DERMATOSES IN THE ELDERLY: BIOCHEMICAL AND HAEMATOLOGICAL STUDIES, WITH SPECIAL REFERENCE TO SERUM PROTEINS

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INTRODUCTION.

An increasing proportion of the population is reaching the higher age groups, but prolonged life is of little value when accompanied by disease. More attention is therefore necessary to the study of normal and pathological states in the elderly.

In the literature, reports have appeared which suggest that a condition of "geriatric nutritional eczema" occurs in elderly, under-nourished subjects. This diagnosis did not seem to be wholly justified for reasons which will be given later. Further study appeared to be necessary and attention has therefore been directed towards the investigation of nutritional states in elderly subjects who were suffering from disease of the skin.

No single, exact yardstick exists by which nutritional states can be measured and in general the assessment of under-nourishment is complicated and unsatisfactory. Nevertheless, history and clinical examination have been supplemented by biochemical and haematological investigations in fifty-two subjects, in an attempt to evaluate the nutritional states and to determine whether high protein diets are indicated in elderly subjects who are suffering from the more common diseases of the skin.

PART I. ASPECTS OF PROTEIN METABOLISM.

The word protein, or the older and now discarded term "proteid", is derived from the Greek protos, meaning first, which signifies its relative importance in living tissues. Mulder (1849), stated that protein was the most important of all known substances in the organic kingdom. Protoplasm consists very largely of protein which is the highly complex substance found in all living cells, in their nuclei, in blood, in hormones and in enzyme systems. Proteins all contain carbon, hydrogen, nitrogen and oxygen, most contain sulphur and a few contain other elements such as phosphorous, iron and iodine. At the beginning of the present century it seemed that the study of nutrition offered little scope for new discoveries of a fundamental nature, but the investigation of the physical chemistry of the proteins, the use of the ultracentrifuge, the electrophoretic procedure of Tiselius and the studies of X-ray photography of large molecules have opened up new biological concepts.

A. Dietary Protein. The ultimate source of all body protein is dietary protein which is absorbed from the alimentary tract as amino-acids of which some twenty-three have been isolated and of which at least eight are essential. Geiger (1947), has shewn that all of the essential amino-acids must be/

be present simultaneously for protein synthesis and that "incomplete" amino-acid mixtures are not stored in the body, but are further metabolised in an irreversible manner. Each protein in the body has its own individual amino-acid composition and lack of one amino-acid may cause breakdown of other tissue proteins to supply it. It is now clear that protein metabolism is one of dynamic rather than static equilibrium and is constantly undergoing rapid and perhaps profound changes of many kinds. Whipple (1942), has shewn that protein stores exist in the tissues and that an ebb and flow of protein between cells and plasma takes place. The site of protein storage is not known but the liver, subcut-:aneous tissue, reticulo-endothelial system and plasma have all been suggested as protein reservoirs. Schoenheimer et al. (1939), using isotopic amino-acids, have shewn that skin and muscle have the greatest share in uptake of dietary nitrogen. Ratner et al. (1940), have shewn that the amino-acids from the intestine are absorbed to form protein of the skin. According to Urbach (1946), albumin, globulin, mucin, keratin, elastin, collagen and melanin are the principal protein components of the skin, which is also dependent on the essential amino-acid methionine for its sulphur content.

B. Plasma Proteins. It is known that plasma proteins consist/

consist of albumin, alpha, beta and gamma globulin and fibrinogen. The latter takes part in clotting mechanisms and is largely removed from plasma in the process of clotting. The fluid which remains is known as serum. The globulin fraction takes part in antibody production and Cannon (1945), reviewing the role of proteins and resistance to infection, stated that in this, the gamma fraction is significant. Cannon et al. (1944) demonstrated that beef serum gamma globulin was highly efficient in regeneration of serum proteins and ipse facto that antibody production must require an ample supply of essential amino-acids either in the diet or in the protein reserves. Wissler et al. (1946) showed that antibody production was impaired in protein depleted rats. Schoenheimer et al. (1942), using isotopic amino-acids, showed that antibodies depend on dietary nitrogen. Krebs (1946) reported low values of gamma globulin in a girl who was suffering from malnutrition. Antibody production and resistance to infection was diminished. White and Docherty (1946) suggested that lymphocytes are a storehouse of beta and gamma globulin, but this has not been confirmed.

Starling (1896) first formulated the conception of the part played by plasma colloids in exerting osmotic or oncotic pressure and Epstein (1917) adapted the hypothesis to explain the oedema in "chronic parenchymatous nephritis". Albumin, since/

since it has a much smaller molecule than globulin, is considered to he more important in maintaining the osmotic pressure of the blood. The modern conception of the forces operating in equilibrium according to Keys et al. (1950), is that the colloid osmotic pressure of the blood, together with the hydrostatic pressure of the tissue fluid, is equal to the colloid osmotic pressure of the tissue fluid, together with the hydrostatic pressure of the blood. Various formulae have been suggested for the calculation of serum colloid osmotic pressure, but since these cannot take such factors as peri-capillary circulation and lymph flow into account, they are not altogether satisfactory. The question of oedema and serum proteins will be considered in more detail in Part II.

C. Protein and Vitamins. Protein metabolism is linked with certain vitamins. These may be concerned in enzyme systems which are related to intermediary reactions of amino-acids. Conger and Elvehjem (1941) found that dermatitis in rats on a pyridoxine deficient diet occurred more readily when there was excess protein in the diet and it would seem that intermediate reactions have priority on pyridoxine before it can be utilised for metabolism of the epidermis. Again, ascorbic acid is involved in the correct metabolism of phenylalanine and tyrosine.

In/

In general, vitamin deficiencies may be related to the protein level in the diet and conversely adequate vitamin intake is necessary for the utilisation of protein.

D. Protein, Vitamins and Blood Formation. Protein and vitamins are also concerned in the formation of blood, for adequate supplies of protein are required to build up the globulin or protein portion of haemoglobin and Vitamins A, C, K and P are concerned respectively with leucocyte formation, marrow stimulation, coagulation and capillary permeability.

Haemoglobin formation, however, according to Whipple (1942), takes precedence over the formation of plasma proteins in the body and body stores must be grossly deficient over a long time before delay in haemoglobin formation takes place. Nevertheless it has been shewn by Whipple et al. (1946) that proteins and certain amino-acids added to the diet caused a much greater increase in haemoglobin production than iron alone, while with excess iron intake, haemoglobin formation was poor until adequate protein was given.

In conclusion, protein is essential in metabolism. General protein deficiency may result from lack of a single amino-acid and dietary deficiency of protein may be expected to result in extensive metabolic upset in which infection, anaemia and oedema may occur. The skin takes part in the metabolic/

metabolic reactions involving the incorporation of dietary nitrogen and is also dependent on at least one essential amino-acid.

It would seem reasonable to suppose that disease of the skin may result directly from deficiencies of dietary protein and that skin disease per se may produce disturbances of protein metabolism. More detailed consideration of the possible relationship between disease of the skin and hypoproteinaemia will be given in reviewing the literature in Part III.

Apart from the more direct connection between skin and protein metabolism, it may be postulated that protein deficiency gives rise to oedema, which in turn may result in disease of the skin, since oedema is not infrequently associated with eczema. It might be expected that dietary deficiency of protein would lead to a fall in serum protein concentration with resulting oedema, but this decrease may be masked by haemoconcentration or by a response from the reserve protein stores. A great volume of literature on the subject of malnutrition, serum protein concentrations and oedema has appeared and will now be reviewed.

PART II. MALNUTRITION, SERUM PROTEINS AND OEDEMA.

Protein deficiency in the diet has generally been assumed to involve a parallel lowering of serum protein and this in turn to the production of oedema. Two world wars have stimulated research into the pathology of starvation which is as yet not completely understood.

According to Cuthbertson (1945), in the absence of all food the body's supply of energy is, after the first day or two, derived from its store of protein and fat and even in partial inanition, body protein will be used up and the extent to which the different organs will be depleted is almost certainly inversely proportional to their import-:ance in survival.

A. Investigations on Animals. Experimental work on starvation has been carried out on animals and while such investigations are artificial and cannot replace controlled observations on human beings, they would seem to be signif-:icant. Addis et al. (1936) found that in rats which had fasted for seven days, the several tissues contributed to the total protein loss in the following proportions:- muscles, skin and skeleton 62 per cent., liver 16 per cent., alimentary tract, spleen and pancreas 14 per cent., blood 6 per cent., kidneys 1 per cent., heart 0.5 per cent. and

other/

other organs 0.5 per cent. They found, however, that liver, heart and kidneys lost relatively more of the original protein content than did the muscles, skin and skeleton. This is not in agreement with Cuthbertson's views. Shelburne and Egloff (1931) produced oedema in three dogs by plasmapheresis and by feeding a protein deficient diet. Administration of sodium chloride and sodium bicarbonate increased the oedema, although it tended to decrease again after a few days. Weech et al. (1933) produced oedema in dogs by plasmapheresis and by low protein diet. With diet depletion, oedema was found when the plasma albumin fell below 2.0 to 1.5 gm. per cent. Globulin tended to increase. Sunderman and Dohan (1941), studying starvation in depancreatised dogs, found a significant fall in plasma protein, but no constant change in serum total base. Cannon et al. (1944) found low serum proteins in rats, after they had been depleted of dietary protein for sixty to ninety-four days, but found no evidence of injury to the mechanism which produced serum protein. Benditt et al. (1946) carried out detailed investigations on rats which received protein deficient diets. The serum protein concentration and the total circulating serum protein were significantly lower in the depleted rats, as were also the blood volume, erythrocyte volume, plasma volume, haemoglobin concentration, haematocrit and mean corpuscular haemoglobin/

haemoglobin concentration values.

B. Famine Oedema. Famine oedema or nutritional oedema has a place in most medical text-books and the field of literature on the subject is vast. Maver (1920) gave an extensive review of the literature and in general, the majority of earlier reports agreed that malnutrition resulted in diminished serum proteins with resulting nutritional oedema. Bruckman et al. (1930) investigated nine patients who were suffering from malnutrition and found low serum total protein and serum albumin concentrations. Serum globulin concentrat-:ions were normal and the authors concluded that albumin is diminished in starvation, but that globulin may be high if infection is present. Oedema was present when albumin was less than 3.0. gm. per cent. Liu et al. (1931a.), studying the influence of dietary regimes on nitrogen balance, plasma proteins and oedema in two cases of nutritional oedema in young persons, stated that nutritional oedema is definitely related to the level of the plasma proteins which can be easily influenced by the protein intake. The same workers (1931b.) reported that changes in the level of plasma proteins induced by alkalis or acids are slight and inconstant. Chang (1932) found low serum total protein concentrations in seven cases of nutritional oedema. The initial concentrations varied from 3.46 to 5.77 gm. per cent. Youmans et al. (1932a.) and/

and (1932b.) studied thirty-one cases of nutritional oedema and found normal serum total protein levels, but in general, low albumin and high globulin concentrations. The seasonal nature of the oedema snd the question of vitamin deficiency raises speculation regarding the diagnosis in their patients. Again, as the patients were sitting when blood samples were removed, the protein concentrations would be higher in comparison with other workers' results obtained in recumbent patients. Vaughan et al. (1945) found low plasma proteins in prisoners-of-war who were suffering from oedema. In only one subject was there a disturbance of the albumin globulin ratio. In ten subjects with generalised oedema the total protein was 3.84 $\pm$ 0.93 gm. per cent. and the albumin 2.09 $\pm$ 0.46 gm. per cent. In cases without gross oedema the total protein was 4.99 $\pm$ 0.78 gm. per cent. and the albumin 2.79 $\pm$ 0.43 gm. per cent. Lipscombe (1945) found that in Belsen cases slight to gross hunger oedema was present. In the latter, a small number of observations revealed low plasma proteins.

Burger et al. (1945) found all degrees of oedema in starvation but did not investigate the serum proteins. Edge (1945) noted at least forty subjects with oedema in three hundred prisoners-of-war. Little change in red blood cells was noted and serum proteins were not investigated.

Gupta/

Gupta (1946) reported low plasma proteins in ten prisoners-of-war, the lowest concentration being 4.3 gm. total protein per cent. Oedema was present in four cases but was not proportional to the protein concentrations. Macrocytic and microcytic anaemia were both seen. Price (1946) found oedema common among one thousand prisoners-of-:war. Stapleton (1946) described delayed oedema in prisoners-of-war. This appeared after the diet had returned to normal. Bennet (1946), Smith (1946) and Scott McGregor (1946) all described the occurrence of famine oedema.

Mollison (1946) studied serum proteins in forty-three Belsen inmates and found the concentrations in oedematous cases on the average to be lower than in non-oedematous cases, although in many subjects there was a lack of correlation between the serum total protein concentration and the degree of oedema. He reported one case which, in spite of considerable protein deficiency and a serum total protein concentration of 4.2 gm. per cent., had retained quite a large amount of body fat which was seen on cutting down on veins. In the male subjects, the average serum total protein was 5.2 gm. per cent. and in the females, the average was 5.0 gm. per cent. In the presence of oedema, blood volume either bore the usual relation to body weight or was relatively lower.

Leyton (1946) reported that among British and Russian prisoners, the average plasma protein concentration was lower in the Russians who received the poorer diet, being 6.5 gm. per cent. in the former and 5.3 gm. per cent. in the latter. Oedema began when the plasma proteins were less than 5.3 gm. per cent. and was universal when they were under 4.6 gm. per cent. Walters et al. (1947a.), in a detailed investigation of prisoners-of-war, found a reduction in the serum total protein concentration which was due to diminution of the albumin fraction. The mean serum total protein concentration was 5.4 gm. per cent. and the mean serum albumin concentration was 2.6 gm. per cent. The same authors (1947b.) reported little change in the plasma volume in a selected group of these subjects. Murray (1947) noted great emaciation but no famine oedema among inmates of Sandbostel concentration camp. Pollack (1947), describing starvation in prisoners-of-war, pointed out that a slow adjustment took place, through stages of oedema, pellagra and beri-beri to death, or to emaciation without oedema. Oedema was not infrequent during intermediate stages leading up to a recovery from the emaciated state and was usually, but not always, associated with low serum proteins. Pevny (1947) reported on inmates of a German concentration camp where oedema appeared in prisoners after they had been three weeks/

weeks on the starvation diet. In some instances well-nourished people carried on fairly well, then suddenly developed oedema. Denz (1947) found the total plasma proteins to be diminished in oedema, varying from 4.9 to 5.24 gm. per cent., while the albumin varied from 2.58 to 3.81 gm. per cent. The author stated that, with regard to oedema, no true "critical" level of plasma proteins existed. Anderson and Altman (1951) reported low absolute and relative values of albumin and high absolute and relative values of gamma globulin in nine children who were suffering from nutritional oedema.

A number of other workers have raised doubts concerning the role of proteins in oedema. Dicker (1948), working on rats which had been fed low protein diets, found tissue oedema present after seven days, but found no change in plasma nitrogen content, albumin globulin ratio or colloid osmotic pressure of plasma. After fourteen days, those levels fell. He concluded therefore, that the onset of oedema in malnutrition is independent of changes either in albumin globulin ratio or in colloid osmotic pressure. No correlation could be found between the degree of hypoproteinaemia, the fall of colloid osmotic pressure and the magnitude of the extracellular phase of tissues. Howard et al. (1946) studied nitrogen and mineral balances during starvation/

starvation in two healthy young men after they had been given a period of five days' bed rest. In an abrupt two day fast, the protein concentration diminished in one subject and increased in the other. With more prolonged fasting, the concentrations in both subjects increased by approximately 0.5 gm. per cent. Uehlinger (1947) pointed out that the oedema of starvation cannot be fully explained, since in a number of cases a decrease of albumin cannot be found. Nevertheless he considered that no better hypothesis than that of lowered protein osmotic pressure was available to explain oedema in malnutrition.

Sinclair (1948), reporting on the Oxford Nutrition Survey in Holland and Germany, found no significant difference between the mean values of serum protein and of serum albumin in subjects with nutritional oedema as compared with those without nutritional oedema. Moreover, the mean values fell within the normal range. He did, however, find a qualitative difference in the serum protein, the mean nitrogen of which was lowered.

Commenting on famine oedema, Himsworth (1948) stated that it could not be explained entirely on the basis of Starling's hypothesis. Himsworth suggested that the normal transient water retention resulting from ingestion of salt dilutes the plasma and if by undernutrition the plasma cannot/

cannot be restored rapidly to normal levels, the diminished osmotic pressure would permit oedema to form. Beattie et al. (1948) studied nutritional oedema in thirty-eight subjects and found hypoproteinaemia in four instances. In those four subjects oedema was present and the total protein concentrat-;ion was less than 5.0 gm. per cent. In one of those subjects the albumin concentration was under 2.5 gm. per cent. The authors noted oedema in a number of the other subjects in whom the total protein concentration was above 5.0 gm. per cent. They found no change in the absolute plasma volume in starvation and suggested that nutritional oedema may occur with or without hypoproteinaemia.

Keys et al. (1950) carried out an extensive experiment in chronic malnutrition and concluded that famine oedema is due to retention of the pre-starvation level of extracellular fluid. They stated, however, that lowered serum proteins may foster the development of oedema. The absolute plasma volume tended to remain constant or increased slightly in chronic starvation.

It is evident then, that oedema is common in malnutrition, but the role of the serum proteins in this connection is not yet clear. The majority of workers have found lowered serum proteins in association with oedema, but a number of authors in recent years have failed to demonstrate such a relation-:ship./

relationship. No marked changes in plasma volume have been noted in chronic malnutrition in the human, but some authors have reported a diminution in plasma volume in animals.

In the field of dermatology, some authors have assumed that lowered serum proteins were associated with oedema and that the intracellular and intercellular oedema of eczema might in some instances be due to lowered osmotic pressure.

The literature regarding the role of hypoproteinaemia in some of the commoner diseases of the skin will now be discussed.

PART III. PROTEIN DEFICIENCY AND DISEASE OF THE SKIN.

It has been shewn that some doubt exists as to whether lowered serum proteins are the main cause of oedema and it follows therefore, that any hypothesis which seeks to explain the etiology of eczema on the basis of oedema due to decreased colloid osmotic pressure, must also be regarded with some reservation. Nevertheless it has also been suggested that loss of protein in desquamated epithelial scales and in serous and purulent exudates may cause an excessive drain on serum protein. Again if there be a histamine release phenomenon in eczema, it is conceivable that increased capillary permeabil-:ity could result in lowered serum proteins.

A. Investigations on Animals. Conclusions drawn from experimental work on animals may not have a wide application, but some evidence can be drawn from such work that protein is important in skin and wound healing.

Clark (1919) shewed that a low protein diet in dogs increased the period necessary for the healing of experimental wounds. The "quiescent" period of wound healing was dimin-:ished in dogs which were fed a high protein diet, so that although the stage of epidermal regrowth was not hastened, the time necessary for final complete healing was shortened. Baker and Carrel (1926) demonstrated that it is the protein fraction of embryo tissue juice which contains the growth-stimulating/

growth-stimulating substance responsible for the continuous growth of fibroblasts in vitro. Harvey and Howes (1930) found an increase in velocity of growth of fibroblasts in healing wounds in the stomach of a rat which was fed on a high protein diet. Weech et al. (1933) found that dogs, fed on a diet deficient in protein, developed oedema, seborrhoeic dermatitis and leg ulcers. The authors did not consider that vitamin deficiency was likely to be a factor in this experiment. Thompson et al. (1938) shewed wound disruption or failure to heal in eight out of eleven dogs which were suffering from protein deficiency. Madden et al. (1943) reported that skin ulceration and scaling erythema of the abdomen and chest developed in dogs which were kept on a low protein mixture after plasmapheresis. Cystine was found to have a stimulating action on formation of plasma proteins, but this effect was not maintained after a few days.

B. Surgical Reports on Wound Healing. Reports by workers in the field of surgery are not in complete agreement. Koster and Shapiro (1940), in a study of fifty-eight surgical patients, found that eighty-six per cent. with disrupted and infected wounds had a serum albumin level under 3.75 gm. per cent., while only twenty per cent. of those with well-healing wounds shewed such low levels. Hartzell et al. (1941) reviewing literature regarding wound healing and vitamin C, stated/

stated that protein and vitamin C were low in wound disruption.

Taylor et al. (1943) found hypoproteinaemia in sixty-three patients who were suffering from burns. Detailed studies of nitrogen metabolism were made in ten cases. Protein deficiency was judged to be the result of tissue destruction, infection and increased nitrogen demand for new tissue formation.

Koster and Kasman (1942) found that, in forty patients who were suffering from wound disruption, the serum albumin level was lower than in forty patients who had well-healed wounds. The authors were cautious in drawing conclusions, since not all subjects who had disrupted wounds had low serum albumin values.

On the other hand, Abbott and Mellors (1943) stated that haemoconcentration in dehydration may conceal protein deficiency and that the results of nitrogen balance studies would bear a closer relation than serum protein concentrations to wound healing or dehiscence. One case of wound disruption, in which the serum protein concentration was over 6.0 gm. per cent. when nitrogen balance was negative and in which bed sores healed when the serum protein concentration was 5.0 gm. per cent. but when nitrogen balance had become positive, was cited in support of this view.

C. Observations/

C. Observations in Dermatoses. Apart from reports on experimental and surgical wound healing, the view that protein deficiency may be associated with disease of the skin has been adopted by a number of writers. Joseph (1930) used insulin in five non-diabetic subjects with decubitus ulcers, in an attempt to improve their general nutrition. Clements (1936) reported that a high protein diet diminished the incidence of tropical ulcers. Talbott and Coombs (1940) investigated thirty-four subjects who were suffering from pemphigus and grouped them into ten acute and twenty-four chronic types. In all subjects in the former group, the serum total protein concentration was less than 5.2 gm. per cent. and in seven subjects in the second group the serum total protein concentration was less than 5.5 gm. per cent. Altschuler et al. (1943) published a report on ten subjects who were suffering from indolent ulcers of the legs. A number of those patients had varicosity of veins. "The blood level of amino-acids was raised and the ulcers improved on treatment with protein hydrolysates." No details of blood levels were given in this report. Mulholland et al. (1943) investigated the plasma proteins in thirty-five subjects who were suffering from decubitus ulcers. The Barbour Hamilton method was used for total protein estimations and the Greenberg method was used for albumin and globulin/

globulin estimations. Controlled nitrogen balance studies were carried out in eight cases. No subject had a total protein concentration above 6.35 gm. per cent. and seventeen had a concentration under 5.5 gm. per cent. High calorie, high amino-acid diets were given with good results. Mulvehill (1944), using the method of Greenberg and Kingsley and estimating the albumin fraction by salting out the globulin with 22.2 per cent. sodium sulphate, investigated the serum proteins in eighty-one subjects who were suffering from a variety of dermatological conditions. A lowered albumin level was found in pemphigus vulgaris, pemphigus foliaceus and pemphigus vegetans, but the author reported normal values in exfoliative dermatitis, herpes zoster, scabies, dermatitis medicamentosa (sulphonamide), lichen planus, seborrhoea, psoriasis, erythema multiforme bullosum, discoid lupus erythematosus, atopic eczema, pediculosis corporis, dermatitis herpetiformis and pityriasis rosea. The number of subjects in each group was therefore too small for the findings to be significant. Peters (1945) reported good results from oral administration of cystine and cysteine in exfoliative dermatitis. He suggested that the loss of epithelium may have caused a deficiency of sulphur-containing amino-acids and that the oedema in his subjects may have been caused by a low concentration of serum protein due to the demand/

demand on body protein to supply cystine. Goldsmith (1947) reported a striking clinical improvement from oral administrat-:ion of protein hydrolysate in two cases of exfoliative dermatitis. Lund and Levenson (1945) stated that protein deficiency may be an important factor in the etiology of gastro-intestinal, leg and decubitus ulcers, but gave no evidence supporting this statement. Glaser and Markson (1945) reported a case of exfoliative erythrodermia, with toxaemia, intermittent oedema and hypoproteinaemia, which was considered to be a possible example of Leiner's disease. In Leiner's disease, according to those authors, low proteins are generally reported and may be due to liver damage or loss of serum in exudates. In four out of five cases of infantile eczema, the authors found low serum proteins which varied from 4.5 to 5.7 gm. per cent. It is known, however, that the serum proteins concentrations in infancy are low, up to the age of three years.

Drant (1946) investigated serum proteins in six cases of Besnier's eczema and reported that immunisation with autogenous serum was efficaceous when the albumin level was high and the globulin low, but obtained poor results when the albumin level was low and the globulin high. Starck (1946) drew attention to the resemblance between extensive burns and bullous dermatoses, but disagreed with Mulvehill who/

who suggested that the serum protein concentration might be used as a diagnostic aid in differentiating dermatitis herpetiformis, pemphigus and erythema multiforme bullosum. Starck found low protein values in one case of pemphigus vulgaris, in one case of pemphigus foliaceus, in one case of pemphigus vegetans and in two cases of dermatitis herpetiformis. Urbach (1946) stated that high protein diet is important in burns, pemphigus, diabetic, varicose and decubital ulcers. Guy et al. (1948) were the first to suggest the diagnosis of "geriatric nutritional eczema" for a condition which occurred in elderly patients. Those patients presented varying degrees of oedema of the legs, associated with a chronic, erythematous, papular, vesicular, pustular, exudative or squamous inflammation of the skin. Cardiac decompensation and renal disease were not found and varicose veins of sufficient degree of severity to cause the oedema were not present. The authors reported that in all cases they found a lowered serum total protein concentration with a reversal of the albumin globulin ratio and suggested that low intake of protein, impaired masticatory powers and impaired assimilation were responsible for the condition. No detailed results of the investigations were recorded and the authors gave no consideration to the fact that normal serum protein concentrations may be low in the elderly.

Later,/

Later, Guy (1950) modified his view and suggested that the diagnosis of protein deficiency does not depend on the finding of a low serum total protein or a reversal of the albumin globulin ratio and that protein deficiency with the type of cutaneous manifestations already described under "geriatric nutritional eczema" may occur in younger patients because of faulty synthesis of plasma proteins by disease of the liver. The author described five subjects who improved as a result of increased protein intake. Of those five, one case designated "geriatric nutritional eczema" had a serum total protein concentration of 5.7 gm. per cent., one case of acute hepatitis and eczema of legs had a serum total protein concentration of 5.88 gm. per cent. and in one case of varicose ulcer and eczema the serum total protein concentration was 4.4 gm. per cent. Laboratory technique was not stated and the final protein values were not recorded. The author admitted that single estimations may be misleading and that the subjects were treated largely on clinical suspicion.

Discussing this report, Wohl stated that he had investigated a number of subjects who had chronic ulcers of the legs. These subjects showed a serum albumin concentration of less than 4.0 gm. per cent. which he considered to be the lower limit of normal. The subjects also showed a lowered

capacity/

capacity to manufacture antibodies. The value given for the lower limit of normal serum albumin does not receive general support. Tashian stated that twenty years previously, he had been impressed with the effect of proteins and amino-acids used as local applications to chronic ulcers.

Keim et al. (1948) reviewed the role of hypoproteinaemia in dermatitis and reported one case of arsenical exfoliative dermatitis in which treatment with intravenous plasma, injections of protein hydrolysate and a high protein diet caused a rapid improvement in the skin condition. No serum protein values were stated. The authors also reported one case of chronic disseminated neurodermatitis treated with high protein diet, intravenous plasma and protein hydrolysate. The condition improved at first but the subject died some months later from hepatitis. Initial serum protein concentrations were not recorded. In addition, six subjects with exudative dermatitis and "significantly lowered" plasma proteins received the same treatment. Of those, two died, one deteriorated, one became worse immediately, but improved two days later and two were much improved. Again no serum protein values were recorded. The employment of intravenous transfusions in subjects who are capable of taking food by mouth is not justifiable and the occurrence of hepatitis following/

following such treatment cannot be ignored. Lane and Rockwood (1949) reviewed two thousand patients who were over sixty years of age and found that eczema was present in only 8.8 per cent. and that only eleven cases of ulcer were noted. The eczema appeared to be more intractable than in younger patients. No single etiological factor was discovered. The review was not representative of the general population and no laboratory findings were stated. Kennedy et al. (1950) mentioned protein deficiency as a factor in the etiology of skin disease in old age, but no laboratory findings were given in their review of eight hundred and seventy-eight patients who were over the age of forty. Walker and Benditt (1950) found diminished serum albumin in lupus erythematosus and in scleroderma and an increase in gamma globulin in these conditions. Dietary deficiency, disturbance of albumin synthesis in the liver due to liver disease or loss of protein by the kidneys were all suggested as possible etiological factors.

D. Famine and Disease of the Skin. When the great volume of literature on the effects of starvation in prisoners-of-war is considered, little mention of skin disease is found. This may be due partly to the fact that most observers were not interested primarily in skin manifestations. Vandervelde and Cantineau (1919) reported on two hundred prisoners who were/

were suffering from oedema. Dry, painful skin and frequent secondary pyogenic infection was noted, while dark pigmented patches on the face were apparent in a few cases. Burger et al. (1945) found that in starvation the skin of the face often showed a dun pigmentation. In other parts of the body the skin was dry and showed a loss of elasticity. Gangrene of skin was common. Scorbutic signs were common in older subjects. Lipscombe (1945) found that specific vitamin deficiency syndromes were absent, but dry skin with foll-:icular keratosis was often noted. Sepsis was present in many forms, sores and abscesses were common, but infective dermatitis was surprisingly rare. No scurvy was noted. Edge (1945) saw no scurvy among three hundred prisoners-of-war. Gupta (1946) found dry, scaling skin in three subjects and bed sores in one subject among ten prisoners. Price (1946) found oedema and jungle sores common among one thousand British prisoners. Leyton (1946) noted that the skin was yellowish but that ulcers and furuncles were not unduly common among British and Russian prisoners-of-war. Mollison (1946) noted no skin conditions, apart from koilonychia and angular stomatitis, in forty-three Belsen prisoners. Pevny (1947) found that in inmates of a concentrat-:ion camp, the skin was pale, shiny and dry, hair and nails grew slowly and greying of hair was rapid. Scurvy, pyoderma, follicular/

follicular keratoses, nodular and lichenoid lesions, and pigmented warts were all common.

Gell (1948) cited German practitioners who reported a marked increase in superficial skin lesions as a result of undernutrition in the civil population. Keys (1948) described the skin in starvation as thin, dry, scaly, inelastic, pallid, greyish or cyanotic, with occasional follicular keratotic lesions on the extensor surfaces of the limbs and pigmented patches which were most frequently noted on the face.

In conclusion then, experimental work on animals suggests that lowered serum proteins are related to delayed skin and wound healing and to disease of the skin. The majority of surgical workers are in agreement with this conception, but a few are more guarded in their conclusions. Reports from workers in the field of dermatology are on the whole of doubtful quality, but suggest that ulceration, eczema and bullous eruptions are associated with lowered serum protein concentrations. In the literature dealing with starvation in prisoners, some evidence is noted that superficial skin infection occurs in malnutrition.

PART IV. SUMMARY AND APPRECIATION OF LITERATURE.

The serum proteins are of vital importance in the structure and physiology of the body and the skin takes part in the metabolic processes involving dietary nitrogen. Since the skin is rich in protein, deficiency of amino-acids in the diet may result in derangement of the skin metabolism. Disease of the skin with concommitant loss of protein in purulent and serous exudates and in desquamated scales, may cause a drain on body protein.

The demand for amino-acids in repair of damaged skin may be sufficient to result in a pathological depletion of protein, especially when protein intake is already low. This may be more likely to occur among the aged population as a result of poverty, ignorance or disease and because of defective absorption, assimilation or utilisation of food.

Again, oedema is one manifestation of malnutrition and it is known that oedema may result in disease of the skin. It is less certain, however, that the serum proteins are significantly lowered in malnutrition and in oedema, although the majority of workers are of this opinion.

