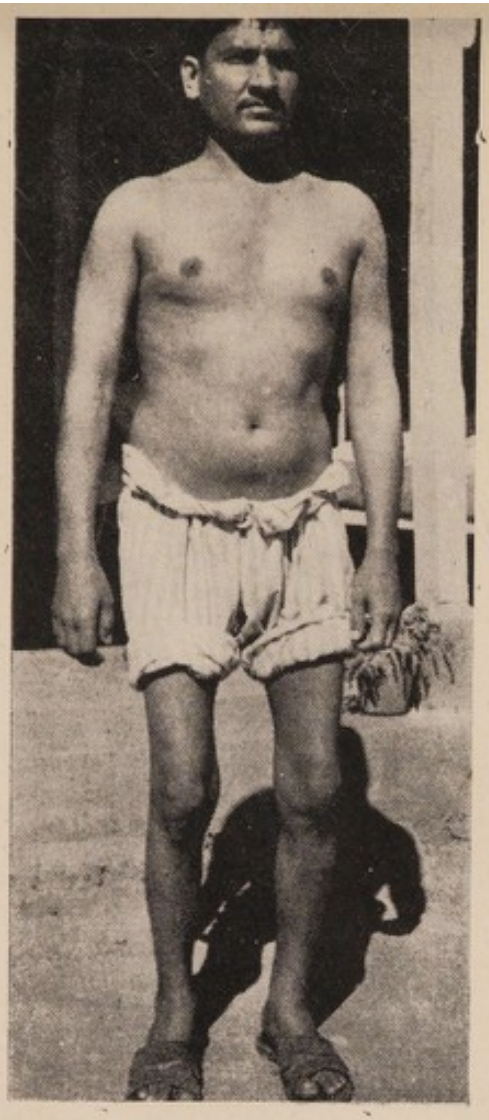
The following cases are cited as examples of the former syndrome :—

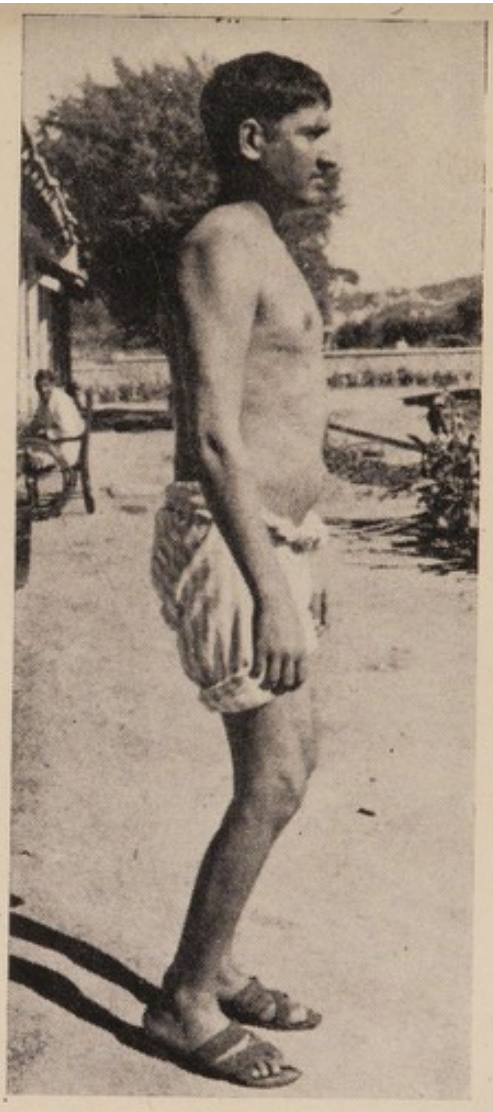
(*a*) *Serial No.* 19.—Sep. Hazir Khan, 2/10 Baluch. Regt., age 25. After his capture in Singapore this man remained on fatigue duties until June 1943 when an attack of dysentery began, which lasted for 2 months and during which symptoms of beri-beri developed. In December 1943 he had a severe attack of malaria during which he became oedematous, lost all sensation below the waist and began to suffer from hesitancy of micturition with dribbling incontinence. During the last few months of his imprisonment he received some vitamin therapy which relieved his oedema, but his neurological condition was unchanged at the time of his repatriation.

On examination his state of nutrition was fair, while the blood picture and chemistry showed no important abnormality except that the serum albumin was only 2·95 gm./100 c.c. and the A/G ratio 0·8/1. The tongue showed moderate ariboflavinosis. Vision was reduced to finger counting at 6 feet and there was marked temporal pallor of the discs. There was hypoaesthesia over the distribution of the 5th nerve, the corneae being markedl insensitive. Bilateral weakness of the 7th nerve of lower motor neurone type was shown by his inability to close his eyes completely or to whistle. The remaining cranial nerves showed no defect. The arms showed impaired position sense below the elbows with loss of light touch and pin-prick sensation up to the middle of the forearms. Sensory ataxia was present with loss of tendon reflexes. On the trunk there was impaired appreciation of light touch and pain below the 6th dorsal segment, with complete loss of all forms of sensation below the 10th dorsal segment. Nevertheless weak abdominal reflexes were obtained. There was weakness of all muscle groups of the legs with absent tendon reflexes and flexor plantar responses. The patient was unable to write, but could just feed himself; he was quite unable to walk, or even stand, unaided. The C.S.F. contained 1 lymphocyte per cmm. and 40 mgm. protein/100 cc. with a negative Wassermann Reaction. The stomach contained free acid.

The patient was treated with oral and parenteral vitamin B_1 and riboflavin for 2 weeks and by liver extract injections 4 cc. every second day throughout his stay in hospital. Re-educational exercises were also given. After 21 weeks treatment he was transferred on compassionate grounds to a hospital near his home. At this time he was able to walk well with the aid of sticks, but ataxia of all four limbs persisted. The optic discs had lost their former pallor; vision was 6/24 in each eye and the fields of vision had widened to about 50% of normal. Slight paraesthesia persisted over the face and cornea and only very slight facial weakness remained. In the arms, vibration sense and joint sense were still lost below the elbows while light touch and pin-prick were appreciated down to the lower ⅓ of the forearms. Over the trunk light touch and pin-prick were impaired below the 8th dorsal segment and vibration sense was lost below this level. Joint sense was completely lost below the knees. Tendon reflexes were still absent, with flexor plantar responses.

Although this man showed much improvement, impairment of function of the long sensory tracts of the cord was still obvious after 5 months treatment.





Flexor Contractures of Knees.

SEPOY RIKHI RAM, P.O.W. FROM SINGAPORE. PERIPHERAL NEURITIS (BERI-BERI).

(*b*) *Serial No.* 33.—Ambulance Sep. Binda, I.A.M.C., age 45. This man was held captive in Singapore where he developed beri-beri in 1943. Within 4 months he developed anorexia, a sore tongue and continuous diarrhoea; he became oedematous and remained bed-ridden until his repatriation.

On examination he was rational but depressed; the tongue was pale and slightly atrophic, the abdomen was very tympanitic and the stools loose and frothy. The only cranial nerve defect was central paraesthesia of the face. Vibration and position sense were lost below the elbows, but light touch and pain sensation only over the hands. There was a sensory ataxia of the arms with a defective finger-nose test, the tendon reflexes were lost. Vibration sense was lost below the 8th dorsal segment, and there was a remarkable loss to all forms of sensation down the centre of the abdomen, attributed to peripheral neuritis of the intercostal nerves. Abdominal reflexes were just obtained. Light touch was lost below the knees and pain and position sense lost over the feet. Knee and ankle jerks could not be obtained and the plantar reflexes were flexor. The C.S.F. showed no abnormality, the Wassermann Reaction was negative and small amounts of free acid were present in the gastric juice.

He was treated with nicotinic acid, with relief of the alimentary dysfunction and with 4 cc. liver extract every second day. After 3 months treatment he was able to walk well with slight ataxia, and only showed loss to light touch over the hands. The tendon reflexes, however, had not returned. Central paraesthesia persisted over the abdomen. Vibration sensation was lost below the 12th dorsal segment and joint sensation was still absent over the feet. Appreciation of pain had been regained, but light touch was still lost over the legs below the knees. Knee and ankle jerks were still absent and the Babinski reflex negative.

In this patient a syndrome suggestive of nicotinic acid deficiency was associated with degeneration of the dorsal columns of the cord together with peripheral neuritis. The neurological signs did not improve, although specific therapy rapidly relieved the alimentary symptoms. Signs suggesting neuritis of the intercostal nerves were seen in other cases besides this patient.

(*c*) *Serial No.* 27.—Sep. Asbik Ali, R.I.A.S.C., age 25. This man developed dysentery while a prisoner in Singapore in April 1942 and in August of that year he began to feel burning in the soles of his feet, while his vision became dim in November 1942. In July 1944 he again had dysentery followed by anasarca. Neuritic pains spread throughout the limbs and he became helpless. In February 1945 articulation and deglutition became difficult and, on account of this and the ataxia of his arms, he had to be fed until the end of April, when improvement followed injections of a preparation of rice polishings.

On examination he was intelligent, alert, but had a poor memory; he was thin but free from oedema while his blood picture and chemistry were normal. Normal amounts of free acid were found on gastric analysis. Neurological examination showed gross ataxia of all four limbs causing complete inability to walk or stand unaided. The fundi showed marked temporal pallor of the discs, with localised pigmentary disturbance outside the temporal margin and general retinal atrophy. Vision was reduced to counting fingers at 12 feet; perimetry could not be carried out. The area of supply of the trigeminal nerve was insensitive; there was facial weakness of the peripheral type and, although the palate was seen to rise in the midline, the voice was toneless and monotonous. The arms showed loss to vibration and position sense below the elbow and to light touch and pin-prick below the middle of the forearms. Tendon reflexes were inactive. The trunk showed loss of vibration sensation below the 6th dorsal segment but the abdominal reflexes were present. Light touch and pin-prick were felt over the legs, although vibration and position sense were lost, but there was complete anaesthesia over the distal half of the feet. The knee jerks were present, the ankle jerks absent and the plantar responses flexor. The C.S.F. was normal and the W.R. negative.

After 4½ months' stay in hospital, during which he received parenteral liver extract, 4c.c. every second day, and re-educational exercises, he was just able to walk

if supported by one hand, and the ataxia of the upper limb was much less evident. The optic fundi still showed temporal pallor of the disc, while the retinal arteries were diminished in calibre with some cuffing. No improvement in the visual acuity or in the fields of vision was evident. The corneae were still relatively insensitive, but all facial weakness had disappeared. Light touch and pain sensation were appreciated to the ends of the fingers, but joint sense and vibration sense were still defective over the hand. On the trunk vibration sense now appeared to be lost below the 10th dorsal segment. Over the legs light touch and pain appreciation had been completely regained, but position sense, even in the large joints, was very defective. Knee jerks were present and the ankle jerks were still absent while the plantar reflexes remained flexor.

This case thus showed gross sensory ataxia and optic atrophy with little peripheral neuritis, but did not appear deficient in either riboflavin or nicotinic acid. He made only slight recovery after 4 months observation.

The spastic type of syndrome is illustrated by the following cases :—

(*d*) *Serial No.* 50.—Sep. Noor Khan, H.K.S. Regt., R.A., age 23. This man was captured and held prisoner in Singapore where he remained at work until April 1944, when he became breathless on slight exertion. The limbs became progressively weaker, but abnormal sensations did not occur. Dimness of vision began about 8 months later, after which he became deaf and soon was too weak to leave his bed.

Examination on his repatriation at the end of October 1945 showed general wasting, but all systems, except the C.N.S., were healthy. He was deaf and obviously had a severe visual defect. The optic discs showed marked temporal pallor, visual acuity was reduced to counting fingers at 6 feet and there was marked concentric contraction of the visual fields. There appeared to be general retinal atrophy, with a localised pigmentary disturbance round the temporal margin. Bilateral nerve deafness was so severe that he could only just understand words shouted into either ear. There was no defect of the remaining cranial nerves. The arms were thin, with wasting of the intrinsic muscles of the hand ; sensation was normal save for the loss of appreciation of light touch over the fingers. There was no ataxia and all tendon reflexes were much exaggerated. The trunk showed apparent loss of vibration sense below the 6th dorsal segment ; the abdominal reflexes were present. There was wasting of all the muscle groups of the legs, most marked distally, but tone was increased. Light touch and position sense were lost over the feet, the knee jerks were much exaggerated and transient patellar clonus was elicited. The ankle jerks were similarly increased and sustained clonus was readily induced. Since the extensor hallucis longus was involved in the general foot drop, an extensor plantar response could not be obtained, but there was marked fanning of the toes on attempting to elicit the Babinski reflex. The blood picture and chemistry were normal. The C.S.F. had 2 lymphocytes per cmm. and 40 mgm. protein per 100 cc. with a negative W.R.

He was treated with parenteral liver extract, 4 cc. every second day, and received massage and re-educational exercises. However, 22 weeks later his condition showed sensory recovery, but no improvement in motor power. Visual acuity had improved to 6/36 and the fields were slightly wider, but there was no alteration in the temporal pallor of the optic nerve-head. Hearing was unchanged. Power in the arms had increased, but spasticity with increased reflexes remained. The only persisting sensory abnormality was loss of vibration sense below the middle of the left leg. Severe spastic paresis, however, remained with exaggerated tendon reflexes and patellar and ankle clonus. He was transferred at this stage to a hospital near his home.

4. (*iii*) *Marasmus with "Captivity Amblyopia"*.—Before discussing the above neurological syndromes, it is appropriate to describe the ophthalmoscopic appearances observed in those patients who had a visual defect during captivity, which condition, while still of uncertain aetiology, has been termed ' Captivity Amblyopia '.

This condition may be seen in the absence of gross vitamin deficiency states; it has been observed in association with ariboflavinosis, and with nicotinic acid deficiency, and may accompany any of the above-mentioned neurological syndromes. In its production, therefore, evidence of any specific vitamin deficiency cannot be adduced. The commonest finding has been bilateral temporal pallor of the disc with which is usually associated a crescentic zone of pigmentary disturbance consisting of a reticulated white or pinkish pattern superimposed on a bluish-black background of choroidal pigment. This fades off into more normal looking retina which is itself usually somewhat pale and atrophic. This zone is adjacent to the temporal margin of the disc. Those cases whose visual defect was most marked usually showed total pallor of the disc, while a white 'cuff' surrounded the vessels for a considerable distance from the disc margin. However, in only one case was gross attenuation of the vessels observed; his retina appeared markedly thinned while the optic disc appeared completely atrophic. It seemed that the diminished vascular supply was merely the result of retinal atrophy, since both fundi gave an identical picture and there was no general vascular disease. His initial visual acuity was less than 6/60 and did not improve during two months observation.

A third type of abnormality consisted of marked enlargement of the physiological cup, which was occasionally so gross that its edge reached the disc margin on the temporal side and even seemed to extend beyond it in rare cases. The vessels were seen to plunge abruptly over the edge of the enlarged and deepened pit, thence to be seen indistinctly running over its floor, completely out of focus.

It is not claimed that these changes are specific, but that they represent pathological degrees of normal variations; they were always associated with marked visual and field defects and were never observed among the rest of the 2,000 patients examined, who had no visual complaint.

Perimetry showed constantly a marked concentric contraction of the visual fields, but in contrast with the findings of Spillane and Scott (1945) and Dansey-Browning and Rich (1946), central scotomata could only be demonstrated in one case. It is not considered, however, that this was a true result, since the meagre capacity of the part-time ophthalmological unit posted to the hospital was completely overwhelmed by the large numbers of patients, approximately 100, requiring perimetry. The patients themselves, being mostly illiterate and only understanding one of several languages, were not generally favourable subjects on which to carry out so delicate a subjective test.

Discussion.—From the preceding description of the neurological sequelae of prolonged imprisonment on a defective diet, it will be seen that our experience runs parallel with that of Spillane and Scott (1945) who, however, in the group of German prisoners from camps in the Middle East, failed to find much supporting evidence of avitaminosis and were doubtful as to the aetiology of the conditions. The cases reported here form too small a group for any inference as to aetiology to be drawn, but it should be noted that, of the patients with 'Captivity Cord Syndrome', 3 out of 4 had had symptoms of beri-beri, one of ariboflavinosis and one of both ariboflavinosis and nicotinic and deficiency.

The neurological abnormalities included:

(*a*) Deficiency polyneuritis (dry beri-beri).

(*b*) Optic atrophy.

(*c*) Paraesthesia over the trigeminal nerve distribution.

(*d*) Facial nerve weakness of a peripheral type.

(*e*) Nerve deafness.

(*f*) Palatal and laryngeal paresis.

(*g*) Sensory ataxia, involving upper as well as lower limbs.

(*h*) Pyramidal tract degeneration causing spastic weakness of the four limbs.

5. *Macrocytic (Nutritional) Anaemia only.*—The occurrence of grave macrocytic anaemia in the absence of general malnutrition is not infrequently found among vegetarian troops who have been living for at least 6 months under Field Service conditions. Macrocytic anaemia was very common among the repatriated prisoners, but it was always accompanied by wasting and other signs of deficiency. The following two cases illustrate the condition of macrocytic anaemia unaccompanied by marasmus.

(*a*) *Serial No.* 46.—Sep. Driver Godhu Ram, R.I.A.S.C., age 29. This man, a vegetarian Jat, went on active service to Burma in 1941. In February 1945 he was admitted to hospital for treatment of 'anaemia' and, after a stay of 6 weeks, was sent to a Convalescent Depot for one month, and rejoined his unit. He returned to India with his unit, but was shortly drafted to Singapore where he arrived on September 15th, 1945. He was admitted to hospital there on December 18th, 1945, owing to anaemia and diarrhoea and was found to have a R.B.C. of $1{\cdot}46\times10^6$ cells/cmm., a Hb. of 8·5 gm./100 c.c. and an indefinite exudate in his stool.

On examination, on his arrival in India, he stated that his diet had consisted of atta, dal, rice and green vegetables with lime-juice and milk, enough to add to his tea and to make one cup daily. He was thin, very anaemic, but not jaundiced and was free from oedema. His tongue and stools were normal. His optic fundi showed numerous retinal haemorrhages with a sub-hyaloid haemorrhage in the right eye. The pulse beat was weak; a soft systolic murmur was heard at the apex and the B.P. was 100/40. The tip of the spleen was palpable. Investigation showed:—R.B.C. $0{\cdot}68\times10^6$ cells/cmm., Hb. 3·3 gm./100 c.c. M.C.V. 135 c.μ, M.C.H.C. 36%, serum protein 5·14 gm./100 c.c. plasma volume 2960 c.c., blood volume 3260 c.c.

He was given an immediate transfusion of 2 pints of citrated blood and injections of liver extract, 4 cc. daily. The results of transfusion form an interesting study. The calculation of total circulating haemoglobin and total circulating plasma protein showed that all f the infused haemoglobin remained in the circulation 24 hrs. later, but that all the infused plasma protein had disappeared (see Section VI). After the transfusion recovery was extremely rapid and at the end of 7 weeks, treatment, the findings were:—R.B.C. $47{\cdot}4\times10^6$ cells/cmm., Hb. 17·25 gm./100 cc., M.C.V., 101 c.μ., M.C.H.C. 35% and serum protein 6·34 gm./100 cc. His general condition was excellent and he had gained 35 lbs. in weight.

In this case a qualitative rather than a quantitive protein deficiency appeared to have been the causal factor.

(*b*) *Serial No.* 49.—Sep. Ram Singh, 7 Rajput Regt., age 29. This vegetarian Jat was stationed in Bengal in 1943, and returned home on one month's leave in May, 1944. On his return to his unit he took part in hard jungle training and soon became so anaemic that he had to be admitted to hospital where, during 2 months treatment, he received 4 blood transfusions of one bottle each. In September 1945 he was posted to Singapore, where in October he developed anorexia and ate little food other than biscuits. In February 1946, he reported sick on account of fever and was again found to have become very anaemic.

On examination on his arrival in India he was found to be well nourished, but extremely anaemic. His tongue was normal and there was no diarrhoea. His optic fundi showed scanty retinal haemorrhages. There was no oedema, the heart was not enlarged and the B.P. was 118/40. The spleen and liver were both enlarged, but there was neither ascites nor enlarged superficial veins. Laboratory investigation showed:—R.B.C. $0{\cdot}76\times10^6$ cells/cmm., Hb. 3·05 gm./100 c.c. M.C.V. 131 c.μ, M.C.H.C. 30·5%, W.B.C. 3,800/cmm., serum protein 5·33 gm./100 c.c. plasma volume 3380 cc., blood volume 3760 cc. A blood transfusion was begun at once, but had to be terminated when he had received 1½ pints on account of a severe febrile reaction. From a calculation of the total circulating haemoglobin and total circulating plasma protein before and after the transfusion it was shown that 24 hours later all the infused haemoglobin remained in the circulation, but that most of the infused plasma protein had left it. The explanation of this phenomenon is uncertain;

it would appear that the acquisition of more cells and haemoglobin allows the transfer from the circulation of a proportion of the plasma protein to tissues in need of it.

Discussion.—These cases call for comment on several important points. Of great importance to the economy of the Indian Army is the fact that vegetarian troops may develop a dangerous degree of macrocytic anaemia, while on Active Service Scale of rations, even although they are not living under battle conditions, and that the disease frequently recurs.

Lack of wasting shows that the caloric intake was sufficient, the anaemia being apparently due to the extremely low intake of animal protein. No other debilitating illness had occurred in either case, iron was not deficient and both men were free from intestinal parasites. It is significant to reflect that these men, who had come from a well-supplied area long after the cessation of hostilities, showed a graver degree of anaemia than any of the prisoners, returned after 3½ years captivity in Japanese prison camps!

The occurrence of severe anaemia without clinical evidence of hypoproteinaemia suggests that the protein deficiency in the diet is largely a qualitative one. Nevertheless, serum protein estimations do show a slight deficiency of total circulating plasma protein and some degree of tissue protein lack is suggested by the apparent rapid removal of transfused protein out of the circulation, in such cases.

6. *Dietary Treatment.*—The specific treatment used in the several types of case has been stated in the clinical accounts of each. Two types of diet were employed. A special low residue bland type of diet called the "Marasmus Diet" was used for those patients who had little appetite, sore tongues and impaired digestive function. This diet supplied approximately 3,800 calories (see Table 57 of the Appendix). When appetite and digestion improved a very full diet containing 158 gm. protein was given; this diet supplied 5,300 calories (see Table 58 of the Appendix). All patients received three major meals daily, with extra milk, tea, fruit, sweets, and biscuits interpolated. They also received "Multavite" tablets and an extract of rice-polishings. There seemed to be few periods during the day in which their mouths were empty.

SECTION III

HAEMATOLOGICAL INVESTIGATION.

1. *Findings.*—Complete haemograms were done on a representative series of cases shortly after they were admitted to hospital and again on the same patients before they were discharged. The patients selected included representatives of each of the different clinical groups described in Section II. In general it may be said that they are typical of the repatriated Indian prisoner of war who was ill enough to require hospital treatment. A summary of the results is given in table 3a, together with a summary of the results obtained from the control series. Details of each individual case can be found in tables 27, 28 and 29 of the Appendix for the P.O.W. patients and in table 36 for the controls.

As can be seen from the tables the majority of the patients studied were anaemic. The mean haemoglobin concentration on admission was 10·5 gm./100 c c. compared with 14·3 gm./100 c.c. on discharge and 15·2 gm./100 cc. for the control cases. The characteristics of the anaemia were remarkably constant. It was macrocytic and normochromic. The average M.C.V. on admission was 119 c.μ. and 94 cμ. on discharge, compared with 86·5 c.μ. for the control group. Also the average M.C.H. was considerably raised, 39 $\gamma\gamma$ on admission, and 31 $\gamma\gamma$ on discharge compared with the control figure of 29 $\gamma\gamma$. The mean difference between the figure observed on admission and that on discharge for the haemoglobin concentration, R. B. C. concentration, haematocrit, M.C.V. and M.C.H. was in each case statistically significant. The M.C.H.C. was, however, never reduced. On admission the mean M.C.H.C. was 32·8 % and 33·0 % on discharge, compared with 33·5 % in the control group. Thus there was no suggestion of the 'dimorphic' type of anaemia described by Trowell (1943). It is in fact remarkable that subjects whose diet was as meagre as that of the patients described should have no signs of iron deficiency. This may in part be due to the fact that iron was available from wasted tissues—the percentage loss in body weight was considerable (see Section V)—or because, in this type of anaemia, with a great reduction in blood volume (see Section V), the total circulating haemoglobin is very low, and hence the demand for iron is greatly reduced. In the majority of cases a blood film was made; this showed the usual picture seen in nutritional macrocytic anaemia—macrocytosis, anisocytosis, poikilocytosis and the usual number of primitive forms.

2. *Discussion.*—The characteristics of the anaemia in these patients are in marked contrast to those of the anaemia of the patients from Belsen described by Mollison (1946) and in those from Lamsdorf reported by Edge (1945). In Belsen the anaemia was normochromic and normocytic. In the Indian prisoners released from Japanese hands the anaemia was normochromic, but very macrocytic. Thus in both series of cases there was no evidence of iron deficiency, but in the Indian prisoners there was evidence of a deficiency of the 'factor' responsible for 'nutritional macrocytic anaemia' or 'tropical macrocytic anaemia,' not seen in the Europeans imprisoned in Belsen. In other respects the two series are comparable; both showed roughly the same degree of loss of body weight, both had the same degree of anaemia, both suffered from a comparable degree of protein deprivation as illustrated in the plasma protein concentration and the degree of oedema. It appears that, either the Indian is more susceptible than the European to nutritional macrocytic anaemia, or alternatively, that the inmates of Belsen received some factor in their diet not accessible to the Indian prisoners. Unfortunately the haematological observations of Mitchell and Black (1946) on British troops released from Japanese prison camps are too meagre to be of much assistance. They state, however, that "compared with their other deficiencies the patients were not markedly anaemic" and that "the average colour index was high." It would thus appear that such anaemia as existed was of the macrocytic type.

Table 3a

Summary of the Haematological Findings of Patients on Admission and on Discharge, together with the Findings in the Control Series.

	Prisoners of War.					Controls.	
	No. Patients	On Admission Mean ± S.D.	On Discharge Mean ± S.D.	Difference Mean ± S.D.	Significance Difference (P)	No. Patients.	Mean ± S.D.
Hb. Concentration (gm./100 cc.) ..	27	10.5±2.4	14.3±1.7	3.7±2.2	Less than 0.01	9	15.2±1.9
R. B. C. Concentration (10^6 cells/cmm.)	27	2.74±0.63	4.64±0.88	1.94±0.62	less than 0.01	9	5.28±0.48
Haematocrit (%) ..	27	31.9±6.0	43.3±1.5	11.4±5.5	less than 0.01	9	45.5±4.0
M. C. V. (c. μ). ..	27	119.0±14.8	94.3±8.1	24.7±16.1	less than 0.01	9	86.5±8.0
M. C. H. ($\gamma\gamma$) ..	27	39.2±6.7	31.1±2.9	8.1±6.6	less than 0.01	9	29.1±4.3
M. C. H. C. (%) ..	27	32.8±2.5	33.0±1.5	0.2±2.4	greater than 0.05	9	33.5±2.4

3. *Response to treatment.*—All patients with macrocytic anaemia were treated with injections of liver extract. The extract used was the Indian preparation "T.C.F." a sheep's liver extract prepared by the Teddington Chemical Factory Ltd., Bombay. It was usually given in doses of 4 c.c. every second or third day. The response was invariably good. Figure 1 shows the rate of recovery in two typical cases. Such follow-up haematological data were obtained in a great number of patients and, in those patients whose recovery was not complicated by blood or plasma transfusion, these data are set out in table 30 of the Appendix.

One feature of the recovery process which is of interest is that the haemoglobin concentration, and sometimes the R.B.C. concentration (though not so often, as the anaemia rapidly became less macrocytic,) frequently fell during the initial stages of treatment. This was at first puzzling and disturbing, but a re-assuring answer to the problem is given in the section on blood and plasma volume (Section V.)