It would appear that the diagnosis of "geriatric nutritional eczema" has been suggested without full appreciation of such factors as normal physiological variations/

variations due to age and to posture and from examination of an inadequate number of cases.

It therefore seemed necessary that further investigation be carried out to determine whether the commoner dermatoses which occur in elderly subjects are associated with malnutrition and whether laboratory evidence of this can be obtained by investigating the serum proteins and the cellular elements of the blood. The methods employed in the present investigation will now be described.

PART V. METHODS.

Blood specimens for investigation were obtained from subjects in the post-absorptive state, at least three hours after a meal.

The majority of subjects were investigated soon after admission to hospital and then again when the skin had healed. All repeated investigations in each subject were performed on blood obtained at the same hour of day and under the same conditions of bed rest, except in the cases where postural observations were carried out. Specimens for two groups of postural change observations were obtained as follows. Group A subjects were investigated while fasting in bed at 8.0a.m. and then again while still fasting, but seated at 9.0a.m., after having been allowed up to walk and sit in the ward for one hour. Group B subjects were ambulant on admission and were investigated while seated on the day of admission and then again at 24 hour intervals for a few days, during which time they were confined strictly to bed.

Five ml. of blood were withdrawn with minimal stasis from a brachial vein into a dry syringe and mixed with solid anticoagulant as recommended by Wintrobe, (i.e. 0.2ml. of a mixture of 2% potassium oxalate and 3% of ammonium oxalate.) This blood was used for erythrocyte and leucocyte/

leucocyte counts and for packed cell volume and haemoglobin estimations.

The needle was left in the vein and 10 ml. of blood allowed to run into a dry test-tube for protein, total serum base and thymol turbidity estimations. Blood for urea estimation and for Wassermann Reaction was then collected from the needle. Films for staining were made with blood obtained from the lobe of the ear.

Blood counts were performed and Wintrobe tubes set up for the erythrocyte sedimentation rate and for the packed cell volume determination within half-an-hour of obtaining the blood specimen.

Erythrocyte and leucocyte counts were made using a Neubauer counting chamber. Red cell dilution was 1 in 200 and the corpuscles in 80 small squares were counted. White cell dilution was 1 in 20 and the corpuscles in the whole of the cross-ruled area of the counting chamber were enumerated.

Blood films were stained with Leishman's stain and 200 cells counted in the differential cell count.

Packed cell volumes were measured by centrifuging the Wintrobe tubes at approximately 2,500 revolutions per minute for half-an-hour and then for a further ten minutes. Identical results were almost constantly obtained in a number of/

of duplicate estimations. Discrepancies were never greater than 0.5 per cent. No correction for trapped plasma was made.

Erythrocyte sedimentation rates were carried out by setting up the Wintrobe tubes in the true vertical position which was found by spirit level. Readings were taken at the end of the first and second hours.

Haemoglobin estimations were performed within one hour of withdrawal of blood. 0.2 ml. of blood were diluted to 25 ml. with 0.05 per cent. ammonia and the deflection read in an EEL photoelectric colorimeter. An Ilford tricolour green filter (404) was used. EEL readings were converted to haemoglobin in gm. per cent. from a standard calibration curve prepared by the senior staff of the Department of Biochemistry. Colour indices were calculated using 14.8 gm. haemoglobin as 100 per cent. of normal and five million red blood corpuscles as 100 per cent. of normal for males and females. The mean corpuscular haemoglobin, the mean corpuscular volume and the mean corpuscular haemoglobin concentration were calculated according to the method of Whitby and Britton (1950).

The blood for serum investigations was allowed to clot for half-an-hour, centrifuged at approximately 2,500 revolutions per minute for 10 minutes and the serum withdrawn into a clean, dry test-tube and placed in the refrigerator.

No/

No haemolysed serum was used. Serum protein, thymol turbidity and serum total base estimations were performed on the serum within two days.

Serum proteins were estimated by the Biuret method of Gornall et al. (1949). Some difficulty was experienced in the initial estimations because of the tendency for the solutions for the estimation of the albumin fraction to crystallise after having been centrifuged. The method was therefore modified by placing them in a water bath at 40°F. for 10 minutes before centrifuging. Trial, daily, duplicate estimations on serum kept for five days in the refrigerator gave EEL scale readings as follows:-

Total Protein.	1st day.	2nd day.	3rd day.	4th day.
Scale Reading.	22.8; 22.7	22.8; 22.8	22.6; 22.5	22.6; 22.5
Albumin. Scale Reading.	13.2; 13.2	13.1; 13.2	13.4; 13.6	13.2; 13.2

Therefore no gross changes were apparent in serum kept for a few days in the refrigerator. This is in agreement with the findings of Gornall et al. (1949) and of other workers.

All estimations were performed in duplicate. Almost all recorded duplicate readings were in agreement within 0.2 on the EEL scale, although a few differed by 0.4.

When/

When greater discrepancies occurred, the estimation was repeated.

Biuret reagent was prepared by the Senior Clinical Biochemist. Each new batch of Biuret reagent was tested by repeated estimations which were in close agreement with results obtained from estimations using the previous Biuret solution, as shown below.

Case No.	Date.	Biuret Solution No.	EEL Scale Reading.	Biuret Solution No.	EEL Scale Reading.
1	8. 6.50	1	28.2; 28.2	2	28.0; 28.2
10	29. 5.50	1	26.0; 26.0	2	26.2; 26.2
17	3. 6.50	1	22.0; 21.8	2	21.6; 21.4
18	20. 7.50	1	11.5; 11.5	2	11.5; 11.5
23	14. 9.50	2	26.4; 26.8	3	26.6; 26.8
23	14. 9.50	2	17.6; 17.6	3	17.6; 17.6
26	16. 9.50	2	23.4; 23.4	3	23.4; 23.0
26	16. 9.50	2	14.6; 14.8	3	14.6; 14.5
39	11. 4.51	3	25.6; 25.4	4	25.4; 25.4
39	16. 4.51	3	23.2; 23.2	4	23.0; 22.8
40	13. 4.51	3	22.4; 22.6	4	22.4; 22.2
41	20. 4.51	3	18.6; 18.6	4	18.6; 18.4

Total/

Total protein estimation on a specimen of serum was carried out by the Micro-Kjeldahl method by the Senior Clinical Biochemist. Saline dilutions of this serum were then prepared and mixed with 8 ml. of biuret reagent and photoelectric readings taken after 30 minutes. Results are expressed in the following table.

0.9% Saline.								
ml.	2.0	1.85	1.7	1.55	1.4	1.2	1.0	0.8	0.6
Serum.									
ml.	0	0.15	0.3	0.45	0.6	0.8	1.0	1.2	1.4
EEL Scale Reading.									
	0	4.4	8.4	12.8	17.2	22.4	27.4	33.4	38.2
Total Protein.	Calculated from Micro-Kjeldahl result *								
Gm.%	0	1.128	2.256	3.384	4.512	6.016	7.52*	9.024	10.528

A graph, plate No. 1, (Volume 1, page 38), was then prepared, incorporating photoelectric readings and calculated protein values for each dilution. This was used to convert all photoelectric readings obtained in the investigation of cases, to protein in gm. per cent. Results are presented in Volume 2, Table 5, pages 127 to 140.

Glassware was steeped in cleaning mixture overnight (10% potassium/

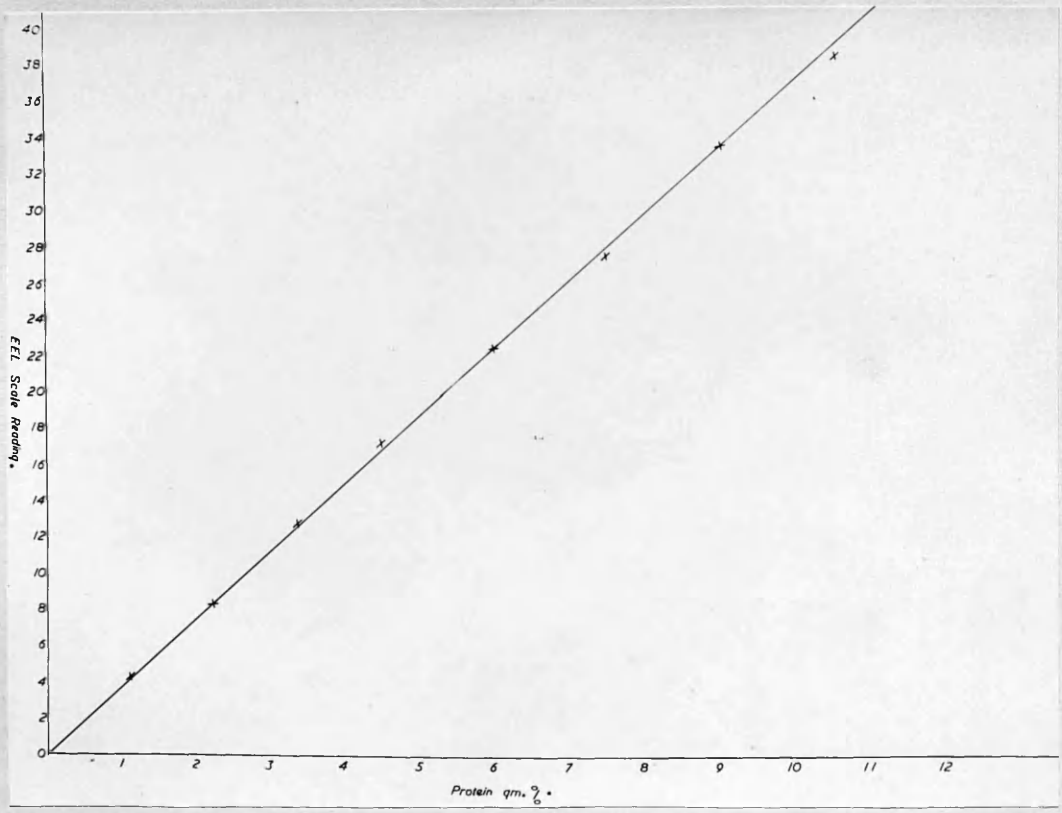

Plate 1. Shewing graph used for converting EEL scale readings to protein in gm. per cent.

potassium dichromate in 25% sulphuric acid), rinsed in running water 20-30 times, steeped in distilled water overnight and oven-dried. Pipettes were marked and each pipette kept for the same stage in all estimations. They were cleaned with running water by suction pump for 30 minutes, rinsed in distilled water and oven-dried. At intervals they were steeped in cleaning mixture overnight before cleaning with running water and distilled water.

Serum total base estimations were performed by the method of Sunderman (1942) using the Sunderman Conductivity Bridge. All readings were taken at 25° and the cell constant was checked on several occasions throughout the course of the investigations. Serum total base was calculated from the formula

$$T.B. = 10.19\ (K \times 10^3) - \frac{69.45}{Pr.} + 35.16$$

where T.B. equals serum total base in milli-equivalents per litre, $K \times 10^3$ = specific conductance x 10^3 at 25° and Pr. = Gm. protein per 100 ml. Results are presented in Volume 2, Table 6, pages 141 to 144.

Repeated estimations were performed on four different samples of serum and readings showed an error of less than one per cent. of total base, as follows.

Serum No.	Date.	Serum Conductivity.	Serum Total Base m.Eq./l.
1	12. 3.51	12.28	148.9
"	13. 3.51	12.28	148.9
"	16. 3.51	12.2	148.1
2	13. 3.51	12.5	151.7
"	16. 3.51	12.42	150.8
3	14. 3.51	12.4	149.7
	15. 3.51	12.3	148.6
	16. 3.51	12.3	148.6
4	28. 2.51	12.5	152.0
	29. 2.51	12.4	150.9
	30. 2.51	12.45	151.4

Thymol turbidity tests were performed by the method of MacLagan (1944).

Fractional test meals were performed, using gruel, as the method. Specimens were withdrawn half-hourly. Quantitative estimations of free and total acid were made according to the method in Notes in Clinical Laboratory Methods, page 67. Histamine was not employed. It was considered that the gruel response would approximate at least/

least as closely to the normal physiological food response. Again the propriety of administering even small doses of histamine to elderly people, some of whom were suffering from extensive skin conditions, was doubtful. Furthermore, previous reports on gastric acidity in dermatoses had not been based on histamine meals. Finally, it was intended to determine what effect histamine release phenomena in dermatoses may produce on gastric secretion.

Urine was examined for albumin by the heat test, for sugar by Benedict's test and for fixation of specific gravity, as they are described in Notes in Clinical Laboratory Methods.

PART VI. DISCUSSION.

Assessment of the findings in any investigation of the elderly is rendered more complex by the diverse pathological processes which may be found together in any one subject. Furthermore, in the elderly, difficulty arises in deciding what are normal as distinct from pathological findings. In diagnosis, no great reliance can be placed upon ancillary aids such as the electrocardiograph, since minor degrees of cardiac abnormality are to be expected in a high proportion of cases investigated by this method. Again, in disease of the skin, exact diagnosis is not always certain and several etiological factors may be present concomitantly.

A. Clinical Aspects. Subjects were selected for investigation first by reason of age and with two exceptions, (numbers fifty-one and fifty-two), they were all over sixty years of age. A further selection was then made to include the more common dermatoses in which the skin of the lower limbs was affected. In addition five subjects, in whom the diagnosis of pemphigus was made and three subjects, diagnosed as suffering from bullous drug eruptions, were investigated. The skin diseases which were present in the fifty-two subjects may be divided into the following groups: varicose conditions, including varicose eczema and varicose ulcers, - eighteen cases;/

cases; infectious eczematoid dermatitis - seventeen cases; scurvy - nine cases; pemphigus and bullous drug eruptions - eight cases. A distinction between infectious eczematoid dermatitis and varicose eczema in the elderly cannot be truly precise, but the diagnosis of infectious eczematoid dermatitis was made when no definite varicosity of leg veins was present and when no history of deep vein thrombosis was obtained. The diagnosis of scurvy was made on clinical grounds. It was not possible to carry out ascorbic acid saturation tests in all nine cases and the seven saturation tests recorded are not altogether satisfactory, since careful collection of urine spec-:imens in elderly subjects was sometimes difficult. To simplify procedures, all subjects were given a standard dose of 700 mgm. vitamin C during the saturation tests and no calculation was made for body weight. The tests, however, do shew a fair degree of vitamin C deficiency. The diagnosis of pemphigus vulgaris was made in three subjects and pemphigus foliaceus was diagnosed in two subjects. No single, absolute point of differentiation exists between pemphigus vulgaris and dermatitis herpetiformis. Tzanck's test is of doubtful value. It is considered, however, that the diagnosis of pemphigus vulgaris was certain in two subjects. In case 26, it was perhaps less certain, but taken as a whole, the clinical picture seemed to warrant its inclusion in the pemphigus/

pemphigus group. Case reports are presented in Volume II, pages 1 to 111.

The general medical condition was assessed in each subject and this will now be considered. Detailed discussion of blood findings will be reserved for future consideration.

Hypertension was present in nineteen subjects. Arbitrary levels of 180 mm. Hg. systolic pressure and of 100 mm. Hg. diastolic pressure have been taken as upper limits of normal. Howell (1949) found that in elderly subjects, high systolic and large pulse pressures were frequently encountered. In seventy-five per cent. of subjects, the author found that the systolic pressure was over 160 mm. Hg. and in twenty-five per cent. of subjects he found it to be over 200 mm. Hg. Hyper-:tension per se plays no part in the production of oedema or in the alteration of serum protein concentrations. Mufson (1932) found no constant relation between brachial and capillary pressures in essential hypertension. No oedema was present in his subjects and serum proteins were normal. Keys et al. (1950) stated that the arterial blood pressure has little significance in oedema formation, since there is a large fall in pressure in arterioles. Venous pressure however has a direct importance and where some degree of congestive cardiac failure was present in association with hypertension, as in cases 10, 25, 26 and 44, the oedema may have been due to increased venous pressure.

The/

The nineteen cases in which hypertension was present may be grouped as follows: varicose conditions - nine cases; infectious eczematoid dermatitis - seven cases; pemphigus - two cases; drug eruption - one case. As might be expected, no scurvy was present in this group. Wiener (1947) stated that hypertension may be associated with arteriosclerosis of vessels in the dermis and therefore may be an etiological factor in pruritus and eczema. However, since arteriosclerosis may appear in local, isolated areas of the dermis and is seldom if ever generalised, this opinion has not received general support.

Signs of mild, congestive, cardiac failure with oedema were present in four subjects (numbers 10, 25, 26, and 44). No enlargement of liver was detected. No gross abnormalities in the serum protein concentrations were present. In the subject with pemphigus vulgaris in this group (number 26), the gross oedema and dyspnoea disappeared within a week after admission to hospital. The condition appeared to be a general toxaemia due to pemphigus.

Thyroid dysfunction was present in one subject (number 18). Oedema and diminished serum proteins were due to metabolic upset. Some degree of cardiac failure was also present.

Chronic bronchitis was present in six subjects (numbers

9,/

9, 20, 32, 40, 47 and 49) and in two of these (numbers 9 and 49) emphysema was also noted. No instance of congestive cardiac failure was found in association and no alteration in serum protein concentrations, apart from possible increase in globulin, would result from the pulmonary condition.

Albuminuria was present in four subjects (numbers 22, 25, 27 and 52) and pyuria was noted in four cases (numbers 5, 8, 11 and 13). The serum proteins were not below normal limits in those subjects with the exception of number 22 (pemphigus vulgaris) where the concentration was at the lower limit of normal. In this instance, albuminuria was probably associated with the pemphigoid skin condition. Senile renal arterio-:sclerosis may have been the cause of the albuminuria in the other three subjects.

Slight glycosuria was present in two subjects, (numbers 37 and 52), but sugar tolerance tests were normal. Therefore no gross changes in serum protein concentrations would result from dysfunction of the pancreas.

Blood urea results showed a moderate increase above 40 mgm. per cent. in twelve subjects. Stewart (1947) found no unusual values in blood urea and urea clearance tests in the elderly. Howell and Piggot (1948) investigated blood urea values in one hundred and three elderly subjects and found that in thirty-one per cent. of subjects, the blood urea was over 40 mgm./

40 mgm. per cent. and that, in six per cent. of subjects, the blood urea was over 50 mgm. per cent. The upper limit of normal may be considered as 50 mgm. per cent. In the present investigation, subjects 5 and 37 showed a blood urea of 57 mgm. per cent. and 52 mgm. per cent. respectively. The former subject was aged 93 years and it is not considered that the blood urea level of 57 mgm. per cent. indicated any significant renal dysfunction. In the latter subject, no abnormality was detected in the urine and the blood urea of 52 mgm. per cent. is not regarded as a significant finding. A low serum protein concentration was present in only one of these twelve subjects (number 50). The blood urea level of 44 mgm. per cent. in this subject cannot be regarded as significant.

In conclusion then, the presence of oedema may be explained on the grounds of cardiac failure in five subjects (numbers 10, 18, 25, 26 and 44). Diminished serum proteins may have been due to thyrotoxicosis in one subject (number 18) and to albuminuria in one subject (number 22).

B. The Biochemical and Haematological Observations.

Before proceeding to examine the data presented in Tables I, IIA, IIB, III and IV, volume II, it is necessary to define the limits of physiological variation as accurately as possible. Assessment of serum protein and haematological values is complicated by the fact that variations due to ingestion/

ingestion of food, changes of temperature, differences of age, physical activity and alterations of posture have been reported. With regard to the first factor, the investigations were designed to rule out gross variations and it will not receive further consideration. No provision could be made to obviate differences due to temperature changes, but as subjects were not under investigation during extreme changes of temperature, variations from this cause would be of small degree and need not be considered. The subjects, with two exceptions, were all over sixty years of age and were not confined strictly to bed, except in the few instances where postural changes were investigated. It is therefore necessary to discuss the normal serum protein and haematological findings in the elderly and the variations which occur due to postural changes.

(1.) Serum Proteins in the Elderly. Ten normal controls, all over the age of sixty years, were investigated after a preliminary period of three days' bed rest. The results are presented in Table III, Volume II, page 121, The controls were normal in the sense that they had no skin disease and that no gross signs of disease were detected on clinical examination. They were all hospital patients who had recovered from slight cerebral thrombosis or who were suffering from mild senile dementia or who had been admitted

to hospital largely because of poor home circumstances. While the controls cannot be considered to be truly repres-:entative of the elderly population in general, they had all been in hospital for several months during which time they had been taking an adequate diet.

The average values found in the ten controls were as follows: total serum protein 6.56 gm. per cent., serum albumin 4.19 gm. per cent., and serum globulin 2.37 gm. per cent. The range of values was as follows: total serum protein 6.2 to 7.2 gm. per cent., albumin 3.9 to 4.45 gm. per cent. and globulin 1.9 to 3.2 gm. per cent.

Few reports on blood proteins in the aged have appeared in the literature. Jones (1887), investigating the specific gravity of blood in different age groups, found lower values in the elderly. The inference is that serum protein values are lower in the higher age groups. In the report of the Medical Research Council (1945), the average serum total protein value for nine subjects over sixty years of age was 6.572 gm. per cent., with a range of 6.17 to 6.97 gm. per cent. Bing et al. (1947), using Henriques and Klausen's method, did not find any variation in the serum total proteins in higher age groups in males, although after the age of thirty-five years, the albumin concentrations were somewhat diminished. In females over the age of thirty-five years, the authors/

authors
found a greater deviation from normal serum protein concentrations.

Bock (1948), using the method of Henriques and Klausen, found a significant fall in the albumin fraction with increasing age, and that this fall was more marked in females. He also found an increase in the globulin fraction. In both sexes, the serum total protein varied from 5.5 to 7.4 gm. per cent. and the albumin ranged from 3.2 to 5.2 gm. per cent. Olbrich (1948), using the micro-Kjeldahl method, found that total protein values decreased with advancing years. The range of values for plasma protein varied from 4.02 to 7.14 gm. per cent.

Detailed results may be tabulated as follows:

Author.	Number of cases.	Age	Sex.	T.Pr. Range and/or Mean Gm.%	Alb. Range and/or Mean Gm.%
Bing.	34	over 35	F.	Range (5.6 - 8.2	Range (3.5 - 5.4
			M.	Range (6.3 - 8.1	Range (3.9 - 5.4
Bock.	76	(60 (95	(M. (F.	Range (5.5 - 7.4	
	41	over 60	F.	6.64	Range (60-95 yrs.
		over 80	F.	6.25	(3.2 - (4.8
	35	over 60	M.	6.63	Range (60-95 yrs.
		over 80	M.	6.33	(3.2 - (5.2
Olbrich.	41	over 60	F.	5.51	3.30
	37	over 60	M.	5.15	3.18
M.R.C.	9	over 60	M.(F.(	6.57	-
Present writer.	10	over 60	M.(F.(	6.56	4.19

The/

The inmates of Queensberry House who were studied by Olbrich may not have been representative of the general population, and the average serum protein values given by the other authors are in agreement with the average values found in the ten controls in the present investigation. The range of values found by Bock would therefore appear to be relevant to the present investigation and this range has been adopted to determine the lower limits of normal, since it has been obtained from a significant number of cases. Accordingly, the lower limit of normal in the elderly, may be taken as 5.5 gm. per cent. for serum total protein and as 3.0 gm. per cent. for serum albumin.

(2) Postural Variations affecting the Blood. Data regarding changes which occurred in the blood when alteration of posture took place are presented in Tables 2A and 2B, Volume 2, pages 119 and 120, and in Plate 2, Volume I, pages 53 and 54. The changes which occurred in blood values in fast-:ing subjects, after they had been allowed up for one hour, are shown in Table 2A. A significant increase in serum protein took place after the upright posture had been assumed for one hour. A fall in the serum total base occurred, but this was not great enough to be significant. The increase in serum total protein varied from 0.25 to 0.85 gm. per cent., the increase in serum albumin varied from 0.15 to 0.67 gm. per cent. and the increase in serum globulin varied from 0.04

to/

Plate 2. Shewing variation in blood values one hour after upright posture was assumed. Hb. = haemoglobin gm. per cent., R.B.C. = red blood cells millions per c.mm., P.C.V. = packed cell volume ml. per cent., T.Pr. = serum total protein gm. per cent., Alb. = serum albumin gm. per cent., Glob. = serum globulin gm. per cent., S.T.B. = serum total base m.Eq./l.

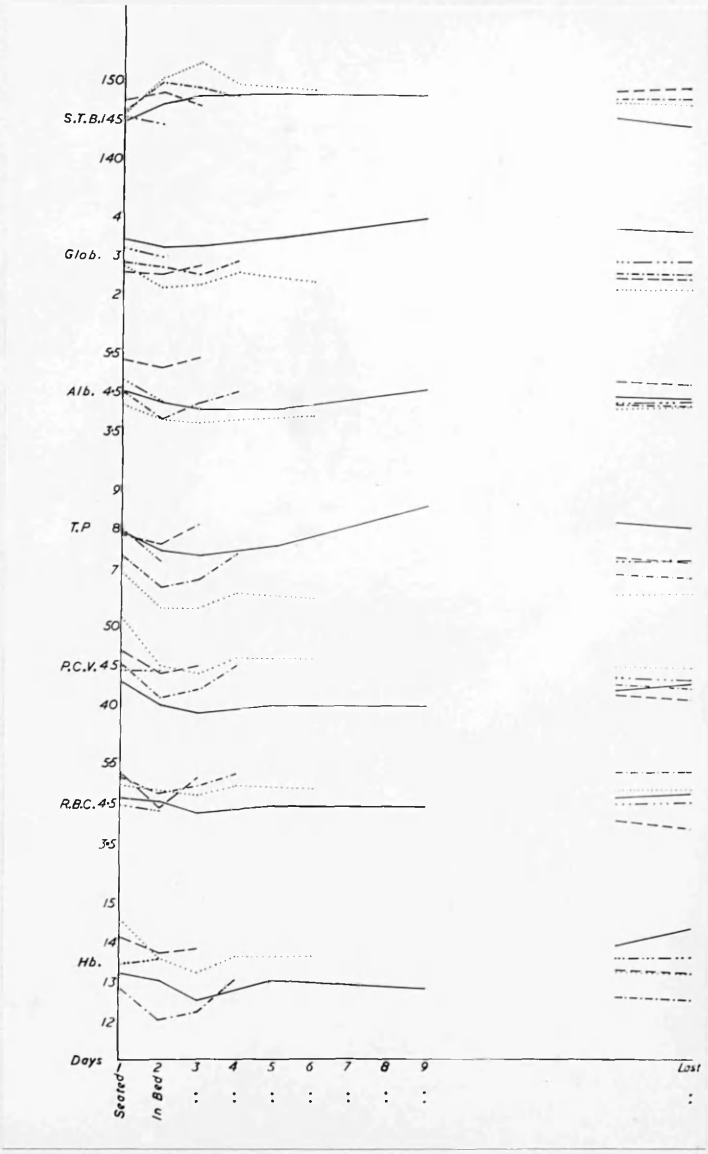

Plate 2 (Continued). Shewing daily variation in blood values when recumbent posture was assumed.
Hb. = Haemoglobin gm. per cent., R.B.C. = red blood cells millions per c.mm., P.C.V. = packed cell volume ml. per cent., T.Pr. = serum total protein gm. per cent., Alb. = serum albumin gm. per cent., Glob. = serum globulin gm. per cent., S.T.B. = serum total base m.Eq./l.

to 0.48 gm. per cent. The average increase was as follows: serum total protein - 0.535 gm. per cent.; serum albumin - 0.3 gm. per cent.; and serum globulin 0.316 gm. per cent.

The changes which occurred in blood values as a result of bed rest are shown in Volume II, Table 2B, and in Plate 2, Volume I, page 54. The subjects were investigated on admission while they were in the sitting position. They were then confined strictly to bed and investigated at daily intervals for a few days and then at longer intervals until the skin had healed. A significant fall in serum protein took place after twenty-four hours of bed rest, A rise in serum total base occurred, but this was not a significant increase. After the first twenty-four hours of bed rest, the decrease in serum total protein varied from 0.23 to 0.9 gm. per cent., the decrease in serum albumin varied from 0.2 to 0.7 gm. per cent. and the decrease in serum globulin varied from 0.03 to 0.55 gm. per cent. The average decrease was as follows: serum total protein - 0.65 gm. per cent.; serum albumin - 0.43 gm. per cent.; and serum globulin 0.22 gm. per cent. The values rose again as the period of bed rest continued and, in three subjects, reached the level found at the initial investigation. It is not possible to state whether such a rise to the "sitting" level may be a constant finding in complete bed rest. It is doubtful

whether all the subjects of this investigation continued to remain strictly in bed during the final days of investigation.

In both groups of postural studies, subjects with oedema showed no greater variation than those with no oedema. It would appear that haemodilution occurs during the early stages of bed rest and that haemoconcentration occurs on resumption of the upright position. The small but consistent changes, in haemoglobin and red cell percentages and in packed cell volumes which took place in both groups of postural studies, tend to support this conclusion. Stewart and Rourke (1938), Stead and Ebert (1941), Lyons et al. (1944) and others have shown that changes in haematocrit values failed to re--flect quantitatively, changes in plasma volume. Nevertheless, according to Ebert and Stead (1941), although accurate estimations of plasma volume changes cannot be made from haematocrit readings, it would seem reasonable to assess the direction of change in plasma volume from the change in haematocrit reading and haemoglobin concentration.

Similar changes in the blood due to alteration of posture have been reported by other workers. Thompson et al. (1928) showed that a decrease in total plasma volume of 11 per cent. and an increase in total protein of 0.5 to 1.4 gm. per cent. took place in the standing position. Youmans et al. (1934) demonstrated/

demonstrated that standing for one hour caused an increase of 18 to 40 per cent. in serum total protein concentration. Perera and Berliner (1943) found that a significant change occurred in a few minutes, although a longer period was required for a maximal effect. They found an average fall in serum total protein of 0.8 gm. per cent. after rest in the horizontal position. Renbourn (1947) showed that horizontal rest for thirty minutes caused a mean fall of 0.35 gm. per cent. plasma protein and that bed rest for 48 hours produced a mean fall of 0.65 gm. per cent. plasma protein. Spealman et al. (1947) found that plasma proteins increased in amount when the upright position was maintained and decreased in the recumbent position. Widdowson and McCance (1950) showed that mean serum total proteins fell 0.48 gm. per cent. after two hours bed rest and that they had not returned to normal after three days.

It is evident then, that in any individual subject, variations in serum total protein up to 0.9 gm. per cent., in serum albumin up to 0.7 gm. per cent. and in serum globulin up to 0.55 gm. per cent., cannot be attributed to pathological changes and that an increase of this order cannot be considered due to a response to treatment or to a recovery from malnutrit-:ion, since postural variations provide an adequate explan-:ation. Again, in any group of subjects, an average increase in/

in serum total protein up to 0.7 gm. per cent., in serum albumin up to 0.5 gm. per cent. and in serum globulin up to 0.4 gm. per cent. cannot be considered as significant of a response to treatment. These values for individual and for group variations have been taken as the maximum individual and average variations noted from Tables 2A and 2B.

The average haematological variations which occurred with postural alterations were as follows: Volume II, Table 2A, haemoglobin - 0.43 gm. per cent.; red blood corpuscles - 0.313 millions per c.mm.; and packed cell volume - 2.3 ml. per cent.: Volume II, Table 2B, haemoglobin - 0.44 gm. per cent.; red blood corpuscles - 0.33 millions per c.mm.; and packed cell volume 3.0 ml. per cent.

The variations in red cell numbers was not significant, but the change in haemoglobin and in packed cell volume was sufficiently large to be a significant finding. Average increases of more than 0.5 gm. haemoglobin per cent. and of more than 3.0 ml. per cent. in packed cell volume are therefore necessary before they can be attributed to a response to treatment in any group of subjects.