4. SUMMARY.

(1) The great majority of Indian prisoners repatriated from Japanese prison camps were anaemic.

(2) The characteristics of the anaemia were high M.C.V., high M.C.H. and normal M.C.H.C. The anaemia was thus orthochromic and macrocytic. This is in contrast to the type of anaemia described in prison camps in Europe such as Belsen and Lamsdorf.

(3) The anaemia improved rapidly on treatment with 'T.C.F.' liver preparation.

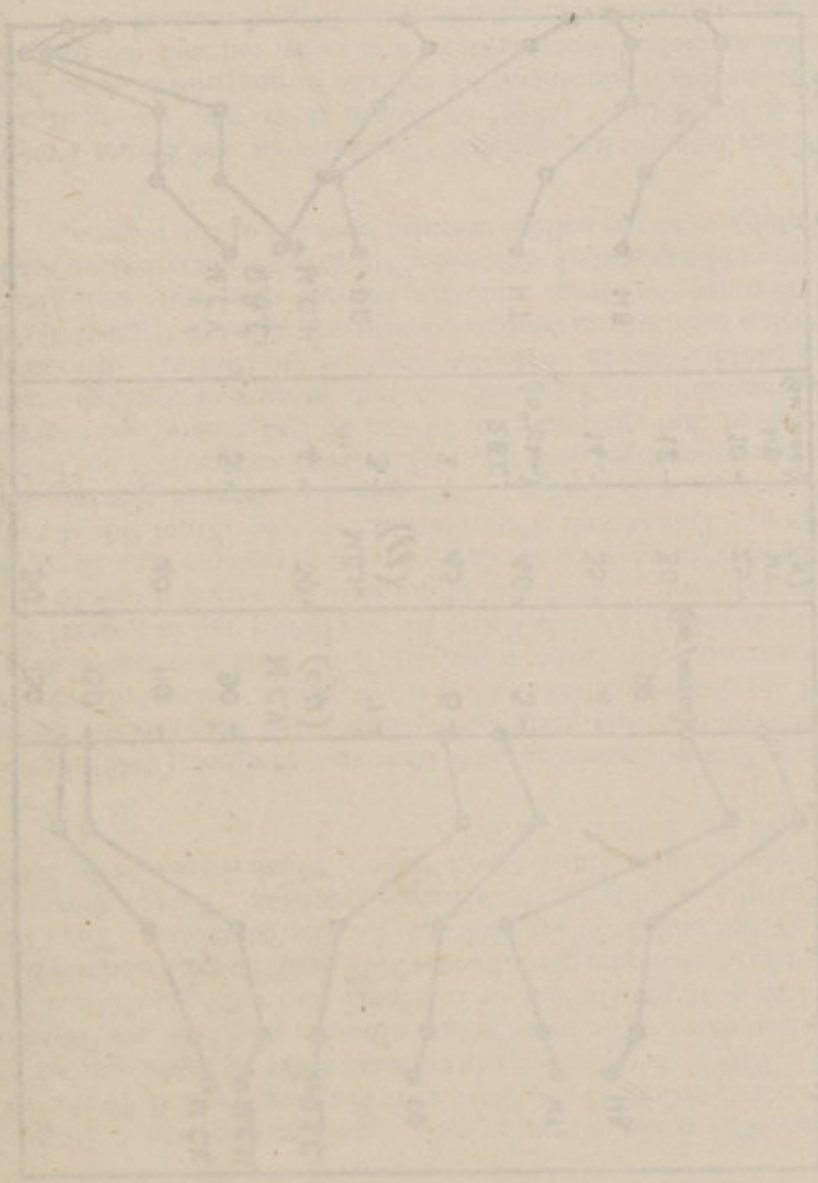


Table 3b

Summary of Biochemical Findings of Patients on Admission and on Discharge, together with the Findings in the Control Series.

	No. Patients.	Prisoners of War.		Controls.			
		On Admission Mean ±S.D.	On Discharge Mean ±S.D.	Difference Mean ±S.D.	Significance of Difference (P)	No. Patients.	Mean ± S.D.
Serum Protein (gm./100 cc.)	27	5.40±0.96	6.83±0.44	1.41±1.15	less than 0.01	9	6.89±0.35
Serum Albumin (gm./100 cc.)	27	2.63±0.92	4.11±0.50	1.48±0.94	less than 0.01	9	4.59±0.49
Serum Globulin (gm./100 cc.)	27	2.77±0.78	2.72±0.72	0.05±0.84	greater than 0.05	9	2.30±0.30
Ratio A/G. (x : 1)	27	1.1±0.6	1.6±0.4	0.6±0.7	less than 0.01	9	2.0±0.5
Serum Calcium (mgm/100 cc).	27	8.5±1.5	10.8±0.8	2.2±1.5	less than 0.01	9	11.4±0.5
Serum Inorganic Phosphorus (mgm./100 cc).	24	3.9±1.1	4.4±0.6	0.5±0.1	between 0.05 and 0.02	9	4.0±0.3
Serum Phosphatase (King-Armstrong Unit).	24	16±8	20±12	4±10	between 0.05 and 0.02	9	16±4

Table 4

Relation of Serum Protein Concentration to Oedema.

Patient No.	Serum Protein (gm./100 cc).	Serum albumin (gm./100 cc).	Serum Globulin (gm./100 cc).	Clinical Condition.
24	2.88	0.75	2.13	Gross generalised œdema and marked ascites.
2	4.05	1.01	3.04	do.
6	4.20	1.46	2.74	Generalised œdema and ascites.
15	4.78	1.20	3.58	do.
22	5.23	1.67	3.56	do.
26	5.10	1.98	3.12	do.
47	3.33	1.96	1.37	do.
5	4.18	2.00	2.18	do.
3	4.81	2.44	2.37	No œdema.
7	4.46	2.40	2.06	do.
18	5.34	2.10	3.24	do.
25	5.17	2.48	2.69	do.
31	5.30	2.75	2.55	do.
34	5.24	3.75	1.49	do.
35	5.30	4.07	1.23	do.
36	5.13	2.56	2.56	do.

hence a relative increase in the 'free' or ionised calcium in the plasma. It is now generally recognised that it is a reduction in the ionised calcium of the plasma which predisposes to tetany.

6. *Serum Inorganic Phosphorus.*—There was no consistent change in the serum inorganic phosphorus (Table 3b.). The mean on admission was 3.9 mgm./100 cc. and 4.4 mgm./100 cc. on discharge. This difference is of doubtful significance (P between 0.02 and 0.05) and the mean on admission (3.9 mgm./100 cc.) was certainly not significantly different from the mean of the control group (4.0 mgm./100 cc.).

7. *Serum Phosphatase.*—Table 3b also shows that there was no consistent change in the serum phosphatase. Again the difference between the mean on admission (16 units) and the mean on discharge (20 units) was of doubtful significance (P between 0.02 and 0.05) and again there was certainly no significance between the mean on admission and the mean of the control group.

The absence of significant change in the serum inorganic phosphorus or serum phosphatase is evidence in favour of the view that the low serum calcium is not related to a vitamin D deficiency.

8. *Other Biochemical Findings.*—In addition, the following investigations were done on some of the patients:—serum urea, plasma prothrombin, bleeding time, clotting time, plasma fibrinogen, vitamin C saturation test and plasma bisulphite binding powers. With the exception of one case, in which there was an impaired vitamin C saturation, all these tests invariably gave figures well within the normal range. It must be borne in mind, however, that the patients had all received treatment, including massive vitamin therapy, immediately following their release and throughout the whole of the time they were in transit to India.

Serum total fat, serum cholesterol, faecal fat and fasting blood sugar estimations will be discussed in the section on tests of absorption from the gastro-intestinal tract (Section VIII); blood volume and plasma volume data will be given in Section V and fractional test meal findings in Section VII.

9. *Response to Treatment.*—The data obtained from serial follow-up biochemical estimations on such patients in whom recovery was not complicated by transfusion are presented in Table 35 of the Appendix. It can be seen that, during the patients' stay in hospital, all the biochemical abnormalities were corrected.

10. SUMMARY.

(1) Indian prisoners repatriated from Japanese prison camps usually had a low serum protein concentration. This reduction in serum protein was almost entirely in the albumin fraction, and hence the A/G ratio was also low.

(2) The degree of oedema was more closely related to the serum albumin concentration than to the serum total protein concentration.

(3) The serum calcium was decreased, in some cases extremely so, but there was no significant change in the serum inorganic phosphorus or serum phosphatase. There was no evidence of latent tetany.

(4) Other tests for vitamin deficiencies were negative.

(5) All the biochemical abnormalities improved rapidly with treatment in hospital.

SECTION V

PLASMA VOLUME AND BLOOD VOLUME INVESTIGATION.

1. *General.*—In a selected group of patients further information was obtained concerning the plasma and total circulating volume. It has generally been assumed that in malnutrition the plasma volume and total circulating volume decreases, but the extent of this fall, as far as is known, has never before been measured, nor has the rate or pattern of recovery been observed.

The plasma volume was determined by the Evan's blue dye technique and from a knowledge of the plasma volume and the haematocrit, the total circulating volume was calculated. The concentration of haemoglobin, red blood cells and serum protein (albumin and globulin) was measured by the usual techniques and, from these figures and the plasma or blood volume, the total amount of each constituent in the circulation was calculated. All the figures were, for comparison purposes, then referred to unit body weight, unit surface area, and unit body height. Measurements were made on the patients soon after they were admitted to hospital and thereafter at 3-4 weekly intervals. The data obtained on admission and shortly before discharge are summarised in tables 5-10. Full details of each individual patient can be found in tables 42-47 of the Appendix. The results obtained from the control series are also given in tables 5-10. Details of individual control cases can be found in the Appendix Tables 36-41. Also the figures obtained from another small series of patients, studied shortly after admission but upon whom for administrative or technical reasons it was impossible to obtain follow-up data, are also given in Tables 5-10. The details of individual cases in this series can be found in the Appendix Tables 48-53.

Tables 5-10 only give the findings on admission and shortly before discharge. They show that, after a period of treatment in hospital, the patients approached normal in every respect, but they do not show the rate at which recovery took place, nor the relative rate of recovery of the different blood constituents. For instance the plasma volume returned to normal very quickly (0-4 weeks); this was followed by the blood volume and the total circulating plasma protein (2-12 weeks) and it was not until much later (8-16 weeks) that such factors as the body weight, the total circulating haemoglobin, the haemoglobin concentration, the plasma protein concentration and the albumin/globulin ratio returned to normal. Details of this recovery process will be mentioned from time to time in the text and illustrated by particular cases. Since the phases of recovery differ slightly from patient to patient in their time relations, average figures would flatten out the points it is desired to emphasise. For the full details of each particular patient studied, the reader is referred to the Appendix Tables 42-47.

2. *Haematology* (Table 5).—The haematological findings in this series differed in no essential respects from those of the larger series reported in Section III (Table

Table 5

Blood Volume Investigation. Haematological Findings.

	Number of Patients	Mean Hb. Concentration (Gm/100 cc) (±S.D.)	Mean R.B.C. Concentration (10^6 cells/cmm) (±S.D.)	Mean Haematocrit (%) (±S.D.)	Mean M.C.V. ($c\mu$) (±S.D.)	Mean M.C.H. ($\gamma\gamma$) (±S.D.)	Mean M.C.H.C. (%) (±S.D.)
A. Control Series.	9	15.2±1.9	5.28±0.48	45.5±4.0	86.5 ±8.0	29.1 ±4.3	33.5 ±2.4
B. Patients on Admission	12	9.8±2.0	2.52±0.39	30.4±4.8	121.4 ±16.2	39.2 ±8.0	32.0 ±2.7
C. Patients in Group B on discharge.	12	13.6±0.7	4.41±0.57	41.6±3.0	95.4 ±8.7	31.3 ±3.5	32.8 ±1.9
D. Cases on Admission not followed up further.	5	7.3±3.1	.....	23.5 ±10.3	121.4 ±12.8	38.2 ±4.5	31.5 ±1.8

3a). There was an orthochromic macrocytic anaemia which responded well to treatment. The M.C.V. and M.C.H. rapidly fell and the M.C.H.C. remained within normal limits throughout. It was noticed, however, that the haemoglobin concentration and the haematocrit and, much more rarely, the R.B.C. concentration continued to fall for a period of up to 4 weeks, and then suddenly rose rapidly to normal values.

3. *Serum Proteins* (Table 6).—The serum protein findings were also similar to the larger series reported in Section IV (Table 3b). The total serum protein concentration was low on admission, the reduction being confined to the albumin fraction. During treatment the serum albumin concentration progressively rose parallel to the total protein concentration and the ablumin/globulin ratio consequently increased. But before increasing, the total serum protein concentration, like the haemoglobin concentration, frequently decreased for a short time. It then returned to normal, rapidly at first, but later more slowly. We believe that it is this decrease in plasma protein, which may occur during the first few weeks of treatment, that is responsible for the cases of 'delayed oedema' which are sometimes observed in patients suffering from protein deprivation (see section II).

4. *Body Weight* (Table 6).—It was obvious that the body weight of the patients studied was much below normal. The average weight on admission was 45.8 kgm. This had risen, after an average stay in hospital of three months, to 61.7 kgm., an increase of almost 16 kgm. (over 2½ stone). The average weight on discharge of 61.7 kgm. is greater than the average weight of 54.8 kgm. of the control group, even when allowance is made for the difference in average height. The average reduction in weight was to 75% of the original weight. This figure is, however, misleading. Many of the patients were grossly oedematous on admission and, during the first four weeks in hospital as the oedema disappeared, the body weight fell so that the reduction in body tissue other than water was much more than 25%. When the body weight eventually began to rise, the increase was exceedingly rapid, frequently being of the order of 7 lbs. per week. This initial fall in body weight was also noticed in the British personnel (Mitchell and Black (1946)).

5. *Body Height* (Table 6).—The average body height of the patients was 169 cm. This is higher than that of the control group (165 cm.). The patients studied had obviously been, therefore, men of fine physique before they were imprisoned.

6. *Surface Area* (Table 6).—This was calculated from the body weight and height by Du Bois formula. The area, as so calculated, was low on admission and during treatment, it at first fell slightly and then returned slowly to normal.

7. *Plasma Volume.* (Table 7).—The mean plasma volume of the patients on admission was 2446 cc. which is slightly lower than that of the control series, 2600 cc., but the difference is of doubtful significance. The mean figure is also less than that

Table 6

Blood Volume Investigation, Plasma Protein and Physical Measurement Finding.

Group.	Number of Patients.	Mean Serum Protein (gm/100 cc.) (±SD)	Mean Serum Albumin (gm/100 cc.) (±S.D)	Mean Serum Globulin (gm/100 cc.) (±S.D)	Mean Ratio A/G (X : I) (±SD)	Mean Body Weight (kgm.) (±SD)	Mean Height (cm.) (±SD)	Mean Surface Area (sq. m.) (±SD).
A. Control Series	9	6.89±0.35	4.59±0.49	2.30±0.30	2.0±0.5	54.8±5.2	165±6	1.59±0.10
B. Patients on Admission.	12	5.24±0.74	2.63±0.77	2.61±0.72	1.1±0.5	45.8±4.1	169±7	1.50±0.10
C. Patients in Group B on Discharge.	12	6.69±0.42	4.25±0.49	2.44±0.42	1.8±0.4	61.7±5.7	169±7	1.71±0.12
D. Cases on Admission not followed up further.	5	4.82±0.65	3.01±0.45	1.81±0.35	1.7±0.3	44.2±5.8	166±3	1.45±0.08

reported by Mollison (1946) for the Belsen cases but the scatter, 1880 cc. to 2940 cc., covers much the same range. The cases on admission could, from the plasma volume viewpoint, be readily divided into two groups :—

(1) Those with a very low plasma volume, an extreme hypoproteinaemia and only a moderate anaemia.

(2) Those with a plasma volume above normal, with only a moderate hypproteinaemia and a marked anaemia.

If the plasma volume was originally decreased, it returned to normal quickly (0-4 weeks) and then rose to well above the normal figure (2-12 weeks), and it was only later (8-16 weeks), that it fell again to normal levels. In all the cases studied the plasma volume was the first figure to return to normal.

The plasma volume referred to the body weight in kilograms (PV/Kg.) was above normal when the patients were admitted. This means that the relative decrease in plasma volume was not as great as that of the body weight. During recovery (PV/Kg.) invariably rose to figures far in excess of normal and then gradually fell again to normal values. This is because, during the early weeks of recovery, the plasma volume increased much more rapidly than the body weight; the plasma volume reached normal values after 0-4 weeks, but the body weight not until 8-16 weeks.

The plasma volume referred to unit surface area (PV/Sq.M.) was much more constant. It was within normal limits when the patients were admitted and remained so when they were discharged. During the initial recovery phase (0-4 weeks) it rose slightly on occasion, but the percentage rise was small compared with that of PV/Kg.

The plasma volume referred to unit body height (PV/cm.) was low on admission, 14.4 cc./cm. compared with 17.6 cc./cm. for the same patients on discharge and 15.7 cc./cm. for the control series. Like the absolute plasma volume, PV/cm. first rapidly increased to above normal and then decreased to normal values.

8. *Blood Volume* (Table 7).—The mean total circulating volume of 3514 cc. observed on admission was considerably lower either than that of the control series, 4779 cc., or that of the same patients on discharge, 5090 cc. This mean figure is of the same order as that reported by Mollison (1946) for the Belsen cases. The low total circulating volume gradually returned to normal (2-12 weeks). This return to normal was not so rapid as that of the plasma volume (0-4 weeks), but it was much

Table 7

Blood Volume Investigation. Plasma Volume and Blood Volume Findings.

Group	Number of Patients.	Mean Plasma Volume (cc.) (±SD)	Mean Plasma Volume per kgm. (cc/kgm.) (±SD)	Mean Plasma Volume per sq. m. (cc/sq.m.) (±SD)	Mean Plasma Volume per cm. (cc/cm.) (±SD)	Mean Blood Volume (cc) (±SD)	Mean Blood Volume per kgm. (cc/kgm.) (±SD)	Mean Blood Volume per sq. m. (cc/sqm.) (±SD)	Mean Blood Volume per cm. (cc/cm. (±SD)
A. Control Series.	9	2600± 229	47.5± 2.9	1634± 95	15.7± 1.2	4779± 370	86.5± 4.8	3004± 131	29.0±1.7
B. Patients on Admission.	12	2446± 328	53.6± 6.2	1625± 182	14.4± 1.7	3514± 413	76.9± 7.4	2334± 212	20.7±2.0
C. Patients in Group B on Discharge.	12	2977± 458	48.1± 5.0	1737± 94	17.6± 2.3	5090± 669	82.5± 7.0	2973± 288	30.0±3.4
D. Cases on Admission not followed-up further.	5	2430± 453	56.1± 3.1	1670± 244	14.6± 2.5	3158± 232	73.9± 10.9	2186± 168	19.0±1.0

quicker than the return to normal of many of the other factors measured. Unlike the plasma volume, there was not a period when the figures for the total circulating volume were greatly in excess of normal.

The blood volume even referred to unit body weight (BV/Kg.) was, on the average, lower than normal (76.9 cc./kgm. on admission, compared with 82.5 cc./kgm. on discharge and 86.5 cc./kgm. for the control series). Mollison (1946) quoting normal values of Gibson and Evans (1937) states that "blood volume in severely undernourished subjects is not reduced in proportion to body weight." In 10 out of the 12 of our cases so studied, BV/Kg. on admission was less than that on discharge and, in 15 out of the total of 17 patients, BV/Kg. on admission was lower than the mean of the control group, all the estimations being done by exactly the same technique. Application of Fisher's 't' test gives : P is less than 0.01 for the difference between the means of the patients on admission (group B) and those of the control group (group A), and P is less than 0.02 for the difference between the means of the patients of group D and the control group. It therefore seems clear that the total circulating volume is decreased to a slightly greater extent than the other body tissues. BV/Kg. returned to normal after first going through a period when it was above normal. This was due to the fact that, on recovery, the total circulating volume increased more rapidly than the body weight.

The blood volume referred to unit surface area (BV/Sq.M.) was reduced relatively much more than BV/Kg. During treatment BV/Sq.M. rose steadily to normal values.

When referred to unit body height, the blood volume (BV/cm.) was lower still. A normal Indian (control series) had 29.0 cc. blood/cm. height, but the repatriated P.O.W. on admission had only 20.7 cc./cm., whereas on discharge he had 30.0 cc./cm. BV/cm. improved rapidly on treatment.

9. *Total Circulating Haemoglobin* (Table 8).—It has previously been seen that there is frequently a period when the haemoglobin concentration falls before it finally commences to rise. If the haemoglobin concentration is multiplied by the blood volume, the total amount of haemoglobin in the circulation can be calculated. It was found that the total circulating haemoglobin rose steadily throughout the whole of the recovery period even although, in the initial stages, the haemoglobin concentration may have fallen, for it is at this time, when the haemoglobin concentration is falling, that the increase in blood volume is greatest. These points are well illustrated in Figure 2 which gives the haemoglobin concentration, total circulating haemoglobin and total circulating volume changes in two typical cases. This is

Table 8

Blood Volume Investigation. Total Circulating Haemoglobin Findings.

Group.	Number of Patients.	Mean Haemoglobin Concentration (gm/100 cc) (±SD)	Mean Total Circulating Hb. (gm.) (±SD)	Mean Total Circulating Hb. per kgm. (gm/kgm). (±SD)	Mean Total Circulating Hb. per sq.m. (gm/sq. m.) (±SD)	Mean Total Circulating Hb. per cm. (gm/cm). (±SD)
A. Control Series	9	15.2±1.9	730±117	13.4±2.5	460±71	4.4±0.7
B. Patients on Admission.	12	9.8±2.0	345±79	7.5±1.7	229±50	2.0±0.4
C. Patients in Group B on Discharge.	12	13.6±0.7	692±85	11.2±1.1	405±39	4.1±0.4
D. Cases on Admission not followed up further.	5	7.3±3.1	225±87	5.5±2.6	159±67	1.3±0.5

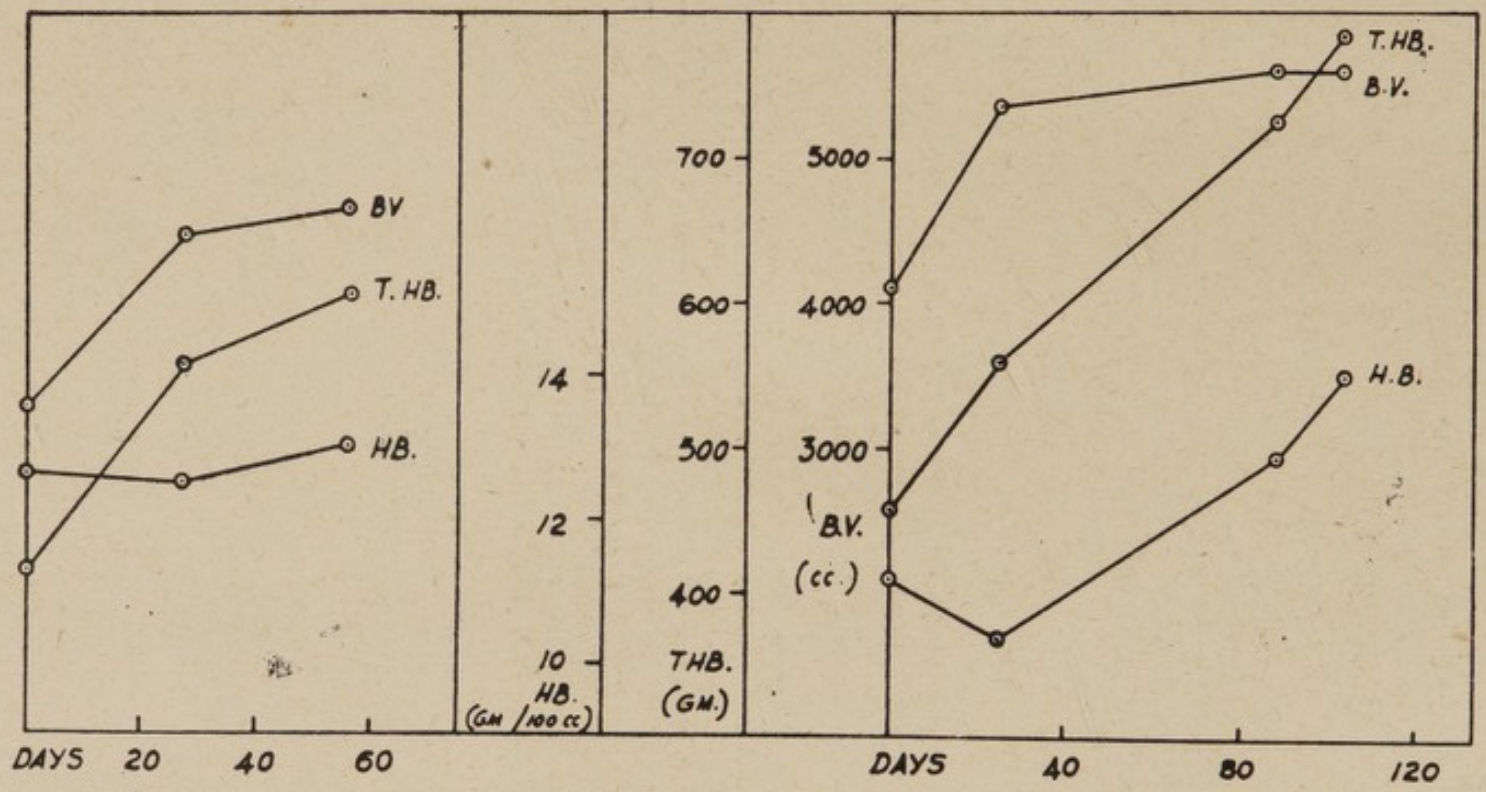


FIGURE 2 - CHANGES IN HAEMOGLOBIN CONC. (HB), TOTAL CIRCULATING HAEMOGLOBIN (T. HB), AND BLOOD VOLUME IN TWO TYPICAL CASES.

important from the clinical viewpoint, for it means that a patient may be producing haemoglobin into the circulation at a surprisingly high rate—in the two cases illustrated haemoglobin was being formed at the rate of 40.5 and 28 gm./week respectively—and yet the haemoglobin concentration is actually falling. It follows therefore that, if the haemoglobin concentration of an under-nourished patient falls during the first few weeks of treatment, it does not necessarily mean that the patient's condition is deteriorating. He may still be elaborating haemoglobin at a very satisfactory rate.

The total circulating haemoglobin on admission was exceedingly low, 345 gm. compared with 730 gm. for the control series, but by the time the patients were fit for discharge it had risen to 692 gm. The total circulating haemoglobin per kilogram body weight was also greatly decreased on admission, signifying that the loss of haemoglobin was relatively much greater than the loss of other body tissues. On treatment this rose rapidly at first and then more slowly.

The total circulating haemoglobin per square metre surface area was reduced relatively much more. On admission the figure was 229 gm./sq. m., compared with 405 gm./sq.m. for the same patients on discharge and 460 gm./sq.m. for the control series. This figure gives some idea of the disability under which the patients were suffering during imprisonment, because the haemoglobin requirement is closely parallel to the surface area. On treatment the total circulating haemoglobin per square metre returned to normal steadily.

When referred to unit body height the total circulating haemoglobin was greatly reduced. On admission the repatriated prisoners only had 2.0 gm. haemoglobin per centimetre height, whereas the figure for the control group was 4.4 gm./cm. Presumably this latter figure is a fair estimate of the total circulating haemoglobin per centimetre body height of the patients before they were imprisoned. On treatment the total circulating haemoglobin per centimetre body height gradually increased.