(3.) Serum Total Protein Observations. Results are presented in Volume II, Tables 1 and 2A. Of the fifty-two subjects who were investigated, three showed a total protein level/

level of 5.0 gm. per cent. or less (cases 18, 41 and 50). Oedema was present in all three. In case 18, infectious eczematoid dermatitis and thyrotoxicosis, the serum total protein concentration was 4.75 gm. per cent. The increase in catabolism due to thyroid dysfunction accounts for the low protein value. A significant increase in serum protein took place as improvement occurred in the general condition. Extra protein in the form of "Casinal" was given in the diet and may have played some part in the clinical improvement. After treatment with thiouracil, a further increase in the serum protein concentration took place, but as the patient was sitting at the time of this investigation, the increase was not significant. In case 41, varicose eczema was com-:plicated by a multiform sulphonamide eruption and the serum total protein value of 5.0 gm. per cent. may be explained as the result of the increase in capillary permeability which apparently occurs in this drug eruption. In case 50, pemphigus vulgaris, the serum total protein concentration of 4.95 gm. per cent. was in agreement with findings by other workers who have investigated similar cases. The increase of serum total protein to 5.8 gm. per cent. when aureomycin and "Casinal" were administered, paralled the improvement in the general condition of the patient and the disappearance of oedema and bullae, but cannot be considered to be a significant increase./

increase. The marked improvement in the skin condition was maintained in this patient, but the general condition deteriorated steadily.

Four subjects, (numbers 16, 22, 31 and 32), showed a serum total protein value between 5.5 and 5.0 gm. per cent. Of these, three were cases of scurvy with oedema, and one was a case of pemphigus vulgaris with no oedema. In the last case, it is interesting to note that "Casinal" had no effect on the serum total protein which fell from 5.6 to 5.2 gm. per cent. and that it did not prevent a recurrence of bullous lesions. At the initial investigation, a course of "Stovarsol" had just been completed and no bullae were present.

In eleven subjects, (numbers 1, 5, 17, 26, 35, 36, 37, 39, 44, 46 and 47), the serum total protein was between 5.6 and 6.0 gm. per cent. The cases may be listed as follows: infectious eczematoid dermatitis - five cases, of which three showed oedema; varicose eczema - two cases, of which one showed oedema; scurvy - three cases, of which two showed oedema; and one case of pemphigus vulgaris with oedema.

In thirteen subjects, numbers 2, 3, 4, 7, 14, 21, 34, 38, 40, 45, 48, 51 and 52) the serum total protein was between 6.0 and 6.5 gm. per cent. The cases may be listed as follows: infectious eczematoid dermatitis - four cases, of which two showed oedema; varicose conditions - four cases, of which two/

two showed oedema; scurvy - three cases, of which one showed oedema; and pemphigus - two cases, none of which showed oedema.

In twenty-one subjects, (numbers 6, 8, 9, 10, 11, 12, 13, 15, 19, 20, 23, 24, 25, 27, 28, 29, 30, 33, 42, 43 and 49), the serum total protein was above 6.5 gm. per cent. The cases may be listed as follows: varicose conditions - twelve cases, of which five showed oedema; infectious eczematoid dermatitis - seven cases, of which five showed oedema; drug eruptions - two cases, none of which showed oedema.

Plate 3, page 62, illustrates the serum total protein concentrations in the various groups which were investigated.

To summarise the findings then, in uncomplicated infectious eczematoid dermatitis and in uncomplicated varicose conditions, normal serum total protein concentrations were found. The values in varicose conditions tended to be higher than in the other groups. The serum total protein concentrat-:ions in scurvy and in pemphigus vulgaris tended to be low. Of the five subjects in whom the serum total protein concentrat-:ion was below the lower limit of normal, (5.5 gm. per cent.), two were suffering from scurvy, one from pemphigus vulgaris, one from varicose eczema complicated by a multiform sulphonamide eruption and one from infectious eczematoid dermatitis complicated by thyrotoxicosis.

No/

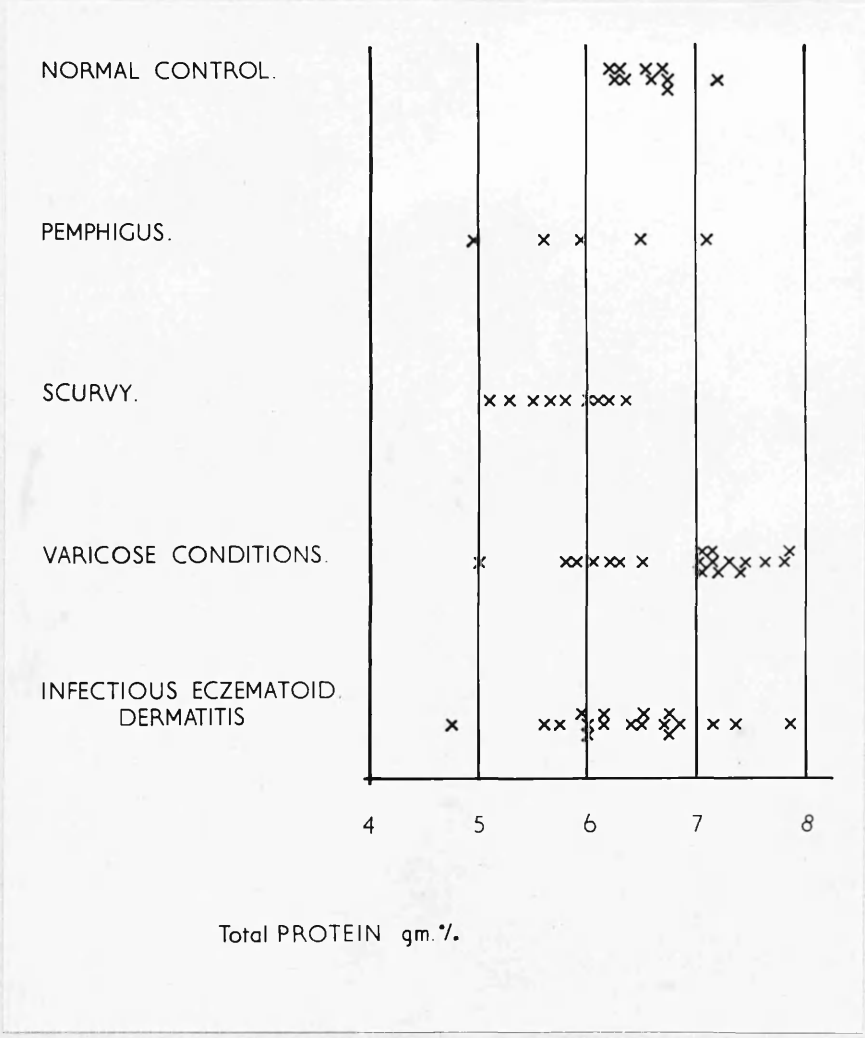

Plate 3. Shewing serum total protein scatter. (gm. per cent.)

No relation between serum total protein concentration and oedema was apparent, since oedema occurred at all levels of protein, but oedema was present in all subjects in whom the serum total protein was under 5.0 gm. per cent. and in three of the four subjects in whom the value was between 5.5 and 5.0 gm. per cent.

(4.) Serum Albumin Observations. Results are presented in Volume II, Tables I and 2A. With regard to the serum albumin, only one subject (number 22) showed a concentration under 3.0 gm. per cent. The diagnosis was pemphigus vulgaris. No oedema was present.

In ten subjects, (numbers 1, 16, 18, 21, 32, 35, 37, 41, 46 and 50) the serum albumin was between 3.0 and 3.5 gm. per cent. The cases may be listed as follows: infectious eczematoid dermatitis - three cases, which all showed oedema; scurvy - four cases, three of which showed oedema; pemphigus - two cases, one of which showed oedema; and one case of multiform drug eruption with oedema.

In eighteen subjects, (numbers 2, 3, 4, 5, 8, 14, 17, 25, 26, 31, 33, 34, 36, 38, 39, 40, 44 and 47), the serum albumin was between 3.6 and 4.0 gm. per cent. The cases may be listed as follows: infectious eczematoid dermatitis - ten cases, five of which showed oedema; scurvy - four cases, three of which showed oedema; varicose conditions - three cases, two of which/

which showed oedema; and one case of pemphigus with oedema.

In twenty-three subjects, (numbers 6, 7, 9, 10, 11, 12, 13, 15, 19, 20, 23, 24, 27, 28, 29, 30, 42, 43, 45, 48, 49, 51 and 52), the serum albumin was 4.1 gm. per cent. or above. The cases may be listed as follows: varicose conditions - 15 cases, six of which showed oedema; infectious eczematoid dermatitis - four cases, three of which showed oedema; two cases of drug eruption with oedema; one case of pemphigus with no oedema; and one case of scurvy with no oedema.

Plate 4, page 65, illustrates the serum albumin concentrations in the various groups which were investigated. In only one case, pemphigus vulgaris, was the serum albumin concentration below the lower limit of normal. The serum albumin concentrations tended to be low in scurvy, infectious eczematoid dermatitis and pemphigus vulgaris. The highest values were found in varicose conditions. No relation between serum albumin concentration and oedema was apparent, since oedema was found at all levels of serum albumin. However, the number of subjects with oedema in each group diminished, at increasing albumin concentrations.

(5.) Serum Globulin Observations. Results are presented in Volume II, Tables I and 2A. Plate 5, page 66, illustrates the serum globulin concentrations in the various groups which were investigated.

The/

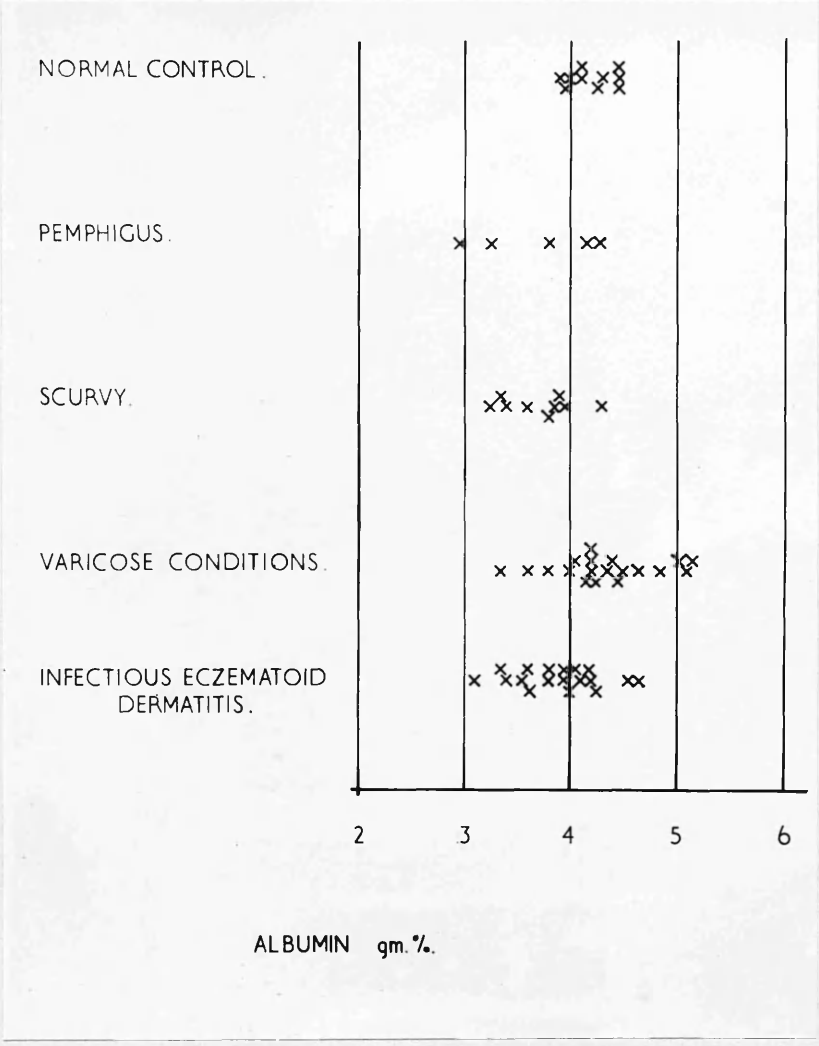

Plate 4. Shewing serum albumin scatter (gm. per cent.)

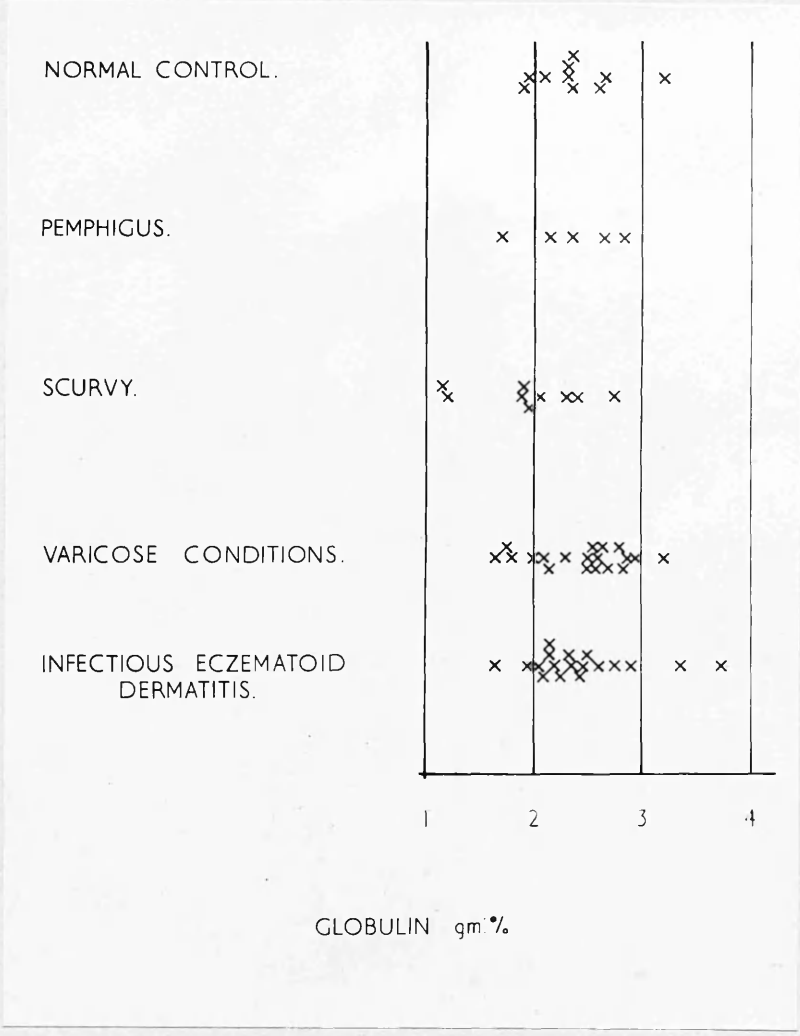

Plate 5. Shewing serum globulin scatter. (gm. per cent.)

The lowest values were found in two subjects who were suffering from scurvy. In general the serum globulin concentrations tended to be high. This may have been due to infection, but the subjects who were suffering from chronic bronchitis, with the exception of number 20, did not show any marked increase in serum globulin. Varicose conditions tended to have higher globulin levels than the other groups. No apparent relation was present between serum globulin concentration and oedema.

Subjects have been grouped into those with oedema and those with no oedema and are listed in Volume II, Table 7, pages 145 to 150. It is evident that a wide scatter in protein values was present in all groups, although the average serum protein values were lower in the male subjects with oedema than in those with no oedema. In the male subjects with oedema, the average serum total protein and serum albumin values were moderately increased after treatment. This may have been due to the fact that six subjects received protein supplemented diets.

In general then, oedema cannot be explained on the basis of low serum protein in the subjects of this investigation. It is probable that mechanical factors such as diminished tissue elasticity, impaired lymphatic drainage and local venous congestion were responsible for oedema in the uncom-

:plicated/

uncomplicated cases of infectious eczematoid dermatitis and varicose eczema. Evans et al. (1943) demonstrated that senile skin showed a marked difference in the connective tissue beds when compared with skin from younger individuals. Greater shrinkage occurred in the connective tissue in younger specimens. Oedema formation therefore takes place more readily in the elderly subject.

(6.) Haematological Observations. Table 3, Volume II, page 121, presents the average values for ten normal controls which were: haemoglobin - 13.2 gm. per cent.; packed cell volume - 44 ml. per cent.; mean corpuscular volume - 95 cubic microns; and mean corpuscular haemoglobin concentration - 30 per cent. In control number six, a moderate degree of hypochromic, normocytic anaemia was present. In control number seven, the total leucocyte count was at the upper limit of normal, but apart from this, the leucocytes showed no significant aberrations. In general then, a degree of macrocytosis was apparent in the control series. Iron deficiency was not a prominent feature, although mean corpuscular haemoglobin concentrations were all at the lower limit of normal.

Many workers have reported on blood findings in the aged and reports regarding haemoglobin levels are not in agreement. This is no doubt due in many instances to the differences in methods/

methods employed. Newman and Gitlow (1943), using Sahli's method, found the mean haemoglobin in fifty men who were aged over sixty-five years, to be 12.65 gm. per cent. and the mean haemoglobin in fifty women who were aged over sixty-five years, to be 14.5 gm. per cent. They found no great differences in the other haematological values in these subjects but the mean corpuscular volume was 97.7 cubic microns in the males and 90 cubic microns in the females. Olbrich (1947) found the mean haemoglobin in forty-one males who were over sixty years of age to be 13.9 gm. per cent. and in forty-seven females aged over sixty years, he found the mean haemoglobin to be 13.2 gm. per cent. The method used was that of Haldane and Gowers. This worker found the mean corpuscular volume to be 88.9 cubic microns for all subjects.

Howell (1948) did not find any change in haemoglobin levels in old age.

These authors all quote many other workers whose findings vary markedly. However, it would seem that the numbers of red and white blood cells in the elderly are not significantly different from those found in younger subjects. Haemoglobin may be rather lower in the elderly, but many authors give increased values. Most authors report some degree of macrocytosis in the red blood cells of the aged. In elderly subjects, the normal range for mean corpuscular volume would appear/

appear to be higher than in younger subjects and the term "normocytic" will therefore be used when the mean corpuscular volume is less than 115 cubic microns. The term "hypochromic" will be used when the mean corpuscular haemoglobin concentrat-:ion is less than 28 per cent. Anaemia, in the sense of a diminished quantity of circulating red cells, will be consid-:ered to be present when the packed cell volume is less than 40 ml. per cent.

Reviewing the haematological findings in the subjects who were investigated, (Volume II, Tables 1, 2A, 2B and 4.) it is evident that no gross iron deficiency was present: the mean corpuscular haemoglobin concentration was at the lower limit of normal in all cases. The lowest value found, 27 per cent., was in case 46 in which a macrocytic hypochromic anaemia was present. Iron was administered by mouth in a number of subjects, but no significant increase in the mean corpuscular haemoglobin concentration took place. This is, however, no certain proof that the concentration was at the normal levels for the elderly, since response to intravenous iron was not investigated.

A tendency towards macrocytosis was present in most subjects who were investigated: the mean corpuscular volume lay between 90 and 100 cubic microns in the majority of cases.

Normochromic normocytic anaemia was present in sixteen subjects,/

subjects, (numbers 2, 3, 8, 14, 16, 22, 25, 29, 31, 32, 33, 35, 37, 42, 50 and 51). The cases may be listed as follows: scurvy - seven cases; infectious eczematoid dermatitis - six cases; pemphigus vulgaris - two cases; and one case of varicose ulcer.

Advanced age is not associated with any significant changes in the white blood cells and the normal range may be taken as 4,000 to 11,000 cells per c.mm. A moderate leucocytosis was present in three subjects, (numbers 19, 22 and 26) and in the control number 7. Two of the subjects were suffering from pemphigus vulgaris and one from varicose eczema. Moderate leucopenia was present in six subjects, (numbers 29, 37, 41, 44, 45 and 51). They may be listed as follows: varicose conditions - three cases; scurvy - two cases; and one case of multiform drug eruption.

A neutrophil polymorphonuclear leucocytosis was present in case 19 (varicose eczema), case 26 (pemphigus vulgaris), case 38 (varicose ulcer) and in control number 7.

Eosinophylia was present in case 42 (infectious eczematoid dermatitis), case 50 (pemphigus vulgaris) and in case 52 (pemphigus foliaceus).

No pathological increase in the total lymphocytes or in the monocytes was present.

In conclusion then, the majority of subjects who were suffering/

suffering from scurvy showed a normochromic normocytic anaemia. Similar anaemia was present in one third of the subjects who were suffering from infectious eczematoid dermatitis.

(7.) Erythrocyte Sedimentation Rate Observations.

Erythrocyte sedimentation rates were performed by Wintrobe's method and the upper limit for normal values in males will be taken as 20 mm. at the end of the first hour and in females will be taken as 30 mm. at the end of the first hour. Olbrich (1948), using the Westergreen method, concluded that the sedimentation rate increases with advancing years within the limits of 9.0 - 14 mm. in the first hour. A divergence of opinion exists as to whether the sedimentation rate ought to be corrected for anaemia and the question is still undecided. Sedimentation rates were determined in forty-two subjects and, with no correction for anaemia, were found to be raised in twenty-seven cases. After the skin had healed, the sedimentation rates had fallen to normal levels in six subjects and had fallen only slightly in eight subjects. Seven subjects showed no change in the sedimentation rate when the skin had returned to normal, two showed an increase and in four subjects the investigation was not repeated. In nine subjects the sedimentation rate was at the upper limit of normal and when the skin had healed, three showed an increased rate, three showed no appreciable change and in three subjects the investigation was not repeated. In six/

six subjects the sedimentation rates were normal, but when the skin had healed the sedimentation rate had increased significantly.

It would appear that the sedimentation rate was not related to the skin condition in the subjects who have been investigated. This conclusion is in agreement with that of Strickler and Aliensis (1947) who stated that a raised erythrocyte sedimentation rate reflected some constitutional disturbance rather than the state of activity of the skin. It is probable that in the elderly subjects of the present study, minor sub-clinical infective processes may have been the cause of the raised sedimentation rates. In all six subjects in whom chronic bronchitis was found on clinical examination, the sedimentation rates were significantly raised and it is probable that minor degrees of respiratory infection may have been present in the other cases where the sedimentation rates were raised above normal. Table 8, Volume II, page 151, shows the initial sedimentation rates, found in all subjects, corrected for anaemia by means of the corpuscular volume according to the method of Hynes and Whitby (1938). With a corrected sedimentation rate, twenty-one subjects shewed raised values. They may be listed as follows: varicose conditions - eight cases; infectious eczematoid dermatitis - seven cases; scurvy - three cases; and pemphigus - three/

- three cases. Four of the six subjects who were suffering from chronic bronchitis appeared in this group.

Plate 6, page 75, illustrates the relationship between red cell and haemoglobin concentrations, packed cell volumes, serum total protein, serum albumin and serum globulin concentrations and erythrocyte sedimentation rates. All values on the same vertical line were obtained from a single specimen of blood. One hundred and ten different blood specimens obtained in the course of the present invest-:igation are represented. It can be seen that the red cell and haemoglobin concentrations and the packed cell volumes were closely related. The serum total protein concentration was not generally closely related to the packed cell volume. The serum albumin concentration was more closely related to the serum total protein concentration than was the serum globulin concentration. The erythrocyte sedimentation rate shewed no close relationships. In a few instances a raised serum globulin concentration was present when the sedimentation rate was low and vice versa. This was not in agreement with Olbrich's findings.

(8). Thymol Turbidity Observations. In no case was the thymol turbidity found to be above the normal range of 1 to 4 units. One single test for liver function cannot be considered sufficient to exclude the presence of liver pathology.

Matsunobu/

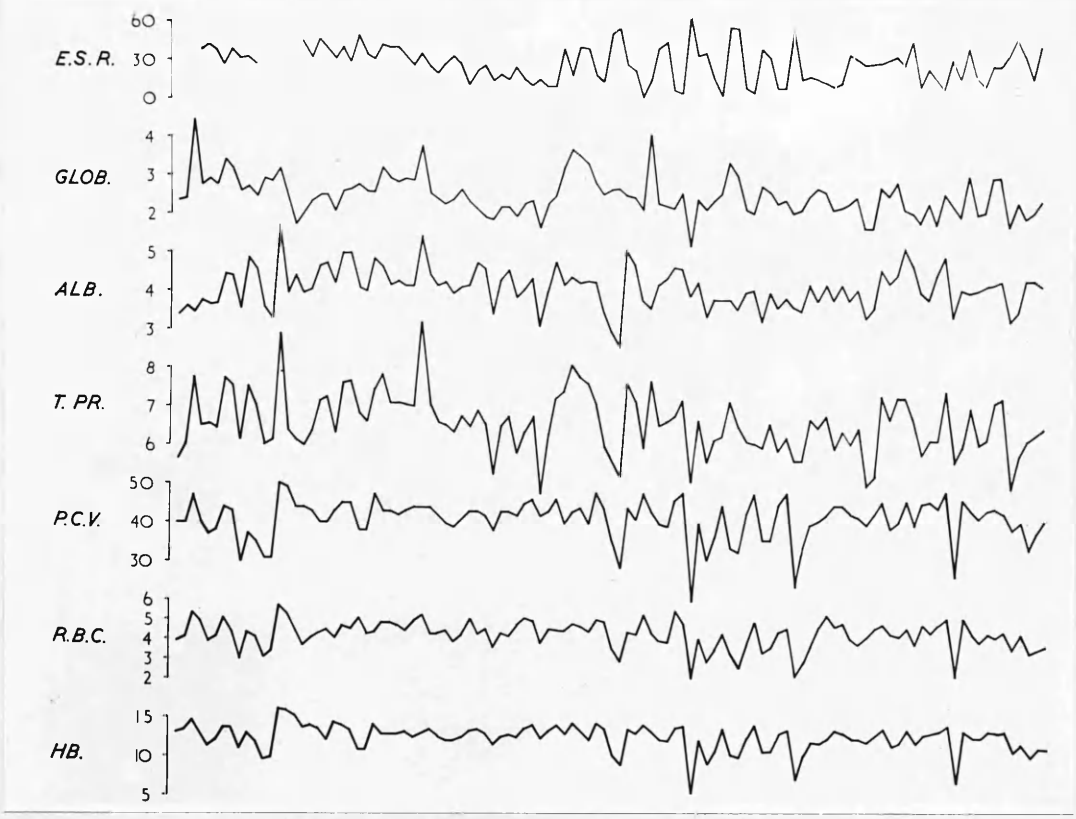

Plate 6. Shewing relationship between haemoglobin gm. per cent. (Hb.), red blood cells millions per c.mm. (R.B.C.), packed cell volume ml. per cent. (P.C.V.), serum total protein gm. per cent. (T.Pr.), serum albumin gm. per cent. (Alb.), serum globulin gm. per cent. (Glob.) and erythrocyte sedimentation rate mm. in first hour (E.S.R.)

Matsunobu (1930), using a tetrachlorophenolphthalein method, found a disturbance of liver function in twenty-three per cent. of eczema cases. Many investigators have studied liver function in relation to diseases of the skin, but the methods employed are not now considered to be satisfactory. According to Ricketts (1951) liver tests within the normal range are not synonymous with the absence of liver pathology, since one-tenth of normal liver parenchyma can carry on adequate function. MacLagan (1944) suggested that the thymol turbidity may indicate the amount of circulating gamma globulin. If this is so, then no significant increase in gamma globulin was present in the subjects of the present investigation.

The albumin globulin ratio showed no significant abnor-:malities in the series of patients investigated, although a temporary reversal was noted in cases 1, 22 and 26. No relationship to sedimentation rate or to thymol turbidity was apparent.

(9.) Serum Total Base Observations. The serum total base is the sum of the sodium, potassium, calcium and mag-:nesium in the blood serum and the normal range according to Sunderman (1942) is approximately from 143-150 m. Eq./l. No range for elderly subjects has been found in the literature. In fourteen subjects in the present investigation, the serum total/

total base was found to be above the normal level. In one subject only, (number 18), the increase was significant in degree. In this subject, the diagnosis was thyrotoxicosis. An increase in serum total base in this disease has been reported by other workers. The total value for potassium, calcium and magnesium in the serum in health may be taken as approximately 13 m. Eq./l. and in disease, variations in the amounts of those bases in the serum are of a small order in comparison with the serum total base value. The serum total base may therefore be taken as a measure of the serum sodium. On this basis, the serum sodium was within normal limits in the subjects in whom total base estimations were performed, with the exception of number 18. Urbach (1946) stated that studies of blood chemistry give no reliable index to the chemistry of the skin and therefore no conclusions regarding the electrolyte content of the skin may be drawn from the present investigation. Marson (1950) reported that the serum sodium range in subjects over the age of sixty years was distinctly lower than in younger individuals. If this is true, then in the present investigation, the serum sodium levels were high. No definite relationship between high serum total base values and oedema was present. Of the fourteen subjects who had serum total base values above 150 m. Eq./l., only seven showed oedema.

(10.)/

(10.) Osmotic Pressure Observations. The values for osmotic pressure, according to the nomogram by Wells et al. (1933), are given in Volume II, Table 9, pages 152 to 157. In general, the osmotic pressure was within normal limits. One low value was found in case 18 (thyrotoxicosis) and in case 22 (pemphigus vulgaris) the osmotic pressure fell to a low level during the course of treatment. Calculations of osmotic pressure are perhaps of doubtful value, since the equations represent the situation at equilibrium and do not take the factors of lymph flow and peri-capillary circulation into account. Various formulae have been suggested for the calculation of osmotic pressure and great divergence of opinion is evident among authors on this subject.

(11.) Fractional Test-Meal Observations. Fractional test-meals were carried out on forty-three subjects. Histamine was not employed for reasons stated under "Methods" and therefore true achlorhydria cannot be assumed to have been present in any subject. For the purposes of discussion, the term "achlorhydria" will be used when free hydrochloric acid was absent in the gruel fractional test-meal. The term hyperchlorhydria will be reserved for those cases in which the free hydrochloric acid was above 50 ml. N/10 per cent. Test-meals in which the free hydrochloric acid was between 10 and 50 ml. N/10 per cent. will be considered normal.

Normal/

Normal values for free hydrochloric acid were found in fifteen subjects. Hyperchlorhydria was present in seven subjects and "achlorhydria" was found in twenty-one subjects. The subjects in which "achlorhydria" was present may be further subdivided into those in which "masked" free hydrochlor-:ic acid was present and those in which no evidence of free acid secretion was apparent. Those subjects in which a sudden rise in the total acid was observed, without any apparent secretion of free acid, are assumed to be instances of false "achlorhydria", in which a slight secretion of free hydrochloric acid was masked by factors such as saliva, mucus or regurgitated intestinal juice. Ten cases of this type were observed among the twenty-one cases of "achlorhydria". Plate 7,pages 80 & 81,illustrates the test-meal charts which were obtained from investigation of the forty-three subjects. "Achlorhydria" was present in 49 per cent., hyperchlorhydria in 16 per cent. and normal free acid curves in 35 per cent. of subjects.