10. *Total Circulating R. B. C.* (Table 9).—It has previously been seen that the R. B. C concentration usually rises slowly. There was rarely the fall observed in the haemoglobin concentration, since the macrocytosis was at the same time becoming progressively less. Multiplying the R.B.C. concentration by the blood volume gives the total number of red cells in the circulation. On admission the average for the repatriated prisoners was 8.8×10^{12} cells, whereas the figure for the control series was 25.3×10^{12} cells and the figure for the patients on discharge was 22.2×10^{12} cells. The total number of circulating red cells returned steadily to normal with treatment.

Table 9

Blood Volume Investigation Total Circulating Red Blood Cell Findings.

Group.	Number of Patients.	Mean R.B.C. Concentration (10^6 cells/(cmm) (±SD)	Mean Total Circulating R.B.C. (Cells x10^{12}) (±SD)	Mean Total Circulating R.B.C. per kgm. (cells x10^{10}/kgm) (±SD)	Mean Total Circulating R.B.C. per sq. m. (cells x10^{12}/sq. m.) (±SD)	Mean Total Circulating R.B.C. per cm. (cells x10^{10}/cm) (±SD)
A. Control Series	9	5.28±0.48	25.3±3.7	46±4	15.9±1.7	15.3±2.0
B. Patients on Admission.	12	2.52±0.39	8.8±1.4	19±3	5.9±0.9	5.2±0.8
C. Patients in Group B on Discharge.	12	4.41±0.57	22.2±2.6	36±5	13.1±1.7	13.1±1.6
D. Cases on Admission not followed up further.	5	1.93±0.82	6.0±2.5	15±8	4.3±2.0	3.7±1.5

The total number of circulating red cells referred both to unit body weight and unit surface area was also greatly reduced, as also was the total number of circulating red cells referred to unit body height. The figure of 5.9×10^{10} cells/cm. on admission can be compared with 15.2×10^{10} cells/cm. in the control series. All these figures approached normal after treatment in hospital.

11. *Total Circulating Plasma Protein* (Table 10).—As previously seen, the serum protein concentration, after a period when there was little improvement, rose, at first rapidly and then more slowly, until normal values were reached. The rise was almost entirely in the albumin fraction so that there was a progressive improvement in the albumin/globulin ratio. By multiplying the serum protein concentration by the plasma volume, an estimate is obtained of the total circulating plasma* protein. It is seen that this was 128 gm. when the patients were admitted to hospital, compared with 199 gm. on discharge and 180 gm. for the control group. It is also seen that, even although many of the patients were admitted in a state of acute protein deprivation, the decrease in total circulating plasma protein was not as great relatively as that of the total circulating haemoglobin because neither was the reduction in plasma volume as great as the reduction in blood volume, nor was the reduction in serum protein concentration as great as that of the haemoglobin concentration. The improvement in the total circulating plasma protein was both dramatic and rapid. Normal figures were reached at the end of 2-12 weeks, much earlier than normal figures for the total circulating haemoglobin.

Table 10

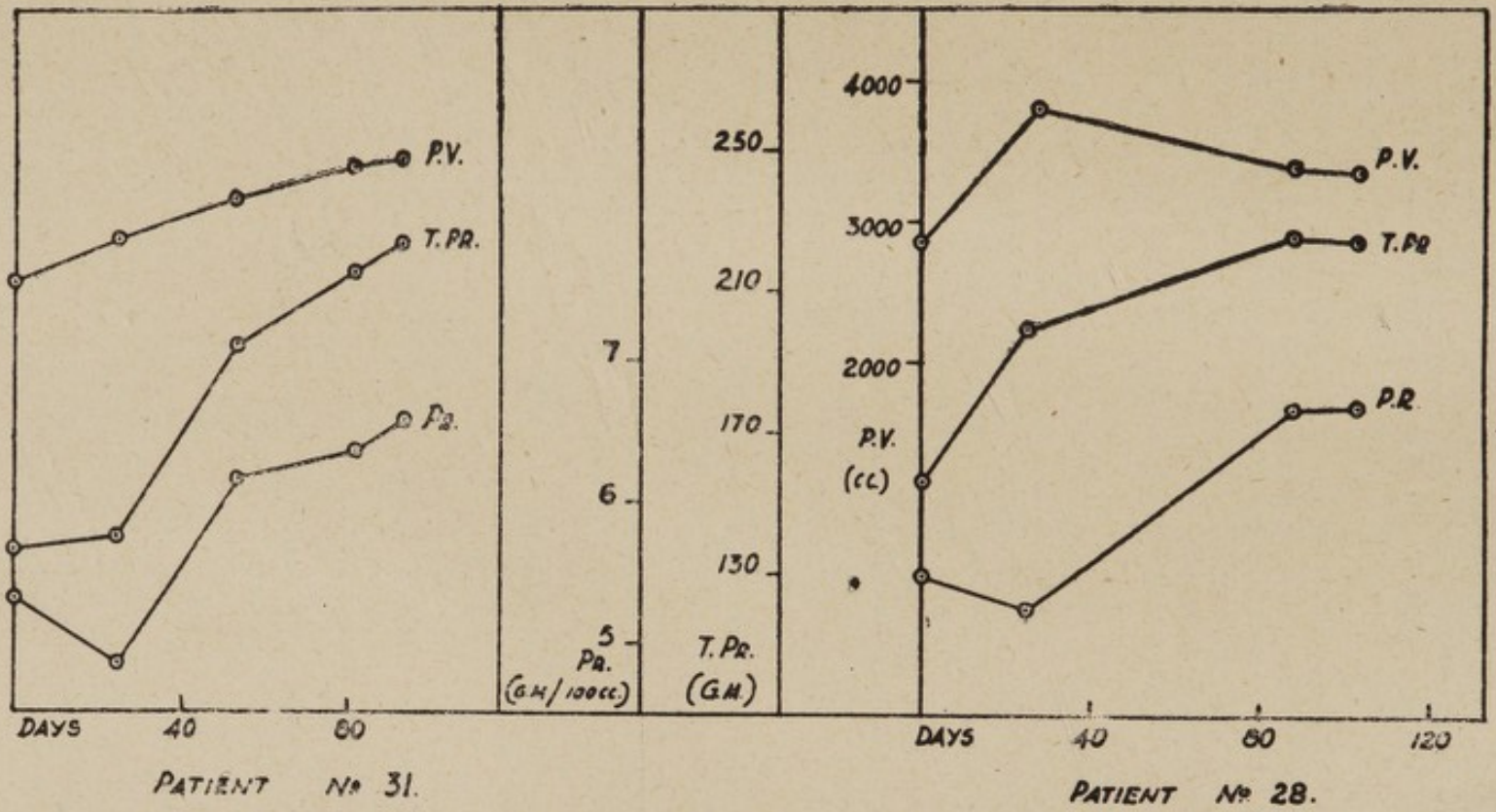
Blood Volume Investigation. Total Circulating Plasma Protein Findings.

Group.	Number of Patients.	Mean Plasma Protein Concentration. (Albumin + Globulin) (gm/100 cc) (±SD)	Mean Total Circulating Plasma Protein. (Albumin + Globulin) (gm) (±SD)	Mean Total Circulating Plasma Protein per kgm. (Albumin + Globulin) (gm/kgm.) (±SD)	Mean Total Circulating Plasma Protein per sq. m. (Albumin + Globulin) (gm/sq. m.) (±SD)	Mean Total Circulating Plasma Protein per cm. (Albumin + Globulin) (gm/cm) (±SD)
A. Control Series	9	6.89±0.35 (4.59±2.30)	180±21 (120±60)	3.3±0.3 (2.2±1.1)	113±10 (75±38)	1.09±0.11 (0.73±0.36)
B. Patients on Admission.	12	5.24±0.74 (2.63±2.61)	128±33 (64±64)	2.8±0.7 (1.4±1.4)	85±20 (43±42)	0.75±0.17 (0.38±0.37)
C. Patients in Group B on Discharge.	12	6.69±0.42 (4.25±2.44)	199±31 (125±74)	3.2±0.4 (2.0±1.2)	117±15 (74±43)	1.17±0.17 (0.74±0.43
D. Cases on Admission not followed up further.	5	4.82±0.65 (3.01±1.81)	120±34	2.7±0.7	82±22	0.72±0.20

The total circulating plasma protein increased steadily in all cases even although, in many, the serum protein concentration actually fell at first. The initial rise in plasma volume was so rapid that there was always an increase in total circulating plasma protein. Figure 3 illustrates these particular points in two typical cases. As in the case of the haemoglobin, it does not necessarily follow that, because the serum protein concentration falls, the patient is not progressing. In patient 28 (Figure 3) serum protein was being produced at the rate of 12 gm./week and yet the serum protein concentration actually fell.

The total circulating plasma protein referred to unit body weight (Pr/Kg.) was but slightly reduced on admission, indicating that the plasma protein was not reduced much more than other tissues. Because of the rapid rise in total circulating plasma protein and the slower rise in body weight, Pr/Kg. quickly rose to figures well

*Strictly speaking, total circulating serum protein.



PATIENT № 31.

PATIENT № 28.

FIGURE 3.- CHANGES IN PLASMA PROTEIN CONC. (PR.), TOTAL CIRCULATING PLASMA PROTEIN (T PR) AND PLASMA VOLUME (P.V.) IN TWO TYPICAL CASES.

above normal before it fell again to normal levels Pr/sq.m. and Pr/cm. were also both reduced, and both rose very rapidly to normal values.

It is seen that the reduction in the total circulating plasma protein was entirely in the albumin fraction. The total circulating albumin was only 50% of normal, whereas there was no change in the total circulating globulin. On recovery, at first both fractions increased, but the albumin increased more rapidly than the globulin. Then, as time progressed, the increase in total circulating albumin became more rapid and that of total circulating globulin less rapid until finally, in many cases, the total circulating globulin, which by now had risen to figures above normal, commenced to decrease towards normal values again.

When referred to unit body weight, surface area or body height, the figures for the total circulating albumin and globulin do not reveal many points of especial interest. It will be noticed, however, that, since the total circulating globulin was normal on admission when the body weight was reduced, the total circulating globulin, when referred to unit body weight, was above normal (1.4 gm./kgm. as opposed to 1.1 gm./kgm.). As the body weight increased, the total circulating globulin per kilogram body weight fell.

12. *Stages of Recovery*.—From the foregoing it is possible to reconstruct the general picture of the pattern of recovery in a protein deficient patient. For the sake of ease of description this recovery process has been divided into three stages. These are of course arbitrary and show very considerable variations in their time relations. The majority of the patients studied, however, demonstrate these stages clearly and the remainder would, we feel confident, have demonstrated them, had blood volume determinations been done at sufficiently frequent intervals. It must also be remembered that in some of the patients the recovery process had already commenced during the period of their evacuation to India; in these Stage I had been passed before they were admitted to hospital.

Stage I (0-4 *weeks*).—This is characterised by the rapid rise in plasma volume to normal. During this period the body weight at first falls, as oedema disappears, and then may slowly rise. The blood volume rises steadily, but at such a rate that, although the haemoglobin concentration together with the haematocrit usually falls, the total circulating haemoglobin actually increases. The R.B.C. concentration usually increases slightly although the haemoglobin concentration may have fallen, since the patient is rapidly becoming less macrocytic. The total circulating R.B.C. increases, as does the total circulating plasma protein. Because of the rapid rise in plasma volume, the plasma protein concentration changes but little; it sometimes falls slightly and sometimes increases. The increase in total circulating protein is in both fractions, but more albumin is formed than globulin, so that the albumin/globulin ratio increases slightly.

Stage II (2-12 *weeks*).—This is characterised by the rapid rise of both the total circulating volume and the total circulating plasma protein to normal. The plasma volume, which had attained normal values in Stage I, increases rapidly to values well above normal. There is also a rapid increase in body weight and in total circulating haemoglobin because both the haemoglobin concentration, which fell in Stage I, and the blood volume are increasing rapidly. Both the haematocrit and the R.B.C. concentration, and hence the total circulating R.B.C., are also increasing. The increase in the total circulating protein is in both fractions, but the albumin fraction increases much more rapidly than the globulin; the total circulating globulin does, however, frequently reach figures well in excess of normal. The albumin/globulin ratio continues to increase.

Stage III (8-16 *weeks*).—This stage marks the transition from Stage II to normal findings. It is characterised by the return of the plasma volume, which rose to above normal in Stage II, back to normal and by the maintainance of the total circulating volume at normal levels. This is accompanied by a rise in the haemoglobin concentration, R.B.C. concentration and haematocrit and, therefore, of the total circulating haemoglobin and total circulating R.B.C. There is a steady rise in the

body weight and a rise in the plasma protein concentration but, because of the fall in plasma volume, the total circulating plasma protein remains the same. There is, however, still a rise in the total circulating albumin, which is balanced by a corresponding fall in the total circulating globulin. This is reflected in the continued increase of the albumin/globulin ratio.

The above description, which is of necessity an over-simplification is summarised in Table II, and the salient features of the 'Stages of Recovery' are represented schematically in Figure 4.

13. *Rate of Recovery.*—Knowing the total circulating haemoglobin, the total circulating R.B.C. and the total circulating plasma protein at any given stage in the recovery process, it is possible to judge the rate of recovery in terms of the rate of production of haemoglobin, R.B.C. or plasma protein.

Table 12 gives the change in total circulatiug haemoglobin, R. B. C. and plasma protein in a series of patients whose progress was not complicated by transfusion, at the time of maximum recovery. It is seen that the average rate of haemoglobin production for the series was 7.9 gm./day (or roughly, if the total circulating volume remains constant, 1% on the Haldane scale per day). The average rate of R.B.C. production was 27×10^{10} cells per day. This reduced to the astonishing figure of 3.1 million cells every second. The average rate of plasma protein production was 2.3 gm./day. Thus, even although every tissue of the body must be suffering from the effects of protein deprivation, over 10 gm.* of the daily dietary protein intake is converted either to haemoglobin or plasma protein.

Table 11

Stages of Recovery in Protein Deficiency.

	Stage I (0-4 weeks)	Stage II (2-12 weeks)	Stage III (8-16 weeks)
Body Weight.	Decreases.	Increases Rapidly	Increases to Normal.
Plasma Volume.	Increases Rapidly to Normal.	Increases Rapidly to Above Normal.	Decreases to Normal.
Blood Volume.	Increases.	Increases to Normal.	No Change.
Hb. Concentration (and Haematocrit).	Decreases or No Change.	Increases.	Increases to Normal.
Total Circulating Haemoglobin.	Increases.	Increases Rapidly.	Increases to Normal.
R. B. C. Concentration.	Increases Slightly or No Change.	Increases.	Increases to Normal.
Total Circulating R.B.C.	Increases.	Increases Rapidly.	Increases to Normal.
Plasma Protein Concentration.	No Change or Slightly Increases.	Increases Rapidly.	Increases Slightly to Normal.
Total Circulating Plasma Protein.	Increases.	Increases Very Rapidly to Normal.	No Change.
Total Circulating Albumin.	Increases.	Increases Rapidly.	Increases to Normal.
Total Circulating Globulin.	Increases Slightly from Normal.	Increases to Above Normal.	Decreases to Normal.
A/G Ratio.	Increases slightly.	Increases.	Increases to Normal.

*This is neglecting the non-haemoglobin protein content of the packed cell volume which may be considerable.

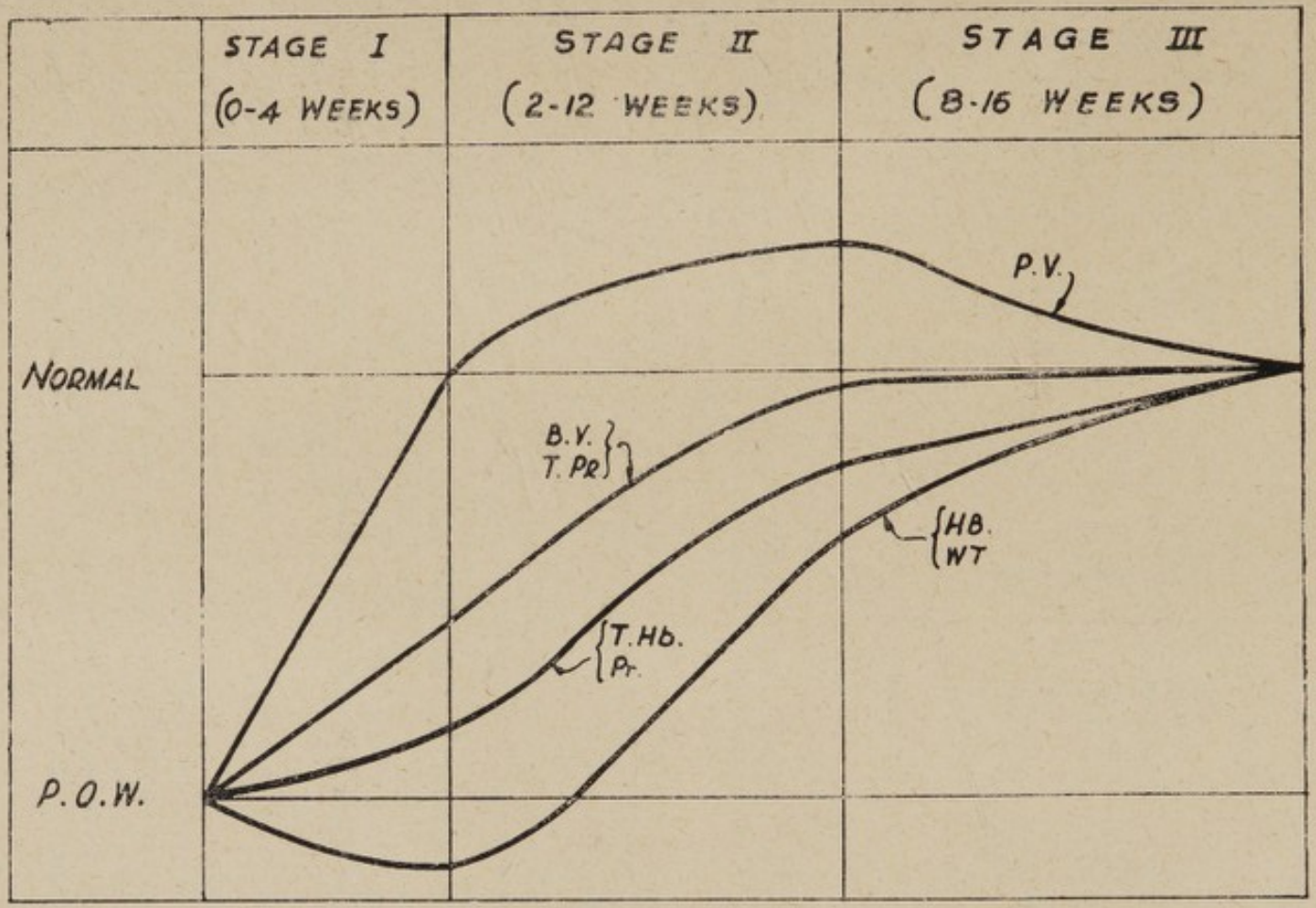


FIGURE 4:- SCHEMATIC REPRESENTATION OF THE STAGES OF RECOVERY. P.V., PLASMA VOLUME. B.V., BLOOD VOLUME. HB., HAEMOGLOBIN CONC. PR., PLASMA PROTEIN CONC. TPR., TOTAL CIRCULATING PLASMA PROTEIN. T.HB., TOTAL CIRCULATING HAEMOGLOBIN. WT., BODY WEIGHT.

Table 12

The Rate of Production of Haemoglobin, R.B.C., and Plasma Protein

Patient No.	Time Observed (Days)	Change in Total Circulating			Change per day in Total Circulating.		
		Haemoglobin (gm)	R.B.C. (Cells $\times 10^{12}$)	Plasma Protein (gm)	Haemoglobin (gm/day).	R.B.C. (Cells $\times 10^{10}$/day)	Plasma Protein (gm/day)
5	35	235	9.5	110	6.7	27.1	3.1
7	25	144	6.7	73	5.8	26.8	2.9
10	28	295	12.3	77	10.5	43.8	2.8
24	18	140	3.3	31	7.8	18.3	1.7
25	30	180	7.3	47	6.0	24.3	1.6
26	25	153	5.8	68	6.1	23.2	2.7
28	25	101	4.6	43	4.0	18.4	1.7
31	29	354	12.2	55	12.2	42.1	1.9
32	33	352	11.1	49	10.7	33.6	1.5
34	21	205	6.2	47	9.8	29.5	2.2
36	25	181	3.8	70	7.2	15.2	2.8
				Mean =	7.9	27.2	2.3

14. *Summary.* (1) Plasma and blood volume observations were made on a series of prisoners repatriated from Japanese prison camps and also on a control series of apparently healthy Indians.

(2) The chief findings in the prisoners at the time they were admitted to hospital were :—an orthochromic macrocytic anaemia ; a reduction in the total serum protein concentration which was confined almost entirely to the albumin fraction, hence a reduction in the albumin/globulin ratio ; a reduction in body weight ; a reduction in plasma volume of doubtful significance, an increase in plasma volume referred to unit body weight, no change referred to unit surface area and a decrease referred to unit body height ; a reduction in total circulating volume which was also reduced when referred to unit body weight, unit surface area and unit body height ; a reduction in the total circulating haemoglobin, total circulating R.B.C. and total circulating plasma protein which was also reduced when referred to unit body weight, unit surface area and unit body height. The reduction in total circulating plasma protein was entirely in the albumen fraction.

(3) All the above findings returned to normal with treatment in hospital.

(4) The haemoglobin concentration, and sometimes the plasma protein concentration, often fell during the first four weeks of treatment in hospital. Although this was so, the increase in plasma and total circulating volume was so great that the total circulating haemoglobin and plasma protein was increased. Therefore it does not necessarily follow that, because the haemoglobin concentration or serum protein concentration falls during the initial stages of treatment, the patient is not making satisfactory progress.

(5) The rate of recovery of each of the above factors was followed and, from this, the pattern of recovery was reconstructed. The recovery process has been divided into three arbitrary stages which have been described in detail.

(6) The rate of recovery in terms of the rate of production of haemoglobin, R.B.C. and plasma protein was also observed.

SECTION VI

THE EFFECT OF TRANSFUSION IN PROTEIN DEFICIENCY.

1. *Transfusion of Plasma.*—It has been mentioned in Section II that plasma transfusion often proves a life saving measure in extreme protein deficiency. The benefit of protein transfusion to the patients in Belsen has been stressed by Janet Vaughan (1945) and to prisoners repatriated from Lamsdorf by Edge (1945). The warning of Lipscomb (1945) underlined by Prior that " the greatest care is necessary to avoid overloading the circulation " has been heeded, but the clinical results of plasma transfusion have been uniformly so good that it is hoped that the above warning will not discourage further intravenous plasma therapy for patients suffering from protein deprivation.

Since the results were so encouraging, it was thought that a further investigation might prove profitable. In two patients, therefore, suffering from hypoproteinaemia, plasma volume determinations were done both before and after transfusion and, in one of these, this procedure was repeated twice more. The results obtained are given in Table 13.

On January 21 patient 47 had a plasma protein concentration of 3.93 gm (confirmed chemically) and a plasma volume of 2000 cc., giving a total circulating plasma protein of 79 gm. (Normal for Indians=180 gm.). His clinical condition was poor. There was a generalised oedema and ascites. On January 22 he was given an infusion of 65 gm. plasma protein in 500 cc. sterile water. The clinical condition improved immediately, the oedema disappeared and the free fluid in his abdomen became less. This is demonstrated in the reduction in his weight from 42.3 kgm. to 40.0 kgm. a loss of 5 lbs. Despite this, however, the plasma protein concentration had fallen from 3.93 gm/100 cc. to 3.42 gm /100 cc., a fall of 13%, and the haemoglobin concentration, R.B.C. concentration, and haematocrit had fallen even further, 27%, 26% and 28% respectively. The plasma volume had increased from 2000 cc. to 3310 cc. so that the total circulating plasma protein had risen from 79 gm. to 113 gm. This means that, although 65 gm of protein were infused, only 34 gm,, or 52% of that given, had remained in the circulation 24 hours later. Presumably the remaining 31 gm (48%) was taken up by other protein deficient tissues. There was no evidence of peripheral circulatery failure or increased capillary permeability.

The behaviour of Patient 24 proved much more interesting. On October 10 he was admitted to hospital in a critical condition (See photograph p. 10-c). He was grossly oedematous and his abdomen was distended with much free fluid. His plasma protein concentration was only 2.88 gm /100 cc. (confirmed chemically) and his plasma volume was 2460 cc. giving a total circulating plasma protein of 71 gm. On October 13 he was given an infusion containing 70 gm. protein. His haemoglobin concentration, R.B.C. concentration and haematocrit fell by 15%, 12% and 10% respectively, whilst his plasma volume rose from 2460 cc. to 2640 cc. and his plasma protein concentration from 2.88 gm./100 cc. to only 3.01 gm./100 cc. This gives a total circulating plasma protein of 80 gm. That is, of the 70 gm. protein infused, only 9 gm. or 13% remained in the circulation. Again it must be assumed that the remainder 61 gm. (87%) left the circulation and was either taken up by other protein depleted tissues or metabolised. Clinically the patient's condition was still critical and the gross oedema and ascites persisted. On the next day (Oct. 16) he was therefore given a further infusion of 70 gm plasma protein. On October 18 there was a slight rise in the haemoglobin concentration, R.B.C. concentration and haematocrit and an even greater rise in plasma protein concentration and a slight increase in plasma volume. The patient's clinical condition had slightly improved and the total circulating plasma protein had risen from 80 gm. to 109 gm. That is, of the 70 gm. protein given in the second infusion, 29 gm. or 41% had remained in the circulation

while 41 gm. or 59% had left it. On October 24 the patient was given a further infusion, this time of 75 gm. protein. The effect of this third infusion was dramatic. There was a marked diuresis and the oedema and ascites became very much less. This is reflected in the patient's weight, which was 44.2 kgm. on admission and had fallen on the day after the third infusion to 40.5 kgm. a fall of 3.7 kgm. or approximately 8 lbs. (See photograph p. 10-c). The plasma volume had risen to 3540 cc. and the plasma protein concentration to 5.44 gm./100 cc. This gives a total circulating plasma protein of 193 gm. or an increase of 84 gm. As 75 gm. were infused, it can be assumed that all of this remained in the circulation. Since it has been shown that, during recovery, the average rate of plasma protein production may be 2.3 gm./day, this figure is not unreasonable.

Table 13

Effect of Plasma Protein Infusion on Patients 47 and 24

Date.	Hb. Concentration (gm/100 cc).	R. B. C. concentration (10^6 cells/cmm).	Haematocrit (%).	Serum Protein (gm/100 cc).	Serum Albumin (gm/100 cc).	Serum globolin (gm/100 cc).	Ratio A/G (x : 1).	Plasma Volume (cc).	Total circ. Plasma Protein (gm).	Body weight (kgm).
	PATIENT 47.									
21-1-46	10.2	2.48	30.5	3.93	2.29	1.64	1.4	2000	79	42.3
22-1-46	Transfusion									
23-1-46	7.4	1.84	21.0	3.42	1.78	1.64	1.1	3310	113	40.0
						∴gained	34	(52 %)		
						Transf used		65		
						∴Left circulation		31	(48 %)	
	PATIENT 24.									
10-10-45	8.8	2.85	29.8	2.88	0.75	2.13	0.35	2460	71	44.2
13-10-45	Transfusion									
15-10-45	7.5	2.50	26.9	3.01	1.10	1.91	0.58	2640	80	
						∴gained		9 (13%)		
						Transf used		70		
						∴ Left circulation		61	(87%)	
16-10-45	Transfusion									
18-10-45	8.2	2.49	29.8	3.87	1.39	2.48	0.56	2810	109	
						∴gained		29 (41 %)		
						Transf used		70		
						∴Left circulation		41 (59 %)		
24-10-45	Transfusion									
25-10-45	7.6	2.64	27.5	5.44	2.09	3.35	0.63	3540	193	
						∴gained		84 (100 %)		
						Transf used		75		
						∴Left circulation		Nil.		

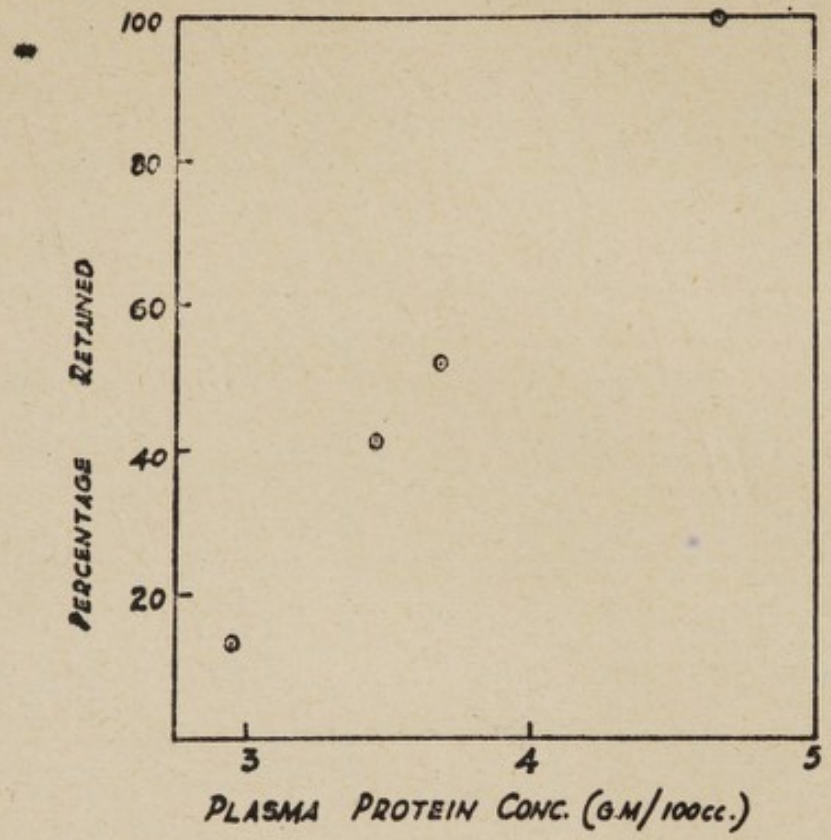
It is thus seen that the amount of protein retained in the circulation after an infusion depends upon the plasma protein concentration or, possibly, upon the total circulating plasma protein. If the degree of protein deprivation is severe, with consequent hypoproteinaemia and low total circulating plasma protein, a high percentage of infused protein leaves the circulation. As the degree of protein deprivation becomes less, and both the plasma protein concentration and the total circulating plasma protein becomes more, the percentage of protein that leaves the circulation becomes progressively less. The relevent data are presented in Table 14, and the important points shown in Figure 5. It will be seen that there is a good correlation between the percentage of infused protein retained in the circulation and both the mean plasma protein concentration and the mean total circulating plasma protein.

2. *Transfusion of Whole Blood.*—The effect of transfusion of whole blood to three patients suffering from nutritional macrocytic anaemia was also studied. It must, however, be stressed that these three patients were not repatriated prisoners and the biochemical and haematological findings have not been included in the consolidated tables of Sections III and IV. All three presented quite a different clinical picture from that of the repatriated prisoners in whom wasting and hypoproteinaemia predominated. The most pronounced feature in these cases was the anaemia, although there was, in addition, apparently a mild degree of super-imposed protein deficiency. All the patients were sufficiently anaemic to require immediate blood transfusion and so the opportunity was taken of estimating the plasma volume both before and after the transfusion. The results are shown in Table 15.

Patient 49 presented the typical picture of nutritional macrocytic anaemia when he was admitted on February 16. His haemoglobin concentration was 3.1 gm./100 cc., R.B.C. concentration 0.76×10^6 cells/cmm. and his haematocrit 10%, giving an M.C.V. of 131 c.μ. His serum protein was 5.33 gm./100 cc. so that, besides having a macrocytic anaemia, he was suffering from a mild degree of hypoproteinaemia. The plasma volume was 3380 cc. and the blood volume 3760 cc., giving a total circulating haemoglobin of 115 gm. (Normal for Indians=730 gm.) and a total circulating plasma protein of 180 gm. On February 17 he was transfused with 1000 cc. citrated blood, the actual haemoglobin concentration of which was 12.4 gm./100 cc. The haematocrit was 37 and the plasma protein concentration 5.8 gm./100 cc., so that he received 124 gm. haemoglobin and 37 gm. protein. On February 18, the day after the transfusion, the haemoglobin concentration, R.B.C. concentration and haematocrit had practically doubled themselves, but there was hardly any increase in plasma protein concentration. The plasma volume was the same as before the transfusion and, because of the increase in haematocrit, the total circulating volume was increased. The total circulating haemoglobin was now 241 gm. This represents a gain to the circulation of 126 gm. haemoglobin and 3 gm. plasma protein. The quantities infused were 124 gm. haemoglobin and 37 gm. protein. Thus, while almost all the infused haemoglobin remained in the circulation, 34 gm. out of the 37 gm. infused protein had left it.

Patient 48 behaved in an almost identical manner. His condition when he was admitted to hospital on January 30 was similar; he had a severe macrocytic anaemia and a mild degree of hypoproteinaemia (Table 15). On January 31 he was transfused with 50 gm. haemoglobin and 19 gm. protein. There was, as in the case of Patient 49, a rise in the haemoglobin concentration, R.B.C. concentration and haematocrit, but there was actually a fall in the plasma protein concentration. There was no change in the plasma volume and, because of the increase in haematocrit, a slight increase in the total circulating volume. When a balance sheet of the total circulating haemoglobin and plasma protein was prepared it was found that, of the 50 gm. haemoglobin infused, 49 gm. remained in the circulation. In fact after the transfusion the total circulating plasma protein was 5 gm. less than before it. This represents a total loss from the circulation of 24 gm. protein.

Patient 46 behaved in an even more striking fashion. When he was admitted to hospital on January 19 condition was again similar to that of patient 49 and patient



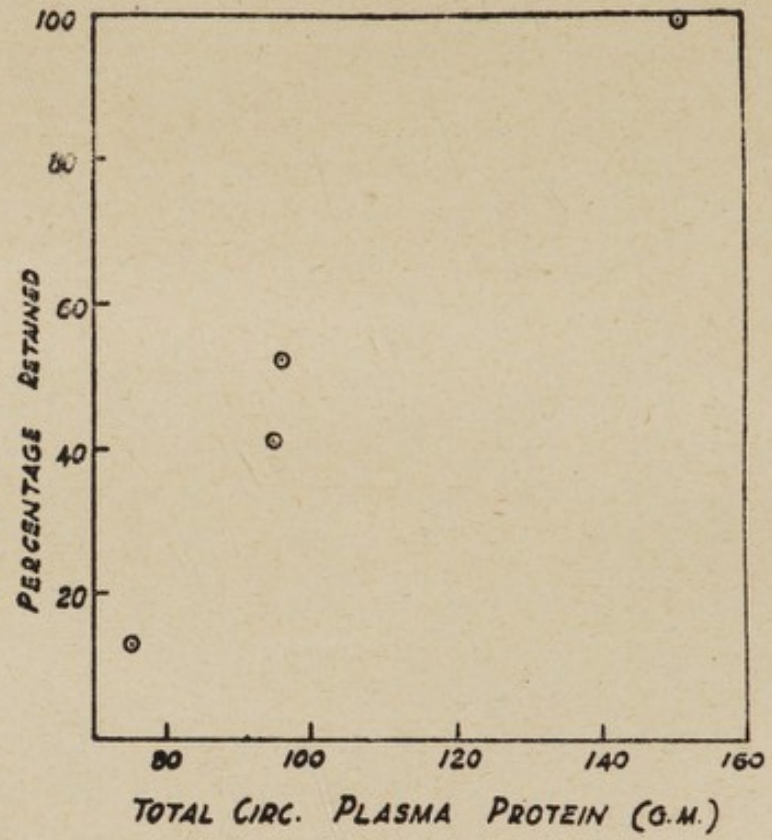
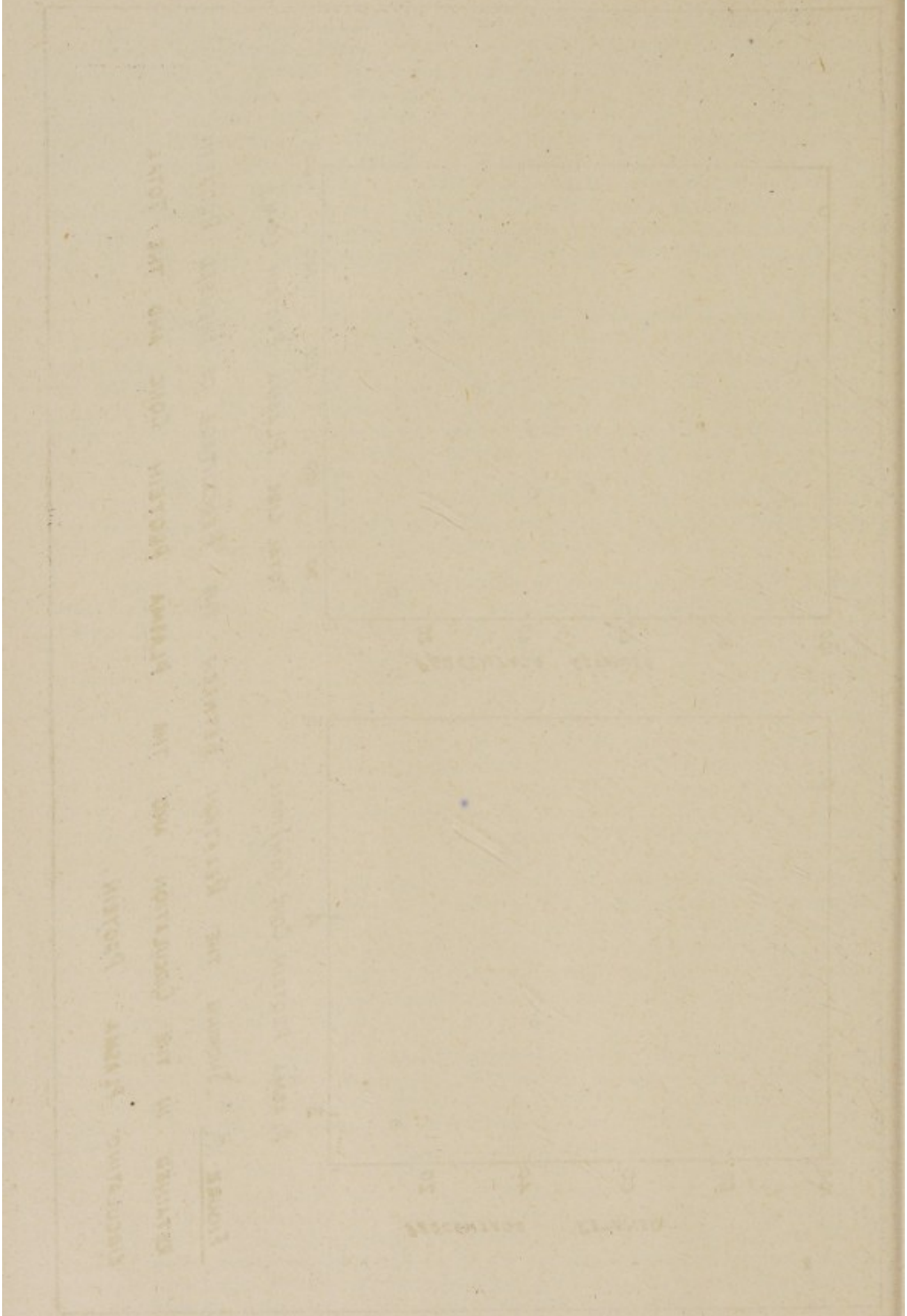


FIGURE 5: SHOWING THE RELATION BETWEEN THE PERCENTAGE OF INFUSED PROTEIN RETAINED IN THE CIRCULATION AND THE PLASMA PROTEIN CONC. AND THE TOTAL CIRCULATING PLASMA PROTEIN.



48 ; he had a severe macrocytic anaemia and a mild degree of hypoproteinaemia (Table 15). On January 20 he was transfused with 86 gm. haemoglobin and 27 gm. protein, and the observations repeated on January 21. There was the usual rise in haemoglobin concentration, R.B.C. concentration and haematocrit, but no change at all in the serum protein concentration. Also the plasma volume had, in this case, fallen from 2960 cc, to 2490 cc. It is seen that, of the 86 gm. haemoglobin infused, 81 gm. was left in circulation 24 hours' later, but that, as for patient 48, none of the

Table 14

Effect of mean plasma protein concentration and mean total circulating plasma protein on the retention of protein in the circulation after a plasma protein infusion.

Patient No.	Mean Plasma Protein Concentration. (gm/100cc)	Mean Total Circulating Plasma Protein (gm)	Percentage of Infused Protein which has left the Circulation.	Percentage of Infused Protein remaining in the Circulation.
24	2.95	75	87%	13%
24	3.44	95	59%	41%
47	3.68	96	48%	52%
24	4.66	151	0.%	100%

Table 15

Effect of blood transfusion on patients 49, 48 *and* 46.

Date.	Hb. Concentration. (gm/100 cc)	R.B.C. Concentration (10^6 Cells/cmm)	Haematocrit (%)	Serum Protein (gm./100 cc)	Serum Albumin (gm./100 cc)	Serum globmin (gm/100 cc).	Ratio A/G (x:1)	Plasma Volume (cc).	Blood Volume (cc)	Total Cir. Haem. (gm)	Total Circulation Plasma Protein (gm)
PATIENT 49.											
16-2-46	3.1	0.76	10.0	5.33	3.07	2.26	1.36	3380	3760	115	180
17-2-46	Transfused.										
18-2-46	5.9	1.43	17.1	5.40	2.87	2.53	1.1	3380	4080	241	183
						Therefore gained				126	3
						Transf used				124	37
						Therefore left circulation				0	34
PATIENT 48											
30-1-46	3.9	1.13	12.0	5.30	3.58	1.72	2.1	2940	3340	130	156
31-1-46	Transfused.										
2-2-46	5.2	1.48	15.5	5.13	3.37	1.76	1.9	2940	3480	179	151
						Therefore gained				49	5
						Transf used				50	19
						Therefore left circulation				1	24
				PATIENT 46							
19-1-46	3.3	0.76	10.0	5.30	3.02	2.25	1.35	2960	3290	107	157
20-1-46	Transfused.										
21-1-46	6.2	2.02	18.0	5.30	4.07	1.23	3.3	2490	3040	188	132
						Therefore gained				81	25
						Transf used				86	27
						Therefore left circulation				5	52

19 gm. infused protein remained in circulation. In this case the total circulating plasma protein was 25 gm. less than before the transfusion. This represents an overall loss to the circulation of 52 gm. protein.

That this is not the usual response to transfusion is shown in the case of patient 6 (Table 16). On October 17, after a month in hospital, this patient was still anaemic. His haemoglobin concentration was 6.3 gm./100 cc., his R.B.C. concentration 2.47×10^6 cells/cmm. and his haematocrit 23.8%. This gave an M.C.V. of 96.4 $c\mu$., so that he was no longer macrocytic; his M.C.V. on admission was 132 $c\mu$. Also, his plasma protein was now 6.28 gm./100 cc. compared with 4.20 gm./100 cc. on admission. Thus the patient was anaemic, but neither macrocytic nor hypoproteinaemic. On October 16 he was transfused with 160 gm. haemoglobin and 51 gm. plasma protein. The haemoglobin concentration, R.B.C. concentration and haematocrit rose as usual, but the plasma protein concentration and the plasma volume both rose also. A balance sheet of the total circulating haemoglobin and plasma protein shows that all of the 160 gm. haemoglobin infused remained in the circulation and 48 gm. of the 51 gm. protein. This is very different from the behaviour of patients 49, 48 and 46 where most of the haemoglobin was retained, but the plasma protein left the circulation.

3. *Discussion.*—It has been shown that, in the case of uncomplicated protein deficiency, only a small part of a plasma protein infusion is retained in the circulation. It also appears that there is a definite relation between the percentage of infused protein retained in the circulation and both the plasma protein concentration and the total circulating plasma protein. In conditions of severe protein deficiency it is quite understandable that protein should leave the circulation, either for the benefit of other protein depleted tissues or for metabolic purposes.

In addition it has been shown that, when a blood transfusion is given to a patient suffering from extreme tropical macrocytic anaemia with apparently only slight evidence of protein deficiency, whereas the haemoglobin of the infused blood remains in the circulation, the plasma protein is no longer present 24 hours later and, in some cases, part of the plasma protein that was originally present leaves the circulation as well. An explanation of this perplexing phenomenon is difficult. One suggestion is that before the transfusion plasma protein remained in the circulation at the expense of other tissues in order to keep up some such factor as the total circulating volume or the blood viscosity. This would imply that, in such patients, there is also a degree of protein deficiency much more severe than that which the serum protein concentration would lead one to believe. Then, when either the total circulating volume or blood viscosity can be maintained with the extra red cells provided in the infusion, the infused plasma protein, and in some cases that which is already present in the circulation, is available for other needs. On this view, the loss of protein from the circulation after blood transfusion in nutritional macrocytic anaemia is similar to the loss of protein that occurs in severe hypoproteinaemia.

4. *SUMMARY.*

(1) When a protein infusion was given to patients with severe hypoproteinaemia only a small part of the infused plasma protein was found in the circulation 24 hours later.

(2) The percentage of infused protein retained was related to the mean serum protein concentration and to the total circulating plasma protein.

(3) When a blood transfusion was given to patients suffering from extreme tropical macrocytic anaemia with apparently only slight evidence of protein deficiency, the haemoglobin of the infused blood remained in the circulation, but the plasma protein was no longer present 24 hours later and, in some cases, some of the plasma protein that was originally present in the circulation had also left it.

(4) When a blood transfusion was given to an anaemic patient who was neither macrocytic nor hypoproteinaemic, both the haemoglobin and the plasma of the infused blood was retained in the circulation 24 hours later.

Table 16

Effect of blood transfusion on patient 6.

Date.	Hb. Concentration (gm/100 cc)	R.B.C. concentration (10^6 x Cells/cmm)	Haematocrit (%)	Serum Protein (gm/100 cc)	Serum Albumin (gm/cc)	Serum globulin (gm/100cc)	Ratio A/G (x : 1)	Plasma Volume (cc)	Blood Volume (cc)	Total Circ. Hb. (gm)	Total Circulation Phasma Protein (gm)
17-10-46	6.3	2.47	23.8	6.23	2.70	3.53	0.77	2700	3540	221	168
18-10-46	Transfused.										
19-10-46	8.8	2.63	30.0	6.85	2.99	3.84	0.78	3150	4500	396	216
						Ther	efore	gained		175	48
						Tran	sfused			160	51
						Ther	efore l	eft circ	ulation	0	3

SECTION VII

FRACTIONAL TEST MEAL INVESTIGATIONS.

1. *Findings.*—Fractional Test Meals were done on a series of 21 patients soon after they were admitted to hospital and again before they were discharged. The meal consisted of 12 ozs. oatmeal gruel. Samples were withdrawn at 15 minutes intervals and, if there was no free acid in the gastric juice after 60 minutes, 1 mgm. histamine was given by subcutaneous injection.

The findings are summarised in Table 17. Of the 21 patients studied, 2 had a histamine resistant achlorhydria, 5 had no free acid after the gruel meal but responded to histamine, one had a hypochlorhydria, 3 had a delayed emptying time and 10 were normal. After the period of treatment in hospital not one patient failed to produce free acid even without histamine, 3 had a hypochlorhydria, one had a delayed emptying time and the remainder were normal. These findings are similar to those of the control series, the results of which are also given in Table 17.

2. *Effect of Specific Therapy on Fractional Test Meal Findings.*—An attempt was made to gauge the effect of treatment on such patients as initially had a deficiency of gastric function. A record was kept of the drugs each patient received and the fractional test meal was repeated. In some cases this was done at frequent intervals. The acid response curve has been divided into five types *viz.* (1) histamine resistant achlorhydria, (2) achlorhydria responding to histamine, (3) hypochlorhydria (less than 20 cc. N/10 free acid per 100 cc.), (4) normal free acid, but delayed emptying time and (5) normal curve. Any change from type (1) in the direction of type (5) was recorded as an improvement and any change in the opposite direction as a deterioration.

The full results are given in Table 18 and can be conveniently summarised in Table 19. It is seen that nicotinic acid, in the dosage employed and by the route employed, had no effect on the fractional test meal findings. Of the 8 tests done, there was an improvement in 2, a deterioration in 3, and no change in 3. Riboflavin on the other hand, caused a dramatic improvement. Patients who produced no free acid even after histamine injection responded to histamine, those who produced no free acid after the gruel meal now did so, and patients with hypochlorhydria and a delayed emptying time became normal. Table 19 shows that of the 17 tests done, there was an improvement in 15, a deterioration in none, and no change in 2. These figures are obviously significant.

Three tests were done with liver extract alone and, like riboflavin, the response was good ; in all three there was an improvement. Also a few tests were done with nicotinic acid, riboflavin and liver extract in different combinations. In each case, as was to be expected, there was a marked improvement.

3. *Discussion.*—It appears that both liver extract (which contains riboflavin) and riboflavin itself have a beneficial effect on gastric function as judged by fractional test meal findings. As far as we know this is the first time it has been shown that riboflavin increases the ability of the gastric mucosa to produce acid. A further clinical trial of riboflavin in other conditions in which achlorhydria is a frequent finding would be of considerable interest. It must be pointed out, however, that patients who received riboflavin alone, although their fractional test meal findings improved, did not maintain a general clinical improvement (Section II) nor did they show any improvement in the glucose tolerance test.

4. *SUMMARY.*

(1) Fractional test meals were done on a series of 21 P.O.W. patients soon after they were admitted to hospital. Of these 2 had a histamine resistant achlorhydria, 5 had no free acid after the gruel meal but responded to histamine, one had a hypochlorhydria, 3 had a delayed emptying time and 10 were normal.

(2) After treatment in hospital all the patients produced free acid even without histamine. One had a hypochlorhydria and one a delayed emptying time; the remainder were normal.

(3) Nicotinic acid had no effect on the fractional test meal findings.

(4) There was a marked improvement in the fractional test meal findings while the patients were receiving riboflavin.

(5) There was also an improvement in the few cases studied while the patient was receiving either liver extract, liver extract plus nicotinic acid, liver extract plus riboflavin, or nicotinic acid plus riboflavin.

Table 17

Fractional test meal findings on P.O.W. patients on admission compared with a control series on healthy Indians.

	No. Examined.	No. with Achlorhydria histamine resistant.	No. with Achlorhydria responding to histamine.	No. with Hypochlohydria.	No. with Delayed Emptying Time.	
Patients on Admission	21	2	5	1	3	10
Patients on discharge ..	21	0	0	3	1	17
Control Series ..	9	0	0	1	3	5

Table 18

Effect of the treatment on fractional test meal findings.

+, Achlorhydria, Histamine Resistant.
++, Achlorhydria responding to Histamine.
+++, Hypochlorhydria (less than 20 cc. N/10 free acid per 100 cc.).
++++, Normal Free acid, Delayed Emptying.
+++++, Normal Curves.

Patient No.	Treatment	Dose.	Duration (Days).	Type of Curve.	
				Before.	After.
15	Riboflavin.	4 mg/day parenterally ..	3	+	++
18	„	4 mg/day parenterally ..	3	++	++
18	„	4 mg/day parenterally 12 mg/day by mouth ..	6	+++	+++++
25	„	9 mg/day parenterally ..	7	++	+++++
25	„	9 mg/day parenterally ..	14	++	+++++
28	„	4 mg/day parenterally .. 12 mg/day by mouth	7	+++	++++
28	„	12 mg/day by mouth ..	7	+++	+++
28	„	12 mg/day by mouth ..	14	+++	++++

Table 18—*contd.*

Patient No.	Treatment.	Dose.	Duration (Days).	Type of Curve.	
				Before.	After.
31	Riboflavin	12 mg/day parenterally ..	7	++++	+++++
32	„	4 mg/day parenterally .. 12 mg/day by mouth	7	+	++
32	„	4 mg/day parenterally .. 12 mg/day by mouth	17	+	+++
33	„	12 mg/day by mouth ..	7	++++	+++++
33	„	12 mg/day by mouth ..	14	++++	+++++
33	„	12 mg/day by mouth ..	21	++++	+++++
34	„	4 mg/day parenterally .. 12 mg/day by mouth	7	+++	+++++
35	„	4 mg/day parenterally .. 12 mg/day by mouth	7	++	+++
36	„	4 mg/day parenterally .. 12 mg/day by mouth	4	++	+++
				44	66
15	Nicotinic Acid	150 mg/day by mouth ..	3	++	+
15	„	300 mg/day by mouth ..	7	+	++
15	„	300 mg/day by mouth ..	14	+	+
18	„	150 mg/day by mouth ..	3	+++	++
18	„	300 mg/day by mouth ..	7	++	++
18	„	300 mg/day by mouth ..	14	++	+++
28	„	300 mg/day by mouth ..	7	++++	+++
32	„	300 mg/day by mouth ..	7	+	+
				16	15
15	Liver Extract	12 cc/day parenterally ..	3	++	+++
18	„	12 cc/day parenterally ..	3	++	+++
18	„	12 cc/day parenterally ..	7	++	+++++
				6	11
6	Nicotinic Acid + Liver Extract	200 mg/day by mouth .. 4 cc/day parenterally	18	+	+++++

Table 18—(*concld.*)

Patient No.	Treatment.	Dose.	Duration (Days).	Type of Curve.	
				Before.	After.
15	Riboflavin + Liver Extract	4 mg/day parenterally .. 12 mg/day by mouth. 4 cc/3 days parenterally	11	+	+++++
15	Riboflavin + Liver Extract	do, ..	17	+	+++++
18	Nicotinic Acid + Riboflavin	200 mg/day by mouth .. 2 mg/day parenterally	11	++	+++
				5	18

Table 19

Summary of the effect of treatment on fractional test meal findings.

Treatment.	No. Tests.	No. Better.	No. Worse.	No. No Change.
Nicotinic Acid ..	8	2	3	3
Riboflavin ..	17	15	0	2
Liver Extract ..	3	3	0	0
Nicotinic Acid .. + Liver Extract	1	1	0	0
Riboflavin .. + Liver Extract	2	2	0	0
Nicotinic Acid .. + Riboflavin	1	1	0	0

SECTION VIII

TESTS OF ABSORPTION FROM THE GASTRO-INTESTINAL TRACT.

1. *Introduction.*—It has generally been believed that starvation is invariably accompanied by a failure of absorption from the gastro-intestinal tract. Thus, for example, Magee (1945) states that "failure of absorption is an essential lesion in starvation. Evidence pointed to a progressive decline in the effeciency of absorption with increase of fasting period".

The majority of the Indian prisoners repatriated from Japanese prison camps were undoubtedly suffering from starvation and many of them from extreme protein deficiency, but, by the time they had been evacuated to India, they were remarkably free from any clinical evidence of deficiency of absorption. In fact one of the most striking features of this investigation has been the rarity with which diarrhoea was encountered. All the reports of the conditions in Belsen have stressed the prevalence of diarrhoea (*e.g.* The Lancet (1945), Collis (1945), Lipscomb (1945) and Mollison (1946)) and apparently diarrhoea was also common in Lamsdorf (Edge (1945)) and Auschwitz (Adelsberger (1946)). In his paper Edge (1945) actually speaks of starvation diarrhoea. The point we wish to emphasise is that, in Indian Troops at any rate, diarrhoea and the accompanying defects of absorption are not necessarily part of the starvation syndrome. Our observations were made when the prisoners, arrived in India. It is possible that the incidence of diarrhoea among the Indian prisoners may have been higher at the time of their release. However, Mitchell and Black (1946), studying British Troops liberated from Japanese prison camps, reported that 23% "had a degree of diarrhoea at one time or another," an incidence which appears to be much lower than that of the European camps where a similar degree of starvation was encountered.