Other workers have reported somewhat similar results in the aged. Vanzant et al. (1932) found "achlorhydria" in 28 per cent. of women and in 23 per cent. of men at the age of sixty years. Rafsky et al. (1947) found low values for free hydrochloric acid in 45 per cent. of subjects who were over the age of sixty-five years. Bockus (1950) stated that/

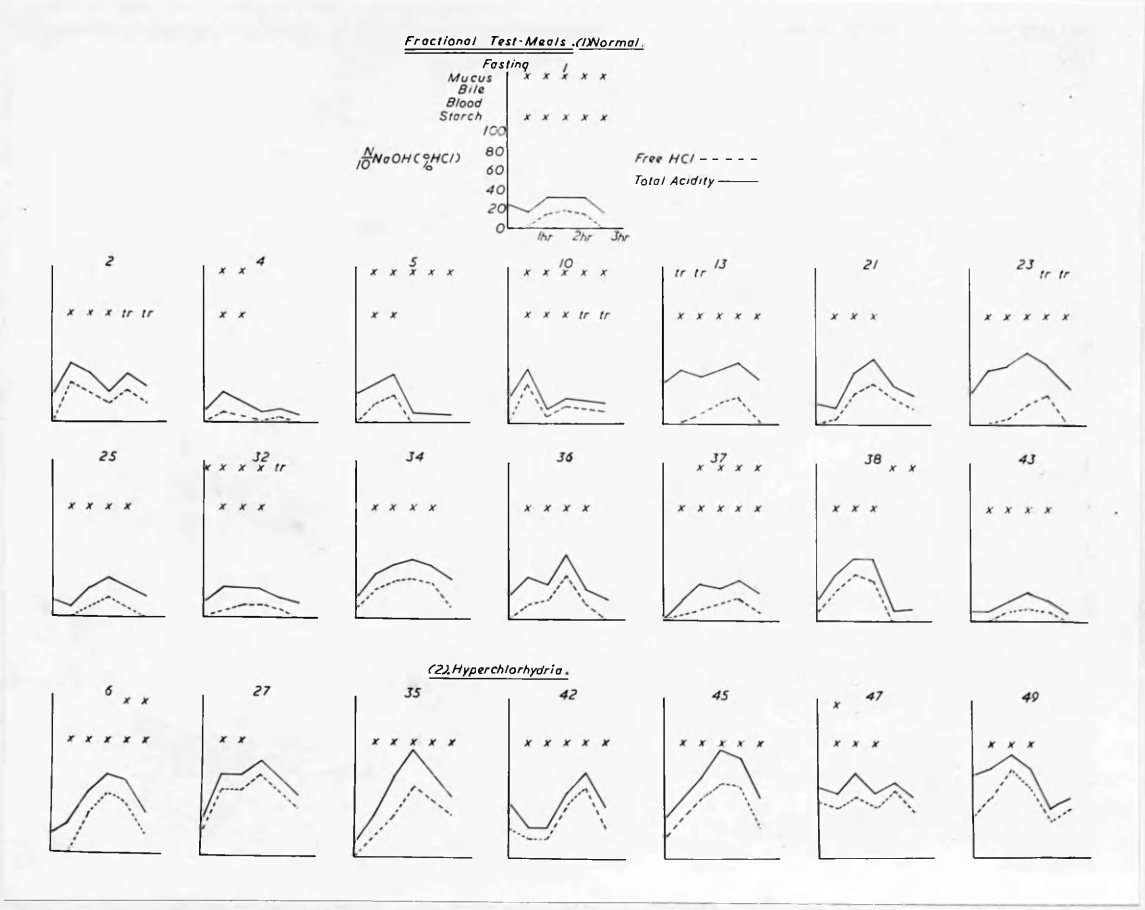

Plate 7. Shewing Fractional Test-Meal charts.

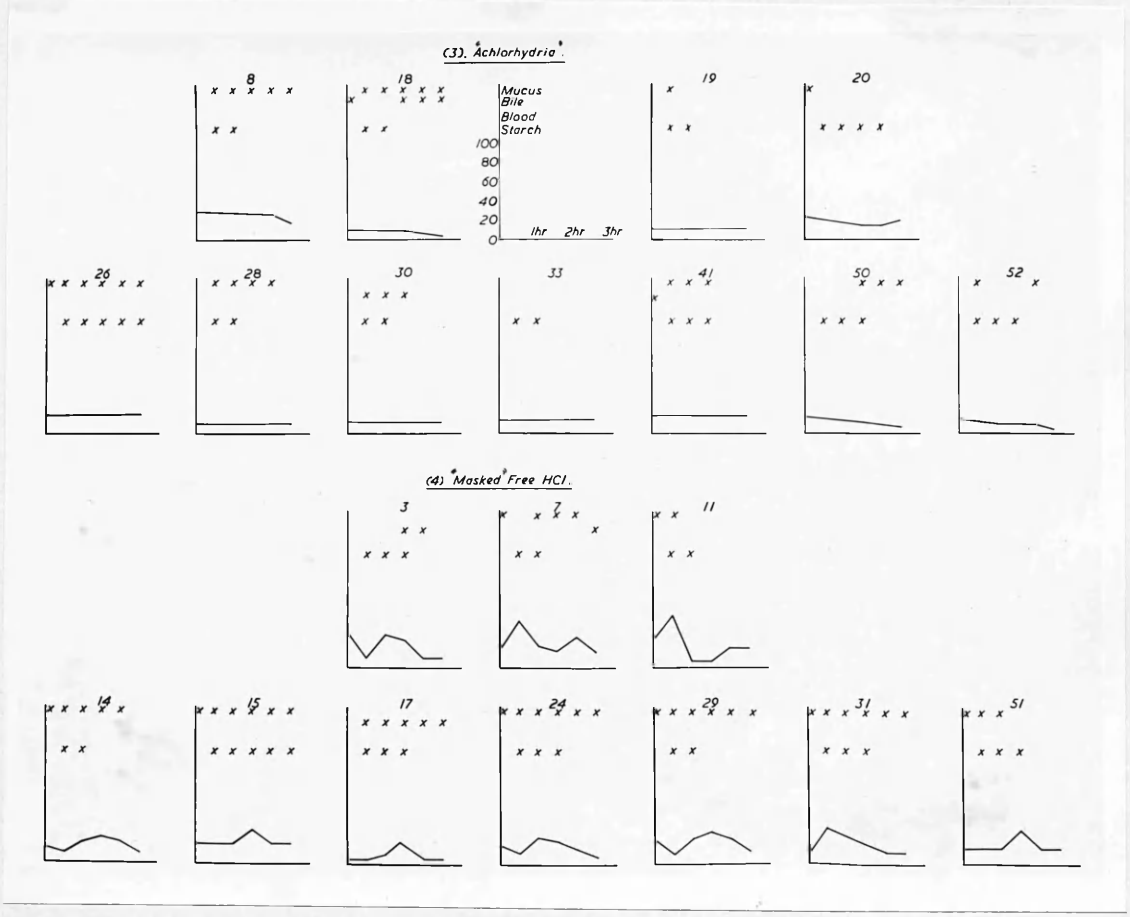

Plate 7. Shewing Fractional Test-Meal charts.
(Continued.)

that "achlorhydria" was present in 35 per cent. of subjects over sixty years of age. When histamine response was elic-:ited, 25 per cent. showed achlorhydria.

When the test-meal results in the present investigation are considered in relation to the skin manifestations, the following observations may be made. Signs of sensitisation eczema were present in all subjects with hyperchlorhydria, in less than 50 per cent. with normal acid curves, in 40 per cent. with "masked" free acid curves and in 18 per cent. with "achlorhydria". The association of hyperchlorhydria with acute skin manifestations was not absolute in this series, and drug eruptions and cases of pemphigus were noted in which "achlorhydria" was present. Brown (1925) found that hyperchlorhydria was the prevailing condition in acute wide-:spread dermatoses. Brown et al. (1935) reported that hypochlorhydria was commonly found in chronic dermatoses. Voss and Voss (1937) found hypochlorhydria in chronic condit-:ions and hyperchlorhydria in acute inflammatory conditions. Two hundred cases were investigated and the response to alcohol was used as the method. The authors quoted a number of other workers, some of whom believed that gastro-intestinal dysfunction was the cause of the skin condition, while the others considered that a coincident upset of skin and gastro-:intestinal tract occurred, as the result of some single toxic factor/

factor or from general constitutional "inferiority".

Male subjects who showed free hydrochloric acid present in test-meals and those who showed "achlorhydria" are listed in Volume II, Table 10, page 159.

It can be seen that a fairly wide scatter in serum protein and haematological values was present in both groups and the average protein values were somewhat higher in the subjects with "achlorhydria". No significant difference was present in the mean corpuscular haemoglobin concentration. Similar findings were present in the female subjects listed in Volume II, Table 10, page 160. It may be that the presence of free hydrochloric acid in the stomach is not so important in the digestion of protein as has hitherto been supposed. Beazell (1941) came to this conclusion after investigating protein digestion in the stomach. On the other hand free hydrochloric acid may have been secreted in response to protein meals in the subjects with "achlorhydria".

(12.) Dietetic Observations. An attempt has been made to assess the quality of each subject's average diet during the year prior to investigation. Such an assessment could not be altogether satisfactory, since it depended entirely upon the history given by each individual. Subjects have been grouped into those with an apparently adequate diet and those with an inadequate diet. The general bodily habitus/

habitus was also estimated by clinical examination and a division into well-nourished and under-nourished groups has been made. Actual weight records were not kept because of practical difficulties, but in any case, weight changes are of limited value since it is known that increases of body weight up to approximately ten pounds may occur as the result of an accumulation of "occult" or "hidden" oedema which is not accompanied by demonstrable clinical signs of oedema. Diet, bodily habitus and disease in the subjects of this investigation may be tabulated as follows:

	Adequate Diet.	Inadequate Diet.	Clinically under-nourished.	Clinically well-nourished.
Infectious Eczematoid Dermatitis.	9	8	9	8
Varicose Conditions.	16	2	4	14
Scurvy.	None.	9	5	4
Pemphigus.	4	1	2	3
Drug Eruptions.	3	None.	1	2

It can be seen that no relation to diet or bodily habitus was apparent in the subjects with infectious eczematoid dermatitis. In the majority of varicose conditions, the subjects were clinically well-nourished and had/

had subsisted on adequate diets. In all subjects with scurvy, the diet had been deficient, but almost half of the subjects appeared to be moderately well-nourished.

According to Peters and Eisenman (1933), protein depletion and hypoproteinaemia may coexist with obesity in subjects who had been subsisting on inadequate protein diets.

The clinically under-nourished and clinically well-nourished subjects are listed in Volume II, Table 11, pages 161 to 165. A fairly marked scatter was apparent in all serum protein and haematological values. The average serum total protein values were higher in the females than in the males and the average serum total protein values in the well-nourished subjects of both sexes were significantly higher than in the under-nourished groups. No significant change was present in the average serum protein and haematological values in the female subjects after the skin had healed. A significant increase in the average serum total protein, serum albumin and serum globulin values was present in the under-nourished male subjects after the skin had healed. The haemoglobin, red cell and packed cell volume averages also showed a significant increase in those subjects when treatment was completed.

Although it has been demonstrated that blood levels tend to rise when bed rest continues and although it is to be/

be expected that the values obtained in subjects who have just been confined to bed will be lower than values obtained in the same subjects after a period of a few weeks' strict bed rest, it is not surprising that where such strict bed rest is not enforced, the average values at the beginning and at the end are approximately the same, since postural changes will tend to be cancelled out.

The increase in serum protein and in haematological values found in the under-nourished male subjects when the skin had healed may have been due to treatment. In this group eight subjects received "Casinal" with added glucose during treatment. "Casinal" consists of casein in the form of its calcium salt. It is said to contain all the essential amino-acids and one ounce of the powder provides approximately twenty-six gm. of protein. Two ounces were administered daily, together with added glucose, to a number of the subjects who were investigated.

Five subjects, with infectious eczematoid dermatitis, in whom the diet was supplemented in this fashion, are listed in Volume II, Table 12, page 166. A significant increase in the serum total protein, serum albumin, serum globulin, haemoglobin, and red cell percentages and in the packed cell volume results was present at the end of treatment. The average mean corpuscular volume had diminished. It is

possible/

possible that a change in the plasma volume may have occurred but this seems unlikely since in the subjects with infectious eczematoid dermatitis who received no additional protein, listed in Volume II, Table 13, page 167, no change was noted in the average values at the end of treatment. There is, therefore, presumptive evidence that protein supplement increased the serum protein values and the formation of red cells which also tended to be less macrocytic at the end of treatment.

All uncomplicated cases of infectious eczematoid dermat-:itis are listed in Volume II, Table 14, pages 168 and 169. No significant difference between males and females was present in the average serum protein and haematological values on admission and the values were within normal limits. After the skin had healed, the average values in the female cases, only one of whom received extra protein in the diet, showed no significant change. In the male cases, an increase in the serum total protein, serum albumin, haemoglobin, red cell and packed cell volume averages was present at the end of treatment. This may have been due to the protein supple-:mented diet which three of these subjects received.

The nine cases of scurvy are listed in Volume II, Table 15, page 170. The average values for serum proteins, haemoglobin, red cells and packed cell volume results/

results at the end of treatment showed an increase. Those subjects with scurvy, who received a protein supplemented diet, are listed separately from those who received only iron and vitamin C. (Volume II, Table 16, pages 171 and 172. A significant increase took place in the average serum total protein, serum albumin, serum globulin, haemoglobin, red cell and packed cell volume values in the subjects who received a protein supplemented diet. A much smaller increase in the average values took place when no protein supplement was exhibited. In the latter group of subjects, the average number of days of treatment was twenty-nine; in the former group, the average number of days of treatment was twenty-three. It would appear, therefore, that a high protein diet is important, in addition to vitamin C, in the treatment of scurvy. Iron may not be required in treatment and the mean corpuscular haemoglobin concentration was not significantly low in the subjects who were investigated. Iron was administered, however, since iron stores may have been depleted and it was considered advisable to give a readily available supply for new red cell formation. Eczema was frequently noted in association with scurvy, but could not be attributed solely to diminished serum proteins and to inadequate diets, since parasitic infestation and local trauma were frequent in scorbutic subjects.

Uncomplicated/

Uncomplicated varicose conditions are listed in Volume II, Table 17, pages 173 & 174. The average values for serum total protein and serum albumin were higher than in the cases of infectious eczematoid dermatitis. When the skin had healed no significant change in values took place, but the average serum total protein value had increased slightly in male subjects after treatment. Case 11 showed a marked increase in serum total protein at the end of treatment and this partly accounts for the average increase.

In conclusion then, in under-nourished subjects the serum protein concentrations were lower than in well-nourished subjects. In infectious eczematoid dermatitis and in scurvy, administration of protein supple-:ments in the diet increased the serum protein concentrat-:ions and the formation of red blood cells.

SUMMARY AND CONCLUSIONS

(1.) Literature regarding serum proteins, malnutrition, oedema, protein deficiency and disease of the skin has been reviewed.

(2.) One hundred and thirty-seven investigations of serum protein concentrations and of haematological values have been carried out in fifty-two subjects, fifty of whom were over sixty years of age. Eighty-two estimations of serum total base have been carried out in thirty-five subjects. One hundred and thirty-four thymol turbidity values have been determined in fifty-two subjects and gruel fractional test-meals have been carried out in forty-two subjects.

Ten normal control subjects who were over sixty years of age have also been investigated. Variations in the blood due to alteration of posture have been demonstrated in eleven subjects.

Eighteen subjects with varicose conditions, seventeen with infectious eczematoid dermatitis, nine with scurvy and eight with pemphigus or bullous drug eruptions were invest-:igated.

(3.) The erythrocyte sedimentation rate was not related to the state of activity of the skin.

(4.)/

(4.) Thymol turbidity estimations were within the normal range in all subjects.

(5.) In uncomplicated varicose conditions and in uncomplicated infectious eczematoid dermatitis the serum total base was within the normal range.

(6.) No close relationship between serum protein concentrations and oedema was found. Oedema occurring in uncomplicated infectious eczematoid dermatitis and in uncomplicated varicose conditions in elderly subjects may be explained by local tissue infection and by mechanical factors such as diminished tissue elasticity, impaired lymphatic drainage and local venous congestion.

(7.) Significant low concentrations of serum total protein were not found in uncomplicated varicose conditions or in uncomplicated infectious eczematoid dermatitis. Serum total protein concentrations below normal were found in two subjects with scurvy, in one subject with pemphigus vulgaris, in one subject with varicose eczema and drug eruption and in one subject with infectious eczematoid dermatitis and thyrotoxicosis.

(8.) The serum albumin concentration was below normal in one subject with pemphigus vulgaris.

Low concentrations of serum albumin were commonly found in scurvy and in infectious eczematoid dermatitis.

Normochromic,/

Normochromic, normocytic anaemia was present in the majority of scorbutic subjects and in one third of subjects with infectious eczematoid dermatitis.

No relationship was evident between "achlorhydria" and serum protein concentrations or haematological values.

(9.) Some evidence was presented that high protein diets were of value in restoring the blood picture to normal in scorbutic subjects and in those with infectious eczematoid dermatitis. Scurvy and infectious eczematoid dermatitis presented some features in common, with respect to the serum albumin concentrations and to the haematological values. Malnutrition may have been a factor in both groups, but it is not possible to state that malnutrition played a definite part in the production of infectious eczematoid dermatitis.

(10.) Administration of a high protein diet to scorbutic subjects and to selected elderly subjects who are suffering from infectious eczematoid dermatitis is suggested as a necessary addition to other routine forms of treatment.

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BIBLIOGRAPHY.

Abbott, W.E. and Mellors, R.C. (1943). Arch. Surg. Chicago. 46:277.

Addis, T., Poo, L.J. and Lew, W. (1936). J. biol. Chem. 115:111.

Altschuler, S.S., Salryun, M., Schneider, N. and Satriano, D. (1943). J. Amer. med. Ass. 121:163.

Anderson, C.G. and Altmann, A. (1951). Lancet. 1:203.

Baker, L.E. and Carrel, A. (1926). J. exp. Med. 44:387.

Beattie, J., Herbert, P.H. and Bell, D.J. (1948). Brit. J. Nutrit. 2:47.

Beazell, J.M. (1941). Amer. J. Physiol. 132:42.

Benditt, E.P., Straube, R.L. and Humphreys, E.M. (1946). Proc. Soc. Exp. Biol., N.Y. 62:189.

Bennet, J. (1946). Lancet. 1:852.

Bing, J., Naeser, J., Rasch, G. and Rojel, K. (1947). Acta med. Scand. 126:351.

Bock, J. (1948). J. Gerontol. 3:119.

Bockus, H.L. (1950). Postgraduate Gastroenterology, Philadelphia:Saunders.

Brown, W.H. (1925). Brit. J. Derm. 37:214.

Brown, W.H., Smith, M.S. and McLachlan, A.D. (1935). Brit. J. Derm. 47:181.

Bruckman, F.S., D'esopo, L.M. and Peters, J.P. (1930). J. clin. Invest. 8: 577.

Burger, G.C.E., Sanstead, H.R. and Drummond, J. (1945). Lancet. 2:282.

Cannon, P.R. (1945). J. Amer. med. Ass. 128: 360.

Cannon, P.R., Humphreys, E.M., Wissler, R.W. and Frazier, L.E. (1944). J. clin. Invest. 23: 601.

Chang, H.C. (1932). Proc. Soc. exp. Biol., N.Y. 29: 829.

Clark, A.H. (1919). Bull. Johns Hopk. Hosp. 30: 117.

Clements, F.W. (1936). J. Amer. med. Ass. 106: 139 (abstr.)

Conger, T.W. and Elvehjam, C.A. (1941). J. biol. Chem. 138: 555.

Cuthbertson, D.P. (1945). Proc. Roy. Soc. Med. 35:388.

Denz, F.A. (1947). Quart. J. Med. 16: 1.

Dicker, S.E. (1948). Proc. Roy. Soc. Med. 41: 548.

Drant, P. (1946). Penn. med. J. 49:1331.

Ebert, R.V. and Stead, E.A. (1941). J. clin. Invest. 20: 317.

Edge, J.R. (1945). Lancet. 2: 317.

Epstein, A.A. (1917). Med. Rec. 91: 128.

Evans, R., Cowdry, E.V. and Nielson, P.E. (1943). Anat. Rec. 86: 545.

Geiger, E. (1947). J. Nutrit. 34: 97.

Gell, P.G.H. (1948). Proc. Roy. Soc. Med. 41:323.

Glaser, K. and Markson, S.M. (1945). J. Pediat. 26: 367.

Goldsmith, N.R. (1947). Arch. Derm. Syph., N.Y. 55:397.

Gornall, A.G., Bardawill, C.J. and David, M.M. (1949). J. biol. Chem. 177:751.

Gupta, L.M. (1946). Brit. med. J. 1:643.

Guy, W.B. (1950). Arch. Derm. Syph., N.Y. 61:261.

Guy, W.H., Jacob, F.M., and Guy, W.B. (1948). Arch. Derm. Syph., N.Y. 57:822.

Hartzell, J.B., Winfield, J.M. and Irvin, J.L. (1941). J. Amer. med. Ass. 116:669.

Harvey, S.C. and Howes, E.L. (1930). Ann. Surg. 91:641.

Himsworth, H.P. (1948). Proc. Roy. Soc. Med. 41:549.

Howard, J.E., Bigham, R.S., Eisenberg, H., Wagner, D. and Bailey, E. (1946). Bull. Johns Hopk. Hosp. 78:282.

Howell, T. (1948). Geriatrics. 3:346.

Howell, T. (1949). Geriatrics. 4:281.

Howell, T. and Piggot, A.P. (1948). J. Gerontol. 3:124.

Hynes, M. and Whitby, L.E.H. (1938). Lancet. 2:249.

Jones, E.L. (1887). J. Physiol. 51:205.

Joseph, B. (1930). Ann. Surg. 92:318.

Keim, H.L., Frisch, D.C. and Palmer, A.E. (1948). Arch. Derm. Syph., N.Y. 58:314.

Kennedy, C.B., Hennington, V.M., Ross, R. and Hartwell, R.M. (1950). South med. J. 43:128.

Keys, A. (1948). J. Amer. med. Ass. 138:500.

Keys, A., Brozek, J., Henschel, A., Mickelsen, O. and Taylor, H.L. (1950). The Biology of Human Starvation, Vol. II. St. Paul: North Central Publishing Company.

Koster, H. and Kasman, L.P. (1942). Arch. Surg. Chicago. 45: 776.

Koster, H. and Shapiro, A. (1940). Arch. Surg. Chicago. 41: 723.

Krebs, E.G. (1946). J. Lab. clin. Med. 31: 85.

Lane, C.G. and Rockwood, E.M. (1949). New Engl. J. Med. 241: 772.

Leyton, G.B. (1946). Lancet. 2: 73.

Lipscombe, F.M. (1945). Lancet. 2: 313.

Liu, S.H., Chu, H.I., Wang, S.H. and Chung, H.L. (1931a.). Proc. Soc. exp. Biol., N.Y. 29: 250.

Liu, S.H., Chu, H.I., Wang, S.H. and Chung, H.L. (1931b.). Proc. Soc. exp. Biol., N.Y. 29: 252.

Lund, C.C. and Levenson, S.M. (1945). J. Amer. med. Ass. 128: 95.

Lyons, R.H., Jacobsen, S.D. and Avery, N.L. (1944). Amer. J. med. Sci. 208: 148.

MacLagan, N.F. (1944). Brit. J. exp. Path. 25: 234.

Madden, S.C., Carter, J.R., Kattus, A.A., Miller, L.L. and Whipple, G.H. (1943). J. exp. Med. 77: 277.

Marson, F.G.W. (1950) Proc. Roy. Soc. Med. 43: 935.

Matsunobu, T. (1930). Jap. J. Derm. Urol. 30:825. Abstr. p.86.

Maver, M.B. (1920). J. Amer. med. Ass. 74: 934.

Mollison, P.L. (1946). Brit. med. J. 1: 4.

Mufson, J. (1932). Amer. J. med. Sci. 183: 632.

Mulder, G.J. (1849). The Chemistry of Vegetable and Animal Physiology. Edinburgh and London: Blackwood.

Mulholland, J.H., Tui, C., Wright, A.M., Vinci, V. and Shafiroff, B. (1943). Ann. Surg. 118: 1015.

Mulvehill, W. (1944). Arch. Derm. Syph., N.Y. 49: 327.

Murray, R.O. (1947). Lancet. 1: 19.

Notes on Clinical Laboratory Methods. (1947). 5th ed. Glasgow:Smith.

Newman, B. and Gitlow, S. (1943). Amer. J. med. Sci. 205: 677.

Newman, B. and Gitlow, S. (1943). Amer. J. med. Sci. 206: 622.

Olbrich, O. (1947). Edinb. med. J. 54: 306.

Olbrich, O. (1948). Edinb. med. J. 55: 100.

Perera, G.A. and Berliner, R.W. (1943). J. clin. Invest. 22: 25.

Peters, B.A. (1945). Lancet. 1: 264.

Peters, J.P. and Eisenman, A.J. (1933). Amer. J. med. Sci. 186: 808.

Pevny, V. (1947). Schweiz. med. Wschr. 77: 1306.

Pollack, H. (1947). J. Amer. diet. Soc. 23:410.

Price, R.K. (1946). Brit. med. J. 1: 647.

Rafsky, H.A., Newman, B. and Joliffe, N. (1947). J. Lab. clin. Med. 32: 118.

Ratner, S., Schoenheimer, R. and Rittenberg, D. (1940). J. biol. Chem. 134: 653.

Renbourn, E.T. (1947). J. Hyg., Cam. 45: 456.

Ricketts, W.E. (1951). Amer. J. med. Sci. 221: 287.

Schoenheimer, R., Ratner, S. and Rittenberg, D. (1939). J. biol. Chem. 130: 703.

Schoenheimer, R., Ratner, S., Rittenberg, D. and Heidelberger, M. (1942). J. biol. Chem. 144: 541.

Scott McGregor, R.G. (1946). Lancet. 1: 852.

Shelburne, S.A. and Egloff, W.C. (1931). Arch. intern. Med. 48: 51.

Sinclair, H.M. (1948). Proc. Roy. Soc. Med. 41: 541.

Smith, D.A. (1946). Lancet. 1: 852.

Spealman, C.R., Newton, M. and Post, R.L. (1947). Amer. J. Physiol. 150: 628.

Spec. Rep. Ser. med. Res. Coun., Lond. (1945). 252.

Stapleton, T. (1946). Lancet. 1: 850.

Starck, V. (1946). Acta derm. - venereol., Stockh. 26: 418.

Starling, E.H. (1896). J. Physiol. 19: 312.

Stead, E.A. and Ebert, R.V. (1941). Amer. J. Physiol. 132: 411.

Stewart, C.P. (1947). Brit. med. J. 2: 569.

Stewart, J.D. and Rourke, G.M. (1938). J. clin. Invest. 17: 413.

Strickler, A. and Aliensis, A.E. (1947). Urol. cutan. Rev. 51:401.

Sunderman, F.W. (1942). J. biol. Chem. 143: 185.

Sunderman, F.W. and Dohan, F.C. (1941). Amer. J. Physiol. 132: 418.

Talbott, J.H. and Coombs, F.S. (1940). Arch. Derm. Syph., N.Y. 41: 359.

Taylor, F.H.L., Levenson, S.M., Davidson, C.S., Browder, N.C. and Lund, C.C. (1943). Ann. Surg. 118: 215.

Thompson, W.D., Ravdin, I.S. and Frank, I.L. (1938). Arch. Surg. Chicago. 36:500.

Thompson, W.O., Thompson, P.K. and Dailey, M.E. (1928). J. clin. Invest. 13: 447.

Uehlinger, E. (1947). Helv. med. Acta. 14: 584.

Urbach, E. (1946). Skin Diseases, Nutrition and Metabolism. London: Heineman.

Vandervelde, M. and Cantineau, M. (1919). J. Amer. med. Ass. 73: 1229 (abstr.)

Vanzant, F.R., Alvarez, W.C., Eusterman, G.B., Dunn, H.L. and Berkson, (1932). Arch. intern. Med. 49: 345.

Vaughan, J., Dent, C. and Pitt Rivers, R. (1945). Proc. Roy. Soc. Med. 35: 388.

Voss, H. and Voss, F. (1937). Munch. med. Wschr. 84: 1486.

Walker, S.A. and Benditt, E.P. (1950). J. invest. Derm. 14:113.

Walters, J.H., Rossiter, R.J. and Lehmann, H. (1947a.). Lancet. 1:205.

Walters, J.H., Rossiter, R.J. and Lehmann, H. (1947b.). Lancet. 1:244.

Weech, A.A., Snelling, C.E. and Goettsch, E. (1933). J. clin. Invest. 12:193.

Wells, H.S., Youmans, J.B. and Miller, D.G. (1933). J. clin. Invest. 12:1103.

Whipple, G.H. (1942). Amer. J. med. Sci. 203:477.

Whipple, G.H., Robscheit-Robbins, F.S. and Miller, L.L. (1946). Ann. N.Y. Acad. Sci. 47:317.

Whitby, L.E.H. and Britton, C.J.C. (1950). Disorders of the Blood, 6th ed. London:Churchill.

White, A. and Dougherty, T.F. (1946). Ann. N.Y. Acad. Sci. 46:859. Cited in Editorial (1948). J. Amer. med. Ass. 138:890.

Widdowson, E.M. and McCance, R.A. (1950). Lancet. 1:539.

Wiener, K. (1947). Skin Manifestations of Internal Disorders. London:Kimpton.

Wissler, R.W., Woolridge, R.L., Steffee, C.H. and Cannon, P.R. (1946). J. Immunol. 52:267.

Youmans, J.B., Bell, A., Donley, D. and Frank, H. (1932a.). Arch. intern. Med. 50:843.

Youmans, J.B., Bell, A., Donley, D. and Frank, H. (1932b.). Arch. intern. Med. 51: 45.

Youmans, J.B., Wells, H.S., Donley, D., Miller, D.G. and Frank, H. (1934). J. clin. Invest. 13: 447.

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Case 1. Infectious eczematoid dermatitis - Hypertension.

Mr. J.H., aged 77 years, was admitted to Stobhill Hospital on 1st February, 1950, complaining of an eruption on his legs of four months' duration.

Personal History: He lived alone and prepared his own food. His diet was inadequate, consisting mainly of bread and tea.

Previous History; Recurring eczema of the face.

History of Present Illness; The patient could give no detailed description of the onset of the eruption. No history of trauma was obtained, and he had not been confined to bed before admission. He gave no history of breathlessness, but had noticed swelling of the legs since the onset of the skin condition.

General Examination; The patient was a thin old man. No cyanosis was present. Pitting oedema was present up to the knees, but no sacral oedema was evident. No venous engorgement was present and no varicose veins were noted. The patient was edentulous and upper dentures only were worn.

Skin: The legs up to the knees were affected by foul smelling areas of superficial ulceration, larger areas of weeping eczema, and scattered pustular and scaling lesions. The skin over the dorsa of the feet was thickened and pigmented. No purpuric lesions were present.

Case 1. (Continued).

Cardio-Vascular System:

The pulse was irregular in rhythm due to the presence of extra systoles. The arterial walls were thickened and tortuous. The apex beat was palpable four inches from the mid-sternal line in the fifth left intercostal space. Heart sounds were of fair quality. Soft systolic murmurs, not conducted but persisting on change of posture, were present at mitral and aortic area. Blood pressure was $\frac{200}{110}$mm. Hg.

Examination of the other systems revealed no abnormality.

Investigations:

Chemical Examination of Urine - Specific Gravity 1015. Reaction - Acid. No Albumin, no Sugar. Microscopical Examination - No abnormality detected.

Blood urea 32 mgm. per cent. Urea Clearance Test 100 per cent. of Normal.

Wassermann Reaction - Negative.

Electocardiogram Report: "Left Axis shift. Ventricular ectopic beats."

Skiagram - Chest "some cardiac enlargement and a bronchitic type of chest".

Progress and Treatment:

1. 2.50. Ferri et ammon. cit. grains 30 t.i.d. Vitamin C 100 mgm. t.i.d. "Multivite" tablets 1 t.i.d. "Casinal"

Case 1. (Continued).

2 ozs. daily. Eusol soaks as local applications to legs.

5. 2.50. 1% Ichthyol Calamine liniment as local applications to legs.

8. 2.50. Bed sores developed.