A few of the patients did, however, have slight evidence of absorptive defect and complained of diarrhoea when they arrived in India, but these were very much in the minority. The patients who form the subjects for the studies described in the remainder of this section represent the sum total of all those who, from the 2000 prisoners examined, had any clinical evidence of impaired absorption. As is pointed out in Section II, all such patients had, in addition, clinical evidence of nicotinic acid deficiency and the diarrhoea is considered part of the nicotinic acid deficiency syndrome. For this reason 'starvation diarrhoea' is a misleading term, for the diarrhoea is not the result of starvation *per se*. In fact, as Mitchell and Black (1946) point out, increasing the dietary intake may cause an increase in the severity of the diarrhoea.

Patients with diarrhoea usually had evidence of steatorrhoea as well, so absorption tests and faecal fat estimations were done soon after they were admitted to hospital and again before they were discharged. The results are presented, not because they are typical of Indian prisoners repatriated from Japanese prison camps, but merely for the sake of completeness.

2. *Glucose Tolerance Curves.*—

(*a*) *General.*—Glucose Tolerance tests were done on a number of patients soon after they were admitted to hospital. In the majority of these there was no impairment of glucose tolerance, but in 11, and these included all the cases with clinical evidence of absorptive defect, there was an impairment. In these patients the test was repeated, in some cases at frequent intervals. For purposes of comparison glucose tolerance tests were also done on the control series. The complete figures are given in Tables 54, 55 and 56 of the Appendix, and the curves shown graphically in Figure 6. It can be seen at a glance that the curves of the patients on admission

tend to start at a lower level, tend to be flatter, and tend to take longer to return to the fasting value, than either the curves of the same patients on discharge or those of the control series. This impairment of glucose tolerance function is thought to be due to a defective absorption from the gastro-intestinal tract.

In a more detailed analysis the curves were judged by the following criteria :— (1) The fasting blood sugar concentration. (2) The rise in blood sugar concentration, *i.e.* the difference between the maximum blood sugar concentration and the fasting level, (3) the time of the maximum blood sugar concentration, (4) the difference between the 3-hour blood sugar concentration and the fasting level.

(*b*) *Fasting Blood Sugar.*—Table 20 gives the fasting blood sugar concentration of the 11 patients shortly after they were admitted to hospital and again before they were discharged, together with the fasting blood sugar concentration of the control group done by the same method. The fasting blood sugar concentration on admission was 91 mgm./100 cc. while on discharge it was 107 mgm./100 cc. representing an average increase of 16 mgm./100 cc. The increase in fasting blood sugar was observed in every patient with the exception of one, in whom there was no change, and is obviously significant statistically. Also the fasting level of the control group was on the average even higher than that of the patients just before they were discharged. It is possible, therefore, that this low fasting blood sugar concentration observed in repatriated prisoners is a result of their poor dietary history.

(*c*) *Rise in Blood Sugar Concentration.*—Table 21 shows that the average rise in blood sugar concentration of the 11 patients after 50 gm. of glucose by mouth was 24 mgm./100 cc. on admission and 38 mgm./100 cc. on discharge. This represents an increase of 14 mgm./100 cc. during the period of treatment in hospital. This increase is just significant statistically (between 0.02 and 0.05) and was observed in 8 out of the 11 patients studied.

(*d*) *Time of the Rise.*—Table 22 shows that, of the 11 patients investigated on admission, the rise was at the ½ hour period in 3, at the 1hour period in 5 and at the 1½-hour period in 3. This is in contrast to the control series where the rise was at the ½ hour period in 7 out of the 8 subjects studied. After treatment in hospital, however, there was little improvement in the glucose tolerance as judged by the time of the rise for, on discharge, of the 11 patients studied, in only 3 was the rise at the ½ hour period ; in 7 it was at the 1 hour period and in one at the 1½-hour period.

(*e*) *Difference between the 3-hour Blood Sugar Concentration and the Fasting Level.*—Table 23 shows that when the patients were admitted to hospital the 3-hour blood sugar concentration was on the average 6 mgm./100 cc. higher than the fasting level whereas, before they were discharged, the 3-hour blood sugar concentration was 13 mgm./100 cc. lower than the fasting level, compared with the control series where it was 20 mgm./100 cc. lower. This improvement in the difference between the 3-hour blood sugar concentration and the fasting level was observed in 10 out of the 11 patients studied while in the remaining one there was no change. It is obviously of significance statistically.

(*f*) *Summary of the Effect of Treatment in Hospital on the Glucose Tolerance Test.*—Table 24 gives a summary of the effect of treatment in hospital on the glucose tolerance curve as judged by each of the four criteria already mentioned. The fasting blood sugar concentration improved in 10 out of 11 patients, the rise in blood sugar in 8 out of 11, but the time of the maximum blood sugar concentration in only 4 out of the 11. The difference between the 3-hour concentration and the fasting level improved in 10 out of the 11.

(*g*) *Effect of Specific Therapy on the Glucose Tolerance.*—The effect of nicotinic acid, riboflavin and nicotinic acid and liver extract together on the glucose tolerance curve, as judged by (1) the height of the rise above the Fasting level and (2) the difference between the 3-hour blood sugar concentration and the fasting level is given in Table 25. The results are conveniently summarised in Table 26. It is seen that riboflavin had no effect whatever on the glucose tolerance. Judged by the height of the rise, there was an improvement in 6 out of the 7 tests done while the patients

were receiving nicotinic acid, but judged by the difference between the 3-hour and the fasting level there was an improvement in only 4 out of the 7. In 5 tests the patient received nicotinic acid and liver extract together and, judged by both criteria, there was an improvement in every test.

Table 20

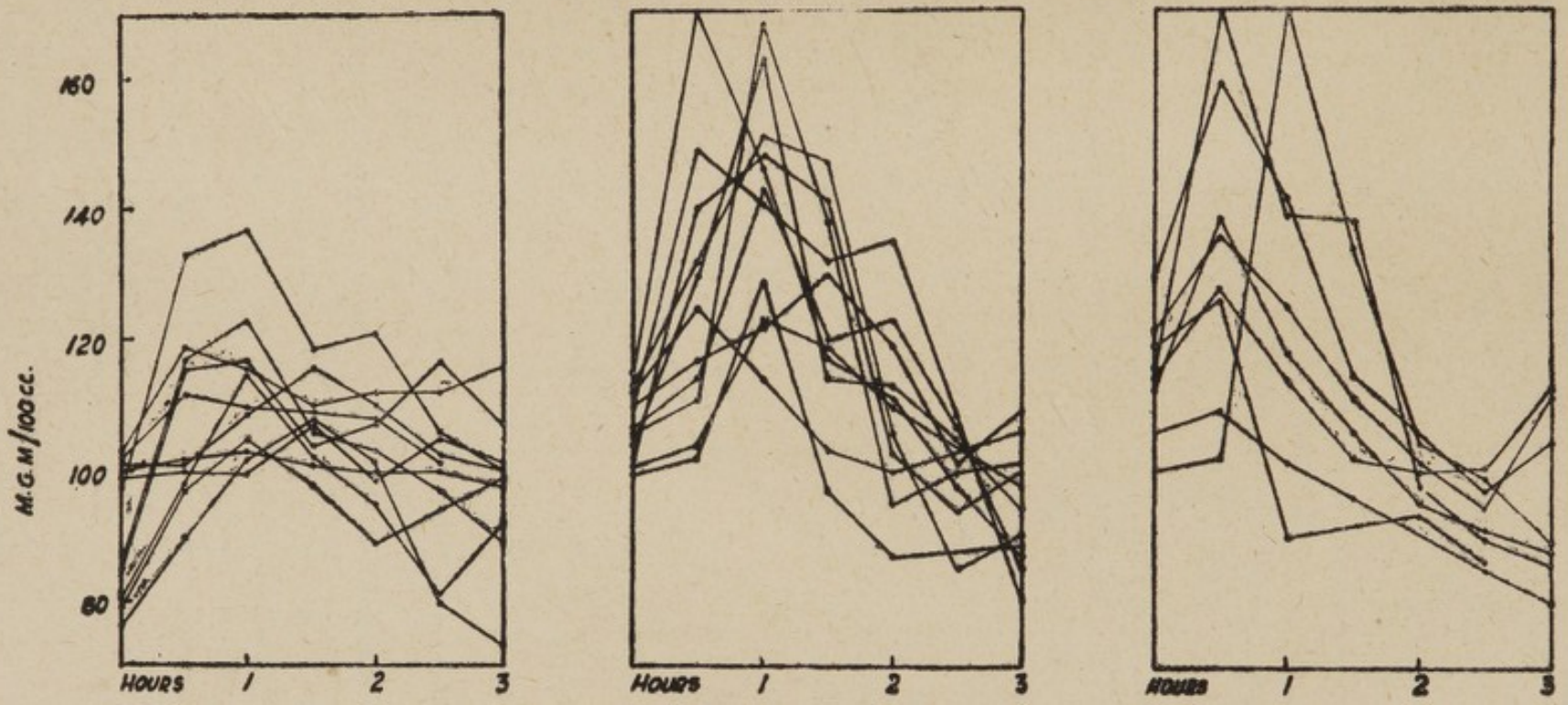
Fasting Blood Sugar (mgm./100 cc.).

Prisoners of War.				Controls.	
Patient No.	On Admission	On Discharge	Difference	Patient No.	Blood Sugar
2	86	116	+24	N37	119
6	85	113	+28	N38	106
11	97	115	+18	N39	100
15	76	106	+30	N41	112
18	79	106	+27	N42	116
25	100	102	+ 2	N43	121
26	80	102	+22	N44	129
28	100	100	0	N45	115
31	101	111	+10		
32	100	112	+12		
36	99	102	+ 3		
Mean ..	91	107	16		115

Table 21

Glucose Tolerance Test. The rise in blood sugar (mgm./100 cc.) i.e. the difference between the maximum blood sugar concentration and the fasting level.

Prisoners of War.				Controls.	
Patient No.	On Admission	On Discharge	Difference	Patient No.	Rise in Blood Sugar
2	37	20	—17	N37	7
6	33	38	+ 5	N38	3
11	40	56	+16	N39	72
15	32	62	+30	N41	26
18	26	37	+11	N42	58
25	4	46	+42	N43	15
26	35	21	—14	N44	30
28	13	29	+16	N45	11
31	6	38	+32		
32	16	13	— 3		
36	20	61	+41		
Mean ..	24	38	14		28



ON ADMISSION. ON DISCHARGE. CONTROLS

FIGURE 6: GLUCOSE TOLERANCE CURVES (50 GM GLUCOSE) OF PATIENTS ON ADMISSION AND ON DISCHARGE COMPARED WITH THOSE OF THE CONTROL SERIES.

Table 22

Glucose Tolerance Test. Time of Maximum Blood Sugar Concentration.

	No. Patients	No. at ½ hr. period	No. at 1 hr. period	No. at 1½ hr. period
On Admission ..	11	3	5	3
On Discharge ..	11	3	7	1
Controls ..	8	7	1	0

Table 23

Glucose Tolerance Test. Difference Between the 3-hour Concentration and the Fasting level (mgm./100 cc.).

Prisoners of War				Controls	
Patient No.	On Admission	On Discharge	Difference	Patient No.	3-hr. Conc.-Fasting Conc.
2	3	—1	4	N37	—33
6	22	—23	45	N38	—26
11	3	—28	31	N39	—11
15	—3	—5	8	N41	—24
18	20	—21	41	N42	—28
25	—2	—2	0	N43	—10
26	12	—3	15	N44	—25
28	1	—12	13	N45	—3
31	—1	—31	32		
32	0	—6	6		
36	17	—8	25		
Mean ..	6	—13	20		—20

Table 24

Effect of Treatment in Hospital on the Glucose Tolerance Test as judged by different Criteria.

Criterion	No. Patients	No. Better	No. Worse	No. no change
Fasting Blood Sugar Level ..	11	10	0	1
Rise in Blood Sugar *i.e.* the Difference between the Maximum Blood Sugar Concentration and the Fasting Level ..	11	8	3	0
Time of Maximum Blood Sugar Concentration ..	11	4	4	3
Difference between the 3-hour Concentration and the Fasting Level	11	10	0	1

Table 25

Effect of treatment on the glucose tolerance test as judged by (1) *the maximum height of the curve above the fasting level and* (2) *the difference between the 3-hour blood sugar concentration and the fasting level.* +=*an improvement.* —=*a deterioration.*

Patient No.	Treatment	Dose	Duration	Improvement or No.	
				1	2
15	Nicotinic Acid.	300 mg/day by mouth ..	7	+	—
15	,,	300 mg/day by mouth ..	14	+	+
18	,,	200 mg/day by mouth ..	12	+	+
28	,,	150 mg/day by mouth ..	7	+	—
28	,,	150 mg/day by mouth ..	7	—	+
31	,,	300 mg/day by mouth ..	7	+	+
32	,,	300 mg/day by mouth ..	9	+	—
25	Riboflavin	9 mg/day parenterally ..	14	+	+
28	,,	4 mg/day parenterally .. 12 mg/day by mouth	7	—	—
28	,,	12 mg/day by mouth ..	7	+	+
31	,,	12 mg/day parenterally ..	7	+	+
31	,,	4 mg/day parenterally .. 12 mg/day by mouth	11	—	+
32	,,	4 mg/day parenterally .. 12 mg/day by mouth	7	—	+
32	,,	12 mg/day by mouth ..	7	+	—
32	,,	12 mg/day by mouth ..	58	—	—
36	,,	4 mg/day parenterally .. 12 mg/day by mouth	14	—	+
6	Nicotinic Acid. Liver Extrct	200 mg/day by mouth .. 4 cc/day parenterally	42	+	+
28	Nicotinic Acid+ Liver Extract	150 mg/day by mouth .. 4 cc/day parenterally ..	47	+	+
31	Nicotinic Acid + Liver Extract	300 mg/day by mouth .. 2 cc/day parenterally	58	+	+
36	Nicotinic Acid+ Liver Extract	500 mg/day by mouth .. 150 mg/day parenterally 4 cc/2 days parenterally	8	+	—
36	Nicotinic Acid + Liver Extract	500 mg/day by mouth .. 4 cc/2 days parenterally	55	+	+

Table 26

Summary of the Effect of Treatment on Glucose Tolerance Test Findings.

Treatment	No. Tests	Judged by height of curve above fasting level		Judged by difference between 3-hr. and fasting level	
		No. Better	No. Worse	No. Better	No. Worse
Nicotinic Acid ..	7	6	1	4	3
Riboflavin ..	9	4	5	6	3
Nicotinic Acid .. + Liver Extract.	5	5	0	5	0

The number of tests made in Table 26 was small and the results should be regarded as suggestive only. The figures tested serially by the exact factorial method turned out to be not within the conventional limits of significance.

(*h*) *Discussion.*—Thus it is seen that, whereas riboflavin caused a marked improvement in the fractional test meal findings, in the dosage employed it had no effect on the glucose tolerance test. Nicotinic acid, on the other hand certainly had no effect on the fractional test meal findings but, at any rate judged by some criteria, did have a beneficial effect on the glucose tolerance test. Nicotinic acid in association with the liver extract caused an improvement in the glucose tolerance test in each of the 5 cases studied.

3. *Fat Tolerance Curves.*—

(*a*) *General.*—Fat Tolerance tests were done on a series of 9 patients soon after they were admitted to hospital and again on the same patients before they were discharged. The patients were selected because, clinically, they showed some abnormality of absorption. Each of them had an impaired glucose tolerance curve. They are, therefore, typical of those patients with the worst absorptive defects rather than representative of all Indian prisoners repatriated from Japanese prison camps. The technique of the fat tolerance test used was as follows: The patient, after a period of 12 hours without food, was given a fat meal consisting of 1 tin of twice concentrated unsweetened milk. The fat content of the meal was 22 gm. Meals with a greater fat content were not well tolerated. It was found that the most suitable times for sampling after such a meal were at the end of 2, 3 and 4 hours. An initial fasting sample was also taken. The serum total fat (ether soluble matter) and serum cholesterol were determined in each sample. In addition, an attempt was made to measure the lipoid phosphorus by the method of Harnes (1928), but the results were technically unsatisfactory and have been discarded.

In normal patients, there was a rise in the serum total fat 2-3 hours after the fat meal, and also a rise in the serum cholesterol at about the same time. In the returned prisoners, at the time of admission, the absorptive curve was, in general, flatter and the time taken for the maximum concentration to be reached was longer. Also it was found that the fasting serum cholesterol concentration was greatly reduced, but not the fasting serum total fat. After a period of treatment in hospital averaging 3 months, the absorptive curves approximated to normal.

(*b*) *Fasting Serum Total Fat.*—The average fasting serum total fat was 480 mgm./100 cc. on admission and 490 mgm./100 cc. shortly before discharge (Table 27). There was thus no significant change in the serum total fat and the figures are of the same order as those of the control group (Mean=460 mgm./100 cc.).

(*c*) *Fasting Serum Cholesterol.*—The mean fasting serum cholesterol of the patients soon after they were admitted to hospital was 99 mgm./100 cc. (Table 28). This figure is surprisingly low. Before the patients were discharged the mean fasting serum cholesterol was 136 mgm./100 cc. representing an average increase of 37 mgm./100 cc. This figure is highly significant (P is less than 0.01). The average figure for the control group, 102 mgm./100 cc. is, however, of the same order as that of the patients on admission. The figure is extremely low, but many workers have commented on the low serum cholesterol found in apparently healthy Indians (Raman (1940), Nayer (1942)).

(*d*) *Rise in Serum Total Fat.*—Table 29 shows that after the fat meal of 22 gm. the mean rise in serum total fat was only 66 mgm./100 cc. at the time the patients were admitted to hospital but that, by the time the patients were fit for discharge, the mean rise was 123 mgm./100 cc. This figure is just significant statistically (P is between 0.02 and 0.05). The mean rise observed in the control series was 121 mgm./100 cc.

(*e*) *Rise in Serum Cholesterol.*—The rise in serum cholesterol after the fat meal was small and, as can be seen from Table 30, very irregular. The mean rise soon after admission was 19 mgm./100 cc. and on discharge was 22 mgm./100 cc. The difference is clearly not significant. Also the mean rise of serum cholesterol of the control group was only 11 mgm./100 cc.

(*f*) *Time of the Maximum Serum Total Fat Concentration.*—Of the 9 tests done on the patients soon after they were admitted to hospital the rise in the serum total fat occurred at the 3-hour period in 4 and at the 4-hour period in 5 (Table 31). By the time the patients were fit for discharge, the rise occurred much sooner. In one patient it occurred at the 2-hour period, in 8 at the 3-hour period and in none at the 4-hour period. Of the 8 tests done on the control series the rise occurred at the 2-hour period in 4, at the 3-hour period in 4 and at the 4-hour period in none.

(*g*) *Time of Maximum Serum Cholesterol Concentration.*—The effect of treatment in hospital on the time of the rise in serum cholesterol concentration was even more impressive (Table 32). Of the 9 tests done when the patients were admitted to hospital, the rise occurred at the 2-hour period in none, at the 3-hour period in 4 and at the 4-hour period in 5. At the time of discharge, the rise occurred at the 2-hour period in 6, at the 3-hour period in 2 and at the 4-hour period in one. This compares favourably with the control series where, in 8 tests, the rise occurred at the 2-hour period in 5, at the 3-hour period in one and at the 4-hour period in 2.

(*h*) *Summary of the Effect of Treatment in Hospital on the Fat Tolerance Test.*—Table 33 gives a summary of the effect of treatment in hospital on the fat tolerance curve, as judged by each of the foregoing criteria . It is seen that there was no change in the fasting serum total fat (increased in 5 patients and decreased in 4), but that there was a marked increase in the fasting serum cholesterol (increased in 8 patients, decreased in one). The rise in serum total fat after the standard fat meal was also increased in 7 patients and decreased in 2, whereas there was no effect on the rise in serum cholesterol (increased in 4 patients, decreased in 5). There was also a decrease in the time at which the rise in both serum total fat and serum cholesterol took place (decreased in 6 patients, increased in none and no change in 3 for both serum total fat and serum cholesterol).

Thus it is seen that, after treatment in hospital, there was an improvement in the fat tolerance curves as judged by the height of the rise in serum total fat and the time of the rise of both serum total fat and serum cholesterol. There was also a significant increase in the fasting serum cholesterol. The fat absorption test was not sufficiently precise to gauge the effect of different courses of therapy on absorptive function.

(*i*) *Serum Inorganic Phosphorus.*—During the course of the fat tolerance test the serum inorganic phosphorus was measured in a number of cases. The results are given in Table 34. It was found that, after the meal of 22 gm. fat, the serum inorganic phosphorus nearly always rose. This rise was at the 3-hour, or more often,

the 4-hour period. Neither the height nor the time of the rise observed in the returned prisoners, most of whom had an impaired fat absorption, appeared to differ significantly from that of the control group, where fat absorption was normal. In view of the current theories concerning 'phosphorylation' in the absorption of fat, these observations on the rise in serum inorganic phosphorus might well repay further study. . In one patient (Patient 15) the inorganic phosphorus content of the fat meal was increased to twice the normal value by the addition of inorganic phosphate. The result was not significantly different, so it is unlikely that the rise in serum inorganic phosphorus is merely the result of the absorption of large quantities of inorganic phosphate from the gastro-intestinal tract. Also in two patients (Patients 15 and 18) it was shown, as is well known, that a meal of 50 gm. glucose causes a fall, and not a rise, in inorganic phosphorus. This observed rise in inorganic phosphorus, therefore, is presumably due to the fat in the milk meal and not to its carbohydrate or phosphate content.

4. *Comparison of Different Absorption Tests.*—Glucose Tolerance tests were also done on the 9 patients whose fat absorption was studied. Table 35 shows the degree of impairment of absorption, both on admission and in the same patients on discharge, as judged by, for the fat tolerance test, (1) the height of the rise in serum total fat, (2) the time of the rise in serum total fat, (3) the time of the rise in serum cholesterol and, for the glucose tolerance test, (4) the height of the rise in blood sugar concentration and (5) the difference between the 3-hour blood sugar concentration and the fasting level. The findings in two patients who had unimpaired absorptive function is also given. It is seen that the agreement between the different indices of absorptive function, both of carbohydrate and of fat, is quite good.

5. *Faecal Fats.*—Table 36 gives the results of the faecal fat estimations done in 5 of the patients who showed the worst absorptive defects clinically. The series of cases is far too small to judge the effect of different therapies on the control of the diarrhoea, but it is seen that, by the time the patients were fit for discharge, the steatorrhoea had disappeared.

6. *SUMMARY.*

(1) In marked contrast to the observations made in the European Prison Camps of Belsen and Lamsdorf, diarrhoea and other signs of impaired absorption from the gastro-intestinal tract were rare in repatriated Indian prisoners by the time they reached India.

(2) A few (less than 5% of those who were sufficiently ill to require hospital treatment) did, however, complain of diarrhoea. The following conclusions are based on a study of these patients only, and are not, therefore, representative of all repatriated Indian prisoners.

(3) In such patients there was, on admission to hospital, an impairment of the glucose tolerance test characterised by (*a*) a low fasting blood sugar concentration, (*b*) a low rise in blood sugar concentration after 50 gm. glucose and (*c*) a positive difference between the 3-hour blood sugar concentration and the fasting level.

(4) By the time these patients were fit for discharge from hospital, the glucose tolerance test had returned to normal in all particulars.

(5) While the patients were receiving nicotinic acid there was a slight improvement in the glucose tolerance curve as judged by the height of the curve above the fasting level. Riboflavin, in the dosage used, had no effect. Nicotinic acid, together with liver extract, had a beneficial effect in all 5 cases tried.

(6) There was also, when the patients were admitted to hospital, an impairment of the fat tolerance as judged by both the height and the time of the rise in serum total fat and the time of the rise of serum cholesterol. There was also a low fasting serum cholesterol, but no change in the fasting serum total fat.

(7) After treatment in hospital the fat tolerance returned to normal.

(8) During the course of the fat tolerance test it was found that the serum inorganic phosphorus also rose. Neither the height nor the time of the rise observed in the returned prisoners with impaired fat absorption, was significantly different from that of a control group whose fat absorption was normal.

(9) There was a good agreement between the different tests of absorptive function both of carbohydrate and of fat.

(10) Most patients with diarrhoea also had steatorrhoea which improved during treatment in hospital.

Table 27

Fasting Serum Total Fat (Ether soluble matter) in mgm./100 cc.

Prisoners of War				Controls	
Patient No.	On Admission	On Discharge	Difference	Patient No.	Serum Fat
2	480	450	—30	N37	520
6	460	600	+140	N38	430
11	580	540	—40	N39	530
15	470	490	+20	N40	370
18	570	520	—50	N41	330
25	510	450	—60	N42	440
26	490	500	+10	N43	470
28	420	490	+70	N44	550
36	320	350	+30	N45	480
Mean	480	490	+10		460

Table 28

Fasting Serum Cholesterol (mgm./100 cc.)

Prisoners of War				Controls	
Patient No.	On Admission	On Discharge	Difference	Patient No.	Serum Cholesterol
2	109	111	+2	N37	122
6	87	180	+93	N38	93
11	160	157	—3	N39	117
15	142	163	+21	N40	91
18	88	128	+40	N41	75
25	106	152	+46	N42	94
26	66	122	+56	N43	109
28	69	108	+39	N44	109
36	68	100	+32	N45	98
Mean	99	136	+37		102

Table 29

Fat Tolerance Test. The Rise in Serum Total Fat (mgm./100 cc.) and the Difference between the Maximum Serum Fat Concentration in the Fasting Level.

Prisoners of War				Controls	
Patient No.	On Admission	On Discharge	Difference	Patient No.	Rise in Serum Fat
2	130	60	—70	N38	170
6	70	150	80	N39	30
11	100	160	60	N40	150
15	110	90	—20	N41	110
18	50	120	70	N42	140
25	20	90	70	N43	220
26	50	150	100	N44	50
28	30	90	60	N45	100
36	40	200	160		
Mean	66	123	57		121

Table 30

The Rise in Serum Cholesterol (mgm./100 cc.) i.e. the Difference between the Maximum Serum Cholesterol Concentration and the Fasting Level.

Prisoners of War				Controls	
Patient No.	On Admission	On Discharge	Difference	Patient No.	Rise in Serum Cholesterol
2	31	19	—12	N38	6
6	37	23	—14	N39	3
11	26	43	+17	N40	10
15	6	45	+39	N41	0
18	2	19	+17	N42	15
25	8	7	—1	N43	16
26	26	12	—14	N44	11
28	37	12	—25	N45	28
36	2	16	+14		
Mean	19	22	3		11

Table 31

Fat Tolerance Test. Time of Maximum Total Fat Concentration.

	No. Patients	No. at 2-hr. period	No. at 3-hr. period	No. at 4-hr. period
On Admission ..	9	0	4	5
On Discharge ..	9	1	8	0
Controls ..	8	4	4	0

Table 32

Fat Tolerance Test. Time of Maximum Serum Cholesterol Concentration.

	No. Patients	No. at 2-hr. period	No. at 3-hr. period	No. at 4-hr. period
On Admission ..	9	0	4	5
On Discharge ..	9	6	2	1
Controls ..	8	5	1	2

Table 33

Effect of Treatment in Hospital on the Fat Tolerance Test as judged by Different Criteria.

Criterion	No. Patients	No. Better	No. Worse	No. no change
Fasting Serum Total Fat ..	9	5	4	0
Fasting Serum Cholesterol ..	9	8	1	0
Rise in Serum Total Fat ..	9	7	2	0
Rise in Serum Cholesterol ..	9	4	5	0
Time of Maximum Serum Total Fat Concentration ..	9	6	0	3
Time of Maximum Serum Cholesterol Concentration ..	9	6	0	3

Table 34

Rise in Serum Inorganic Phosphorus (mgm./100 cc.) after a Fat Meal containing 22 gm. Fat.