4. 3.50. Bed sores healing.

8. 3.50. Internal medication discontinued.

2. 5.50. Internal medication resumed.

15. 5.50. Bed sores healed.

17. 5.50. Internal medication discontinued. Skin healed. Slight oedema of feet still present.

Case 2. Infectious eczematoid dermatitis.

Mr. A.G., aged 73 years, an inmate of Foresthall Institution, was seen on 16th of March, 1950, when he complained of an eruption on both legs of twelve years' duration.

Personal History: He had been an inmate of Foresthall since 1937. Diet was inadequate.

Previous History: April, 1946 - Erysipelas. August, 1947 - Cerebral thrombosis from which he had made a good recovery. December, 1947 - Hypostatic pneumonia.

History of Present Illness: Twelve years previously the skin of his legs had become irritable, red, moist and crusted. The condition had persisted in spite of many different forms of local treatment. He had been confined to bed for six weeks prior to this examination. No history of breathlessness was obtained.

General Examination: The patient was a thin old man. No oedema was present. The patient had a solitary carious stump in the lower jaw. No dentures were worn. No varicosity of leg veins was noted and no venous engorgement was present.

Skin: The skin of the right leg was plum-coloured with scattered superficial abrasions and areas of crusted, scaling and moist eczema. The left leg showed some erythematous and crusted areas. An erythemato-papular

Case 2. (Continued).

eruption was present on the face and scalp.

Cardio-Vascular System: Occasional extra systoles were present, but no other abnormalities were noted. Blood pressure was $\frac{160}{70}$mm. Hg.

Examination of other systems revealed no abnormality.

Investigations:

Chemical Examination of Urine - Specific Gravity 1014.
Reaction - Acid. No Sugar, no Albumin.
Microscopical Examination - no abnormality detected.
Blood Urea 25 mgm. per cent.
Wassermann Reaction - Negative.

Progress and Treatment:

16. 3.50. Ferri et ammon. cit. grains 30 t.i.d.
Vitamin C 100 mgm. t.i.d. "Casinal" 2 ozs. daily.
1% Ichthyol Calamine Liniment as local application.
1. 4.50. 5% Crude Coal Tar in Vaseline, as local application.
3. 5.50. Internal medication discontinued. Skin healed.

Case 3. Infectious eczematoid dermatitis.

Mr. C.S., a male patient, aged 72 years, was admitted to Stobhill Hospital on 8th February, 1950, complaining of an eruption on both legs of several months' duration.

Personal History: He lived alone and prepared his own food. Diet was grossly inadequate.

Previous History: None could be obtained.

History of Present Illness: It was not possible to elicit any detailed history. He had not been confined to bed before admission to hospital and gave no history of breathlessness.

General Examination: The patient was an emaciated old man, suffering from pediculosis corporis. Moderate oedema of ankles was present. No varicose veins were evident, and no venous engorgement was present. He was edentulous.

Skin: Linear excoriations and scattered pigmented areas were present on the trunk. Areas of moist and scaling eczema were present on both legs.

Cardio-Vascular System: No abnormalities were detected. Blood pressure was $\frac{140}{80}$mm. Hg.

Examination of other systems revealed no abnormality.

Investigations:

Chemical Examination of Urine - Specific Gravity 1018. Reaction - Acid. No Albumin, no Sugar.

Microscopical Examination - no abnormality detected.

Case 3. (Continued).

Blood urea 19 mgm. per cent.

Wassermann Reaction - negative.

Progress and Treatment:

9. 2.50. Ferri et ammon. cit. grains 30 t.i.d. Vitamin C 100 mgm. t.i.d. Acid hydrochlor. dil., min. 30 t.i.d. "Casinal" 2 ozs. daily. "Multivite" tablets 1 t.i.d. Ichthyol 1% in Calamine Liniment as local application.

1. 3.50. 1% Crude Coal Tar in Zinc Paste as local application.

4. 3.50. Bed sore developed. Oedema gone.

3. 4.50. Skin completely healed.

Case 4. Nummular infectious eczematoid dermatitis. Hypertension.

Mrs. G.P., aged 66 years, was admitted to Stobhill Hospital on 28th February, 1950, complaining of an eruption on arms and legs of several years' duration.

Personal History: She kept house for her husband and her diet seemed adequate.

Previous History: Measles and Scarlet Fever in childhood. Pleurisy - 1934.

History of Present Illness: The eruption had commenced on the dorsal aspect of the hands several years previously. The forearms and the legs became affected later. She had not been confined to bed and gave no history of breath-:lessness.

General Examination: The patient was a small, thin lady. Mucous membranes were pale. No cyanosis or oedema was present. No varicose veins were apparent and no venous engorgement was present. Upper dentures were worn, but the lower jaw was edentulous.

Skin: Nummular patches of moist and scaling eczema were present on the dorsa of the hands and forearms, and on the anterior aspects of both legs.

Cardio-Vascular System: Blood pressure was $\frac{200}{110}$mm. Hg. and arterial walls were moderately thickened, but no other abnormalities were detected.

Case 4. (Continued).

Respiratory System: A few rhonchi were present in both lungs.

Examination of other systems revealed no abnormality.

Investigations:

Chemical Examination of Urine - Specific Gravity 1018.

Reaction - Acid. No Albumin, no Sugar.

Microscopical Examination - no abnormality detected.

Blood urea 41 mgm. per cent.

Wassermann Reaction - negative.

Progress and Treatment:

28. 2.50. 1% Ichthyol Calamine Liniment as local application.

10. 3.50.. 10% Crude Coal Tar in acetone as local application.

30. 3.50. Skin healed.

Case 5. Infectious eczematoid dermatitis.

Mrs. W.M., aged 93 years, was admitted to Stobhill Hospital on 21st March, 1950, complaining of an eruption on the arms and legs of two weeks' duration.

Personal History: She lived by herself and her diet was inadequate.

Previous History: Measles and Diphtheria in childhood.

History of Present Illness: She had scalded her left foot six weeks previously and the skin had broken down. Two weeks before admission an eruption appeared on the arms and legs. She had not been confined to bed. No history of breathlessness was obtained.

General Examination: The patient was an emaciated old lady, with a prolapsed uterus and a left femoral hernia. Oedema was present over the sacral region but no oedema of legs was noted. Mucous membranes were pale. A few teeth were present in the lower jaw, while the upper jaw was edentulous. No varicosity of veins was present and no venous engorgement was detected.

Skin: A moist weeping eczema was present on the legs. Nummular patches of eczema were scattered over both forearms and thighs.

Cardio-Vascular System: No abnormality was noted. Blood Pressure was $\frac{140}{80}$mm. Hg.

Examination of other systems revealed no abnormality.

Case 5. (Continued).

Investigations:

Chemical Examination of Urine - Reaction - acid. Specific Gravity 1010. Albumin - a trace, no Sugar.

Microscopical Examination - A few pus cells present.

Blood urea 57 mgm. per cent.

Wassermann reaction - negative.

Progress and Treatment:

23. 3.50. Ferri et ammon, cit. grains 30 t.i.d. "Casinal" 2 ozs. daily. Vitamin C 100 mgm. t.i.d. "Multivite" tablets 1 t.i.d. Ichthyol 1% in Calamine Liniment, as local application.

4. 4.50. Crude Coal Tar 10% in acetone as local application.

18. 4.50. Skin healed. Slight oedema still present over sacrum.

Case 6. Varicose Eczema.

Miss B.K., aged 68 years, was admitted to Stobhill Hospital on 4th April, 1950, complaining of an eruption on both legs of ten months' duration.

Personal History: She had been an inmate of Foresthall Institution for several years. Diet was fair.

Previous History: Measles, mumps and whooping cough in childhood. Gall bladder operation, 1930.

History of Present Illness: Ten months previously the lower part of the right leg became erythematous and moist eczematous patches appeared. The other leg and the forearms became affected later. No history of trauma was obtained. The patient had not been confined to bed prior to admission and gave no history of breathlessness.

General Examination: The patient was fairly well-nourished. Slight oedema of the ankles was present and there was moderate varicosity of leg veins. No venous engorgement was noted. A few teeth were present in the lower jaw, while the upper jaw was edentulous.

Skin: Weeping eczema was present on the legs and nummular eczematous areas affected the forearms.

Cardio-Vascular System: The pulse was regular in rate and rhythm. Arterial walls were not thickened. No cardiac enlargement was detected. Heart sounds were of good quality. A faint systolic murmur, not conducted but

Case 6. (Continued).

persisting on change of posture, was present at mitral and aortic areas. Blood pressure was $\frac{165}{75}$mm. Hg.

Examination of other systems revealed no abnormality.

Investigations:

Chemical Examination of Urine - Specific Gravity 1005.

Reaction - Acid. No Albumin, no Sugar.

Microscopical Examination - no abnormality detected.

Blood urea - 35 mgm. per cent.

Wassermann Reaction - negative.

Skiagram - "Chest - no lung disease; calcification of Aorta".

Progress and Treatment:

4. 4.51. 1% Ichthyol Calamine liniment as local application.

16. 4.50. 10% Crude Coal Tar in acetone as local application.

25. 4.50. Skin healed. No oedema.

Case 7. Varicose ulcer and varicose eczema.

Mr. J.E., aged 75 years, was admitted to Stobhill Hospital on 5th April, 1950, complaining of an eruption on arms and legs of six months' duration.

Personal History: He lived with his daugher and his diet appeared to be adequate.

Previous History: Scurvy, 1943.

History of Present Illness: Six months previously a small ulcer had appeared on the right leg. Soon afterwards an eruption appeared on both legs and both arms. No history of breathlessness was obtained. He had been confined to bed for a few days before admission.

General Examination: The patient was a thin old man. Slight oedema was present at both ankles and there was varicosity of the leg veins. No engorgement of neck veins was noted. He was edentulous.

Skin: A small, shallow ulcer (2 cm. x 3 cm.) was present above the medial malleolus of the right leg. Moist eczema extended from knee to ankle. Nummular eczematous patches were present on the left leg and on both forearms.

Cardio-Vascular System: No abnormalities were detected. Blood Pressure was $\frac{170}{80}$mm. Hg.

Examination of other systems revealed no abnormality.

Investigations:

Chemical Examination of Urine - Specific Gravity 1022.

Case 7. (Continued).

Reaction - Acid. No Albumin, no Sugar.

Microscopical Examination - no abnormality detected.

Blood urea - 24 mgm. per cent.

Wassermann Reaction - negative.

Skiagram - "Chest and sinuses; no abnormality detected".

Progress and Treatment:

13. 4.50. Acid hydrochlor. dil. minims. 30 t.i.d. orally, 1% Ichthyol Calamine liniment as local application.

14. 5.50. Crude Coal Tar 10% in acetone as local application.

5. 6.50. Skin healed.

Case 8. Infectious eczematoid dermatitis.

Mr. A.L., aged 63 years, was admitted to Stobhill Hospital on 7th April, 1950, complaining of an eruption on both legs of twelve days' duration.

Personal History: He lived alone and prepared his own food. Diet was inadequate.

Previous History: Amputation of left foot during 1914-18 War.

History of Present Illness: Twelve days before admission the skin around the amputation area of the left ankle became swollen and irritable. The wound had not previously given any trouble and there had been no immediate trauma. A few days later the right leg also became affected. He had not been confined to bed before admission and gave no history of breathlessness. He complained of dysuria and frequency of micturition.

General Examination: The patient appeared to be fairly well-nourished. There was no cyanosis, oedema or varicosity of veins and no engorgement of neck veins. A few carious stumps were present in both jaws.

Skin: Both legs, up to the knees, were affected by a moist weeping eczema.

Cardio-Vascular System: The pulse was regular in rate and rhythm, arterial walls were not thickened, and the heart was not enlarged. Heart sounds were of fair quality.

Case 8. (Continued).

A soft, systolic murmur, not conducted, but persisting with change of posture, was heard at the mitral area. The Blood Pressure was $\frac{120}{70}$mm. Hg.

Respiratory System: Scattered rhonchi were heard in both lungs.

Rectal examination revealed the prostate to be slightly enlarged, but not unduly hard.

Physical Examination revealed no other abnormality.

Investigations:

Chemical Examination of Urine - Specific Gravity 1020. Reaction - Acid. Albumin - a trace. No sugar.

Microscopical examination - 6 - 12 pus cells per high power field.

Culture - moderate growth of B. proteus.

Blood urea - 27 mgm. per cent.

Wassermann Reaction - negative.

Progress and Treatment:

7. 4.50. Ferri et ammon. cit. grains 30 t.i.d.

13. 4.50. Sulphatriad grammes 1 t.i.d. plus alkalis. 1% Ichthyol in Calamine Liniment dressings as local application.

20. 4.50. Sulphatriad and alkalis discontinued.

27. 4.50. Urine culture "No growth".

28. 4.50. Skin healed.

Case 9. Varicose ulcer, varicose eczema, chronic bronchitis and emphysema.

Mr. F.O., aged 74 years, was admitted to Stobhill Hospital on 14th April, 1950, complaining of an ulcer on his left leg, of one year's duration.

Personal History: He lived alone and prepared his own food. Diet was inadequate.

Previous History: Pneumonia - 1947.

History of Present Illness: Twelve months previously a small ulcerated area had appeared on the left leg. No history of trauma was obtained. The patient had not been confined to bed before admission. He gave a history of recurring cough especially in winter and of breathlessness on moderate exertion.

General Examination: The patient was moderately well nourished. Mucous membranes were pale. Varicosity of leg veins was present. No oedema and no engorgement of neck veins was noted. He was edentulous, apart from a few carious stumps in both jaws.

Skin: A small superficial ulcerated area (2 cm. x 2 cm.) was present on the left leg behind the medial malloelus. The skin of both legs was erythematous and scattered pustules and excoriations were present.

Cardio-Vascular System: No abnormality was detected. Blood Pressure was 160mm. Hg.

Case 9. (Continued).

Respiratory System: Signs of chronic bronchitis and emphysema were present.

Examination of Other Systems revealed no abnormality.

Investigations:

Chemical Examination of Urine - Specific Gravity 1020.

Reaction - Acid. No Albumin, no Sugar.

Microscopical Examination - No abnormality detected.

Blood Urea - 20 mgm. per cent.

Wassermann Reaction - negative.

Progress and Treatment.

18. 4.50. 1% Ichthyol Calamine Liniment locally.

22. 4.50. Patient left hospital by his own wish.

Case 10. Infectious eczematoid dermatitis. Hypertension and Cardiac Failure.

Mr. P.H., aged 84 years, was admitted to Stobhill Hospital on 21st April, 1950, complaining of an eruption on his limbs of eighteen months' duration.

Personal History: He lived with his sister who provided adequate meals.

Previous History: Measles in childhood. Smallpox 1900. Neoplasm of left ear, 1948, (treated with radium).

History of Present Condition: Eighteen months previously an irritable eruption appeared on the left ankle. Within a few weeks both arms and both legs were affected. No history of trauma was elicited. The patient had not been confined to bed before admission. He complained of breathlessness on slight exertion.

General Examination: The patient was a fairly well nourished old man. Oedema of the legs and sacral region was present. A moderate degree of venous engorgement was evident in the jugular veins. No varicosity of leg veins was apparent. Slight cyanosis was present.

Skin: Weeping eczema was present on both legs from ankles to knees. A small nummular area of vesicular eczema was apparent on the dorsum of the left hand.

Cardio-Vascular System: The pulse was irregular in rate and rhythm due to the presence of frequent extra systoles.

Case 10. (Continued).

Arterial walls were thickened and tortuous. The apex beat was palpable in the fifth left intercostal space, four inches from the mid-sternal line. Heart rate was approximately seventy beats per minute. Heart sounds were soft and of poor quality, but no murmurs were detected. Blood Pressure was $\frac{210}{90}$mm. Hg.

Examination of the other systems revealed no abnormality.

Investigations:

Chemical Examination of Urine - Specific Gravity 1014.

Reaction - Acid. No Albumin, no Sugar.

Microscopical examination - no abnormality detected.

Blood urea - 40 mgm. per cent.

Urea Clearance Test - 82% of normal.

Wassermann Reaction - negative.

Skiagram - "Lung fields; normal. Hypertensive changes in heart and aorta".

Electrocardiogram Report: "Myocardial Disease. Auricular and ventricular ectopic beats. Chest leads indicate some left heart strain".

Progress and Treatment: Local treatment, 1% Ichthyol Calamine Liniment followed by 1% Boric Zinc Oil.

17. 5.50. Digitalis leaf grains ii t.i.d. 10% Crude Coal Tar in acetone as local application to skin.

19. 5.50. Digitalis leaf grains 1 t.i.d.

29. 5.50. Skin healed. No oedema.

Case 11. Varicose eczema.

Mr. H.C., aged 70 years, was admitted to Stobhill Hospital on 19th April, 1950, complaining of a widespread eruption which had commenced on the right leg one month previously.

Personal History: He lived in a hostel and meals appeared to be reasonably adequate.

Previous History: Compaund fracture right leg, 1920.

History of Present Illness: The eruption had commenced on the right leg one month previously. No history of trauma was obtained. Ten days later the left leg, forearms and lower back were affected. The patient had not been confined to bed before admission. He gave no history of breathlessness.

General Examination: The patient was fairly well nourished. Slight oedema was present at the ankles. Some varicosity of leg veins was evident. No engorgement of neck veins was noted. A few carious teeth were present in the lower jaw, while the upper jaw was edentulous.

Skin: A weeping eczematous eruption was present on the legs, forearms and lower back.

Cardio-Vascular System: No abnormality was detected. Blood pressure was $\frac{140}{70}$mm. Hg.

Examination of other systems revealed no abnormality.

Investigations:

Case 11. (Continued).

Chemical Examination of Urine - Specific Gravity 1024. Reaction - Acid. Albumin - a trace. No Sugar. Microscopical examination - Scanty epithelial cells and polymorphonuclear leucocytes. Culture - moderate growth of streptococcus faecalis.

Blood Urea - 25 mgm. per cent.

Wassermann Reaction - negative.

Skiagram - "Chest, no abnormality detected; Sinuses, mucosal thickening in left antrum".

Progress and Treatment. Local treatment, 1% Ichthyol Calamine Liniment.

24. 4.50. Diarrhoea developed. Stool culture - negative. Sulphaguanidine course and alkalis administered.

1. 5.50. Sulphaguanidine and alkalis discontinued.

27. 5.50. Culture of urine - no growth.

11. 6.50. Skin healed. No oedema.

Case 12. Varicose eczema. Hypertension.

Miss M.M., aged 84 years, an inmate of Foresthall Institution, was seen on the 18th of May, 1950, when she complained of an eruption on both legs of some weeks' duration.

Personal History: She had been an inmate of Foresthall Institution since October, 1948. Diet was fair.

Previous History: No illnesses.

History of Present Illness: Some weeks previously a few spots had appeared around both ankles. The condition spread in spite of various local applications. The patient had not been confined to bed. No history of breathlessness was obtained.

General Examination: The patient was fairly well nourished. Marked oedema of the ankles was present but no sacral oedema was present. Varicosity of leg veins was present. No engorgement of neck veins was noted. The patient was edentulous, apart from a few carious stumps.

Skin: A weeping eczematous eruption was present on the legs.

Cardio-Vascular System: The pulse was regular in rate and rhythm. The arterial walls were thickened and tortuous. The apex beat was palpable four inches to the left of the mid-sternal line in the fifth left intercostal space. Heart sounds were of fair quality. A systolic murmur was audible at the mitral and aortic areas and was conducted upwards at the latter area.

Case 12. (Continued).

Blood Pressure was $\frac{175}{95}$mm. Hg.

Investigation of other systems revealed no abnormality.

Investigations:

Chemical Examination of urine - Specific Gravity 1018.

Reaction - Acid. No Albumin, no Sugar.

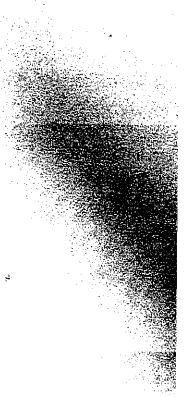
Microscopical examination - no abnormality detected.

Blood Urea - 31 mgm. per cent.

Wassermann Reaction - negative.

Progress and Treatment: 1% Ichthyol Calamine Liniment as local application.

Patient refused further investigation.



Case 13. Infectious eczematoid dermatitis and drug eruption.

Mrs. J.W., aged 75 years, was admitted to Stobhill Hospital on 9th May, 1950, complaining of a generalised eruption which had commenced on the legs three months previously.

Personal History: She had lived alone for the previous ten years and prepared her own meals which appeared to be fairly adequate.

Previous Illnesses: Enteric fever in childhood. "Dermatitis" of the legs from time to time during the previous ten years. Pneumonia, 1948.

History of Present Condition: The patient could not recall how her condition began, but an eruption had appeared ten years previously on her leg and numerous recurrences had taken place. Three months before admission to hospital a few "spots" had appeared on her right leg which became intensely itchy. The eruption spread and the other leg became involved. One month before admission the forearms became affected. At this time she was given sulphonamide tablets by mouth and a generalised eruption appeared on the trunk.

She complained of dysuria. She had not been confined to bed and gave no history of breathlessness.

General Examination: The patient was poorly nourished.

Case 13. (Continued).

Slight oedema of the ankles was present. No venous engorgement was noted. The mucous membranes were pale. The patient was edentulous and upper dentures only were worn.

Skin: A weeping eczematous eruption was present on the arms and legs. A widespread, erythematous, maculo-papular and urticarial eruption was apparent on the trunk.

Cardio-Vascular System: No abnormality was detected apart from occasional extra systoles. Blood pressure was $\frac{135}{85}$mm. Hg.

Examination of the other systems revealed no abnormality.

Investigations:

Chemical Examination of urine - Specific Gravity 1026. Reaction - Acid. Haze of Albumin, no Sugar.

Microscopical examination - 10 to 15 pus cells per high power field.

Culture - heavy growth of B coli.

Blood Urea - 27 mgm. per cent.

Wassermann Reaction - negative.

Skiagram - "Chest, no abnormality detected".

Progress and Treatment: Local treatment, 1% Ichthyol Calamine Liniment, followed by Aluminium acetate 1% in zinc paste.

15. 5.50. Penicillin course commenced.

Case 13. (Continued).
21. 5.50. Penicillin discontinued. 2% Crude Coal Tar in Zinc Paste as local application to skin.

7. 6.50. Skin healed, No oedema. Urine - no abnormality detected.

Case 14. Scurvy. Varicose eczema.

Mr. R.L., aged 75 years, was admitted to Stobhill Hospital on 20th May, 1950, complaining of an ulcer on his right leg of three weeks' duration and an eruption on both legs and both arms.

Personal History: He had been living alone for eight years and prepared his own meals. His diet was deficient in fruit and vegetables. He had not been confined to bed before admission.

Previous Illnesses: Whooping cough in childhood.

History of Present Condition: Three weeks prior to admission, the right leg became swollen and a small ulcerated area appeared. Soon afterwards an eruption appeared on both legs and both arms. The patient gave no history of breathlessness.

General Examination: The patient was a very thin old man. Mucous membranes were pale. Slight oedema of ankles was present. He was edentulous. Some varicosity of leg veins was present. No engorgement of neck veins was noted.

Skin: A small superficial ulcer (2 cm. x 3 cm.) with a clean base and surrounded by purple staining was present on the anterior aspect of the lower third of the right leg. Follicular petechial and macular purpuric lesions were present on both legs and on the dorsa of the hands and arms. Scattered papulo-vesicles and nummular eczematous patches

Case 14. (Continued).

were also apparent on the legs.

Cardio-Vascular System: No abnormality was detected.

Blood Pressure was $\frac{170}{80}$ mm. Hg.

Examination of the other systems revealed no abnormality.

Investigations:

Chemical Examination of urine - Specific gravity 1016.

Reaction - Acid. No Albumin, no Sugar.

Microscopical Examination - no abnormality detected.

Blood Urea - 32 mgm. per cent.

Wassermann Reaction - negative.

Progress and Treatment:

20. 5.50. 1% Ichthyol Calamine Liniment as local application. Ferri et ammon. cit. grains 30 t.i.d. Vitamin C 700 mgm. daily. "Multivite" tablets 1 t.i.d. Acid hydrochlor. dil. minims. 30 t.i.d. "Casinal" 2 ozs. daily.

12. 6.50. Internal treatment discontinued.

16. 7.50. Skin healed. No oedema.

Case 15. Drug eruption. Hypertension.

Mrs. J.C., aged 66 years, was admitted to Stobhill Hospital on 21st May, 1950, complaining of an eruption on her arms and legs of one week's duration.

Personal History: The patient lived alone, but her diet appeared to be satisfactory.

Previous History: Measles in childhood. 1918, Ovarian tumour removed. 1918, Alopecia of scalp (transitory).

History of Present Illness: The patient had been taking phenobarbitone tablets for some weeks before admission to hospital. A few days before admission an eruption appeared on the arms and legs. She had not been confined to bed and gave no history of breathlessness.

General Examination: The patient was a well-nourished lady. Marked oedema of legs up to knees was present. The patient was edentulous and upper dentures only were worn. No venous engorgement was present.

Skin: A maculo-papular erythematous eruption was present on the arms and legs. Large tense bullae (3 cm. x 2 cm.) containing clear fluid were apparent on the legs which also showed macular purpuric lesions and erythematous urticarial plaques.

Cardio-Vascular System: Blood pressure was $\frac{210}{100}$mm. Hg. No other abnormalities were detected.

Examination of other systems revealed no abnormality.

Case 15. (Continued).

Investigations:

Chemical Examination of urine - Specific gravity 1014.

Reaction - Acid. No Albumin, no Sugar.

Microscopical Examination - no abnormality detected.

Blood Urea - 26 mgm. per cent.

Wassermann Reaction - negative.

Skiagram - "Chest, no abnormality detected".

Progress and Treatment:

22. 5.50. 1% Ichthyol Calamine Liniment as local application. Benadryl mgm. 50 t.i.d. orally.

9. 6.50. Skin healed. Benadryl discontinued. No oedema.

Case 16. Scurvy. Varicose ulcer and varicose eczema.

Mr. A.D., aged 71 years, was admitted to Stobhill Hospital on 26th May, 1950, complaining of a general eruption of one month's duration.

Personal History: He had lived in lodging houses during the previous ten years. His diet was grossly inadequate.

Previous Illnesses: Gastric ulcer, 1909. Recurrent ulcer of leg since 1920.

History of Present Condition: One month previously he had noticed a small ulcerated area on the right leg. Soon afterwards the skin of both legs became discoloured and moist weeping areas appeared. The patient had not been confined to bed before admission. He gave no history of breathlessness.

General Examination: The patient was a very emaciated old man. Oedema of both ankles was present. Varicose veins were present in the legs. No engorgement of neck veins was noted. The patient was edentulous, apart from one carious stump in the lower jaw.

Skin: Pediculosis corporis was present. Linear excoriations and eroded papules were evident on the trunk. Follicular petechial haemorrhages and purpuric macules were apparent on the legs. A superficial ulcerated area (5 cm. x 2 cm.) was present on the inner aspect of the right lower leg. Moist eczematous patches were present

Case 16. (Continued).

on both legs.

Cardio-Vascular System: No abnormality was detected.

Blood pressure was $\frac{100}{60}$mm. Hg.

Examination of other systems revealed no abnormality.

Investigations:

Chemical Examination of urine - Specific gravity 1025.

Reaction - Acid. No Albumin, no Sugar.

Microscopical Examination - no abnormality detected.

Skiagram - "Stomach, no abnormality detected".

Faecal occult blood not present.

Blood Urea - 34 mgm. per cent.

Wassermann Reaction - negative.

Progress and Treatment:

26. 5.50. 1% Ichthyol Calamine Liniment as local application. Ferri et ammon. cit. grains 30 t.i.d. Vitamin C 700 mgm. daily. "Multivite" tablets 1 t.i.d. "Casinal" 2 ozs. daily. Acid hydrochlor. dil. minims. 30 t.i.d.

4. 6.50. Vitamin C 100 mgm. t.i.d.

17. 8.50. Skin healed. No oedema.

Case 17. Scurvy. Infectious eczematoid dermatitis. Chronic bronchitis and emphysema.

Mr. B.S., aged 70 years, was admitted to Stobhill Hospital on 2nd June, 1950, complaining of an eruption which had commenced on his legs one month previously.

Personal History: He lived with his daughter, but his diet was deficient in fruit, vegetables and meat.

Previous Illnesses: Pneumonia, 1900. Recurrent attacks of bronchitis.

History of Present Condition: An eruption had appeared on both legs one month previously and the forearms became affected later. No history of trauma was obtained. Breathlessness was present on slight exertion.

General Examination: The patient was plethoric. Marked oedema of the legs was present. No clubbing of fingers was evident. No varicose veins were noted in the legs. No overfilling of jugular veins was present. Upper and lower dentures were worn.

Skin: A weeping eczematous eruption was present on feet and legs. Scattered petechial macules and larger purpuric ecchymoses were apparent on the legs. A papulo-vesicular eruption was present on the hands and forearms.

Cardio-Vascular System: No abnormalities were detected. Blood pressure was $\frac{170}{70}$mm. Hg.

Respiratory System: Signs of chronic bronchitis and

Case 17. (Continued).

emphysema were present.

Examination of other systems revealed no abnormality.

Investigations:

Chemical Examination of urine - Specific gravity 1016.

Reaction - Acid. No Albumin, no Sugar.

Microscopical Examination - no abnormality detected.

Culture - no growth.

Blood urea - 32 mgm. per cent.

Wassermann Reaction - negative.

Ascorbic acid saturation test. Commenced 3. 6.50.

6. 6.50.	Daily excretion of ascorbic acid			- Nil.
7. 6.50.	"	"	"	- 32 mgm. per cent.
8. 6.50.	"	"	"	- 35 mgm. per cent.
9. 6.50	"	"	"	- 56 mgm. per cent.
10. 6.50	"	"	"	-512 mgm. per cent.
11. 6.50	"	"	"	-727 mgm. per cent.

Skiagram - Chest, chronic bronchitic changes in both lungs.

Progress and Treatment:

3. 6.50. Vitamin C 700 mgm. daily. "Multivite" tablets 1 t.i.d. Acid Hydrochlor. dil. min. 30 t.i.d.

11. 6.50. Vitamin C 100 mgm. t.i.d.

13. 7.50. Skin well. Slight oedema still present.

Case 18. Infectious eczematoid dermatitis. Thyrotoxicosis.

Mr. T.C., aged 63 years, was admitted to Stobhill Hospital on 19th July, 1950, complaining of a generalised eruption which had commenced on the left leg two months previously.

Personal History: He lived with his wife and his diet was satisfactory.

Previous History: Scarlet Fever in childhood. 1925, Operation for duodenal ulcer. 1927, Appendicectomy. 1940, Gunshot wound of chest. 1947, "Cardiac trouble".

History of Present Condition: Two months previously the patient sustained an abrasion of the left leg. Local applications did not help the condition and the trunk and arms became affected two weeks before admission to hospital.

The patient was breathless on exertion and excitable. He perspired profusely and had lost weight. He was intolerant of warm weather and suffered from palpitations. He had been in bed for one week prior to admission to hospital.

General Examination. The patient was very thin. Exophthalmos was present, with definite lid retraction. Tremor of the outstretched fingers was obvious, and the skin was moist. Oedema of both legs up to the knees was present, but no sacral oedema was evident. No varicosity of leg veins was noted. No definite thyroid swelling was

Case 18. (Continued).

felt and the trachea was in the mid-line.

Skin: A moist eczematous eruption was present on both legs and on both arms and a papulo-vesicular eruption was apparent on the trunk.

Cardio-Vascular System: The pulse was regular in rate and rhythm. (100 beats per minute). The apex beat was five inches from mid-sternal line in the fifth left intercostal space. The heart sounds were loud, but no murmurs were heard. The blood pressure was $\frac{150}{70}$mm. Hg.