Patient No.		Before Meal	After 2 hours	After 3 hours	After 4 hours	Rise in Inorganic Phosphorus
2	Prisoners of War ..	3.1	3.6	4.0	4.0	0.9
6		2.8	2.9	3.0	3.0	0.2
11		3.9	4.2	4.9	5.3	1.4
11		3.8	4.1	4.0	4.2	0.4
15		3.4	3.4	3.9	3.8	0.5
18		3.0	4.1	4.2	..	1.2
22		3.0	3.0	2.9	3.0	0
24		3.9	4.2	..	3.9	0.3
25		4.6	4.8	5.0	5.1	0.5
26		4.1	4.7	5.0	5.1	1.0
N37	Controls ..	3.7	4.7	4.9	5.1	1.4
N39		4.4	4.6	5.3	5.3	0.9
N40		4.3	4.4	4.6	5.0	0.7
N41		3.7	4.7	5.2	5.0	1.5
N42		3.7	4.1	4.7	5.0	1.3
N43		4.2	4.3	4.9	5.0	0.8
N44		3.5	3.9	4.4	4.6	1.1
N45		3.4	4.0	4.2	4.7	1.3
15	Phosphate Content of Meal Doubled. ..	4.2	4.4	4.9	5.1	0.9
15	Meal of 50 gm. glucose ..	4.3	4.1	3.7	..	—0.6
18		4.6	3.8	4.4	..	—0.8

Table 35

Comparison of Different Tests of Absorption from the Gastro-Intestinal Tract.

		Fat Tolerance Test			Carbohydrate Tolerance Test	
	Patient No. + = ++ = +++ =	Height of Rise in Serum Total Fat. 0.50 mgm/100cc 51–100 „ Above 100 „	Time of Rise in Serum Total Fat. 4-hr. period 3-hr. period 2-hr. period	Time of Rise in Serum Cholesterol. 4-hr. period 3-hr. period 2-hr. period	Height of Rise in Blood Sugar Concentration. 0–20 mgm/100cc 21–40 „ Above 40 „	3-hr. Blood Conc.—Fasting Level. above 5 mgm /100cc 5 to –5 „ below –5 „
On Discharge	25	+	+	+	+	++
	18	+	+	+	++	+
	26	+	+	+	++	+
	36	+	++	++	+	+
	28	+	++	++	+	+
	6	++	+	+	++	+
	11	++	++	++	++	++
	2	+++	+	+	++	++
	15	+++	++	++	++	++
Normal	22	+++	++	+++	+++	+++
	24	+++	+++	+++	+++	+++
On admission	25	++	+	+++	+++	++
	18	+++	++	+	++	+++
	26	++	++	++	++	++
	36	+++	++	++	+++	+++
	28	++	+++	+++	++	+++
	6	+++	++	+++	++	+++
	11	+++	++	++	+++	+++
	2	++	++	+++	+	++
	15	++	++	+++	+++	++

Table 36

Faecal Fats

	On Admission				On Discharge				Difference
Patient No.	Total Fat (gm/100 gm)	Split Fat (gm/100 gm)	Neutral Fat (gm/100 gm)	% Split	Total Fat (gm/100 gm)	Split Fat (gm/100 gm)	Neutral Fat (gm/100 gm)	% Split	Total Fat (gm/103 gm)
6	37.7	29.4	8.3	78	27.6	19.3	8.3	70	10.1
11	46.0	31.1	14.9	68	32.1	22.2	9.9	69	13.9
28	58.3	39.7	18.6	68	22.9	17.2	5.7	75	35.4
31	48.8	37.7	11.1	77	17.9	11.7	6.2	65	30.9
36	82.3	52.3	30.0	64	17.1	17.1	0	100	65.2

SECTION IX

CONCLUSIONS.

1. *Clinical Features.*—Clinically, the cases of marasmus studied fell into 5 main groups.

(*i*) *Those showing marked wasting but little evidence of specific vitamin deficiency.*—These cases had a low blood volume and low blood pressure, but improved very rapidly on a high calorie diet. The incidence of such cases was about 60% of the 2,000 patients examined.

(*ii*) *Those showing evidence of severe hypoproteinaemia with oedema and massive anasarca.*—These cases had a low plasma and blood volume and plasma protein concentration (especially the albumin fraction). They responded dramatically to transfusion of concentrated plasma, which was life-saving in the most severe cases. This was a small group representing about 1% of all cases received.

(*iii*) *Those showing syndromes considered to be due to deficiencies of vitamins of the B_2 group.*—

(*a*) *Riboflavin Deficiency.*—Characterised by:—Angular stomatitis and cheilosis; swelling and apical erosion of individual fungiform and filiform papillae, giving rise to an intensely sore, swollen, and bright red tongue; impaired gastric acid production and gastric motility.

(*b*) *Nicotinic Acid Deficiency*, of which the clinical features were:—Loss of appetite, abdominal distension after food, sometimes vomiting; a small glazed, pale mauve-coloured tongue, which was not very sore, but whose epithelium was markedly atrophic; tympanites and diarrhoea with stools which showed an abnormally high fat content; gastro-intestinal absorption, as demonstrated by the fat and glucose tolerance tests, was impaired.

Response to specific therapy was prompt and complete in each case. The incidence of such cases showing vitamin B_2 group deficiencies was approximately 10%.

(*iv*) *Those showing neurological syndromes.*—

(*a*) *Peripheral Neuritis* (*Beri-beri*).—This group contained a large number of cases, approximately 20% of the whole. They recovered fairly rapidly except where muscular contractures had developed.

(*b*) "*Captivity Cord Syndrome.*"—These cases frequently showed evidence of degeneration of the posterior columns, rarely of the pyramidal tracts and very occasionally of both. Such cases had a normal C.S.F., normal fractional test meal findings and no constant changes in the blood picture. They were not usually associated with the above-mentioned signs of nicotinic acid or riboflavin deficiency, or necessarily with gross wasting. A reasonable degree of recovery was seen in cases of the first type (posterior column involvement), but those showing spasticity (pyramidal tract involvement) did not improve during 3 months observation. Such cases formed approximately 2% of all repatriated prisoners who required hospital treatment.

(*c*) "*Captivity Amblyopia.*"—These were cases showing evidence of optic atrophy with localised retinal changes. This syndrome was sometimes, though not always, associated with syndromes (*a*) or (*b*). Perimetry showed marked concentric contraction of the fields of vision, but central scotomata could not be demonstrated. Considerable recovery occurred in all but the most severe cases. The incidence was about 9% of all cases examined.

(*v*) *Macrocytic (Nutritional) Anaemia Only.*—Conversely, among medical patients evacuated from the Forces of Occupation in the Burma-Malayan Area after cessation of hostilities, many examples of gross riboflavin and nicotinic acid deficiencies have been seen, approximately 20%. Among such patients, cases of grave macrocytic anaemia with no evidence of other nutritional defect, were commonly encountered. The incidence of this condition was about 5%.

2. *Haematological Investigation.*—The repatriated Indian prisoners had an anaemia, the characteristics of which were : high M.C.V., high M.C.H. and normal M.C.H.C. The anaemia was thus orthochromic and macrocytic. This is in contrast to the type of anaemia described in prison camps in Europe such as Belsen and Lamsdorf. The anaemia improved rapidly on treatment with T. C. F. liver preparation.

3. *General Biochemical Investigation.*—The prisoners also had a low serum protein concentration. This reduction was almost entirely in the albumin fraction, and hence the A/G ratio was also low. The degree of oedema was more closely related to the serum albumin than to the serum total protein concentration. In addition the serum calcium was decreased, in some cases extremely so, but there was no significant change in the serum inorganic phosphorus or serum phosphatase. There was no evidence of latent tetany. All other tests for vitamin deficiencies were negative. Such biochemical abnormalities as did occur, improved rapidly with treatment in hospital.

4. *Plasma Volume and Blood Volume Investigation.*—The average plasma volume of the prisoners was lower than that of the control series, but this was of doubtful significance. The plasma volume referred to unit body weight was, however, increased, and there was no change in the plasma volume referred to unit surface area, and a decrease when it was referred to unit body height. The blood volume was greatly reduced and it was also reduced when referred to unit body weight, unit surface area and unit body height. There was also a reduction in the total circulating haemoglobin, total circulating R.B.C. and total circulating plasma protein which was confined to the albumin fraction.

All the above findings returned to normal on treatment in hospital and, by observing the individual rates of improvement, the pattern of recovery has been reconstructed. This recovery process has been divided into three arbitrary stages which are described in the text in some detail. Stage I (0-4 weeks) is characterised by a rapid rise in plasma volume to normal and by a decrease in body weight and haemoglobin concentration. Stage II (2-12 weeks) is notable for the rapid rise in blood volume to normal and the rise in plasma volume to figures in excess of normal. In Stage III (8-16 weeks) normal figures were reached for each of the factors measured.

An important clinical point is that the haemoglobin concentration, and sometimes the plasma protein concentration often fell during Stage I. Although this was so, the increase in plasma and total circulating volume was so great that the total amount of circulating haemoglobin and plasma protein was increased. Therefore it does not necessarily follow that, because the haemoglobin concentration or serum protein concentration falls during the initial stages of treatment, the patient is necessarily not making satisfactory progress.

5. *The Effect of Transfusion in Protein Deficiency.*—When a plasma protein infusion was given to a patient with severe hypoproteinaemia only a small part of the infused protein was found in the circulation 24 hours later. The percentage of infused protein retained was related to the mean serum protein concentration and to the total circulating plasma protein.

When a blood transfusion was given to a patient suffering from extreme tropical macrocytic anaemia with apparently only slight evidence of protein deficiency, the haemoglobin of the infused blood remained in the circulation, but the plasma protein was no longer present 24 hours later and, in some cases, some of the plasma protein that was originally present in the circulation had also left it. On the other hand, when a blood transfusion was given to an anaemic patient who was

neither macrocytic nor hypoproteinaemic, both the haemoglobin and the plasma protein of the infused blood were retained in the circulation 24 hours later.

6. *Fractional Test Meal Investigation.*—Of 21 P.O.W. patients, 2 had a histamine resistant achlorhydria, 5 had no free acid after a gruel meal but responded to histamine, one had a hypochlorhydria, 3 had a delayed emptying time and 10 were normal. After treatment in hospital all the patients produced free acid even without histamine; one had a hypochlorhydria, one had a delayed emptying time and the remaining 19 were normal.

Nicotinic acid had no effect on the fractional test meal findings, but there was a marked improvement while the patients were receiving riboflavin. There was also an improvement in the few cases studied, while the patient was receiving liver extract.

7. *Tests of Absorption from the Gastro-Intestinal Tract.*—In marked contrast to the observations made in the European Prison Camps of Belsen and Lamsdorf, diarrhoea and other signs of impaired absorption from the gastro-intestinal tract were rare in Indian prisoners by the time they had been repatriated to India. A few (less than 5% of those who were sufficiently ill to require hospital treatment) did, however, complain of diarrhoea. The following conclusions are based on a study of these patients only and are not, therefore, representative of all repatriated Indian prisoners. In such patients there was an impairment of the glucose tolerance test characterised by (*a*) a low fasting blood sugar concentration, (*b*) a low rise in blood sugar concentration after 50 gm. glucose and (*c*) a positive difference between the 3-hour blood sugar concentration and the fasting level. By the time the patients were fit for discharge from hospital, the glucose tolerance test had returned to normal in all respects.

While the patients were receiving nicotinic acid, there was a slight improvement in the glucose tolerance curve as judged by the height of curve above the fasting level. Riboflavin, in the dosage used, had no effect. Nicotinic acid, together with liver extract, had a beneficial effect in all 5 cases tried.

In addition there was an impairment of the fat tolerance as judged by both the height and the time of the rise in serum total fat and the time of the rise of serum cholesterol. There was also a low fasting serum cholesterol, but no change in the fasting serum total fat. After treatment in hospital the fat tolerance returned to normal. Most patients with diarrhoea also had steatorrhoea which improved during treatment in hospital.

SECTION X

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SECTION XI

APPENDIX.

Table 27

Haematological Findings of Patients on Admission.

Patient No.	Hb. Concentration (gm/100 cc)	R.B.C. Concentration (10^6 cells /cmm)	Haematocrit (%)	M.C.V. (Cμ)	M.C.H. ($\gamma\gamma$)	M.C.H.C. (%)
1	14.4	3.59	44.0	122.5	40.4	32.8
2	8.8	2.20	30.0	136.2	40.0	29.3
3	10.7	2.40	32.0	133.0	44.6	33.4
4	13.4	3.15	40.0	127.0	42.4	33.4
5	10.8	2.48	33.0	133.0	43.4	32.6
6	7.5	2.05	25.0	122.0	36.6	30.0
7	12.6	2.25	34.0	151.0	56.0	37.1
10	8.9	2.46	28.5	116.0	35.9	31.1
11	11.1	2.36	31.5	133.5	47.1	35.3
15	9.1	1.66	25.5	154.0	54.8	35.7
16	14.0	3.22	38.5	119.8	43.5	36.4
17	12.0	3.66	33.9	92.8	32.8	35.4
18	11.5	3.55	36.0	101.0	32.4	32.0
19	14.5	4.26	44.3	103.5	34.0	32.7
20	8.5	2.26	25.3	112.0	37.6	33.6
21	12.8	3.41	38.0	111.0	37.8	33.7
22	10.6	3.01	31.0	103.0	35.2	34.2
23	14.0	4.08	39.0	95.5	34.3	35.9
24	8.8	2.85	29.8	104.5	30.9	29.5
25	9.3	3.08	32.5	105.5	30.2	28.6
26	11.0	3.01	33.5	111.2	36.5	32.8
28	11.2	2.42	30.5	126.0	46.3	36.8
31	9.5	2.10	28.8	137.1	45.2	33.0
32	5.5	1.96	19.0	97.0	28.1	28.9
34	9.5	2.64	30.0	115.5	36.0	31.1
36	7.8	2.21	26.0	117.8	35.3	30.0
35	6.5	1.60	21.1	132.0	40.6	30.8

Table 28

Haematological Findings of Patients on Discharge.

Patient No.	Hb. Concentration (gm/100 cc)	R.B.C. Concentration 10^6 cells /cmm)	Haematocrit (%)	M.C.V. (Cμ)	M.C.H. ($\gamma\gamma$)	M.C.H.C. (%)
1	16.7	5.65	48.6	86.3	29.5	34.3
2	12.1	3.75	37.8	100.9	32.3	32.0
3	16.3	5.45	49.0	90.0	29.9	33.3
4	13.2	5.38	41.5	77.3	24.6	31.9
5	13.0	4.28	40.0	93.5	30.4	32.5
6	11.5	3.80	36.5	96.0	30.3	31.5
7	13.0	4.20	40.0	95.2	31.0	32.5
10	13.2	4.00	41.0	102.5	33.0	32.2
11	13.1	3.63	39.2	108.0	36.1	33.5
15	15.5	5.26	44.5	84.8	29.6	34.9
16	16.9	5.45	50.0	91.8	31.0	33.8
17	13.5	4.95	41.5	83.9	27.3	32.5
18	13.8	4.64	41.6	90.0	29.9	33.2
19	17.8	6.15	54.0	87.8	28.9	32.9
20	11.5	3.67	37.0	100.9	31.4	31.1
21	15.0	4.74	46.2	97.5	31.7	32.5
22	15.0	5.16	46.0	89.2	29.1	32.6
23	17.3	5.66	50.2	88.8	31.0	34.4
24	12.5	4.55	40.0	88.0	27.5	31.3
25	14.6	5.30	50.0	94.4	27.6	29.2
26	14.0	4.43	42.5	96.0	31.6	32.9
28	14.0	3.79	40.0	105.6	36.9	35.0
31	13.7	3.90	38.0	97.5	35.1	36.1
32	15.4	5.19	42.9	82.7	29.8	36.0
34	13.8	4.23	43.5	102.8	32.7	31.7
35	15.4	4.38	47.0	107.2	35.2	32.7
36	13.1	3.64	39.3	108.0	36.0	33.3

Table 29

Haematological Findings. Difference between Findings on Discharge and on Admission.

Patient No.	Hb. Concentration (gm/100cc)	R.B.C. Concentration (10^6 cells/cmm)	Haemotocrit %	M.C.V. (Cμ)	M.C.H. ($\gamma\gamma$)	M.C.H.C. (%)
1	+ 2.3	+2.06	+ 4.6	—36.2	—10.9	+1.5
2	+ 3.3	+1.55	+ 7.8	—35.3	—7.7	+2.7
3	+ 5.6	+3.05	+17.0	—43.0	—14.7	—0.1
4	— 0.2	+2.23	+ 1.5	—49.7	—17.8	—1.5
5	+ 2.2	+1.80	+ 7.0	—39.5	—13.0	—0.1
6	+ 4.0	+1.75	+11.5	—26.0	—6.3	+1.5
7	+ 0.4	+1.95	+ 6.0	—55.8	—25.0	—4.6
10	+ 4.3	+1.54	+12.5	—13.5	—2.9	+1.1
11	+ 2.0	+1.27	+ 7.7	—25.5	—11.0	—1.8
15	+ 6.4	+3.60	+19.0	—69.2	—25.2	—0.8
16	+ 2.9	+2.23	+11.5	—28.0	—12.5	—2.6
17	+ 1.5	+1.29	+ 7.6	—8.9	—5.5	—2.9
18	+ 2.3	+1.09	+ 5.6	—11.0	—2.5	+1.2
19	+ 3.3	+1.89	+ 9.7	—15.7	—5.1	+0.2
20	+ 3.0	+1.41	+11.7	—11.1	—6.2	—2.5
21	+ 2.2	+1.33	+ 8.2	—13.5	—6.1	—1.2
22	+ 4.4	+2.15	+15.0	—13.8	—6.1	—1.6
23	+ 3.3	+1.58	+11.2	—6.7	—3.3	—1.5
24	+ 3.7	+1.70	+16.2	—16.5	—3.4	+1.8
25	+ 5.3	+2.22	+17.5	—11.1	—2.6	+0.6
26	+ 3.0	+1.42	+ 9.0	—15.2	—4.9	+0.1
28	+ 2.8	+1.37	+ 9.5	—20.4	—9.4	—1.8
31	+ 4.2	+1.80	+ 9.2	—39.6	—10.1	+3.1
32	+ 9.9	+3.23	+23.9	—14.3	+1.7	+7.1
34	+ 4.3	+1.59	+13.0	—12.7	—3.3	+0.6
35	+ 8.9	+2.78	+25.9	—24.8	—5.4	+1.9
36	+ 5.3	+2.43	+13.3	—9.8	+0.7	+3.3

Table 30

Follow-up Haematological Data on Patients whose Recovery was not Complicated by Transfusion.

Patient No.	Date	Day	Hb. Concentration (gm/100 cc)	R.B.C. Concentration (10^6 cells/cmm)	Haematocrit (%)	M.C.V. ($C\mu$)	M.C.H. ($\gamma\gamma$)	M.C.H.C. (%)
1	17. 9.45		14.4	3.59	44.0	122.5	40.4	32.8
	12.10.45	25	12.6	3.90	38.5	99.0	32.4	32.8
	17.11.45	61	15.4	5.20	47.0	90.4	29.6	32.7
	6.12.45	80	16.5	4.84	48.7	100.5	34.1	33.8
	2. 1.46	107	16.7	5.65	48.6	86.3	29.5	34.3
3	17. 9.45		10.7	2.40	32.0	133.0	44.6	33.4
	12.10.45	25	12.8	3.50	38.3	109.0	36.6	33.4
	17.11.45	61	14.6	4.25	43.9	103.2	34.4	33.3
	19.12.45	93	16.2	4.91	46.0	93.7	33.0	35.2
	2. 1.46	107	16.3	5.45	49.0	90.0	29.9	33.3
4	17. 9.45		13.35	3.15	40.0	127.0	42.4	33.4
	11.10.45	24	14.0	3.87	42.0	108.5	36.2	33.4
	19.10.45	32	13.9	4.23	42.8	101.0	32.9	32.5
	14.11.45	58	12.8	5.15	40.0	77.7	24.5	32.0
	10.12.45	84	13.2	5.40	42.0	77.8	24.5	31.4
	22.12.45	96	13.2	5.38	41.5	77.3	24.6	31.9
5	18. 9.45		10.75	2.48	33.0	133.0	43.4	32.5
	27. 9.45	9	10.25	2.17	32.0	147.5	47.4	32.1
	12.10.45	24	10.4	2.92	32.0	109.6	35.5	32.5
	1.11.45	44	12.5	3.50	38.2	109.0	35.7	32.7
	22.11.45	65	13.0	4.28	40.0	93.5	30.4	32.5
7	18. 9.45		12.6	2.25	34.0	151.0	56.0	37.1
	14.10.45	26	12.6	3.225	34.0	105.0	39.1	37.1
	16.10.45	28	12.5	3.17	36.0	113.5	39.5	34.7
	13.11.45	56	13.0	4.20	40.0	95.2	31.0	32.5
10	22. 9.45		8.85	2.46	28.5	116.0	35.9	31.1
	13.10.45	21	10.5	3.475	33.8	97.5	30.3	31.1
	25.10.45	33	10.5	3.85	34.1	95.3	29.3	30.8
	14.11.45	53	13.2	4.00	41.0	102.5	33.0	32.2
11	22. 9.45		11.1	2.36	31.5	133.5	47.1	35.3
	14.10.45	22	11.5	2.62	32.5	124.0	44.0	35.4
	27.10.45	35	12.4	3.025	35.8	118.0	41.0	34.7
	14.11.45	53	12.6	3.50	38.0	108.5	36.0	33.2
	24.11.45	63	13.1	3.63	39.2	108.0	36.1	33.5
15	26. 9.45		9.1	1.66	25.5	154.0	54.8	35.7
	17.10.45	21	10.3	2.13	29.0	136.0	48.4	35.5
	20.11.45	55	10.8	2.96	33.5	113.5	36.5	32.5
	19.12.45	84	12.2	3.87	36.5	94.4	31.5	33.5
	15. 1.46	111	15.5	5.25	44.5	84.8	29.6	34.9
16	2[illegible]. 9.45		14.0	3.22	38.5	119.8	43.5	36.4
	17.10.45	20	14.5	4.10	42.5	103.5	35.4	34.2
	28.11.45	62	16.9	5.45	50.0	91.8	31.0	33.8
17	27. 9.45		12.0	3.66	33.9	92.8	32.8	35.4
	17.10.45	20	11.5	3.34	36.0	107.9	34.4	32.0
	28.11.45	62	13.5	4.95	41.5	83.9	27.3	32.5
18	30. 9.45		11.5	3.55	36.0	101.0	32.4	32.0
	18.10.45	18	10.5	3.36	35.5	105.6	31.3	29.6
	21.11.45	52	12.2	3.51	37.9	108.0	34.8	32.2
	20.12.45	81	13.8	4.64	41.6	90.0	29.9	33.2

Table 30—*concld.*

Patient No.	Date	Day	Hb. Concentration (gm/100 cc)	R.B.C. Concentration (10^6 cells/cmm)	Haematocrit (%)	M.C.V. ($C\mu$)	M.C.H. ($\gamma\gamma$)	M.C.H.C. (%)
19	30. 9.45		14.5	4.26	44.3	103.5	34.0	32.7
	18.10.45	18	14.7	4.28	44.0	103.0	34.4	33.4
	26.11.45	57	16.0	5.82	48.0	82.5	27.5	33.3
	15. 1.46	107	17.8	6.15	54.0	87.8	28.9	32.9
22	5.10.45		10.6	3.01	31.0	103.0	35.3	34.2
	22.10.45	17	11.3	2.81	34.9	124.0	40.3	32.4
	23.11.45	47	13.0	3.80	39.0	102.6	34.2	33.3
	20.12.45	74	15.0	5.16	46.0	89.2	29.1	32.6
25	25.10.45		9.3	3.08	32.5	105.5	30.2	28.6
	23.11.45	29	11.2	4.00	36.5	91.2	28.0	30.7
	14.12.45	50	11.8	4.28	40.5	94.6	27.6	29.1
	31.12.45	67	14.2	5.00	46.5	93.0	28.5	30.6
	5.1.46	72	14.6	5.30	50.0	94.4	27.6	29.2
26	26.10.45		11.0	3.01	33.5	111.2	36.5	32.8
	20.11.45	25	11.6	3.50	37.2	104.5	33.1	31.2
	14.12.45	49	13.0	3.90	38.5	98.8	33.3	33.8
	31.12.45	66	14.0	4.39	41.6	95.0	32.0	33.6
	5.1.46	71	14.0	4.43	42.5	96.0	31.6	32.9
28	1.11.45		11.2	2.42	30.5	126.0	46.3	36.8
	26.11.45	25	10.4	2.69	29.5	109.9	38.7	35.2
	21.12.45	50	9.9	2.55	29.0	113.8	38.8	34.1
	15. 1.46	75	11.6	3.20	31.8	99.4	36.3	36.5
	28. 1.46	88	12.9	3.59	39.7	110.6	35.8	32.5
	11. 2.46	102	14.0	3.79	40.0	105.6	36.9	35.0
31	11.11.45		9.5	2.10	28.8	137.1	45.2	33.0
	5.12.45	24	8.4	1.87	25.8	137.9	45.0	32.5
	3.1.46	53	12.6	3.62	41.5	114.6	34.8	30.4
	1.2.46	82	13.0	3.86	39.1	101.2	33.6	33.2
	12. 2.46	93	13.7	3.90	38.0	97.5	35.1	36.1
32	11.11.45		5.5	1.96	19.0	97.0	28.1	28.9
	14.12.45	31	11.1	3.65	39.0	106.9	30.4	28.5
	3.1.46	53	12.6	4.30	40.5	94.2	29.4	31.1
	1.2.46	82	15.4	5.19	42.9	82.7	29.8	36.0
34	21.11.45		9.5	2.64	30.5	115.5	36.0	31.1
	12.12.45	21	12.2	3.40	38.2	112.2	35.9	31.9
	4.1.46	44	13.8	4.23	43.5	102.8	32.7	31.7
35	22.11.45		6.5	1.60	21.1	132.0	40.6	30.8
	12.12.45	21	9.5	2.30	32.0	139.0	41.3	29.7
	4.1.46	44	15.4	4.38	47.0	107.2	35.2	32.7
36	26.11.45		7.8	2.21	26.0	117.8	35.3	30.0
	18.12.45	22	8.7	2.67	30.3	113.5	32.6	28.8
	13. 1.46	48	10.3	2.85	33.0	116.0	36.2	31.1
	11. 2.46	77	13.1	3.64	39.3	108.0	36.0	33.3

Table 31

Biochemical Findings of Patients on Admission.

Patient No.	Serum Protein (gm/100cc)	Serum Albumin (gm/100cc)	Serum Globulin (gm/100cc)	Ratio A/G (X/I)	Serum Calcium (mgm/100cc)	Serum Inorganic Phosphorus (mgm/100cc)	Serum Phosphatase (King-Armstrong Unit)
1	6.53	4.21	2.32	1.8	5.1	..	30
2	4.05	1.01	3.04	0.3	5.9	..	21
3	4.81	2.44	2.37	1.0	7.6	..	24
4	5.49	3.38	2.11	1.6	10.9	4.5	8
5	4.18	2.00	2.18	0.9	6.6	1.8	22
6	4.20	1.46	2.74	0.5	7.8	2.5	..
7	4.46	2.40	2.06	1.1	7.8	4.1	..
10	7.03	2.58	4.45	0.6	10.4	5.0	23
11	5.47	3.22	2.25	1.4	8.5	3.0	6
15	4.78	1.20	3.58	0.6	8.8	3.4	28
16	6.32	3.96	2.36	1.7	9.2	4.1	10
17	6.52	3.79	2.73	1.4	9.7	5.6	11
18	5.34	2.10	3.24	0.3	9.0	5.4	20
19	6.82	2.95	3.87	0.8	9.5	2.4	10
20	6.50	2.10	4.40	0.5	4.4	2.6	..
21	6.75	3.23	3.52	0.9	9.6	4.1	12
22	5.23	1.67	3.56	0.5	8.1	3.2	21
23	5.46	2.82	2.64	1.1	10.6	4.5	6
24	2.88	0.75	2.13	0.3	8.1	4.1	11
25	5.17	2.48	2.69	0.9	8.8	4.0	11
26	5.10	1.98	3.12	0.7	8.2	4.1	35
28	5.48	2.80	2.68	1.0	8.5	3.9	6
31	5.30	2.75	2.55	1.1	9.0	4.4	11
32	6.33	3.44	2.89	1.2	9.2	4.4	18
34	5.24	3.75	1.49	2.5	9.7	3.3	15
35	5.30	4.07	1.23	3.3	10.0	5.6	8
36	5.13	2.56	2.57	1.0	9.5	3.2	5

Table 32

Biochemical Findings of Patients on Discharge.

Patient No.	Serum Protein (gm/100cc)	Serum Albumin (gm/100cc)	Serum Globulin (gm/100cc)	Ratio A/G (x/1)	Serum Calcium (mgm/100cc)	Serum Inorganic Phosphorus (mgm/100cc)	Serum Phosphatase (King Armstrong Unit)
1	6.84	4.22	2.62	1.6	11.0	..	64
2	7.74	4.86	2.88	1.7	10.7	..	22
3	6.50	4.25	2.35	1.9	11.4	..	32
4	6.78	4.04	2.74	1.5	11.3	3.4	18
5	7.17	2.86	4.31	0.7	10.5	5.8	24
6	7.25	3.62	3.62	1.0	9.0	4.5	..
7	6.26	4.32	1.94	2.2	8.5	4.3	..
10	6.91	3.80	3.11	1.2	10.4	5.0	9
11	6.50	3.74	2.74	1.4	9.8	4.1	16
15	7.35	4.36	3.05	1.4	11.4	4.1	23
16	6.44	4.26	2.18	2.0	10.1	3.7	9
17	7.19	4.49	2.70	1.7	10.4	4.2	11
18	6.84	3.80	3.04	1.3	10.5	4.9	21
19	6.53	4.56	1.97	2.3	11.3	3.5	10
20	7.84	3.13	4.71	0.7	9.6	3.6	..
21	6.50	4.64	1.86	2.5	11.7	4.1	13
22	7.12	3.39	3.73	0.9	10.5	5.2	19
23	6.16	4.29	1.87	2.2	11.2	3.7	13
24	7.53	4.42	3.11	1.4	11.7	5.2	10
25	6.43	4.28	2.15	2.0	11.1	4.5	10
26	6.66	4.45	2.21	2.1	11.1	5.1	26
28	6.66	4.19	2.47	1.7	11.7	4.0	14
31	6.56	3.45	3.11	1.1	11.6	4.2	37
32	7.46	4.96	2.50	2.0	11.1	4.1	14
34	5.98	4.13	1.85	2.2	11.1	4.3	25
35	7.05	4.70	2.35	2.6	11.1	5.3	26
36	6.26	3.87	2.39	1.6	11.0	4.3	13

Table 33

Biochemical Findings. Difference between Patients on Discharge and on Admission

Patient No.	Serum Protein (gm/100cc)	Serum Albumin (gm/100cc)	Serum Globulin (gm/100cc)	Ratio A/G (X/1)	Serum Calcium (mgm/100cc)	Serum Inorganic Phosphorus (mgm/100cc)	Serum Phosphatase (King Armstrong Units)
1	0.31	0.01	+0.30	—0.2	+ 5.9	..	+34
2	3.69	3.85	—0.16	+1.4	+ 4.8	..	—1
3	1.69	1.81	—0.12	+0.9	+ 3.8	..	+8
4	1.29	0.66	+0.63	—0.1	+ 0.4	—1.1	+10
5	2.99	0.86	+2.11	—0.2	+ 3.9	+4.0	+2
6	3.05	2.16	+0.88	+0.5	+ 1.2	+2.0	..
7	1.80	1.92	—0.12	+1.1	+ 0.7	+0.2	..
10	—0.12	1.22	—1.34	+0.6	0.00	0.00	—14
11	1.03	0.52	+0.49	0.00	+ 1.3	+1.1	+10
15	2.57	3.10	—0.53	+1.1	+ 2.6	+0.7	—5
16	0.12	0.30	—0.18	+0.3	+ 0.9	—0.4	—1
17	0.67	0.70	—0.03	+0.3	+ 0.7	—1.4	0.00
18	1.50	1.70	—0.20	+0.7	+ 1.5	—0.5	+1
19	—0.29	1.61	—1.90	+1.5	+ 1.8	+1.1	0.00
20	1.34	1.03	+0.31	+0.2	+ 5.2	+1.0	..
21	—0.25	1.41	—1.66	+1.6	+ 2.1	0.00	+1
22	1.89	1.72	+0.17	+0.4	+ 2.4	+2.4	—2
23	0.70	1.47	—0.77	+1.1	+ 0.6	—0.8	+7
24	4.65	3.67	+0.98	+1.1	+ 3.6	—1.1	—1
25	1.26	1.80	—0.54	+1.1	+ 2.3	+0.5	—1
26	1.56	2.47	—0.91	+1.4	+ 2.9	+1.0	—9
28	1.18	1.39	—0.21	+0.7	+ 3.2	+0.1	+8
31	1.26	0.70	+0.56	0.00	+ 2.6	—0.2	+26
32	1.13	1.52	—0.39	+0.8	+ 1.9	—0.3	—4
34	0.74	0.38	+0.36	—0.3	+ 1.4	+1.0	+10
35	1.75	0.63	+1.12	—0.7	+ 1.1	0.3	+18
36	1.13	1.31	—0.17	+0.6	+ 1.5	+1.1	+8

Table 34

Control Series: Biochemical Findings.

Patient No.	Serum Calcium (mgm/100 cc)	Serum Inorganic Phosphorus (mgm/100 cc)	Serum Phosphatase (King Armstrong Units)
N 37	11.3	4.0	18
N 38	11.7	3.9	17
N 39	11 5	4.3	24
N 40	11.7	4.1	15
N 41	11.9	3.7	14
N 42	11.2	4.1	16
N 43	11.3	4.4	16
N 44	10.3	3.5	9
N 45	11.9	4.2	12

Table 35

Follow-up Biochemical Data on Patients whose Recovery was not complicated by Transfusion.

Patient No.	Date.	Day.	Serum Protein (gm/100 cc)	Serum Albumin (gm/100 cc)	Serum Globulin (gm/100 cc)	Ratio A/G (X/I)	Serum Calcium (mgm/ 100cc)	Serum Inorgan Phos-phorus (mgm 100cc)	Serum Phospha-tase (King Armstrong units)
1	17. 9.45		6.53	4.21	2.32	1.8	5.1	..	30
	29.10.45	42	6.40	2.72	3.68	0.7	10.2	4.8	..
	17.11.45	61	6.70	3.19	3.51	0.9	9.9	4.4	44
	6.12.45	80	6.66	3.92	2.74	1.4	10.7	4.9	53
	2.1.46	107	6.84	4.22	2.62	1.6	11.0	4.5	64
3	17. 9.45		4.81	2.44	2.37	1.0	7.6	..	24
	29.10.45	42	6.25	3.31	2.94	1.1	10.6	5.4	..
	17.11.45	61	5.82	3.07	2.75	1.1	9.9	5.2	22
	19.12.45	93	6.13	4.38	1.75	2.5	11.8	4.4	..
	2. 1.46	107	6.50	4.25	2.25	1.9	11.4	4.6	23
									8
4	17. 9.45		5.49	3.38	2.11	1.6	..	..	..
	29.10.45	42	6.67	3.00	3.67	0.8	10.9	4.5	17
	14.11.45	58	6.40	3.66	2.74	1.5	10.6	3.9	18
	10.12.45	84	7.35	5.25	2.10	2.5	11.6	3.8	..
	22.12.45	96	6.78	4.04	2.74	1.5	11.3	3.4	
5	18 9.45		4.18	2.00	2.18	0.9	6.6	1.8	..
	1.11.45	44	7.18	4.65	2.53	1.9	11.0	6.1	22
	22.11.45	65	6.84	2.21	4.63	0.5	10.5	5.8	24
7	18. 9.45		4.46	2.40	2.06	1.1	7.8	4.1	10
	16.10.45	28	5.94	3.39	2.55	1.3	..	..	..
	5.11.45	48	6.19	3.75	2.44	1.6	8.5	4.3	..
	13.11.45	56	6.26	4.32	1.94	2.2	..	..	..
10	22. 9.45		7.03	2.58	4.45	0.6	..	..	23
	25.10.45	33	6.84	3.00	3.84	0.8	..	..	..
	5.11.45	44	7.23	3.33	3.90	0.9	10.4	5.0	..
	14.11.45	53	6.91	3.80	3.11	1.2	10.4	4.1	9
11	22.9 .45		5.47	3.22	2.25	1.4	8.5	3.0	6
	6.10.45	14	5.30	..	..	..	..	3.7	..
	27.10.45	35	6.50	3.76	2.74	1.4	10.9	..	..
	14.11.45	53	6.50	3.76	2.74	1.4	9.8	4.1	16
15	25. 9.45		4.78	1.20	3.58	0.6	8.8	3.4	28
	5.10.45	9	4.89	..	..	..	..	3.4	..
	5.11.45	40	7.19	2.25	4.94	0.5	9.8	4.6	..
	30.11.45	55	7.25	3.15	4.10	0.8	10.1	4.9	25
	19.12.45	84	6.84	3.56	3.28	1.1	11.6	4.0	25
	15 .1.46	111	7.35	4.30	3.05	1.4	11.4	4.1	23
16	28. 9.45		6.32	3.96	2.36	1.7	9.2	4.1	10
	5.11.45	39	6.16	3.98	2.18	1.8	11.2	4.9	..
	28.11.45	62	6.44	4.26	2.18	2.0	10.1	3.7	9

Table 35—*concld.*

Patient No.	Date	Day	Serum Protein (gm/100 cc)	Serum Albumin (gm/100 cc)	Serum Globulin (gm/100 cc)	Ratio A/G (X/I)	Serum Calcium (mgm/ 100cc)	Inorganic Phosphorms (mgm/ 100cc)	Serum Phosphatase (King Armstrong Units)
17	26. 9.45		6.52	3.79	2.73	1.4	9.7	5.6	11
	6.11.45	40	6.52	2.89	3.63	0.8	11.9	4 6	..
	28.11.45	62	7.19	4.49	2.70	1.7	10.4	4.2	11
18	29. 9.45		5.34	2.10	3.24	0.7	9.0	5.4	20
	6.11.45	37	6.67	2.38	4.29	0.6	10.5	3.5	..
	30.11.45	51	5.50	3.61	2.89	1.2	9.8	4.6	21
	30.12.45	81	6.84	3.80	3.04	1.3	10.7	4.6	21
19	30. 9.45		6.82	2.95	3.87	0.8	9.5	2.4	10
	6.11.45	37	6.44	3.82	2.62	1.5	10.6	3.5	..
	27.11.45	58	6.33	3.72	2.61	1.4	10.6	3.8	9
	15. 1.45	107	6.53	4.56	1.97	2.3	11.3	3.5	10
22	4.10.45		5.23	1.67	3.56	0.5	8.1	3.2	21
	6.11.45	30	6.80	2.78	4.02	0.7	10.9	4.3	..
	23.11.45	47	7.53	3.40	4.13	0.7	11.0	4.8	19
	20.12.45	74	7.12	3.39	3.73	0.9	10.5	5.2	..
25	25.10.45		5.17	2.48	2.69	0.9	8.8	4.0	..
	9.11.45	15	5.82	3.89	1.93	2.0	10.7	4.6	..
	23.11.45	29	5.85	3.85	2.00	1.9	10.8	4.5	11
	14.12.45	50	6.16	4.11	2.05	2.0	11.9	4.6	..
	31.12.45	67	6.33	4.07	2.36	1.8	11.1	4.5	10
	5.1.46	72	6.43	4.28	2.15	2.0	..	..	..
26	26.10.45		5.10	1.98	3.12	0.7	8.2	4.1	..
	9.11.45	14	5.67	2.58	3.09	0.8	10.6	4.3	..
	20.11.45	25	6.50	4.64	1.86	2.5	9.8	5.9	35
	14.12.45	49	6.16	4.47	1.69	2.6	12.3	5.2	..
	31.12.45	66	6.44	3.30	3.14	1.1	11.1	5.1	26
	5.1.46	71	6.66	4.45	2.21	2.0	..	..	..
28	1.11.45		5.48	2.80	2.68	1.0	8.5	3.9	..
	26.11.45	25	5.23	3.15	2.08	1.5	9.5	3.3	6
	21.12.45	50	5.88	..	..	..	9.7	3.8	..
	15. 1.46	75	6.33	4.43	1.90	2.3	9.9	3.9	14
	28. 1.46	88	6.68	3.92	2.76	1.4	..	..	..
	11. 2.46	102	6.66	4.19	2.47	1.7	11.7	4.0	..
31	11.11.45		5.30	2.75	2.55	1.1	9.0	4.4	..
	5.12.45	24	4.83	2.95	1.88	1.6	9.1	3.3	11
	3.1.46	53	6.16	3.04	3.12	1.0	9.9	4.4	37
	1.2.46	82	6.33	3.90	2.43	1.6	..	..	..
	12. 2.46	93	6.56	3.45	3.11	1.1	11.6	4.2	..
32	11.11.45		6.33	3.44	2.89	1.2	9.2	4.4	..
	14.12.45	31	7.10	4.86	2.24	2.2	12.7	4.2	18
	3.1.46	53	6.84	3.69	3.15	1.1	11.1	4.1	14
	1.2.46	82	7.46	4.96	2.50	2.1	..	..	..
34	21.11.45		5.24	3.75	1.49	2.5	9.7	3.3	15
	12.12.45	21	6.34	4.50		2.4	10.6	4.4	..
	4.1.46	44	5.98	4.13	1.85	2.2	11.1	4.3	25
35	22.11.45		5.30	4.07	1.23	3.3	10.0	5.6	8
	12.12.45	21	6.34	4.70	1.64	2.9	10.2	4.5	..
	4.1.46	44	7.05	4.70	2.35	2.0	11.1	5.2	26
36	26.11.45		5.13	2.56	2.57	1.0	9.5	3.2	5
	18.12.45	22	4.62	1.81	2.81	0.7	10.1	3.4	..
	13. 1.46	48	5.81	3.93	1.88	2.1	10.7	4.9	13
	11. 2.46	77	6.26	3.87	2.39	1.6	11.0	4.3	..

Table 36

Control Series: Haematological Findings.

Patient No.	Hb. Concentration (gm/100cc)	R.B.C. Concentration (10^6 cells/cmm)	Haematocrit (%)	M.C.V. (Cμ)	M.C.H. ($\gamma\gamma$)	M.C.H.C. (%)
N 37	17.3	5.515	49.1	89.0	31.3	35.2
N 38	15.3	5.625	48.0	85.5	27.2	31.9
N 39	13.4	4.715	40.0	84.8	28.4	33.5
N 40	14.0	4.91	41.9	85.4	28.5	33.4
N 41	16.7	5.44	50.0	92.0	30.7	33.4
N 42	17.7	5.55	51.1	92.0	31.8	34.6
N 43*	11.3	6.18	41.0	66.4	18.3	27.5
N 44	16.0	4.70	45.0	95.7	34.1	35.6
N 45	15.5	4.85	43.0	88.6	31.9	36.0

*This patient was obviously microcytic and hypochromic (confirmed by blood film and repeat count on another blood sample). He was otherwise healthy and therefore there seems no justification for discarding him from the "Control" series.

Table 37

Control Series : Plasma Protein and Physical Measurement Findings

Patient No.	Serum Protein (gm/100cc)	Serum Albumin (gm/100cc)	Serum Globulin (gm/100cc)	Ratio A/G (X/1)	Body Weight (kgm)	Height (cm)	Area (sq.m.)
N37	7.05	4.64	2.41	1.9	63.1	173	1.76
N38	6.50	4.33	2.17	2.0	56.8	168	1.63
N39	7.19	4.78	2.41	2.0	54.1	164	1.58
N40	6.67	3.90	2.77	1.4	52.8	168	1.59
N41	6.33	3.83	2.50	1.5	49.1	152	1.43
N42	7.11	4.74	2.37	2.0	52.3	169	1.59
N43	6.84	5.26	1.58	3.3	64.6	168	1.73
N44	6.84	4.58	2.26	2.0	49.6	161	1.50
N45	7.52	5.29	2.23	2.3	50.9	159	1.50

Table 38

Control Series: Plasma Volume and Blood Volume Findings.

Patient No.	Plasma Volume (cc)	Plasma-vol. per kgm (cc/kgm)	Plasma vol. per sq.m. (cc/sq.m)	Plasma Vol.per cm (cc/cm)	Blood Volume (cc)	Blood Vol. per kgm (cc/kgm)	Blood Vol. per sq.m (cc/sq.m)	Blood Vol. per cm (cc/cm)
N37	2830	44.6	1610	16.3	5570	88.0	3160	32.1
N38	2610	46.0	1600	15.5	5010	88.5	3080	29.9
N39	2640	48.8	1670	16.1	4400	81.1	2780	26.8
N40	2580	48.9	1630	15.3	4450	84.4	2800	26.5
N41	2180	44.4	1520	14.3	4360	88.8	3050	28.7
N42	2330	44.5	1470	13.8	4770	91.0	3000	28.2
N43	3000	46.6	1730	17.8	5100	78.9	2940	30.4
N44	2550	51.3	1700	15.8	4640	93.6	3090	28.8
N45	2680	52.0	1790	16.8	4710	92.8	3140	29.6

Table 39

Control Series: Total Circulating Haemoglobin Findings.

Patient No.	Hb. Concentration (gm/100 cc)	Total Circulating Hb. (gm)	Total circ. Hb. per kgm (gm/kgm)	Total circ Hb. per sq. m (gm/sq. m)	Total circ. Hb. per cm (gm/cm)
N37	17.3	962	15.2	548	5.5
N38	15.3	768	13.5	471	4.6
N39	13.4	590	10.9	373	3.6
N40	14.0	623	11.8	392	3.7
N41	16.7	729	14.8	510	4.8
N42	17.7	845	16.1	531	5.0
N43	11.3	576	8.9	333	3.4
N44	16.0	742	15.0	495	4.6
N45	15.5	731	14.4	488	4.6

Table 40

Control Series : Total Circulating Red Cell Findings.

Patient No.	R.B.C. Concentration (10^6xCells/mm)	Total Circulating R.B.C. (Cells x 10^{12})	Total Circ. R.B.C. per kgm (Cells x10^{10}/kgm)	Total Circ. R.B.C. per sq. m (Cells x 10^{12}/ sq.m)	Total Circ. R.B.C. per cm (Cells x 10^{10}/ cm)
N37	5.52	30.7	49	17.5	17.7
N38	5.63	28.3	50	17.3	16.8
N39	4.72	20 7	38	13.1	12.6
N40	4.91	21.9	42	13.8	13.0
N41	5.44	23.7	48	16.6	15.6
N42	5.55	26.4	50	16.6	15.6
N43	6.18	31.4	49	18.1	18.7
N44	4.70	21.8	44	14.5	13.6
N45	4.85	22.8	45	15.2	14.2

Table 41

Control Series : Total Circulating Plasma Protein Findings.

Patient No.	Plasma Protein concentration (Alb+glob) (gm./100cc)	Total Circulating Plasma Protein (Alb+ glob) (gm)	Total Circ. Plasma Protein per kgm (Alb+ glob) (gm/kgm)	Total Circ. Plasma Protein per sq. m (Alb+ glob) (gm/sq.m)	Total Circ. Plasma Protein per cm. (Alb+ glob) (gm/cm)
N37	7.05 (4.64+2.41)	200 (131+69)	3.2 (2.1+1.1)	114 (75+39)	1.16 (0.76+0.40)
N38	6.50 (4.33+2.17)	169 (113+56)	3.0 (2.0+1.0)	104 (69+35)	1.00 (0.67+0.33)
N39	7.19 (4.78+2.41)	190 (126+64)	3.5 (2.3+1.2)	120 (80+40)	1.16 (0.77+0.39)
N40	6.67 (3.90+2.77)	172 (101+71)	3.3 (1.9+1.4)	108 (64+44)	1.02 (0.60+0.42)
N41	6.33 (3.83+2.50)	138 (84+54)	2.8 (1.7+1.1)	97 (59+38)	0.91 (0.55+0.36)
N42	7.11 (4.74+2.37)	166 (110+56)	3.2 (2.1+1.1)	104 (69+35)	0.98 (0.65+0.33)
N43	6.84 (5.26+1.58)	206 (158+48)	3.2 (2.4+0.8)	119 (91+28)	1.23 (0.94+0.29)
N44	6.84 (4.58+2.26)	175 (117+58)	3.5 (2.3+1.2)	116 (78+38)	1.09 (0.73+0.36)
N45	7.52 (5.29+2.23)	202 (142+60)	4.0 (2.8+1.2)	134 (94+40)	1.28 (0.90+0.38)

Table 41

Blood Volume Investigation. Haematological Findings.

Patient No.	Date	Day	Hb. Concentration (gm/100 cc)	R.B.C. Concentration (10^6 Cells/cmm	Haematocrit (%)	M.C.V. (cμ)	M.C.H. ($\gamma\gamma$)	M.C.H.C. (%)
4	20.9.45		13.4	3.15	40.0	127.0	42.4	33.4
	10.12.45	81	13.2	5.40	42.0	77.8	24.5	31.4
5	27. 9.45		10.3	2.17	32.0	147.5	47.4	32.1
	22.11.46	57	13.0	4.28	40.0	93.5	30.4	32.5
7	21. 9.45		12.6	2.25	34.0	151.0	56.0	37.1
	16.10.45	25	12.5	3.17	36.0	113.5	39.5	34.7
	13.11.45	53	13.0	4.20	40.0	95.2	31.0	32.5
10	27. 9.45		8.9	2.46	28.5	116.0	35.9	31.1
	25.10.45	28	10.5	3.58	34.1	95.3	29.3	30.8
	14.11.45	48	13.2	4.00	41.0	102.5	33.0	32.2
24	10.10.45		8.8	2.85	29.8	104.6	38.9	29.5
	25.10.45	15	7.6	2.64	27.5	104.0	28.8	27.6
	12.11.45	33	10.0	3.14	35.0	111.5	28.6	31.7
	13.12.45	64	12.5	4.55	40.0	88.0	27.5	31.3
25	24.10.45		9.3	3.08	32.5	106.0	30.3	28.7
	23.11.45	30	11.2	4.00	36.5	91.2	28.0	30.7
	14.12.45	51	11.8	4.28	40.5	94.6	27.6	29.1
	5. 1.46	73	14.6	5.30	50.0	94.4	27.6	29.2
26	26.10.45		11.0	3.01	33.5	111.5	36.5	32.8
	20.11.45	25	11.6	3.56	37.2	104.5	33.1	31.2
	14.12.45	49	13.0	3.90	38.5	98.8	33.3	33.8
	5. 1.46	71	14.0	4.43	42.5	96.0	31.6	32.9
28	1.11.45		11.2	2.42	30.5	126.0	46.3	36.8
	26.11.45	25	10.4	2.69	29.5	109.9	38.7	35.2
	28.12.45	57	12.9	3.59	39.7	110.6	35.8	32.8
	11. 2.45	102	14.0	3.79	40.0	105.6	36.9	35.0
31	11.11.45		9.5	2.10	28.8	137.1	45.2	33.0
	5.12.45	24	8.4	1.87	25.8	137.9	45.2	32.5
	3. 1.46	53	12.6	3.62	41.5	114.6	34.0	30.4
	1. 2.46	82	13.0	3.86	39.1	101.2	33.6	33.2
	12. 2.46	93	13.7	3.90	38.0	97.5	35.1	36.1
32	11.11.45		5.5	1.96	19.0	97.0	28.1	28.9
	14.12.45	33	11.1	3.65	39.0	106.9	30.4	28.5
	3. 1.46	53	12.6	4.30	40.5	94.2	29.4	31.1
	1. 2.46	82	15.4	5.19	42.9	82.9	29.8	36.0
34	21.11.45		9.5	2.64	30.5	115.9	36.1	31.1
	12.12.45	21	12.2	3.40	38.5	112.2	35.9	31.9
	4. 1.46	44	13.8	4.23	43.5	102.8	32.7	31.7
	26.11.45		7.8	2.21	26.0	117.6	35.3	30.0
36	18.12.45	22	8.7	2.67	30.3	113.5	32.6	28.8
	13. 1.46	48	10.3	2.85	33.0	116.0	36.2	31.1
	11. 2.46	77	13.1	3.64	39.3	108.0	36.0	33.3

Table 43

Blood Volume Investigation : Plasma Protein and Physical Measurement Findings.

Patient No.	Date	Day	Serum protein (gm/100 cc)	Serum Albumin (gm/100 cc)	Serum Globulin (gm/100 cc)	Ratio A/G(X:1)	Body weight (kgm)	Height (cm)	Area (sqm)
4	20. 9.45		5.50	3.40	2.10	1.6	39.3	165	1.39
	10.12.45	81	7.35	5.25	2.10	2.5	59.1	165	1.65
5	27. 9.45		4.80	..	..	..	45.0	169	1.49
	22.11.45	57	6.84	2.21	4.63	0.5	60.7	169	1.70
	21. 9.45		4.46	2.40	2.06	1.2	50.1	174	1.60
7	16.10.45	25	5.94	3.39	2.55	1.3	55.1	174	1.67
	13.11.45	53	6.26	4.32	1.94	2.2	63.7	174	1.77
	27. 9.45		7.03	2.59	4.44	0.6	48.4	175	1.58
10	25.10.45	28	6.84	3.00	3.84	0.8	64.3	175	1.78
	14.11.45	48	6.91	3.80	3.11	1.2	69.0	175	1.83
	10.10.45		2.88	0.75	2.13	0.4	44.2	161	1.43
	25.10.45	15	5.44	2.09	3.35	0.6	40.5	161	1.38
24	12.11.45	33	6.76	4.18	2.58	1.6	47.7	161	1.48
	13.12.45	64	6.91	4.09	2.82	1.5	59.1	161	1.61
	11.10.45		5.17	2.48	2.69	0.9	46.8	170	1.52
25	23.11.45	30	5.85	3.85	2.00	1.9	55.3	170	1.64
	14.12.45	51	6.16	4.11	2.05	2.0	59.0	170	1.69
	5. 1.46	73	6.43	4.28	2.15	2.0	61.6	170	1.71
	20.10.45		5.10	1.98	3.12	0.7	42.6	166	1.43
	20.11.45	25	6.50	4.64	1.86	2.5	47.6	166	1.50
26	14.11.15	41	6.16	4.47	1.69	2.6	58.8	166	1.66
	5. 1.46	71	6.66	4.45	2.21	2.0	63.1	166	1.71
	1.11.45		5.48	2.80	2.68	1.0	51.0	180	1.65
	26.11.45	25	5.23	3.15	2.08	1.5	55.0	180	1.72
28	28.12.45	57	6.68	3.92	2.76	1.4	64.2	180	1.82
	11. 2.46	102	6.68	4.19	2.49	1.7	68.8	180	1.88
	11.11.45		5.30	2.75	2.55	1.1	50.6	173	1.59
	5.12.45	24	4.83	2.95	1.88	1.6	51.4	173	1.60
31	3. 1.46	53	6.16	3.04	3.12	1.0	51.7	173	1.61
	1. 2.46	82	6.33	3.90	2.43	1.6	61.0	173	1.73
	12. 2.46	93	6.56	3.45	3.11	1.1	64.1	173	1.77
	11.11.45		6.33	3.44	2.89	1.2	38.7	160	1.34
	14.12.45	33	7·10	4.86	2.24	2.2	43.5	160	1.42
32	3. 1.46	53	6.84	3.69	3.15	1.2	50.9	160	1.51
	1. 2.46	82	7.46	4.96	2.50	2.0	50.6	160	1.50
	21.11.45		5.24	3.75	1.49	2.5	42.9	160	1.41
34	12.12.45	21	6.34	4.50	1.84	2.4	46.1	160	1.45
	4. 1.46	44	5.98	4.13	1.85	2.2	52.2	160	1.52
	26.11.45		5.12	2.56	2.56	1.0	49.6	178	1.61
36	18.12.45	22	4.62	1.81	2.81	0.7	52.3	178	1.66
	13. 1.46	48	5.81	3.93	1.88	2.1	58.8	178	1.73
	11. 2.46	77	6.26	3.87	2.39	1.6	68.8	178	1.85

Table 44

Blood Volume Investigation: Plasma Volume and Blood Volume Findings.