Examination of other systems revealed no abnormality.

Investigations:

Chemical Examination of Urine - Specific gravity 1024. Reaction - Acid. No Albumin, no Sugar.

Microscopical Examination - a few polymorphonuclear leucocytes were present. Culture - no growth.

Blood Urea - 26 mgm. per cent.

Wassermann Reaction - Negative.

Blood Cholesterol - 175 mgm. per cent.

Basal Metabolic Rate - Plus 33%.

Barium Swallow - "No retrosternal goitre."

Progress and Treatment:

20. 7.50. 1% Ichthyol Calamine Liniment as local application. "Casinal" 2 ozs. daily. Ferri et ammon. cit. gr. 30 t.i.d., "Multivite" tablets 1 t.i.d., Acid

Case 18. (Continued).

hydrochlor. dil. min. 30 t.i.d.

18. 8.50. Skin healed. Internal medication discontinued.

No oedema. Tremor and nervousness still present.

28. 8.50. Thiouracil 50 mgm. q.i.d. commenced.

25.10.50. Patient very well.

Case 19. Varicose eczema.

Mr. W.L., aged 63 years, was admitted to Stobhill Hospital on 26th July, 1950, complaining of an eruption on his legs of four months' duration.

Personal History: He had lived in a lodging house for years and his diet was inadequate.

Previous History: No illness.

History of Present Condition: The patient could not give a satisfactory history. He had not been in bed before admission, and gave no history of breathlessness.

General Examination: The patient was a thin old man suffering from pediculosis corporis. Marked oedema of the legs was present. There was no cyanosis. A few carious teeth were present in both jaws. Varicosity of leg veins was apparent. No engorgement of neck veins was noted.

Skin: Linear excoriations and scattered areas of pigmentation were apparent on the trunk. Weeping eczema was present on both legs.

Cardio-Vascular System: No abnormality was detected. Blood pressure was $\frac{170}{90}$mm. Hg.

Examination of other systems revealed no abnormality.

Investigations:

Chemical Examination of Urine - Specific gravity 1020
Reaction - Acid. No albumin, no Sugar.
Microscopical Examination - no abnormality detected.

Case 19. (Continued).

Blood Urea - 23 mgm. per cent.

Wassermann Reaction - Negative.

Progress and Treatment:

26. 7.50. 1% Ichthyol Calamine Liniment.

28. 7.50. Acid Hydrochlor. dil. min. 30 t.i.d.

30. 7.50. Oedema gone.

10. 8.50. Skin healed.

Case 20. Varicose ulcer, Varicose eczema and chronic bronchitis.

Mr. F.E., aged 75 years, was admitted to Stobhill Hospital on 15th August, 1950, complaining of an eruption on his legs of some months' duration.

Personal History: He lived with his wife and his diet was satisfactory.

Previous History: Recurring eczema of legs during previous ten years. Cough, recurring in winter.

History of Present Condition: The condition had recurred after phlebitis of his right leg.

General Examination: The patient was a well nourished old man. Marked varicosity of leg veins was present. Cyanosis, oedema and engorgement of neck veins were not present. The patient was edentulous, apart from a few carious stumps.

Skin: A shallow, superficial ulcer (3 cm. x 3 cm.) was present on the right leg. Moist and scaling eczema with pigmentation was apparent on both legs.

Cardio-Vascular System: No abnormality was detected. Blood pressure was $\frac{120}{60}$mm. Hg.

Respiratory System: Signs of chronic bronchitis were present.

Examination of other systems revealed no abnormality.

Case 20. (Continued).

Investigations:

Chemical Examination of urine - Specific gravity 1024.

Reaction - Acid. No Albumin, no Sugar.

Microscopical Examination - no abnormality detected.

Blood Urea - 28 mgm. per cent.

Wassermann Reaction - Negative.

Progress and Treatment: Eusol soaks as local application followed by 1% Ichthyol Calamine Liniment four days later.

21. 8.50. Acid hydrochlor. dil. minims 30 t.i.d.

24. 9.50. 1% Salicylic Acid in zinc cream as local application to skin.

2.10.50. Skin healed.

Case 21. Pemphigus foliaceus.

Mr. J.M., aged 64 years, was admitted to Stobhill Hospital on 13th August, 1948, complaining of a generalised eruption which had commenced three years previously.

Personal History: He had been two years in hospital at the time of examination. His diet had been adequate.

Previous History: Measles in childhood.

History of Present Condition: In 1930, his face and limbs had become affected by a red, scaling eruption. This cleared up after three years. In 1948, a generalised eruption appeared. No history of breathlessness was obtained.

General Examination: The patient was fairly well nourished. The axillary, inguinal and supraclavicular glands were enlarged. There was no cyanosis, oedema or venous engorge-:ments. Upper and lower dentures were worn.

Skin: A generalised exfoliative eruption was present. The scales tended to be centrally attached. Nicolsky's sign was present.

Cardio-Vascular System: Pulse was regular in rate and rhythm. Arterial walls were not thickened. The heart was not enlarged. Heart sounds were of poor quality. A systolic murmur, not conducted and persisting on change of posture, was present at the mitral area.

Blood pressure was $\frac{130}{70}$mm. Hg.

Case 21. (Continued).

Examination of other systems revealed no abnormality.

Investigations:

Chemical Examination of Urine - Specific gravity 1026.

Reaction - Acid. No Albumin, no Sugar.

Microscopical Examination - no abnormality detected.

Blood Urea - 20 mgm. per cent.

Wassermann Reaction - Negative.

Barium meal - "Stomach, no abnormality detected."

Electrocardiogram Report - "Myocardial disease with some delay in intra-ventricular conduction. Augmented unipolar leads show normal pattern with slurring of the Q.R.S. complexes in all leads, providing further evidence of myocardial disease."

Histological examination of skin section revealed appearances consistent with the diagnosis of pemphigus foliaceus. Skin section and description of section presented. (Volume 11, page 183.)

Progress and Treatment:

22. 8.50. "Stovarsol" gr. iv t.i.d.

27. 8.50. "Stovarsol" discontinued.

16.10.50. Eruption unchanged.

Case 22. Pemphigus Vulgaris.

Mrs. J.K., aged 89 years, was admitted to Stobhill Hospital on 5th July, 1950, complaining of an eruption on the limbs of four weeks' duration.

Personal History: She lived alone and her diet was unsatisfactory.

Previous History: "Biliary" trouble, 1941.

History of Present Condition: Four weeks before admission to hospital "little red spots" appeared on the legs and on the arms. Blisters soon formed on those areas. The patient took no medicines of any kind and had not been confined to bed before admission to hospital. She gave no history of breathlessness.

General Examination: The patient was a very thin old lady. No oedema, cyanosis or venous engorgement was present. The patient was edentulous.

Skin: The trunk, arms, legs and groins were affected by bullous lesions, (one to several centimetres in size). Some lesions were tense and others were flaccid. The contents were clear in some cases and sero-purulent in others. Raw and crusted areas were present here and there on the trunk and limbs. Erythematous halos were not evident around recent primary lesions. Ruptured bullae were present on the mucous membranes of the mouth. Nikolsky's Sign was easily elicited.

Case 22. (Continued).

Cardio-Vascular System: No abnormality was detected.

Blood pressure was $\frac{150}{80}$mm. Hg.

Examination of other systems revealed no abnormality.

Investigations:

Chemical Examination of urine - Specific gravity 1018. Reaction - Alkaline. Trace of Albumin present, no Sugar. Microscopical Examination - a few hyaline casts; no pus cells. Culture - no growth.

Culture from blister fluid - Light growth of staphylococcus aureus (coagulase positive) and staphylococcus albus.

Histological examination of skin section revealed appearances consistent with the diagnosis of pemphigus vulgaris. Skin section and description of section presented. (Volume 11, page 184.)

Blood Urea - 22 mgm. per cent.

Wassermann Reaction - Negative.

Progress and Treatment:

21. 8.50. "Stovarsol" grains 4 t.i.d.

25. 8.50. "Stovarsol" discontinued.

29. 8.50. No new bullae. Ferri et ammon. cit. grains 30 t.i.d. "Casinal" 2 ozs. daily, Vitamin C 100 mgm. t.i.d. "Multivite" tablets 1 t.i.d.

14.10.50. Recurrence of bullous lesions.

26.11.50. Patient died.

Case 23. Varicose ulcer, Varicose eczema and Hypertension.

Mrs. S.C., aged 64 years, was admitted to Stobhill Hospital on 31st August, 1950, complaining of an eruption on her legs of six months' duration.

Personal History: She lived with her daughter and her diet was satisfactory.

Previous History: Eczema of legs, 1947.

History of Present Condition: Six months previously an eruption appeared on the legs. No history of trauma was obtained. A few weeks later a small ulcerated area appeared on the right leg. The patient had not been confined to bed before admission to hospital, and gave no history of breathlessness.

General Examination: The patient was an obese old lady. Both legs were oedematous and varicosity of leg veins was present. No engorgement of neck veins was noted. Upper and lower dentures were worn.

Skin: The lower thirds of both legs showed a weeping eczematous eruption. Areas of scaling and pigmentation were also present. A circular ulcer (2" in diameter) was present over the medial malleolus of the right leg. A purulent exudation was apparent in the base of the ulcer.

Cardio-Vascular System: The pulse was regular in rate and rhythm. The arterial walls were moderately thickened. The left border of the heart was percussed in the fifth

Case 23. (Continued).

left intercostal space five inches from the mid-sternal line. Heart sounds were of good quality and no murmurs were detected. The blood pressure was $\frac{175}{120}$mm. Hg.

Examination of other systems revealed no abnormality.

Investigations:

Chemical Examination of urine - Specific gravity 1020.

Reaction - Acid. No Albumin, no Sugar.

Microscopical Examination - no abnormality detected.

Culture - no growth.

Blood Urea - 28 mgm. per cent.

Wassermann Reaction - Negative.

Progress and Treatment: Eusol soaks to ulcer followed by 1% Ichthyol Calamine Liniment.

14. 9.50. Skin healed. No oedema.

Case 24. Varicose ulcers and Hypertension.

Mr. B.M., aged 78 years, was admitted to Stobhill Hospital on 1st April, 1949, complaining of ulceration of both legs of fifteen years' duration.

Personal History: Prior to admission to hospital he had lived alone and his diet had been inadequate. At the time of examination he had been on adequate diet for fifteen months.

Previous History: Recurring ulceration of legs since 1934.

History of Present Condition: The patient had suffered from recurring ulcers for many years. Treatment with skin grafting had not been successful. He had been confined to bed since admission to hospital. The leg ulcers were extremely resistant to treatment. No history of breathlessness was obtained.

General Examination: 3. 9.50. The patient was apparently well nourished. No oedema was present and there was no cyanosis and no engorgement of neck veins. Varicosity of leg veins was present. The patient was edentulous.

Skin: An extensive ulcer (6" x 5") was present on the right leg and several smaller ulcers (1" x 2") were present on the left leg. Surrounding skin was thickened and sclerotic. A copious, purulent discharge exuded from the ulcers.

Case 24. (Continued).

Cardio-Vascular System: Occasional extra systoles occurred. Arterial walls were thickened. The apex beat was palpable five inches to the left of the mid-sternal line, in the fifth left intercostal space. Heart sounds were of good quality and no cardiac murmurs were audible.

Blood pressure was $\frac{260}{120}$mm. Hg.

Respiratory System: A few fine crepitations were present at both bases.

Examination of other systems revealed no abnormality.

Investigations:

Chemical Examination of urine - Specific gravity 1028.

Reaction - Acid. No Albumin, no Sugar.

Microscopical Examination - no abnormality detected.

Blood Urea - 15 mgm. per cent.

Wassermann Reaction - negative.

Postural change investigations on the patient's blood were carried out and progress notes are not appended.

Case 25. Infectious eczematoid dermatitis. Hypertension. Cardiac Failure.

Mr. H.D., aged 79 years, was admitted to Stobhill Hospital on 7th September, 1950, complaining of an eruption on the legs of six weeks' duration.

Personal History: He lived with his wife and his diet was adequate.

Previous History: Appendicectomy, 1903. Gassed during 1914/1918 War. Cerebral Thrombosis, 1944.

History of Present Condition: In 1946 the patient had suffered from "eczema" of legs. This had cleared up with local treatment. Six weeks before admission to hospital the skin of his legs became scaly and then formed moist weeping areas. He had not been confined to bed. He complained of breathlessness on slight exertion.

General Examination: The patient was a plethoric old man. Oedema of both legs was present. Moderate overfilling of jugular veins was noted. No varicose veins were present. The patient was edentulous and upper dentures only were worn.

Skin: A weeping eczematous eruption was present on both legs. Marked rhinophyma was present.

Cardio-Vascular System: The pulse was regular in rate and rhythm. Vessel walls were thickened. The left border of the heart was percussed five inches from the mid-sternal

Case 25. (Continued).

line in the fifth left intercostal space. The heart sounds were muffled. No murmurs were audible. The blood pressure was $\frac{210}{100}$mm. Hg.

Examination of other systems revealed no abnormality.

Investigations:

Chemical Examination of urine - Specific gravity 1020. Reaction - Acid. Albumin - a trace, no Sugar. Microscopical Examination - a few hyaline casts were present.

Fixation of Specific Gravity - Highest Specific Gravity 1020.

Blood Urea - 20 mgm. per cent.

Wassermann Reaction - Negative.

Skiagram - Chest "the heart is enlarged and shows evidence of hypertension. Lung fields are clear".

Postural change investigations on the patient's blood were carried out and progress notes are not appended.

Case 26. Pemphigus Vulgaris. Hypertension and Cardiac Failure.

Mr. C.M., aged 65 years, was admitted to Stobhill Hospital on 13th September, 1950, complaining of a generalised eruption which had commenced one month previously.

Personal History: He lived with his wife and his diet was adequate.

Previous History: Measles in childhood.

History of Present Condition: About one month prior to admission to hospital "blisters" had appeared on his right foot. The other foot was soon affected. Soon afterwards, the face, trunk and limbs became affected by a generalised erythematous eruption.

The patient complained of breathlessness even at rest, but had not been confined to bed.

General Examination: The patient was very thin. Gross oedema of legs was present and, in addition, a marked pad of sacral oedema. Overfilling of the jugular veins was noted. Slight cyanosis was present. Upper and lower dentures were worn.

Skin: The face, trunk and limbs were affected by an erythemato-papular and eczematous eruption. A few bullae (2 cm. in diameter) were present on the hands and feet.

Case 26. (Continued).

Cardio-Vascular System: The pulse was regular in rate and rhythm, (120 beats per minute). Apex beat was palpable five inches to the left of the mid-sternal line in the fifth left intercostal space. Heart sounds were soft and of poor quality. A soft, systolic murmur, not conducted, but persisting on change of posture, was heard at the mitral and aortic areas. Blood pressure was $\frac{200}{100}$mm. Hg.

Examination of other systems revealed no abnormality.

Investigations:

Chemical Examination of urine - Specific gravity 1024.
Reaction - Acid. No Albumin, no Sugar.
Microscopical Examination - occasional pus cell.
Culture - no growth.
Blood Urea - 23 mgm. per cent.
Wassermann Reaction - Negative.
Fixation of Specific Gravity - Highest Specific Gravity 1026.
Repeated cultures of blister fluid - no growth.
Skiagram - "Chest, no abnormality detected; Sinuses - no abnormality detected."
Electrocardiogram Report - "Sinus tachycardia."

Histological appearances of skin section were consistent with a diagnosis of pemphigus vulgaris. Skin section and description of section presented.(Volume 11, page 185.)

Case 26. (Continued).

Progress and Treatment: Potassium permanganate hand and foot baths and local Eusol soaks. 1% Ichthyol Calamine Liniment applied to other areas.

16. 9.50. "Casinal" 2 ozs. daily. Acid Hydrochlor. dil, min. 30 t.i.d.

20. 9.50. New bullae appeared on neck.

21. 9.50. Bullae appeared on arms and legs.

10.10.50. Skin healed. No oedema. Pulse 60 beats per minute. No breathlessness.

12. 6.51. Recurrence of bullous lesions on trunk.

Case 27. Varicose eczema. Hypertension.

Mr. L.B., aged 64 years, was admitted to Stobhill Hospital on 12th September, 1950, complaining of an eruption on the face and limbs of one month's duration.

Personal History: He lived with his wife and his diet was adequate.

Previous History: Measles and whooping cough in childhood. Ulceration of throat, 1914. Sciatica, 1916. Dermatitis venenata of forearms, 1948.

History of Present Condition: About one month prior to admission, an eruption appeared on the ankles. No history of trauma was obtained. The condition soon spread over the legs and face and forearms were then affected. No history of breathlessness was obtained.

General Examination: The patient was plethoric. No oedema was present at time of investigation. Varicosity of leg veins was marked. No engorgement of neck veins was noted. The patient possessed upper and lower dentures, but these were not worn at meals.

Skin: A weeping eczematous eruption was apparent on the legs. An erythemato-maculo-papular eruption was present on the face. The skin of the forearms was lichenified.

Cardio-Vascular System: Arterial walls were moderately thickened and the blood pressure was $\frac{200}{110}$mm. Hg., but no other abnormality was detected.

Case 27. (Continued).

Examination of other systems revealed no abnormality.

Investigations:

Chemical Examination of Urine - Specific gravity 1022. Reaction - Acid. Albumin present (half to one part Esbach). No Sugar.

Microscopical Examination - a few pus cells present and some debris. Culture - no growth.

Blood Urea - 15 mgm. per cent.

Wassermann Reaction - negative.

Fixation of Specific Gravity - Highest Specific Gravity 1020.

Postural change observations on the blood were carried out and no progress notes are appended.

Case 28. Infectious eczematoid dermatitis. Hypertension.

Mrs. J.L., aged 77 years, was admitted to Stobhill Hospital on 12th September, 1950, complaining of an eruption on her legs of three months' duration.

Personal History: She lived with her daughter and her diet was adequate.

Previous History: Measles, Chickenpox, Scarlet Fever and Whooping Cough in childhood. Operation on gall bladder, 1915. Pneumonia, 1947. Removal of right breast, 1948.

History of Present Condition: About three months before admission to hospital the skin of the left foot became moist and scaly. Both legs were soon affected and an eruption then appeared on the lower abdomen. The patient was not confined to bed before admission to hospital and gave no history of breathlessness.

General Examination: The patient was obese. Slight oedema was present at the ankles. No engorgement of neck veins was present. No varicosity of leg veins was noted. The patient was edentulous and upper dentures only were worn.

Skin: A weeping, scaling and excoriated eczema was present on the legs and a papulo-vesicular eruption was evident on the lower abdomen.

Cardio-Vascular System: The pulse was regular in rate and rhythm. The arterial walls were moderately thickened.

Case 28. (Continued).

No cardiac enlargement was present. Heart sounds were of fair quality. A systolic murmur, not conducted, was heard at the mitral area. The blood pressure was $\frac{210}{100}$mm. Hg.

Examination of other systems revealed no abnormality.

Investigations:

Chemical Examination of urine - Specific Gravity 1020.

Reaction - Acid. No Albumin, no Sugar.

Microscopical Examination - no abnormality detected.

Blood Urea - 29 mgm. per cent.

Wassermann Reaction - Negative.

Progress and Treatment: 1% Ichthyol Calamine Liniment as local application, followed by Argyrol 2% in Zinc Paste.

1.10.50. Acid Hydrochlor. Dil. min. 30 t.i.d.

6.11.50. Skin healed. No oedema.

Case 29. Varicose ulcer. Hypertension.

Mrs. A.M., aged 76 years, was admitted to Stobhill Hospital on 30th September, 1950, complaining of an ulcer on the right leg of four years' duration.

Personal History: She lived with her son and her diet was adequate.

Previous History: No illness.

History of Present Condition: For the previous 20 years the patient had suffered from recurrent leg ulcers. Four years before admission to hospital an ulcer had appeared on the right leg and had persisted in spite of treatment. No history of breathlessness was obtained.

General Examination: The patient was well nourished. There was no oedema. Varicosity of leg veins was present. No engorgement of neck veins was noted. The patient was edentulous, apart from a few teeth in the lower jaw.

Skin: A large ulcer (6 cm. x 2 cm.) was present on the antero-medial aspect of the right lower leg. The base was sloughing and the edge was indurated.

Cardio-Vascular System: The blood pressure was $\frac{210}{90}$mm. Hg., but no other abnormality was present.

Examination of other systems revealed no abnormality.

Investigations:

Chemical Examination of Urine - Specific gravity 1015. Reaction - Acid. No Albumin, no Sugar.

Case 29. (Continued).

Microscopical Examination - no abnormality detected.

Blood Urea - 21 mgm. per cent.

Wassermann Reaction - Negative.

Postural changes on the blood were investigated and no progress notes are appended.

Case 30. Infectious eczematoid dermatitis. Hypertension.

Mrs. R.M., aged 61 years, was admitted to Stobhill Hospital on 25th October, 1950, complaining of an eruption on both legs of five months' duration.

Personal History: She lived with her brother and her diet was adequate.

Previous History: Broncho-pneumonia, 1936.

History of Present Condition: About five months before admission to hospital a number of itching papules appeared on the left leg. The condition spread to involve both legs in spite of treatment. No history of breathlessness was obtained.

General Examination: The patient was plethoric, with marked oedema of both legs. No varicosity of leg veins was noted. No engorgement of neck veins was present.

Skin: A weeping and crusted eczematous eruption was present on both legs.

Cardio-Vascular System: The pulse was regular in rate and rhythm.. The arterial walls were not thickened. The apex beat was percussed in the fifth left intercostal space five inches from the mid-sternal line. Heart sounds were of fair quality. A soft systolic murmur, not conducted, was present at the aortic area. Blood pressure was $\frac{220}{110}$ mm. Hg.

Examination of other systems revealed no abnormality.

Case 30. (Continued).

Investigations;

Chemical Examination of Urine - Specific gravity 1020.

Reaction - Acid. No Albumin, no Sugar.

Microscopical Examination - no abnormality detected.

Skiagram - "Chest, calcified hilar glands; no evidence of active disease in the lungs. Atheroma of the aorta and increase in the cardio/thoracic ratio present."

Blood Urea - 18 mgm. per cent.

Wassermann Reaction - Negative.

Progress and Treatment: One-half per cent. Silver Nitrate in water as soaks applied locally.

28.10.50. Acid Hydrochlor. Dil. min. 30 t.i.d.

25.11.50. Skin healed. No oedema.

Case 31. Scurvy. Varicose eczema.

Mr. R.M., aged 71 years, was admitted to Stobhill Hospital on 5th December, 1950, complaining of discolouration and eczema of his legs of some weeks' duration.

Personal History: He lived alone and his diet was inadequate.

Previous History: Malaria in 1914-18 War. Eczema of the legs during the two years prior to admission.

History of Present Condition: Some weeks before admission to hospital, the legs had become swollen, discoloured and painful. No history of breathlessness was obtained.

General Examination: The patient was a thin old man. Mucous membranes were pale and oedema was present in the feet, ankles and legs. The patient was edentulous. Petechial haemorrhages were present on the hard palate. Varicosity of leg veins was evident. No engorgement of neck veins was present.

Skin: Follicular petechial haemorrhages were apparent on forearms, legs and thighs and larger areas of ecchymosis affected the legs and thighs. A few small areas of moist eczema were also present on the legs.

Cardio-Vascular System: Arterial walls were moderately thickened but no other abnormality was noted.

Blood pressure was $\frac{100}{60}$mm. Hg.

Case 31. (Continued).

Examination of other systems revealed no abnormality.

Investigations:

Chemical Examination of urine - Specific Gravity 1018.

Reaction - Acid. No Albumin, no Sugar.

Microscopical Examination - no abnormality detected.

Blood Urea - 20 mgm. per cent.

Wassermann Reaction - Negative.

Serum bilirubin - 2.2 mgm. per cent.

Ascorbic acid saturation test - Commenced 7.12.50.

7.12.50 - Daily excretion, Nil.

8.12.50 - " " Nil.

11.12.50 - " " 960 mgm. per cent.

Skiagram - Chest, no abnormality detected.

Treatment and Progress:

7.12.50. Vitamin C 700 mgm. daily. Ferri et ammon. cit. grains 30 t.i.d. "Casinal" 2 ozs. daily. "Multivite" 1 tablet t.i.d. Acid Hydrochlor. Dil. min. 30 t.i.d.

11.12.50. Vitamin C 100 mgm. t.i.d.

2. 1.51. Skin healed. No oedema.

Case 32. Scurvy. Chronic bronchitis.

Mr. J.K., aged 69 years, was admitted to Stobhill Hospital on 20th January, 1951, complaining of discolour-:ation and pain in his legs of two weeks' duration.

Personal History: He lived alone and prepared his own meals. His diet was inadequate.

Previous History: Nephrectomy (left kidney), 1930; Scurvy, 1935. Recurring winter cough.

History of Present Condition: About two weeks prior to admission to hospital, the patient noticed discolouration of the skin of the legs, and began to experience pains in the calves. He gave no history of breathlessness.

General Examination: The patient appeared moderately well-nourished. Mucous membranes were pale. Oedema was present in both legs. Varicosity of leg veins was evident. No engorgement of neck veins was present.

Skin: Purpuric ecchymoses and small petechial haemorrhages were present in both legs. Moist eczematous areas were also present on the legs.

Cardio-Vascular System: No abnormalities were detected. Blood pressure was $\frac{140}{76}$mm. Hg.

Respiratory System: Signs of chronic bronchitis were present.

Examination of other systems revealed no abnormality.

Case 32. (Continued).

Investigations:

Chemical Examination of Urine - Specific Gravity 1016.

Reaction - Acid. No Albumin, no Sugar.

Microscopical Examination - no abnormality detected.

Blood Urea - 50 mgm. per cent.

Wassermann Reaction - Negative.

Ascorbic acid saturation test - Commenced 22. 1.51.

25. 1.51. - Daily excretion of ascorbic acid - Nil.

30. 1.51. - " " " " - 248 mgm%.

Progress and Treatment:

22. 1.51. Vitamin C 700 mgm. daily. Ferri et ammon. cit. grains 30 t.i.d. Tabs. "Multivite" 1 t.i.d.

30. 1.51. Oedema gone. Vitamin C 100 mgm. t.i.d.

13. 3.51. Skin healed.

Case 33. Infectious eczematoid dermatitis.

Mrs. W.P., aged 66 years, was admitted to Stobhill Hospital on 2nd February, 1951, complaining of an eruption on both legs of some years' duration.

Personal History: She lived with her family. Her diet was deficient of meat.

Previous History: Erysipelas, 1946, 1948.

History of Present Illness: A number of years previously the patient had injured her left leg. An eruption appeared on the leg after this injury and both legs were soon affected. This eruption had persisted in spite of local treatment. No history of breathlessness was obtained.

General Examination: The patient was a thin, pale old lady. Oedema of both feet were present. No varicosity of veins and no venous engorgement was noted. The patient was edentulous.

Skin: Both legs were affected by a weeping, eczematous and crusted eruption.

Cardio-Vascular System: No abnormality was detected. Blood pressure was $\frac{180}{100}$mm. Hg.

Examination of other systems revealed no abnormality.

Investigations:

Chemical Examination of urine - Specific Gravity 1014. Reaction - Alkaline. No Albumin, no Sugar.

Case 33. (Continued).

Microscopical Examination - no abnormality detected.

Blood Urea - 34 mgm. per cent.

Wassermann Reaction - Negative.

Progress and Treatment:

2. 2.51. Ferri et ammon. cit. grains 30 t.i.d.

Vitamin C 100 mgm. t.i.d. 1% Ichthyol Calamine Liniment as local application.

28. 2.51. Skin healed. No oedema.

Case 34. Infectious eczematoid dermatitis.

Mr. J.B., aged 71 years, was admitted to Stobhill Hospital on 3rd February, 1951, complaining of an eruption on his legs and trunk of six months' duration.

Personal History: He lived alone and prepared his own food. His diet appeared to be adequate.

Previous History: No illness.

History of Present Condition: About six months previously an eruption appeared on the left thigh. Since then the condition had progressed till both legs were affected. No history of breathlessness was obtained.

General Examination: The patient was fairly well-nourished. Oedema of both legs was present. No varicosity of leg veins was noted and no venous engorgement was present. His upper jaw was edentulous. Lower incisors only were present.

Skin: A weeping, excoriated, eczematous eruption was present on the legs and arms, while a papulo-vesicular sensitisation was apparent on the trunk.

Cardio-Vascular System: No abnormality was detected. Blood pressure was $\frac{140}{70}$mm. Hg.

Examination of other systems revealed no abnormality.

Investigations:

Chemical Examination of urine - Specific Gravity 1022. Reaction - Acid. No Albumin, no Sugar.

Case 34. (Continued).

Microscopical Examination - no abnormality detected.

Blood Urea - 18 mgm. per cent.

Wassermann Reaction - Negative.

Progress and Treatment:

3. 2.51. 1% Ichthyol Calamine Liniment as local application. Ferri et ammon. cit. gr. 30 t.i.d. Vitamin C 100 mgm. t.i.d.

12. 3.51. Skin healed. No oedema.

Case 35. Scurvy. Varicose eczema.

Mrs. M.M., aged 64 years, was admitted to Stobhill Hospital on 1st February, 1951, complaining of an eruption on her legs of two weeks' duration.

Personal History: The patient lived alone and her diet was inadequate.

Previous History: Varicose ulcers recurring over the past 40 years.

History of Present Condition: About two weeks previously the patient had noticed an eruption on her legs and began to experience pains in the thighs and calves.

General Examination: The patient was a pale, thin lady. Oedema of both legs was present. Varicosity of leg veins was noted. No engorgement of neck veins was present. She was edentulous and wore upper dentures only. No history of breathlessness was obtained.

Skin: Petechial haemorrhages, ecchymoses and weeping eczematous areas were present on legs and forearms. Ecthymatous lesions were apparent on both legs and on the lower back.

Cardio-Vascular System: No abnormality was detected. Blood pressure was $\frac{140}{80}$mm. Hg.

Examination of other systems revealed no abnormality.

Case 35. (Continued).

Investigations:

Chemical Examination of Urine - Specific Gravity 1020.

Reaction - Acid. No Albumin, no Sugar.

Microscopical Examination - no abnormality detected.

Blood Urea - 24 mgm. per cent.

Wassermann Reaction - Negative.

Progress and Treatment:

3. 2.51. Vitamin C 700 mgm. daily. Ferri et ammon. cit. grains 30 t.i.d. "Multivite tablets 1 t.i.d.

1% Ichthyol Calamine Liniment locally.

12. 2.51. Vitamin C 100 mgm. t.i.d.

27. 3.51. Skin healed. No oedema.

Case 36. Varicose eczema. Hypertension.

Mr. F.M., aged 76 years, was admitted to Stobhill Hospital on 13th March, 1951, complaining of an eruption on his legs of four months' duration.

Personal History: He lived with his wife and his diet appeared to be adequate.

Previous History: No illness.

History of Present Condition: About four months previously an eruption began on his right leg. Various local applications did not improve the condition and both legs and forearms were soon affected. No history of breathlessness was obtained.

General Examination: The patient was a thin old man. Varicosity of leg veins was evident, but no oedema was apparent. No engorgement of neck veins was present. He was edentulous, apart from a few stumps in both jaws.

Skin: A weeping eczematous eruption was present on arms and legs.

Cardio-Vascular System: Blood pressure was $\frac{190}{90}$mm. Hg. Arterial walls were palpable. No other abnormality was detected.

Examination of other systems revealed no abnormality.

Investigations:

Chemical Examination of urine - Specific Gravity 1016. Reaction - Acid. No Albumin, no Sugar.

Case 36. (Continued).

Microscopical Examination - no abnormality detected.

Blood Urea - 29 mgm. per cent.