Patient No.	Date	Days	Plasma Volume (cc)	Plasma Volume per kgm (cc/kgm)	Plasma Volume per sq.m. (cc/sq.m)	Plasma Volume per cm. (cc/cm)	Blood Volume (cc)	Blood Volume per/kgm (cc/kgm)	Blood Volume per sq.m (cc/sq-m)	Blood Volume per cm (cc/cm.)
4	20. 9.45		1960	50.0	1410	11.9	3270	83.2	2350	19.8
	10.12.45	81	2380	40.2	1440	14.4	4110	69.7	2490	25.0
5	27. 9.45		2280	64.0	1930	17.1	4240	94.0	2840	25.1
	22.11.45	57	2850	47.0	1670	16.9	4750	78.3	2790	28.1
7	21. 9.45		2160	43.1	1350	12.4	3280	66.3	2050	18.9
	16.10.45	25	2850	51.7	1710	16.4	4450	80.7	2670	25.6
	13.11.45	53	2790	43.8	1580	16.0	4650	73.2	2630	26.7
10	27. 9.45		2860	59.1	1810	16.3	4000	82.7	2530	22.8
	25.10.45	28	4060	63.0	2280	23.3	6180	95.8	3460	35.3
	14.11.45	48	3720	53.9	2030	21.3	6300	91.3	3440	36.0
24	10.10.45		2460	55.7	1710	15.3	3500	79.1	2440	21.7
	25.10.45	15	3540	87.3	2560	22.0	4880	120.0	3540	30.3
	12.11.45	33	3310	69.7	2240	20.6	5100	107.0	3450	31.6
	13.12.45	64	3360	56.9	2090	20.9	5600	94.6	3480	34.7
25	24.10.45		2230	47.8	1470	13.1	3300	70.7	2170	19.4
	23.11.43	30	2760	49.9	1680	16.3	4350	78.6	2650	15.6
	14.12.45	51	2940	49.9	1740	17.3	4940	83.6	2920	29.1
	5. 1.46	73	2610	42.4	1520	15.4	5220	84.9	3210	30.7
26	26.10.45		1590	45.9	1360	11.7	2930	68.6	2040	17.6
	20.11.45	25	2570	54.0	1710	15.5	4100	86.1	2730	24.7
	14.12.45	49	2980	50.8	1790	17.9	4860	82.9	2930	29.3
	5. 1.46	71	2730	43.0	1600	16.4	4750	75.1	2780	28.6
28	1.11.45		2850	55.9	1730	15.8	4100	80.3	2480	22.8
	26.11.45	25	3800	68.1	2210	21.1	5390	96.5	3130	29.9
	28.12.45	57	3390	52.7	1860	18.8	5610	87.1	3090	31.2
	11. 2.46	102	3360	48.8	1790	18.7	5600	81.5	2970	31.1
31	11.11.45		2570	50.9	1620	14.8	3600	71.1	2260	20.8
	5.12.45	24	2880	56.0	1800	16.6	3880	75.2	2420	22.4
	3. 1.46	53	3160	61.2	1960	18.3	5400	105.0	3360	31.2
	1. 2.46	82	3390	55.5	1960	19.6	5580	91.1	3210	32.1
	12. 2.46	93	3430	53.5	1940	19.8	5530	86.5	3140	32.1
32	1.11.45		2400	62.1	1790	15.0	2960	76.5	2210	18.5
	14.12.45	33	2830	64.9	1990	17.7	4640	104.0	3260	29.0
	3. 1.6	53	2900	57.1	1930	18.1	4880	95.8	3220	30.5
	1. 2. 46	82	2420	47.8	1610	15.1	4240	83.9	2820	26.5
34	21.11.45		2260	52.8	1600	14.1	3250	75.9	2310	20.3
	12.12.45	21	2590	56.1	1780	16.2	4210	91.3	2910	26.4
	4. 1.46	44	2510	48.1	1650	15.1	4440	85.0	2920	27.7
36	26.11.45		2770	55.9	1720	15.5	3740	75.2	2320	21.0
	18.12.45	22	3000	57.2	1810	16.9	4310	82.4	2600	24.2
	13. 1.46	48	3600	61.3	2080	20.2	5390	91.9	3110	30.3
	11. 2.46	77	3560	51.8	1920	20.0	5890	85.9	3180	33.0

Table 45

Blood Volume Investigation: Total Circulating Haemoglobin Findings.

Patient No.	Date	Days	Hb. Concentration (gm/100cc)	Total Circulating Hb. (gm)	Total circulating Hb. per kgm. (gm/kgm)	Total circulating Hb. per sq. m. (gm/sq.m.)	Total circulating Hb. per cm. (gm/cm)
4	20. 9.45		13.4	436	11.1	313	2.6
	10.12.45	26	13.2	542	9.2	328	3.3
5	27. 0.45		10.3	434	9.6	291	2.6
	22.11.45	57	13.0	620	10.2	364	3.7
7	21. 9.45		12.6	414	8.3	259	2.4
	16.10.45	25	12.5	558	10.1	333	3.2
	13.11.45	53	13.0	604	9.5	341	3.5
10	27. 9.45		8.9	354	7.3	224	2.0
	25.10.45	28	10.5	649	10.1	364	3.7
	14.11.45	48	13.2	831	12.1	455	4.8
	10.10.45		8.8	308	7.0	216	1.9
24	25.10.45	15	7.6	370	9.2	268	2.3
	12.11.45	33	10.0	510	10.7	344	3.2
	13.12.45	64	12.5	700	11.8	435	4.4
25	24.10.45		9.3	307	6.5	202	1.8
	23.11.45	30	11.2	487	8.8	297	2.9
	14.12.45	51	11.8	583	9.9	345	3.4
	5. 1.46	73	14.6	762	12.4	446	4.5
26	26.10.45		11.0	323	7.6	226	1.9
	20.11.45	25	11.6	476	10.0	317	2.9
	14.12.45	49	13.0	632	10.8	382	3.8
	5. 1.46	71	14.0	663	10.5	387	4.0
28	1.11.45		11.2	459	9.0	278	2.5
	26.11.45	25	10.4	560	10.0	325	3.1
	28.12.45	57	12.9	727	11.3	399	4.0
	11. 2.46	102	14.0	784	11.4	416	4.4
31	11.11.45		9.5	343	6.8	216	2.0
	5.12.45	24	8.4	326	6.3	204	1.9
	3. 1.46	53	12.6	680	13.2	422	3.9
	1. 2.46	82	13.0	725	11.9	419	4.2
	12. 2.46	93	13.7	760	11.9	429	4.4
32	11.11.45		5.5	163	4.2	121	1.0
	14.12.45	33	11.1	515	11.8	362	3.2
	3. 1.46	53	12.6	615	12.1	407	3.8
	1. 2.46	82	15.4	653	12.9	437	4.1
34	21.11.45		9.5	309	7.2	219	1.9
	12.12.45	21	12.2	514	11.1	354	3.2
	4. 1.46	44	13.8	613	11.7	404	3.8
36	26.11.45		7.8	292	5.9	181	1.6
	18.12.45	22	8.7	375	7.1	226	2.1
	13. 1.46	48	10.3	556	9.5	321	3.1
	11. 2.46	77	13.1	770	11.2	416	4.3

Table 46

Blood Volume Investigation: Total Circulating Red Cell Findings.

Patient No.	Date	Days	R.B.C. Concentration (10^6 cells/cmm)	Total Circulating R.B.C. (cells x 10^{12})	Total Circulating R.B.C. per kgm (Cells x 10^{10}/kgm)	Total Circulating R.B.C. per sq.m (Cells x 10^{12}/sq.m)	Total Circulating R.B.C. per cm (Cells x 10^{10}/cm)
4	20. 9.45		3.15	10.3	26	7.4	6.2
	10.12.45	81	5.40	22.2	38	13.4	13.4
5	27. 9.45		2.17	9.2	20	6.2	5.5
	22.11.45	57	4.28	20.4	34	12.0	12.1
7	21. 9.45		2.25	7.4	15	4.6	4.3
	16.10.45	25	3.17	14.1	26	8.5	8.1
	13.11.45	53	4.20	19.5	31	11.0	11.2
10	27. 9.45		2.46	9.8	20	6.2	5.6
	25.10.45	28	3.58	22.1	34	12.4	12.6
	14.11.45	48	4.00	25.2	37	13.8	14.4
24	10.10.45		2.85	9.9	22	6.9	6.1
	25.10.45	15	2.64	12.7	31	9.2	7.9
	12.11.45	33	3.14	16.0	34	10.8	9.9
	13.12.45	64	4.55	25.5	43	15.8	15.8
25	24.10.45		3.08	10.1	22	6.6	5.9
	23.11.45	30	4.00	17.4	31	10.6	10.3
	14.12.45	51	4.28	21.2	36	12.5	12.5
	5. 1.46	73	5.30	27.7	45	16.2	16.3
26	26.10.45		3.01	8.8	21	6.2	5.3
	20.11.45	25	3.56	14.6	31	9.7	8.8
	14.12.45	49	3.90	18.9	32	11.4	11.4
	5. 1.46	71	4.43	21.1	33	12.3	12.7
28	1.11.45		2.42	9.9	19	6.0	5.5
	26.11.45	25	2.69	14.5	26	8.5	8.0
	28.12.45	57	3.59	20.1	31	11.1	11.2
	11. 2.46	102	3.79	21.2	31	11.3	11.8
31	11.11.45		2.10	7.6	15	4.8	4.4
	5.12.45	24	1.87	7.3	14	4.6	4.2
	3. 1.46	53	3.62	19.5	38	12.1	11.3
	1. 2.46	82	3.86	21.6	35	12.5	12.5
	12. 2.46	93	3.90	21.6	34	12.2	12.5
32	11.11.45		1.96	5.8	15	4.3	3.6
	14.12.45	33	3.65	16.9	39	11.9	10.6
	3. 1.46	53	4.30	21.0	41	13.9	13.1
	1. 2.46	82	5.19	22.0	44	14.7	13.7
34	21.11.45		2.64	8.5	20	6.0	5.3
	12.12.45	21	3.40	14.7	32	10.1	9.2
	4. 1.46	44	4.23	18.8	36	12.4	11.8
36	26.11.45		2.21	8.2	17	5.1	4.6
	18.12.45	22	2.67	11.5	22	6.9	6.5
	13. 1.46	48	2.85	15.3	26	8.8	8.6
	11. 2.46	77	3.64	21.4	31	11.6	12.0

Table 47

Blood Volume Investigation: Total Circulating Plasma Protein Findings.

Patient No.	Date	Days	Plasma Protein Concentration (Alb+Glob.) (gm/100cc)	Total Circulating Plasma Protein (Alb.+Glob.) (gm)	Total Circulating Protein per kgm. (Alb.+Glob.) (gm/kgm)	Total Circulating Protein per sq.m. Alb.+Glob.) (gm/sq.m)	Total Circulating Protein per cm. (Alb.+Glob. (gm/cm)
			5.50	108	2.8	78	0.66
4	20.9.45		(4.30+2.20)	(67+41)	(1.7+1.1	(48+30)	(0.41+0.25)
			7.35	175	3.0	106	1.06
	10.12.45	81	(5.25+2.10)	(125+50)	(2.1+0.9)	(76+30)	(0.76+0.30)
5	27. 9.45		4.8	138	3.1	93	0.82
	22.11.45	57	6.84	195	3.2	115	1.16
			4.46	96	1.9	60	0.55
7	21. 9.45		(2.40+2.06)	(52+44)	(1.0+0.9)	(32+28)	(0.30+0.25)
			5.94	169	3.1	101	0.97
	16.10.45	25	(3.39+2.55)	(97+72)	(1.8+1.3)	(58+43)	(0.56+0.41)
			6.26	175	2.8	99	1.01
	13.11.45	53	(4.32+1.94)	(121+54)	(1.9+0.9)	(68+31)	(0.070+0.31)
			7.03	201	4.2	127	1.15
10	27. 9.45		(2.59+4.44)	(74+127)	(1.5+2.7)	(47+80)	(0.42+0.73)
			6.84	278	4.3	156	1.59
	25.10.45	28	(3.0+3.84)	(122+155)	(1.9+2.4)	(69+87)	(0.70+0.89)
			6.91	258	3.8	141	1.47
	14.11.45	48	(3.80+3.11)	(141+117)	(2.1+1.7)	(77+64)	(0.80+0.67)
			2.88	71	1.6	50	0.44
24	10.10.45		(0.75+2.13)	(19+52)	(0.4+1.2)	(13+37)	(0.12+0.32)
			5.44	193	4.8	140	1.20
	25.10.45	15	(2.09+3.35)	(74+119)	(1.8+3.0)	(54+86)	(0.46+0.74)
			6.76	224	4.7	151	1.39
	12.11.45	33	(4.18+2.58)	(138+86)	(2.9+1.8)	(93+58)	(0.86+0.53)
			6.91	233	3.9	145	1.45
	13.12.45	64	(4.09+2.82)	(138+95)	(2.3+1.6)	(86+59)	(0.86+0.59)
			5.17	115	2.6	76	0.67
25	24.10.45		(2.48+2.69)	(55+60)	(1.2+1.4)	(36+40)	(0.32+0.35)
			5.85	162	2.9	99	0.95
	23.11.45	30	(3.85+2.00)	107+55)	(1.9+1.0)	(65+34)	(0.63+0.32)
			6.16	181	3.1	107	1.06
	14.12.45	51	(4.11+2.05)	(121+60)	(2.1+1.0)	(72+35)	(0.71+0.35)
			6.43	168	2.7	98	0.99
	5. 1.46	73	(4.28+2.15)	(112+56)	(1.8+0.9)	(66+32)	(0.66+0.33)
			5.10	99	2.3	69	0.60
26	26.10.45		(1.98+3.12)	(39+60)	(0.9+1.4)	(27+42)	(0.24+0.36)
			6.50	167	3.5	111	1.01
	20.11.45	25	(4.64+1.86)	(119+48)	(2.5+1.0)	(80+31)	(0.72+0.29)
			6.16	184	3.1	111	1.11
	14.12.45	49	(4.47+1.69)	(133+51)	(2.3+0.8)	(80+31)	(0.80+0.31)
			6.66	181	2.9	107	1.09
	5. 1.46	71	(4.45+2.21)	(121+60)	(1.9+1.0)	(71+36)	(0.73+0.36)
			5.48	156	3.1	94	0.88
28	1.11.45		(2.80+2.68)	(80+76)	(1.6+1.5)	(48+46)	(0.44+0.44)
			5.23	199	3.6	116	1.10
	26.11.45	25	(3.15+2.08)	(120+79)	(2.2+1.4)	(70+46)	(0.66+0.44)
			6.68	226	3.5	124	125
	28.12.45	57	(3.92+2.76)	(133+93)	(2.1+1.4)	(73+51)	(0.74+0.51)
			6.68	224	3.3	119	1.25
	11. 2.46	102	(4.19+2.49)	(141+83)	(2.1+1.2)	(75+44)	(0.78+0.47)

Table 47—*concld.*

Patient No.	Date	Days	Plasma Protein Concentration (Alb.+Glob.) (gm/100cc)	Total Circulating Plasma Protein (Alb.+Glob.) (gm)	Total circulating Protein per km. (Alb.+Glob.) (gm/kgm)	Total circulating Protein per sq.m. (Alb.+Glob) (gm/sq.m.)	Total circulating Protein per cm (Alb.+Glob.) (gm/cm)
31	11.11.45		5.3 (2.75+2.55)	136 (71+65)	2.7 (1.4+1.3)	86 (45+41)	0.79 (0.41+0.38)
	5.12.45	24	4.83 (2.95+1.88)	139 (85+54)	2.7 (1.6+1.1)	87 (53+34)	0.80 (0.49+0.31)
	3. 1.46	53	6.16 (3.04+3.12)	194 (96+98)	3.8 (1.9+1.9)	120 (60+60)	1.12 (0.55+0.57)
	1. 2.46	82	6.33 (3.90+2.43)	215 (133+82)	3.5 (2.2+1.3)	124 (77+47)	1.24 (0.77+0.47)
	12. 2.46	93	6.56 (3.45+3.11)	224 (118+106)	3.5 (1.8+1.7)	127 (67+60)	1.29 (0.68+0.61)
32	11.11.45		6.33 (3.44+2.89)	152 (83+69)	3.9 (2.1+1.8)	113 (62+51)	0.95 (0.52+0.43)
	14.12.45	33	7.10 (4.86+2.24)	201 (138+63)	4.6 (3.2+1.4)	141 (97+44)	1.26 (0.87+0.39)
	3. 1.46	53	6.84 (3.69+3.15)	198 (107+91)	3.9 (2.1+1.8)	131 (71+60)	1.24 (0.67+0.57)
	1. 2.46	82	7.46 (4.96+2.50)	181 (120+61)	3.6 (2.4+1.2)	121 (80+41)	1.13 (0.75+0.38)
34	21.11.45		5.24 (3.75+1.49)	118 (85+33)	2.8 (2.0+0.8)	84 (60+24)	0.74 (0.53+0.21)
	12.12.45	21	6.34 (4.50+1.84)	165 (117+48)	3.6 (2.5+1.1)	114 (81+33)	1.03 (0.73+0.30)
	4. 1.46	44	5.98 (4.13+1.85)	150 (104+46)	2.9 (2.0+0.9)	99 (68+31)	0.94 (0.65+0.29)
36	26.11.45		5.12 (2.56+2.56)	142 (71+71)	2.8 (1.4+1.4)	88 (44+44)	0.80 (0.40+0.40)
	18.12.45	22	4.62 (1.81+2.81)	139 (54+85)	2.7 (1.1+1.6)	84 (33+51)	0.78 (0.30+0.48)
	13. 1.46	48	5.81 (3.93+1.88)	209 (141+68)	3.6 (2.4+1.2)	120 (81+39)	1.17 (0.79+0.38)
	11. 2.46	77	6.26 (3.87+2.39)	223 (138+85)	3.2 2.0+1.2)	121 (75+46)	1.25 (0.77+0.48)

Table 48

Blood Volume Investigation—Cases not Followed-up: Haematological Findings.

Patient No.	Hb. Concentration (gm/100cc)	R.B.C. Concentration (10^6 cells/cmm)	Haematocrit (%)	M.C.V. (μ)	M.C.H. ($\gamma\gamma$)	M.C.H.C. (%)
9	8.7	2.77	30.0	108.0	31.2	28.9
12	10.4	2.53	35.0	138.0	41.0	29.9
46	3.3	0.76	10.0	132.1	43.0	32.5
47	10.2	2.48	30.5	123.0	41.2	33.5
48	3.9	1.13	12.0	105.9	34.4	32.5

Table 49

Blood Volume Investigation—Cases Not Followed-up: Plasma Protein and Physical Measurement Findings.

Patient No.	Serum Protein (gm/100cc)	Serum Albumin (gm/100cc)	Serum Globulin (gm/100cc)	Ratio A/G (x : 1)	Body Weight (kgm)	Height(cm)	Area (sq.m)
9	5.43	..	..	..	35.7	168	1.35
12	4.12	..	..	..	40.2	163	1.40
46	5.30	3.02	2.28	1.3	46.0	168	1.50
47	3.93	2.49	1.44	1.7	42.3	161	1.40
48	5.30	3.58	1.72	2.1	52.8	168	1.59

Table 50

Blood Volume Investigations—Cases Not Followed-up: Plasma Volume and Blood Volume Findings.

Patient No.	Plasma Volume (cc)	Plasma Volume per kgm (cc/kgm)	Plasma Volume per sq.m (cc/sq. m)	Plasma Volume per cm (cc/cm)	Blood Volume (cc)	Blood Volume per kgm (cc/kgm)	Blood Volume per sq. m (cc/sq. m)	Blood Volume per cm (cc/cm)
9	2370	66.2	1760	14.1	3390	94.8	2510	20.1
12	1880	46.9	1340	11.5	2890	71.9	2070	17.7
46	2960	64.3	1970	17.6	3290	71.4	2190	19.6
47	2000	47.3	1430	12.4	2880	68.1	2060	17.9
48	2940	55.9	1850	17.5	3340	63.4	2100	19.9

Table 51

Plasma Volume Investigations—Cases Not Followed-up: Total Circulating Haemoglobin Findings.

Patient No.	Hb. Concentration (gm/100 cc)	Total Circulating Hb. (gm)	Total Circulating Hb. per kgm (gm/kgm)	Total Circulating Hb. per sq. m (gm/sq. m)	Total Circulating Hb. per cm (gm/cm)
9	8.7	294	8.2	218	1.7
12	10.4	301	7.5	214	1.8
46	3.3	107	2.3	71	0.6
47	10.2	294	7.0	210	1.8
48	3.9	130	2.5	82	0.8

Table 52

Blood Volume Investigations—Cases Not Followed-up: Total Circulating Red Cell Findings.

Patient No.	R.B.C. Concentration (10^6 Cells/cmm)	Total Circulating R.B.C. (Cells x 10^{12})	Total Circulating R.B.C. per kgm (Cells x 10^{10} kgm)	Total Circulating R.B.C. per sq. m. (Cells x 10^{12}/sq. m)	Total Circulating R.B.C. per cm (Cells x 10^{10}/cm.)
9	2.77	9.4	26	7.0	5.6
12	2.53	7.3	18	5.2	4.5
46	0.76	2.5	5	1.7	1.5
47	2.48	7.1	17	5.1	4.4
48	1.13	3.8	7	2.4	2.3

Table 53

Blood Volume Investigations—Cases Not Followed-up: Total Circulating Plasma Protein Findings.

Patient No.	Plasma Protein Concentration (Alb.+glob). (gm/100cc)	Total Circ. Plasma Protein (Alb.+glob.) (gm)	Total Circ. Plasma Protein per kgm. (Alb.+glob.) (gm/kgm)	Total Circ. Plasma Protein per sq. m (Alb.+glob.) (gm/sq.m)	Total Circ. Plasma Protein per cm. (Alb.+glob.) (gm/cm)
9	5.43	129	3.6	96	0.77
12	4.12	78	1.9	56	0.48
46	5.30 (3.02+2.28)	157 (90+67)	3.4 (2.0+1.4)	105 (60+45)	0.94 (0.54+0.40)
47	3.93 (2.49+1.44)	79 (50+29)	1.9 (1.2+0.7)	56 (36+20)	0.49 (0.31+0.18)
48	5.30 (3.58+1.72)	156 105+51)	3.0 (2.0+1.0)	98 (66+32)	0.93 (0.63+0.30)

Table 54

Sugar Tolerance Curves of Patients on Admission.

Patient No.	Blood Sugar (mgm/100 cc).						
	Fasting	After ½ hour	After 1 hour	After 1½ hours	After 2 hours	After 2½ hours	After 3 hours
2	86	177	123	106	103	97	89
6	85	118	117	104	107	117	107
11	97	133	137	119	121	106	100
15	76	90	102	108	102	80	73
18	79	97	105	98	79	..	99
25	100	102	104	100	100	99	98
26	80	87	115	103	95	80	92
28	100	113	110	108	108	101	..
31	101	101	101	107	99	105	100
32	100	100	109	116	110	101	100
36	99	119	116	110	112	112	116

Table 55

Sugar Tolerance—Curves of Patients on Discharge

Patient No.	Blood Sugar (mgm/100 cc)						
	Fasting	After ½ hour	After 1 hour	After 1½ hours	After 2 hours	After 2½ hours	After 3 hours
2	110	117	122	130	119	101	109
6	113	132	161	147	106	85	90
11	115	171	146	117	111	98	87
15	106	111	168	138	95	100	101
18	106	114	143	120	123	107	85
25	102	140	148	139	103	94	100
26	102	104	123	119	110	..	99
28	100	102	129	97	87	..	88
31	111	149	141	132	135	106	80
32	112	125	114	103	..	100	106
36	102	130	163	115	114	101	94

Table 56

Sugar Tolerance—Curves of Control Series.

Patient No.	Blood Sugar (mgm/100 cc)						
	Fasting	After ½ hour	After 1 hour	After 1½ hours	After 2 hours	After 2½ hours	After 3 hours
37	119	126	90	..	93	86	..
38	106	109	101	96	..	85	80
39	100	102	172	134	104	99	89
41	112	138	118	106	95	91	88
42	116	174	139	138	90	90	88
43	121	136	125	111	99	94	111
44	129	159	141	114	..	96	104
45	115	126	114	102	98	99	112

Table 57

" Marasmus Diet ".

Food	Quantity	Calories	Protein (gm)	Carbohydrate (gm)	Fat (gm)	Iron (mgm)	Calcium (mgm)	Vit. A. (I. U.)	Vit. B_1 (μg)	Riboflavin (μg)	Nicotinic Acid (mgm)	Ascorbic Acid (mgm)
Milk	80oz.	1520	72	112	88	0	320 0	3700	1040	3400	0	0
Eggs	4	210	16	1	16	4	80	1300	200	530	0	0
Rice	8oz.	780	14	179	1	5	20	0	480	180	9	0
Dal	2oz.	210	10	35	3	6	110	100	240	180	1	6
Atta	2oz.	200	7	41	1	4	20	0	260	70	3	0
Sugar	2oz.	220	0	56	0	0	0	0	0	0	0	0
Ghee	2oz.	510	0	0	56	0	0	700	0	0	0	0
Orange Juice	2oz.	30	1	6	0	0	30	100	40	20	0	28
Tea	1/6oz.	0	0	0	0	0	0	0	0	0	0	0
Chicken	8oz.	180	24	0	56	5	10	0	200	..	..	0
Total	..	3860	144	430	221	24	3470	5900	2460	4380	13	34

Table 58

High Calorie Diet.

Food	Quantity	Calories	Protein (gm)	Carbohydrate (gm)	Fat (gm)	Iron (mgm)	Calcium (mgm)	Vit. A. (I.U.)	Vit. B_1 (μg)	Riboflavin (μg)	Nicotinic Acid (mgm)	Ascorbic Acid (mgm)
Milk	80 oz.	1520	72	112	88	0	3200	3700	1040	3400	0	0
Butter	1 oz.	210	0	0	23	0	0	800	0	0	0	0
Ghee	2 oz.	510	0	0	56	0	0	700	0	0	0	0
Eggs	2	100	8	0	8	2	40	600	100	260	0	0
Mutton	5 oz.	220	21	0	15	3	10	100	190	250	10	0
Bread	2 oz.	150	5	31	0	0	10	0	40	20	0	0
Rice or Atta	20oz.	1960	36	448	2	12	60	0	1200	460	22	0
Dal	2 oz.	210	10	35	3	6	110	100	240	180	1	6
Potatoes	4 oz.	60	2	14	0	0	10	0	100	60	1	12
Sugar	2 oz.	0	0	56	0	0	0	0	0	0	0	0
Fresh Fruit	8 oz.	100	1	25	0	1	20	800	60	60	1	16
Fresh Vegetables	8 oz.	50	3	9	0	5	160	1600	110	120	1	56
Total	..	5310	158	730	195	29	3620	8400	3080	4810	36	90