Wassermann Reaction - Negative.

Progress and Treatment:

14. 3.51. Ichthyol Calamine Liniment as local application.

10. 4.51. Skin healed.

Case 37. Scurvy. Psoriasis.

Mr. M.R., aged 73 years, was admitted to Stobhill Hospital on 30th March, 1951, complaining of an eruption on his arms and legs of three months' duration.

Personal History: He lived in a lodging house and his diet was deficient in meat and vegetables.

Previous History: Psoriasis from time to time.

History of Present Condition: About three months previously the patient had noticed an eruption on his legs. Some time later, he began to experience pains in the legs and the skin became discoloured. No history of breathlessness was obtained.

General Examination: The patient was fairly well-nourished. Mucous membranes were pale. No oedema was present. Tightening of hamstrings was evident on flexing the legs. The patient was edentulous. Haemorrhages were evident over the tonsils. No engorgement of veins was noted.

Skin: Petechial haemorrhages were present on the arms and legs. Larger ecchymoses were also evident on the legs. Pigmented areas were apparent on the trunk and limbs. Small psoriatic plaques were present on the knees and elbows.

Cardio-Vascular System: No abnormality was detected. Blood pressure was $\frac{125}{70}$mm. Hg.

Examination of other systems revealed no abnormality.

Case 37. (Continued).

Investigations:

Chemical Examination of urine - Specific gravity 1024.

Reaction - Acid. No Albumin. Sugar - a trace.

Sugar Tolerance Test - within normal limits.

Microscopical Examination of urine - no abnormality detected.

Ascorbic acid saturation test - Commenced 1. 4.51.

3. 4.51. - Daily excretion of ascorbic acid: Nil.

9. 4.51. - " " " " 221 mgm. per cent.

10. 4.51. - " " " " 640 mgm. per cent.

Blood Urea - 52 mgm. per cent.

Wassermann Reaction - Negative.

Progress and Treatment:

1. 4.51. Vitamin C 700 mgm. daily. Ferri et ammon. cit. grains 30 t.i.d. "Multivite" tablets 1 t.i.d.

10. 4.51. Vitamin C 100 mgm. t.i.d.

21. 4.51. "Casinal" 2 ozs. daily.

16. 5.51. Skin healed.

Case 38. Varicose ulcers and Varicose eczema. Hypertension.

Mrs. M.C., aged 62 years, was admitted to Stobhill Hospital on 4th April, 1951, complaining of an eruption on her legs of three weeks' duration.

Personal History: She lived with her husband and her diet seemed adequate.

Previous History: "Kidney trouble", 1914. Acute rheumatism, 1918.

History of Present Condition: The patient had been troubled with varicose veins for some years and about three weeks previously the skin of her right leg had broken down. Soon afterwards an eruption appeared on both legs. No history of breathlessness was obtained.

General Examination: The patient was plethoric. Oedema of both legs was present. Varicosity of leg veins was evident. No engorgement of neck veins was present. Upper and lower dentures were worn.

Skin: A large ulcer (6 in. in diameter) was present on the medial aspect of the left lower leg. Small crusted and ulcerated areas and weeping eczematous patches were present on both legs.

Cardio-Vascular System: Blood pressure was $\frac{230}{120}$mm. Hg., and arterial walls were thickened, but no other abnormalities were noted.

Examination of other systems revealed no abnormality.

Case 38. (Continued).

Investigations:

Chemical Examination of Urine - Specific Gravity 1020.

Reaction - Acid. No Albumin, no Sugar.

Microscopical Examination - no abnormality detected.

Blood Urea - 48 mgm. per cent.

Wassermann Reaction - Negative.

Progress and Treatment: Eusol soaks as local application, followed by 1% Ichthyol Calamine Liniment.

28. 5.51. Skin healed. No oedema.

Case 39. Infectious eczematoid dermatitis.

Mrs. J.C., aged 69 years, was admitted to Stobhill Hospital on 11th April, 1951, complaining of an eruption on her arms and legs of eight months' duration.

Personal History: She lived alone and her diet was inadequate.

Previous History: Haematemesis, 1940.

History of Present Condition: About eight months previously an eruption had appeared on the instep of her left foot. Local applications did not improve the condition and both legs and arms were soon affected. No history of breathlessness was obtained.

General Examination: The patient was a thin old lady. Mucous membranes were pale. She was edentulous. No oedema was present. No varicosity of leg veins was noted, and no venous engorgement was detected.

Skin: A weeping and erythemato-squamous eruption was present on the legs and thighs. A papulo-vesicular sensitisation affected the forearms.

Cardio-Vascular System: No abnormalities were detected. Blood pressure was $\frac{160}{80}$mm. Hg.

Examination of other systems revealed no abnormality.

Investigations:

Chemical Examination of Urine - Specific gravity 1020. Reaction - Acid. No Albumin, no Sugar.

Case 39. (Continued).

Microscopical Examination - no abnormality detected.

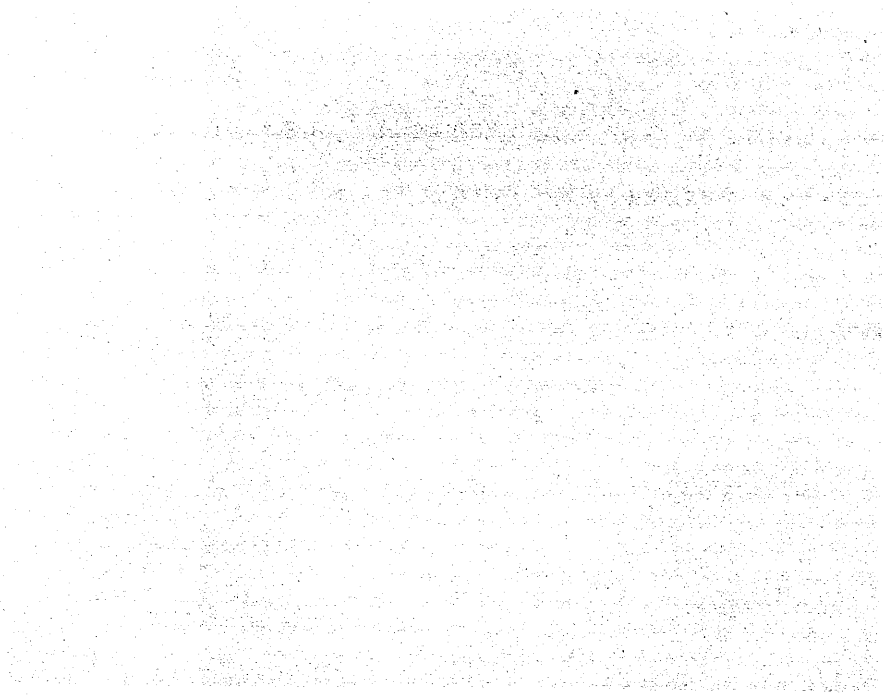
Blood Urea - 42 mgm. per cent.

Wassermann Reaction - Negative.

Progress and Treatment:

12. 4.51. 1% Ichthyol Calamine Liniment as local application.

23. 4.51. Skin healed.



Case 40. Scurvy. Chronic bronchitis.

Mr. P.H., aged 78 years, was admitted to Stobhill Hospital on 12th April, 1951, complaining of an eruption on his legs of one month's duration.

Personal History: He had been an inmate of Foresthall Institution during the previous year. Diet was inadequate.

Previous History: Sycosis Barbae, 1921. Erysipelas, 1943.

History of Present Condition: A few weeks previously the patient had noted a scaling eruption on his legs.

General Examination: The patient appeared moderately well nourished. No oedema was present. No varicosity of veins was noted and no venous engorgement was detected.

Skin: Numerous petechial haemorrhages were present on both legs. A squamous, eczematous, nummular eruption also affected both legs. Follicular keratotic lesions were apparent on the abdomen.

Cardio-Vascular System: No abnormalities were detected. Blood pressure was $\frac{160}{80}$mm. Hg.

Respiratory System: Signs of chronic bronchitis were present.

Investigations:

Chemical Examination of Urine - Specific Gravity 1018. Reaction - Acid. No Albumin, no Sugar.

Microscopical Examination - no abnormality detected.

Case 40. (Continued).

Ascorbic Acid Saturation Test - Commenced 13. 4.51.

16. 4.51. - Daily excretion of ascorbic acid - 38 mgm. per cent.
19. 4.51. - " " " " - 27 mgm. per cent.
23. 4.51. - " " " " - 50 mgm. per cent.
26. 4.51. - " " " " -132 mgm. per cent.
8. 5.51. - " " " " -356 mgm. per cent.

Blood Urea - 44 mgm. per cent.

Wassermann Reaction - Negative.

Progress and Treatment:

13. 4.51. Vitamin C 700 mgm. t.i.d., Ferri et ammon. cit. grains 30 t.i.d., "Multivite" tablets 1 t.i.d.

27. 4.51. Follicular petechial and keratotic lesions gone.

Case 41. Drug eruption. Varicose eczema.

Mr. R.F., aged 68 years, was admitted to Stobhill Hospital on 20th April, 1951, complaining of a generalised eruption of six days' duration.

Personal History: He lived with his wife and his diet was adequate.

Previous History: No illness.

History of Present Condition: About three months previously eczema had appeared on his left leg. Various local applications were prescribed and sulphonamide tablets were also taken. Shortly aftereards an eruption appeared on the face and spread to involve trunk and limbs. No history of breathlessness was obtained.

General Examination: The patient was fairly well nourished. Moderate oedema was present at ankles. Some varicosity of leg veins was noted. No venous engorgement was present.

Skin: A dusky, red, multiform eruption was present on the face, trunk and limbs. A few bullous lesions were present on the legs, where moist eczematous areas were also apparent.

Examination of other systems revealed no abnormality.

Blood pressure was $\frac{150}{80}$mm. Hg.

Investigations:

Chemical Examination of urine - Specific Gravity 1020. Reaction - Acid. No Albumin, no Sugar.

Case 41. (Continued).

Microscopical Examination - no abnormality detected.

Blood Urea - 45 mgm. per cent.

Wassermann Reaction - Negative.

Progress and Treatment:

20. 4.51. Benadryl 50 mgm. q.i.d. 1% Ichthyol Calamine Liniment as local application.

22. 4.51. Oedema gone.

5. 6.51. Skin healed.

Case 42. Infectious eczematoid dermatitis. Hypertension.

Mrs. G.B., aged 74 years, was admitted to Stobhill Hospital on 27th April, 1951, complaining of an eruption on her limbs and trunk of seven weeks' duration.

Personal History: She lived with a daughter, and her diet appeared to be adequate.

Previous History: Compound fracture of left lower leg, 1947.

History of Present Condition: Eczema of leg appeared after fracture. No history of breathlessness was obtained.

General Examination: The patient was a thin old lady. No oedema was present. No varicosity of leg veins was noted and no engorgement of neck veins detected.

Skin: The arms, legs, groins and back were affected by a weeping eczematous eruption. In places marked lichenif-:ication was evident.

Cardio-Vascular System: Occasional extra systoles occurred. The blood pressure was $\frac{260}{120}$mm. Hg. No other abnormalities were detected.

Examination of other systems revealed no abnormalities.

Investigations:

Chemical Examination of Urine - Specific Gravity 1012. Reaction - Acid. No Albumin, no Sugar.

Microscopical Examination - no abnormality detected.

Blood Urea - 31 mgm. per cent.

Case 42. (Continued).

Wassermann Reaction - Negative.

Progress and Treatment:

28. 4.51. Phenobarbitone grains ½ t.i.d. 1% Ichthyol Calamine Liniment followed by Crude Coal Tar 10% in Acetone as local application.

17. 5.51. Skin healed.

Case 43. Varicose eczema. Hypertension.

Mrs. J.H., aged 77 years, was admitted to Stobhill Hospital on 1st May, 1951, complaining of an eruption on both legs of twenty years' duration.

Personal History: She lived alone. Her diet seemed fairly adequate.

Previous History: Operation on gall bladder, 1930.

History of Present Condition: For many years the patient had been troubled with recurring eczema of the legs. No history of breathlessness was obtained.

General Examination: The patient was fairly well nourished. No oedema was present, but varicosity of leg veins was apparent. No engorgement of neck veins was detected.

Skin: Weeping eczema was present on both legs up to the knees.

Cardio-Vascular System: The pulse was regular in rate and rhythm. Arterial walls were moderately thickened. No cardiac enlargement was detected. Heart sounds were of good quality. A soft systolic murmur, not conducted but persisting with change of posture, was present at the mitral area. Blood pressure was $\frac{230}{130}$mm. Hg.

Examination of other systems revealed no abnormality.

Case 43. (Continued).

Investigations:

Chemical Examination of Urine - Specific Gravity 1018.

Reaction - Acid. No Albumin, no Sugar.

Microscopical Examination - no abnormality detected.

Blood Urea - 30 mgm. per cent.

Wassermann Reaction - Negative.

Progress and Treatment:

1. 5.51. 1% Ichthyol Calamine Liniment as local application.

22. 5.51. Skin healed.

Case 44. Varicose eczema. Hypertension, and Cardiac Failure.

Mr. J.W., aged 81 years, was admitted to Stobhill Hospital on 1st May, 1951, complaining of an eruption on his legs of ten years' duration.

Personal History: The patient's home circumstances were satisfactory and his diet was adequate.

Previous History: No illness.

History of Present Condition: The patient could not recall how the eruption began. He gave a history of breathlessness on moderate exertion.

General Examination: The patient was plethoric. Oedema of both legs and varicosity of leg veins was present. Moderate overfilling of neck veins was present.

Skin: A weeping and scaling eczema was evident on both legs. A sensitisation eruption was present on the forearms and shoulders, with scattered areas of lichenification.

Cardio-Vascular System: The pulse was regular in rate and rhythm. Arterial walls were moderately thickened. The heart was slightly enlarged to the left. Heart sounds were of poor quality, and a soft systolic murmur, not conducted, was heard at all areas. Blood pressure was $\frac{240}{110}$ mm. Hg.

Examination of other systems revealed no abnormality.

Case 44. (Continued).

Investigations:

Chemical Examination of Urine - Specific Gravity 1022.

Reaction - Acid. No Albumin, no Sugar.

Microscopical Examination - no abnormality detected.

Blood Urea - 36 mgm. per cent.

Wassermann Reaction - Negative.

Progress and Treatment:

2. 5.51. 1% Ichthyol Calamine Liniment as local application, followed by Crude Coal Tar 10% in Acetone. Phenobarbitone gr. ½ t.i.d.

18. 5.51. Skin healed. No oedema.

Case 45. Varicose eczema.

Mr. P.L., aged 68 years, was admitted to Stobhill Hospital on 1st May, 1951, complaining of an eruption on his limbs and face of some weeks' duration.

Personal History: He had been living in a lodging house for years, but his diet seemed to be reasonably adequate.

Previous History: Recurring eczema of legs during the previous few years. No history of breathlessness was obtained.

General Examination: The patient was fairly well nourished. Varicosity of leg veins was present, but no oedema was evident. No engorgement of neck veins was detected. Upper and lower dentures were worn.

Skin: A weeping eczematous eruption was apparent on the legs and a papulo-vesicular sensitisation was present on the arms and face.

No abnormalities were detected on systemic examination.

Blood pressure was $\frac{130}{70}$mm. Hg.

Investigations:

Chemical Examination of Urine - Specific Gravity 1018.

Reaction - Acid. No Albumin, no Sugar.

Microscopical Examination - no abnormality detected.

Blood Urea - 26 mgm. per cent.

Wassermann Reaction - Negative.

Case 45. (Continued).

Progress and Treatment:

1. 5.51. 1% Ichthyol Calamine Liniment locally to skin, followed by Crude Coal Tar 10% in Acetone.

18. 5.51. Skin healed.

Case 46. Infectious eczematoid dermatitis. Macrocytic Anaemia.

Mrs. B.G., aged 74 years, was admitted to Stobhill Hospital on 4th May, 1951, complaining of a generalised eruption of several weeks' duration.

Personal History: She lived alone and her diet was inadequate.

Previous History: No history obtained.

History of Present Condition: No history obtained.

General Examination: The patient was fairly well nourished. The mucous membranes were very pale. Moderate oedema of ankles was present. No varicosity of veins was noted, and no engorgement of neck veins was present. Pediculosis corporis was evident.

Skin: A weeping and crusted eczema was present on the legs and scalp. Papulo-vesicular sensitisation was apparent on the trunk, where linear excoriations and scattered pigmentation were also present.

Cardio-Vascular System: No abnormalities were detected. The blood pressure was $\frac{166}{70}$mm. Hg.

Examination of other systems revealed no abnormality.

Investigations:

Chemical Examination of Urine - Specific Gravity 1014. Reaction - Acid. No Albumin, no Sugar.

Microscopical Examination - no abnormality detected.

Case 46. (Continued).

Blood Urea - 41 mgm. per cent.

Wassermann Reaction - Negative.

Progress and Treatment:

1% Ichthyol Calamine Liniment locally to skin. Anahaemin 2 cc. twice weekly.

Patient developed broncho-pneumonia and died.

Case 47. Infectious eczematoid dermatitis. Chronic bronchitis.

Mr. T.M., aged 87 years, was admitted to Stobhill Hospital on 7th May, 1951, complaining of a generalised eruption of one month's duration.

Personal History: His home circumstances were satisfactory and his diet appeared to be adequate.

Previous History: Bronchitis from time to time.

History of Present Condition: About one month previously an eruption had appeared on the dorsum of his left hand and, soon afterwards, the arms, legs and back were affected. A history of breathlessness on moderate exertion was obtained.

General Examination: The patient was fairly well nourished. No oedema was present. No varicosity of leg veins was noted and no engorgement of neck veins was present.

Skin: A weeping eczema was present on the arms and legs. Nummular areas of papulo-vesicular sensitisation were present on the lower back and on the forehead. Some erythema was evident at the angles of the mouth.

Cardio-Vascular System: The pulse was regular in rate and rhythm. The heart was not enlarged. Heart sounds were of fair quality. A soft, systolic murmur, not conducted but persisting on change of posture, was present at the mitral

Case 47. (Continued).

area. Blood pressure was $\frac{170}{90}$mm. Hg.

Respiratory System: Signs of chronic bronchitis were present.

No abnormalities were detected on examination of other systems.

Investigations:

Chemical Examination of Urine - Specific Gravity 1024. Reaction - Neutral. No Albumin, no Sugar.

Microscopical Examination - no abnormality detected.

Blood Urea - 25 mgm. per cent.

Wassermann Reaction - Negative.

Progress and Treatment:

7. 5.51. 1% Ichthyol Calamine Liniment as local application.

4. 6.51. Skin healed.

Case 48. Varicose eczema.

Mr. J.M., aged 69 years, was admitted to Stobhill Hospital on 8th May, 1951, complaining of a generalised eruption of one week's duration.

Personal History: Lived with his wife and his diet was adequate.

Previous History: No illness.

History of Present Condition: About one year previously the patient had sustained a burn on the foot. This was followed by an eruption which cleared up after some weeks. About one week before admission to hospital the condition recurred and spread to involve the face, trunk and limbs. No history of breathlessness was obtained.

General Examination: The patient was a thin old man. Mucous membranes were pale. Varicosity of leg veins was present. No engorgement of neck veins was detected. No oedema was apparent. He was edentulous.

Skin: A weeping eczematous eruption was present on the legs, and papulo-vesicular sensitisation was apparent on the trunk, arms and face.

Cardio-Vascular System: No abnormality was detected. Blood pressure was $\frac{140}{80}$mm. Hg.

Examination of other systems revealed no abnormality.

Case 48. (Continued).

Investigations:

Chemical Examination of Urine - Specific Gravity 1026.

Reaction - Acid. No Albumin, no Sugar.

Microscopical Examination - no abnormality detected.

Blood Urea - 34 mgm. per cent.

Wassermann Reaction - Negative.

Progress and Treatment:

8. 5.51. 1% Ichthyol Calamine Liniment as local application.

21. 5.51. Skin healed.

Case 49. Varicose eczema. Chronic bronchitis and emphysema.

Mr. H.M., aged 67 years, was admitted to Stobhill Hospital on 25th May, 1951, complaining of a generalised eruption of some years' duration.

Personal History: The patient lived alone but his diet seemed reasonably adequate.

Previous History: "Cardiac Failure", 1950.

History of Present Condition: The patient had been troubled by a recurring eruption for some years. Shortly before admission the condition had become worse.

General Examination: The patient was plethoric. Varicosity of leg veins was evident, but no oedema was present. No engorgement of neck veins was present. He was breathless in bed.

Skin: A weeping eczema was present on the legs and nummular areas of papulo-vesicular eczema affected the arms, trunk and face.

Cardio-Vascular System: The pulse was regular in rate and rhythm, and arterial walls were not unduly thickened. The heart was moderately enlarged. Heart sounds were of fair quality and no murmurs were detected.

Blood pressure was $\frac{160}{85}$mm. Hg.

Respiratory System: Signs of chronic bronchitis and emphysema were present.

Case 49. (Continued).

Investigations:

Chemical Examination of Urine - Specific Gravity 1020.

Reaction - Acid. No Albumin, no Sugar.

Microscopical Examination - no abnormality detected.

Blood Urea - 29 mgm. per cent.

Wassermann Reaction - Negative.

Progress and Treatment:

25. 5.51. 1% Ichthyol Calamine Liniment locally to skin followed by 1% Crude Coal Tar in Zinc.

12. 6.51. Skin healed.

Case 50. Pemphigus Vulgaris.

Mr. A.M., aged 76 years, was admitted to Stobhill Hospital on 22nd May, 1951, complaining of a generalised eruption of six weeks' duration.

Personal History: He lived with his wife and his diet was adequate.

Previous History: Operation on gall bladder, 1945.

History of Present Condition: The eruption began on the arms about six weeks previously and spread to involve the trunk and legs about four weeks later. No history of breathlessness was obtained.

General Examination: The patient appeared fairly well-nourished. Upper and lower dentures were worn. Marked oedema of ankles was present. No varicosity of veins and no engorgement of neck veins was present.

Skin: A generalised erythema was present, while on the axillae, groins, flexures of limbs and hands and feet, bullae were evident. Lesions varied in size from one to several centimetres. Raw and crusted areas were present where bullae had ruptured. A few smaller bullous lesions were apparent on the trunk. Mucous membrane of mouth was affected. Nikolsky's Sign was present.

Cardio-Vascular System: No abnormality was detected. Blood Pressure was $\frac{150}{90}$mm. Hg.

Case 50. (Continued).

Examination of other systems revealed no abnormality.

Investigations:

Chemical Examination of Urine - Specific Gravity 1022.

Reaction - Acid. No Albumin, no Sugar.

Microscopical Examination - no abnormalities detected.

Skiagram: "Chest - no abnormality detected."

Blood Urea - 44 mgm. per cent.

Wassermann Reaction - Negative.

Fluid from bullae:- Culture, no growth.

Histological appearances of skin section were compatible with a diagnosis of Pemphigus Vulgaris.

Skin section and description of section presented.(Volume 11, page 186.)

Progress and Treatment:

31. 5.51. Temperature 101°F. Patient semi-comatose. Retention of urine. Indwelling catheter applied. "Aureomycin" 500 mgm. t.i.d. and "Dibexan" 2 capsules t.i.d. orally.

7. 6.51. Patient much improved. Bullae have disappeared. Temperature normal. No oedema. Scarring widespread on trunk and limbs.

7. 8.51. Patient has remained free from gross skin lesions, but a few abortive bullae have appeared from time to time. General condition steadily deteriorating.

Case 51. Scurvy.

Mr. B.S., aged 49 years, was admitted to Stobhill Hospital on 31st May, 1951, complaining of an eruption on his legs and pains in his calves of some weeks' duration.

Personal History: The patient lived alone and his diet was grossly inadequate.

Previous History: Scurvy - three previous attacks. Bronchitis (recurrent).

General Examination: The patient was a thin, pale individual, looking old for his years. He was edentulous. No oedema was present and no varicosity of veins was apparent. No engorgement of neck veins was present.

Skin: A follicular, petechial eruption was present on both legs, with larger purpuric, macular and ecchymotic areas.

Cardio-Vascular System: No abnormalities were detected. Blood pressure was $\frac{90}{54}$mm. Hg.

Examination of other systems revealed no abnormality.

Investigations:

Chemical Examination of Urine - Specific Gravity 1020. Reaction - Acid. No Albumin, no Sugar.

Microscopical Examination - no abnormality detected.

Blood Urea - 41 mgm. per cent.

Wassermann Reaction - Negative.

Case 51. (Continued).

Ascorbic Acid Saturation Test, commenced 1. 6.51.

4. 6.51. - Daily excretion of ascorbic acid - Nil.

5. 6.51. - " " " " - Nil.

6. 6.51. - " " " " - 323 mgm.

8. 6.51. - " " " " - 544 mgm.

Skiagram: "Chest - no abnormality detected."

Progress and Treatment:

1. 6.51. Vitamin C 700 mgm. daily. Ferri et ammon. cit. grains 30 t.i.d. "Multivite" tablets 1 t.i.d.

11. 6.51. Skin lesions cleared up, apart from staining.

Case 52. Pemphigus foliaceus. Hypertension.

Mrs. W.L., aged 57 years, was admitted to Stobhill Hospital on 11th June, 1951, complaining of a generalised eruption of three months' duration.

Personal History: She lived with her husband and her diet was adequate.

Previous History: Diphtheria in childhood. Arthritis, (1947), for which she received gold injections which were followed by a generalised dermatitis. This cleared up completely. Herpes Zoster Ophthalmicus 1949.

History of Present Condition: About three months previously an eruption had appeared on the groins. This soon became generalised. No history of breathlessness was obtained.

General Examination: The patient was well-nourished and no oedema was present. No engorgement of veins was noted.

Skin: A generalised exfoliative eruption was present. The scales were fine, papery and attached in the centre. Nikolsky's Sign was present.

Cardio-Vascular System: The pulse was regular in rate and rhythm. Arterial walls were not unduly thickened. The heart was not enlarged. Heart sounds were of good quality. A soft systolic murmur was present at the aortic area, and the second sound at the mitral area was accentuated.

Blood pressure was $\frac{250}{100}$ mm. Hg.

Case 52. (Continued).

Examination of other systems revealed no abnormality.

Investigations:

Chemical Examination of Urine - Specific Gravity 1020.

Reaction - Acid. Albumin - a trace. Sugar present.

Microscopical Examination - a few hyaline casts, red blood cells and pus cells present.

Blood Urea - 41 mgm. per cent.

Wassermann Reaction - Negative.

Sugar Tolerance Test - Normal.

Fixation of Specific Gravity - Highest Specific Gravity 1020.

Histological appearances of skin section were compatible with a diagnosis of Pemphigus foliaceus.

Skin section and description of section presented.(Volume 11, page 187.)

Progress and Treatment: Aureomycin 500 mgm. t.i.d. "Dibexan" 2 t.i.d. Some improvement apparent.

TABLES I, 2 AND 3

LIST OF ABBREVIATIONS

T. Pr. = Total Protein.

Alb. = Albumin.

Glob. = Globulin.

A/G Ratio. = Albumin/Globulin Ratio.

Hb. = Haemoglobin.

R.B.C. = Red Blood Cells.

C.I. = Colour Index.

P.C.V. = Packed Cell Volume.

M.C.V. = Mean Corpuscular Volume.

M.C.H. = Mean Corpuscular Haemoglobin.

M.C.H.C. = Mean Corpuscular Haemoglobin Concentration.

W.B.C. = White Blood Cells.

M.Eq./l. = milli Equivalents per litre.

E.S.R. = Erythrocyte Sedimentation Rate.

Thym. Turb. = Thymol Turbidity.

Alk. = Potassium Citrate, Sodium Bicarbonate aa gr. $\overline{XXX}$ 4 hourly.

"SG." = Sulphaguanidine gm. vi and gm. iii 6 hourly.

HCL. = Acid Hydrochlor. Dil. m. $\overline{XXX}$ t.i.d.

Fe. = Ferri et. Ammon. Citrat. gr. $\overline{XXX}$ t.i.d.

TABLES I, 2 AND 3

LIST OF ABBREVIATIONS (Continued)

Vit. C.	=	Redoxon mgm. 700 daily.
Pr.	=	Protein = "Casinal" 2 ounces daily.
"M."	=	"Multivite" tabs. 2 t.i.d.
+	=	present.
–	=	Not done.

T A B L E I.

Age	Sex	Case No.	Date of Admission	Date of Investigation	T.Pr. Gm.%	Alb. Gm.%	Glob. Gm.%	A/G Ratio	Hb. Gm.%	R.B.C. Millions per c.mm.	C.I.	P.C.V. ml.%	M.C.V. cubic microns	M.C.H. micro-micro-grams	M.C.H.C. %	W.B.C. per c.mm.	E. S. R. mm. 1st Hr.	E. S. R. mm. 2nd Hr.	Thym. Turb. units	Test Meal Free Acid	Exam. of Urine	Oedema	Internal Treatment
77	M.	1	1. 2.50	1.2.50	5.75	3.4	2.35	1.4	13.0	3.92	1.1	40	102	33	33	9,200	-	-	3	+	N.A.D.	+ +	Fe.,Vit.C., "M", Pr.
		"	"	18.2.50	6.0	3.6	2.4	1.5	13.3	4.16	1.1	40	96	32	33	4,400	-	-	-		"	+ +	" " "
		"	"	8.3.50	7.86	3.45	4.41	0.8	14.7	5.31	0.9	47	88.5	28	31	6,200	-	-	-		"	+	Discontinued.
		"	"	7.4.50	6.5	3.75	2.75	1.4	13.0	4.84	0.9	40	83	27	32	5,600	38	50	1		"	+	"
		"	"	27.4.50	6.55	3.65	2.9	1.3	11.3	3.9	1.0	37	95	29	31	3,600	43	53	1		"	+	"
		"	"	2.5.50	6.45	3.67	2.78	1.3	12.1	4.15	1.0	38	92	29	32	5,000	37	52	1		"	+	Fe.,Vit.C., "M", Pr.
		"	"	17.5.50	7.85	4.45	3.4	1.3	13.6	5.06	0.9	44	87	27	31	6,000	25	46	1		"	+	" " "
		"	"	8.6.50	7.55	4.4	3.15	1.4	13.6	4.38	1.0	43	98	31	32	7,800	38	54	1		"	+	Discontinued
73	M.	2	1937	16.3.50	6.15	3.55	2.6	1.4	11.0	3.1	1.2	30	97	35	37	4,400	32	48	1	+	N.A.D.	Nil	Fe., Vit.C., "M", Pr.
		"	"	3.5.50	7.55	4.85	2.7	1.8	12.9	4.35	1.0	37	85	30	35	5,200	33	50	1		"	Nil	Discontinued.
		"	"	24.5.50	7.05	4.58	2.47	1.9	11.9	4.09	1.0	35	86	29	34	5,800	26	49	1		"	Nil	"
72	M.	3	8.2.50	9.2.50	6.5	3.6	2.9	1.2	9.6	3.07	1.1	31	101	31	31	4,200	-	-	1	Nil	N.A.D.	+	Fe.,Vit.C.,"M", HCL.Pr.
		"	"	4.3.50	6.15	3.3	2.85	1.2	9.8	3.39	1.0	31	91	29	32	4,600	-	-	-		"	Nil.	" " " " "
		"	"	3.4.50	8.9	5.7	3.2	2.0	16.0	5.71	0.9	50	88	28	32	7,600	-	-	1		"	Nil.	" " " " "
66	F.	4	28.2.50	13.3.50	6.4	3.95	2.45	1.6	15.8	5.35	1.0	49	92	29	32	9,200	-	-	1	+	N.A.D.	Nil.	Nil.
		"	"	30.3.50	6.15	4.4	1.75	2.5	15.1	4.53	1.1	44	97	33	34	8,600	-	-	1		"	Nil.	Nil
93	F.	5	21.3.50	23.3.50	6.0	3.95	2.05	1.9	13.7	3.72	1.2	44	118	37	31	7,600	44	54	1	+	Pus	+	Fe.,Vit.C., "M", Pr.
		"	"	18.4.50	6.35	4.02	2.33	1.7	13.9	4.09	1.1	43	105	34	32	8,600	32	48	2		"	+	" " "
68	F.	6	4.4.50	5.4.50	7.15	4.65	2.5	1.9	13.5	4.36	1.0	40	92	31	34	4,000	47	52	1	+	N.A.D.	+	Nil
		"	"	25.4.50	7.22	4.72	2.5	1.9	12.2	4.46	0.9	40	90	27	31	5,400	39	50	1		"	Nil.	Nil.
75	M.	7	5.4.50	13.4.50	6.3	4.2	2.1	2.0	14.3	4.08	1.2	43	105	35	33	4,000	32	44	1	Nil.	N.A.D.	+	HCL
	"	"	"	4.5.50	7.6	5.0	2.6	1.9	14.0	4.68	1.0	45	96	30	31	6,000	42	50	1		"	Nil.	"
		"	"	5.6.50	7.65	5.0	2.65	1.9	13.4	4.56	1.0	45	99	29	30	7,600	28	45	1		"	Nil	"
63	M.	8	7.4.50	7.4.50	6.85	4.1	2.75	1.5	11.0	5.06	0.7	38	75	22	29	5,000	49	54	1	Nil.	Pus.	Nil.	Fe., Alk., "S"
		"	"	28.4.50	6.6	4.0	2.6	1.5	11.0	4.27	0.9	38	89	26	29	6,200	36	50	2		N.A.D.	Nil.	"S", Alk.,discontinued (20.4.50)
71	M.	9	14.4.50	18.4.50	7.45	4.86	2.59	1.9	14.2	4.37	1.1	47	108	32	30	4,400	30	48	1	-	N.A.D.	Nil.	Nil.

T A B L E I. (CONTINUED)
(2nd Sheet)

Age	Sex	Case No.	Date of Admission	Date of Investigation	T. Pr. Gm.%	Alb. Gm.%	Glob. Gm.%	A/G Ratio	Hb Gm.%	R.B.C. Millions per c.mm.	C.I.	P.C.V. ml.%	M.C.V. cubic microns	M.C.H. micro-micro-grams	M.C.HC %	W.B.C. per c.mm.	E. S. R. mm. 1st Hr.	E. S. R. mm. 2nd Hr.	Thym. Turb. units	Test Meal Free Acid	Exam. of Urine	Oedema	Internal Treatment
84	M.	10	21. 4.50	22. 4.50	7.85	4.65	3.2	1.4	13.0	4.92	0.9	43	87	26	30	4,600	43	48	1	+	N.A.D.	+	
		"	"	(6. 5.50 1.30p.m.	7.1	4.18	2.92	1.4	13.0	4.91	0.9	43	88	26	30	6,200	40	48	1		"	+	
		"	"	(6. 5.50 (8.0p.m.	7.1	4.28	2.82	1.5	13.0	4.69	0.9	42	90	28	31	6,600	41	49	1		"	+	17. 5.50 Digitalis.
		"	"	29. 5.50	7.05	4.15	2.9	1.4	13.2	4.45	1.0	43	97	30	31	6,400	34	47	1		"	Nil.	"
70	M.	11	19. 4.50	24. 4.50	7.02	4.15	2.87	1.4	12.6	4.92	0.9	44	89	26	29	6,000	26	-	1	Nil	Pus.	+	Alk., "SG"
		"	"	11.6.50	9.2	5.43	3.77	1.4	13.0	5.22	0.8	44	84	25	30	7,400	36	48	1		N.A.D.	Nil.	1. 5.50 HCL.
84	F.	12	1948	18. 5.50	7.05	4.47	2.58	1.7	13.6	4.26	1.1	44	103	32	31	6,200	25	45	1	-	N.A.D.	+	Nil.
75	F.	13	9. 5. 50	15. 5.50	6.6	4.18	2.42	1.7	12.8	4.3	1.0	42	98	30	30	5,200	19	40	2	+	Pus.	+	Alk.
		"	"	7.6. 50	6.53	4.25	2.28	1.9	12.3	4.46	0.9	40	90	28	31	8,200	27	40	1		N.A.D.	Nil.	
75	M.	14	20. 5. 50	20. 5.50	6.35	3.95	2.4	1.6	12.3	3.92	1.1	39	99	31	32	9,200	34	50	1	Nil	N.A.D.	+ +	Fe., Vit.C., HCL,Pr.,"M"
		"	"	12.6. 50	6.75	4.1	2.65	1.5	12.6	4.28	1.0	41	96	29	31	3,800	27	42	1		"	+	Discontinued.
		"	"	16. 7.50	6.5	4.15	2.35	1.8	13.4	5.05	0.9	43	85	26	31	5,400	12	25	1		"	Nil	
66	F.	15	21. 5. 50	22. 5.50	6.9	4.75	2.15	2.2	13.6	4.3	1.1	43	100	32	32	5,000	22	44	1	Nil	N.A.D.	+	Benadryl.
		"	"	9. 6.50	6.55	4.6	1.95	2.4	13.0	4.6	1.0	42	91	28	31	4,600	27	47	1		"	Nil.	Discontinued.
71	M.	16	26. 5. 50	26. 5.50	5.27	3.4	1.87	1.8	11.7	3.68	1.1	38	103	32	31	7,200	14	25	1	-	N.A.D.	+	Fe.,Vit.C., M., HCL.,Pr.
		"	"	15. 6.50	6.5	4.3	2.2	2.0	12.7	4.39	1.0	43	98	29	30	4,600	18	30	1	-	"	Nil.	Fe.,Vit.C., Pr.
		"	"	17. 8.50	6.75	4.55	2.2	2.1	12.9	4.22	1.0	43	102	31	30	4,600	15	30	1	-	"	Nil.	" " "
70	M.	17	2. 6. 50	3.6. 50	5.8	3.85	1.95	2.0	12.6	4.79	0.9	42	88	26	30	6,200	26	50	1	Nil	N.A.D.	+ +	Vit.C.,M., HCL.
		"	"	13. 6.50	6.35	4.05	2.3	1.8	13.6	5.14	0.9	45	88	26	30	6,400	16	40	1		"	+	" " " "
		"	"	13. 7.50	6.7	4.33	2.37	1.8	14.15	5.02	1.0	46	92	28	31	8,400	12	26	1		"	+	" " " "

TABLE I. (CONTINUED)
(3rd Sheet)

Age	Sex	Case No.	Date of Admission	Date of Investigation	T.Pr. Gm.%	Alb. Gm.%	Glob. Gm.%	A/G Ratio	Hb Gm.%	R.B.C. Millions per c.mm.	C.I.	P.C.V. ml.%	M.C.V. cubic microns	M.C.H. micro-micro-grams	M.C.H.C %	W.B.C. per c.mm.	Serum Total Base m.Eq/l	E. S. R. mm. 1st hr.	E. S. R. mm. 2nd hr.	Thym Turb Units	Test Meal Free Acid	Exam. of Urine	Oedema	Internal treatment
63	M	18	19.7.50	20.7.50	4.75	3.1	1.65	1.9	12.49	3.88	1.1	42	108	32	30	5,200	158.5	16	31	1	Nil	N.A.D.	+ +	Fe.,Vit.C.,"M".,HCL.,Pr.
		"	"	18. 8.50	6.3	4.0	2.3	1.7	13.3	4.59	1.0	43	94	29	31	6,400	154.5	10	27	1		"	Nil	Discontinued
		"	"	25.10.50	7.25	4.75	2.5	1.9	14.0	4.55	1.0	46	101	31	30	6,800	147.9	10	24	1		"	Nil	Thiouracil (28.8.50)
63	M	19	26.7.50	27.7.50	7.4	4.2	3.2	1.3	13.0	4.54	1.0	40	88	29	32	11,600	147.0	40	50	1	Nil	N.A.D.	+	HCL.
		"	"	10.8.50	8.1	4.4	3.7	1.2	14.35	4.8	1.0	43	90	30	33	7,600	144.7	18	40	1		"	Nil	"
75	M	20	15.8.50	21.8.50	7.8	4.25	3.55	1.2	13.4	4.7	1.0	44	90	28	30	4,800	147.5	41	44	2	Nil	N.A.D.	Nil	HCL.
		"	"	2.10.50	7.65	4.3	3.55	1.3	12.4	4.45	0.9	40	90	28	31	7,000	153.3	40	44	1		"	Nil	"
64	M	21	13.8.48	22.8.50	7.1	4.27	2.83	1.6	14.35	5.06	1.0	48	95	28	30	4,000	147.7	19	38	3	+	N.A.D.	Nil	"Stovarsol"
		"	"	16.10.50	6.02	3.5	2.52	1.4	13.6	4.91	0.9	44	90	28	31	4,400	148.9	13	34	2		"	Nil	
89	F	22	5.7.50	29.8.50	5.6	2.95	2.65	1.1	10.35	3.61	1.0	35	97	29	30	11,200	145.5	51	55	3	-	Alb.	Nil	Fe.,Vit.C.,"M"., Pr.
		"	"	14.10.50	5.2	2.52	2.68	0.9	9.1	2.97	1.0	29	98	31	31	9,400	148.3	55	65	2	-	"	Nil	" " " "
64	F	23	31.8.50	1.9.50	7.62	5.1	2.52	2.0	13.7	4.4	1.1	44	100	31	31	5,400	148.3	28	42	1	+	N.A.D.	+	Nil
		"	"	14.9.50	7.18	4.72	2.46	1.9	13.2	3.87	1.2	41	106	34	32	4,000	149.6	25	48	1		"	Nil	"
65	M	26	13.9.50	16.9.50 8 a.m.	5.95	3.8	2.15	1.8	14.05	5.28	0.9	48	91	27	29	11,400	151.4	2	4	1	Nil	N.A.D.	+ +	Pr., HCL.
		26	"	10.10.50 8 a.m.	7.7	3.6	4.1	0.9	13.4	4.38	1.0	43	98	31	31	12.800	145.8	15	28	2		"	Nil	" "

TABLE I. (CONTINUED)
(4th Sheet)

Age	Sex	Case No.	Date of Admission	Date of Investigation	T.Pr. Gm.%	Alb. Gm.%	Glob. Gm.%	A/G Ratio	Hb Gm.%	R.B.C Millions per c.mm.	C.I.	P.C.V. ml.%	M.C.V. cubic microns	M.C.H. micro-micro-grams	MC.H.C %	W.B.C. per c.mm.	Serum Total Base mEq/l	E. S. R. mm. 1st Hr.	E. S. R. mm. 2nd Hr.	Thym. Turb. Units	Test Meal Free Acid	Exam. of Urine	Oedema	Internal treatment
76	F.	28	12. 9.50	1.10.50	6.52	4.2	2.32	1.8	12.3	4.0	1.0	40	100	31	31	5,200	149.2	40	50	1	Nil	N.A.D.	+	HCL.
		"	"	6.11.50	6.62	4.35	2.27	1.9	12.2	3.96	1.0	39.5	100	31	31	8,800	147.9	44	55	1		"	Nil	
61	F.	30	25.10.50	28.10.50 8 a.m.	6.75	4.55	2.2	2.1	13.8	5.48	0.9	46	84	25	30	5,600	146.1	7	26	1	Nil	N.A.D.	+	HCL.
		"	"	25.11.50 8 a.m.	6.75	4.65	2.1	2.2	13.6	4.63	1.0	47	102	29	29	6,400	146.3	3	10	1		"	Nil	
71	M.	31	5.12.50	8.12.50	5.1	3.9	1.2	3.3	5.6	2.07	0.9	20	97	27	28	4,000	146.8	63	73	1	Nil	N.A.D.	+ +	Fe.,Vit.C.,"M".,Pr.,HCL.
		"	"	2.1. 51	6.65	4.25	2.4	1.8	12.2	4.014	1.0	41	102	30	30	4,600	149.0	33	45	1		"	Nil	" " " " "
69	M.	32	20.1.51	22.1.51	5.5	3.35	2.15	1.6	9.2	2.86	1.1	31	108	32	30	3,000	151.3	35	54	1	+	N.A.D.	+ +	Fe.,Vit.C.,"M".
		"		30.1.51	6.2	3.8	2.4	1.6	10.8	3.54	1.0	37	105	30.5	29	5,200	149.8	17	38	2		"	Nil	" " "
		"		13.3.51	6.37	3.8	2.57	1.5	13.6	4.32	1.0	45	104	31	30	4,800	151.7	3	12	2		"	"	
67	F.	33	2. 2.51	2.2. 51	7.15	3.8	3.35	1.13	10.3	3.19	1.1	34	107	32	30	6,800	151.7	56	60	2	Nil	N.A.D.	+	Fe.,Vit.C.
		"	"	28.2.51	6.55	3.55	3.0	1.18	10.1	2.72	1.1	33	110	34	31	4,000	152.0	55	60	3		"	"	" "
71	M.	34	3. 2.51	3. 2.51	6.15	4.0	2.15	1.86	12.6	3.89	1.09	43	110	32	29	8,200	148.1	9	22	1	+	N.A.D.	+	Fe.,Vit.C.
				12.3.51	6.1	4.05	2.05	1.97	14.15	4.98	1.0	48	96	28	29	8,200	148.9	5	16	1		"	Nil	" "
64	F.	35	1. 2.51	3.2.51	6.0	3.25	2.75	1.18	10.85	3.41	1.07	36	106	32	30	5,600	147.0	40	54	3	+	N.A.D.	+	Fe.,Vit.C.,"M".
				27.3.51	6.6	4.0	2.6	1.54	10.9	3.69	1.0	36	98	30	30	3,200	149.4	32	53	3		"	Nil	" " "
76	M.	36	13.3.51	14.3.51	5.9	3.6	2.3	1.56	13.2	4.49	1.0	45	100	29	29	4,000	149.7	8	20	1	+	N.A.D.	Nil.	
				10.4.51	6.25	3.85	2.4	1.6	13.6	4.68	1.0	48	103	29	28	4,800	151.4	8	26	2				
73	M.	37		1.4.51	5.65	3.6	2.05	1.7	7.4	2.23	1.1	24	108	33	31	1,200	150.3	52	66	2	+	Sugar	Nil	Fe.,Vit.C.
				21.4.51	5.65	3.5	2.15	1.6	10.3	2.93	1.2	33.5	113	35	31	4,600	150.3	15	30	2		N.A.D.	Nil	" " Pr.
				16.5.51	6.7	4.2	2.5	1.68	12.1	3.79	1.1	40	106	32	30	5,200	149.5	17	34	2		"	Nil	" " "
62	F.	38	4.4.51	5.4.51	6.5	3.8	2.7	1.4	12.0	4.77	0.9	41	86	25	29	5,800	149.8	15	38	1	+	N.A.D.	+ +	Nil.
				28.5.51	6.8	4.2	2.6	1.6	12.5	5.33	0.8	42.5	80	23	29	7,200	148.3	13	34	2		"	Nil	

TABLE I. (CONTI
(5th S...

Age	Sex	Case No.	Date of Admission	Date of Investigation	T.Pr. Gm.%	Alb. Gm.%	Glob. Gm.%	A/G. Ratio	Hb. Gm.%	R.B.C. Millions per c.mm.	C.I.	P.C.V. ml.%	M.C.V. cubic microns	M.C.H. micro-micro-grams	M.C.H.C %	W.B.C. per c.mm.	Serum Total Base m.Eq/l	E.S.R. mm. 1st Hr.	E.S.R. mm. 2nd Hr.	Thym. Turb. Units	Test Meal Free Acid	Exam. of Urine	Oedema	Internal Treatment
69	F	39	11. 4.51	12.4.51	5.95	3.8	2.15	1.8	13.6	4.8	1.0	45	94	28	30	6,000	149.8	10	30	3	-	N.A.D.	Nil	Nil
				23.4.51	6.4	4.2	2.2	1.9	13.2	4.89	0.9	45	92	27	29	9,200	147.5	12	37	3	-	N.A.D.	Nil	
78	M	40	12. 4.51	13.4.51	6.1	3.8	2.3	1.7	12.6	4.21	1.0	43	102	30	29	5,400	151.5	34	46	1	-	N.A.D.	Nil	Vit.C., Fe.
				27.4.51	6.5	4.05	2.45	1.65	12.6	3.96	1.1	42	106	32	30	9,000	153.2	30	46	2			Nil	
68	M	41	20.4.51	20.4.51	5.0	3.35	1.65	2.03	12.2	4.29	1.0	40	93	28	30	3,600	148.7	26	45	1	Nil	N.A.D.	+	Benodryl.
				22.4.51	5.25	3.6	1.65	2.18	13.0	4.74	0.9	43	91	27	30	4,600	151.2	26	45	1			Nil	
				5.6.51	7.3	4.6	2.7	1.7	13.8	4.92	0.9	46	93	28	30	4,600	-	27	45	3			Nil	
74	F	42	27.4.51	28.4.51	6.75	4.25	2.5	1.7	11.75	4.46	0.9	39	87	26	30	5,800	149.2	29	46	2	+	N.A.D.	Nil	Phenobarb.
				17.5.51	7.3	4.45	2.85	1.56	12.1	4.32	0.9	41	95	28	30	5,600	147.9	32	47	2			Nil	
77	F	43	1.5.51	1.5.51	7.3	5.15	2.15	2.4	13.6	4.75	0.9	46	97	29	30	5,200	146.8	25	42	1	+	N.A.D.	Nil	Nil
				22.5.51	6.75	4.7	2.05	2.3	12.1	3.94	1.0	40	101	31	30	8,200	147.7	45	54	2			Nil	
81	M	44	1.5.51	2.5.51	5.8	4.0	1.8	2.2	13.2	4.96	0.9	45.5	92	27	29	3,600	150.0	10	22	1	-	N.A.D.	+	Phenobarb.
				18.5.51	6.2	3.9	2.3	1.7	13.4	4.56	1.0	46	101	29	29	4,600	150.3	23	43	1			Nil	
68	M	45	1.5.51	2.5.51	6.2	4.45	1.75	2.54	13.6	4.96	0.9	44.5	90	27	31	3,600	151.4	15	30	1	+	N.A.D.	Nil	Nil
				18.5.51	7.45	4.9	2.55	1.92	14.3	5.25	0.9	48.5	92	27	29	6,800	149.5	8	24	2			Nil	
74	F	46	4.5.51	5.5.51	5.6	3.35	2.25	1.48	7.2	2.31	1.0	27	117	31	27	7,400	152.7	30	46	2	-	N.A.D.	+	Anahaemin.
87	M	47	7.5.51	8.5.51	6.0	4.05	1.95	2.07	13.6	5.02	0.9	46.5	93	27	29	9,600	151.0	15	34	1	+	N.A.D.	Nil	Nil
				4.6.51	7.0	4.0	3.0	1.33	12.8	4.49	1.0	44	98	29	29	6,000	-	41	47	1			Nil	
69	M	48	8.5.51	10.5.51	6.05	4.05	2.0	2.02	12.8	4.06	1.0	42	103	32	30	5,000	151.1	14	28	1	-	N.A.D.	Nil	Nil
				21.5.51	6.2	4.15	2.05	2.0	13.6	4.48	1.0	44	98	30	31	8,000	151.4	9	19	2			Nil	
67	M	49	25.5.51	26.5.51	7.15	4.2	2.95	1.42	13.55	4.33	1.0	44.5	103	31	30	6,800	144.1	24	43	2	+	N.A.D.	Nil	Nil
				12.6.51	7.25	4.3	2.95	1.45	13.6	4.59	1.0	43.5	95	30	31	5,400	-	24	45	3			Nil	
76	M	50	22.5.51	31.5.51	4.95	3.25	1.7	1.91	11.2	3.79	1.0	39	103	30	29	6,800	-	33	50	1	Nil	N.A.D.	+ +	Pr.,Aureomycin.
				7.6.51	5.8	3.5	2.3	1.52	12.2	4.44	0.9	41	92	27	30	6,600	-	46	52	3			Nil	
49	M	51	31.5.51	1.6.51	6.2	4.3	1.9	2.26	10.7	3.59	1.0	34	95	30	31	3,000	148.3	33	50	2	Nil	N.A.D.	Nil	Fe.,Vit.C.
				11.6.51	6.35	5.3	2.05	2.1	11.85	3.76	1.0	38	101	32	31	3,200	-	15	26	2			Nil	
57	F	52	11.6.51	13.6.51	6.5	4.15	2.35	1.76	11.85	3.91	1.0	41.5	106	30	29	6,000	-	40	49	2	Nil	Alb.Sugar a trace	Nil	Aureo-mycin.

T A B L E 2.

POSTURAL CHANGES. (A)

Age	Sex	Case No.	Date of Admission	Date of Investigation	T.Pr. Gm.%	Alb. Gm.%	Glob. Gm.%	A/G. Ratio	Hb Gm%	R.B.C. Millions per c.mm.	C.I.	P.C.V. ml.%	M.C.V. cubic microns	M.C.H. micro-micro-grams	M.C.H.C %	W.B.C. per c.mm.	Serum Total Base m/Eq/l	E.S.R. mm. 1st Hr.	E.S.R. mm. 2nd Hr.	Thym. Turb. Units	Test Meal Free Acid	Exam. of Urine	Oedema
78	M	24	1. 4.49	3. 9.50 8.0 a.m. In Bed	7.05	4.4	2.65	1.6	13.9	5.07	0.9	46	91	27	30	7,800	148.6	21	40	1	Nil	N.A.D.	Nil
				3.9.50 9.0 a.m. seated	7.9	5.07	2.83	1.8	14.35	5.38	0.9	49	91	27	29	10,200	147.6	7	15	1			Nil
79	M	25	7. 9.50	11.9.50 8.0a.m.In bed	7.34	3.62	3.72	1.0	11.7	4.21	0.9	37	88	28	32	5,600	149.4	52	60	1	+	tr. alb.	+
		"	"	11.9.50 9.0a.m seated	7.95	4.05	3.9	1.0	12.3	4.41	0.9	40	91	28	31	8,400	147.6	37	54	1			+
65	M	26	13.9.50	16.9.50 8.0a.m.In bed 9.0a.m. seated.	5.95	3.8	2.15	1.8	14.05	5.28	0.9	48	91	27	29	11,400	151.4	2	4	1	Nil	N.A.D.	+ +
				16.9.50	6.3	3.95	2.35	1.7	14.15	5.33	0.9	50	94	27	29	16,400	150.8	1	2	1			+ +
64	M	27	12.9.50	21.9.50 8.0 a.m. In bed	7.85	5.02	2.83	1.8	14.55	5.29	0.9	47	89	27.5	31	6,400	149.1	2	15	1	+	Alb.	Nil
				21.9.50 9.0 a.m. seated	8.45	5.4	3.05	1.8	15.15	5.69	0.9	50	88	26.6	30	6,400	149.2	4	20	1			Nil
76	F.	29	30.9.50	11.10.50 8.0a.m. In bed	7.2	4.5	2.7	1.6	11.4	4.05	0.95	36.5	90	28	31	2,800	148.7	37	-	2	Nil	N.A.D.	Nil
				11.10.50 9.0a.m.Seated	7.45	4.71	2.74	1.7	11.8	4.38	0.91	38.5	88	27	31	4,800	147.0	46	-	2			Nil
61	F.	30	25.10.50	25.11.50 8.0 a.m.In bed	6.75	4.65	2.1	2.2	13.6	4.63	1.0	47	102	29	29	6,400	146.33	3	10	1	Nil	N.A.D.	Nil
				25.11.50 9.0 a.m.Seated	7.2	4.62	2.58	1.8	14.0	4.92	1.0	48	98	28.5	29	10,400	139.1	5	12	1			Nil

TABLE 2.

POSTURAL CHANGES (B)

Age	Sex	Case No.	Date of Admission	Date of Investigation	Posture	T.Pr. Gm.%	Alb. Gm.%	Glob. Gm.%	A/G Ratio	Hb. Gm.%	R.B.C. Millions per c.mm.	C.I.	P.C.V. ml.%	M.C.V. cubic microns	M.C.H. micro-micrograms	M.C.H.C %	W.B.C. per c.mm.	Serum Total Base m.Eq/l	E.S.R. mm. 1st Hr.	E.S.R. mm. 2nd Hr.	Thym. Turb. Units	Test Meal Free Acid	Exam. of Urine	Oedema	Internal Treatment
63	M	19	26.7.50	26.7.50	Seated	7.9	4.5	3.4	1.3	13.2	4.63	1.0	43	93	29	31	12,400	144.7	20	43	1		N.A.D.	+	HCL
				27.7.50	In bed	7.4	4.2	3.2	1.3	13.0	4.54	1.0	40	88	29	32	11,600	147.0	40	50	1		"	+	
				28.7.50	" "	7.3	4.05	3.25	1.2	12.5	4.29	1.0	39	91	29	32	8,600	148.0	42	52	1		"	+	
				30.7.50	" "	7.56	4.07	3.46	1.1	13.0	4.46	1.0	40	90	29	32	7,600	148.3	47	50	1		"	Nil	
				3.8.50	" "	8.6	4.6	4.0	1.2	12.8	4.44	1.0	40	90	29	32	10,400	148.3	47	50	1		"	Nil	
				10.8.50	" "	8.1	4.4	3.7	1.2	14.35	4.8	1.0	43	90	30	33	7,600	144.7	18	40	1		"	Nil	
				11.8.50	Seated	8.95	4.65	4.3	1.1	13.3	4.51	1.0	43	95	30	31	9,400	142.5	19	36	1		"	Nil	
64	F	23	31.8.50	31.8.50	Seated	7.85	5.3	2.55	2.1	14.1	5.26	0.9	47	89	27	30	4,400	147.5	26	42	1		N.A.D	+	Nil
				1.9.50	In bed	7.62	5.1	2.52	2.0	13.7	4.4	1.1	44	100	31	31	5,400	148.3	28	42	1		"	+	
				2.9.50	" "	8.1	5.35	2.75	1.9	13.8	5.15	0.9	45	87	27	31	4,600	146.7	7	20	1		"	+	
				14.9.50	" "	7.18	4.72	2.46	1.9	13.2	3.87	1.2	41	106	34	32	4,000	149.6	25	48	1		"	Nil	
62	F	38	4.4.51	4.4.51	Seated	7.3	4.5	2.8	1.6	12.8	5.19	0.8	45	87	25	28	10,000	146.3	15	38	1		N.A.D.	+ + +	Nil
				5.4.51	In bed	6.5	3.8	2.7	1.4	12.0	4.77	0.85	41	86	25	29	5,800	149.8	15	38	1		"	+ +	
				6.4.51	" "	6.7	4.2	2.5	1.7	12.2	4.95	0.8	42	85	25	29	6,400	149.1	17	42	1		"	+	
				7.4.51	" "	7.35	4.5	2.85	1.6	13.0	5.22	0.8	45	86	25	27	6,000	148.0	11	30	1		"	Nil	
				28.5.51	" "	6.8	4.2	2.6	1.6	12.5	5.33	0.8	42.5	80	23	29	7,200	148.3	13	34	2		"	Nil	
69	F	39	11.4.51	11.4.51	Seated	6.85	4.15	2.7	1.5	14.55	4.96	1.0	51.	103	29	29	7,800	145.7	6	27	3		N.A.D.	Nil	Nil
				12.4.51	In bed	5.95	3.8	2.15	1.8	13.6	4.8	1.0	45	94	28	30	6,000	149.8	10	30	3		"	"	
				13.4.51	" "	5.95	3.7	2.25	1.6	13.2	4.7	0.9	44	94	28	30	6,600	152.3	15	28	3		"	"	
				14.4.51	" "	6.35	3.8	2.55	1.5	13.6	4.91	0.9	46	94	28	30	6,400	149.5	10	27	4		"	"	
				16.4.51	" "	6.25	3.9	2.35	1.65	13.6	4.89	0.9	46	94	27	29	7,200	148.7	12	32	3		"	"	
				23.4.51	" "	6.4	4.2	2.2	1.9	13.2	4.89	0.9	45	92	30	29	9,200	147.5	12	37	3		"	"	
67	M	49	25.5.51	25.5.51	Seated	7.95	4.8	3.15	1.5	13.4	4.45	1.0	44.5	100	30	30	5,200	145.2	45	49	2		N.A.D.	Nil	Nil
				26.5.51	In Bed	7.15	4.2	2.95	1.4	13.55	4.33	1.0	44.5	103	31	30	6,800	144.2	24	43	2		"	"	
				12.6.51	" "	7.25	4.3	2.95	1.45	13.6	4.59	1.0	43.5	95	30	31	5,400	Not done	24	45	3		"	"	

TABLE 3.

(CONTROLS)

No.	Age	Sex	T.Pr. Gm.%	Alb. Gm.%	Glob. Gm. %	A/G Ratio	Hb Gm.%	R.B.C. millions per c.mm.	C.I.	P.C.V. ml.%	M.C.V. cubic microns	M.C.H. micro-micro-grams	M.C.H.C. %	W.B.C. per c.mm.	Serum Total Base m.Eq./l.	E. S. R. mm.		Thymol Turbidity Units
																1st Hr.	2nd Hr.	
1	82	M	6.75	4.45	2.3	1.93	14.0	4.66	1.0	46	99	30	30	6,200	146.6	20	40	1
2	70	M	6.25	4.3	1.95	2.2	12.6	4.1	1.0	41	100	31	31	6,000	145.3	10	22	1
3	71	M	6.2	4.1	2.1	1.96	12.8	4.84	0.9	43	89	26	28	7,800	150.3	12	36	1
4	72	M	6.6	4.25	2.35	1.8	15.0	4.69	1.1	49	104	32	31	7,800	148.3	6	26	1
5	67	M	6.35	4.45	1.9	2.34	15.3	4.88	1.1	50	102	31	31	6,000	149.3	2	15	1
6	86	F	6.55	3.9	2.65	1.47	10.25	4.52	0.8	39	86	23	26	7,000	145.8	34	50	2
7	60	F	7.2	4.0	3.2	1.25	12.8	4.62	0.9	44	95	28	29	11,600	144.2	11	40	2
8	64	F	6.7	4.1	2.6	1.57	12.6	4.85	0.9	41	85	26	31	9,200	152.2	41	49	2
9	77	F	6.75	4.45	2.3	1.93	14.1	5.15	0.9	47	91	27	30	6,000	148.1	13	35	2
10	84	F	6.3	3.95	2.35	1.7	12.19	4.33	1.0	41	95	28	30	5,800	145.3	40	50	1
Total M & F.			65.65	41.95	23.7		131.6	46.64		441	946	282	297					
Total M.			32.15	21.55	10.6		69.7	23.17		229	504	150	151					
Total F.			33.5	20.4	13.1		61.9	23.47		212	452	132	146					
Average M.& F.			6.56	4.19	2.37		13.2	4.66		44	95	28	30					
Average M.			6.43	4.31	2.12		13.9	4.63		46	101	30	30					
Average F.			6.7	4.08	2.62		12.4	4.69		42	90	26	29					

TABLE 3 (Continued.)

Control No.	Blood urea mgm. %	Wassermann Reaction
1	18	Negative
2	24	"
3	27	"
4	19	"
5	26	"
6	27	"
7	19	"
8	28	"
9	40	"
10	47	"

TABLE 4

LIST OF ABBREVIATIONS.

N.	=	NEUTROPHILS.
E.	=	EOSINOPHILS.
B.	=	BASOPHILS.
L.L.	=	LARGE LYMPHOCYTES.
S.L.	=	SMALL LYMPHOCYTES.
M.	=	MONOCYTES.

TABLE 4

Case No.	Date.	Leucocytes per cu.mm.	Differential Leucocyte count per cent.						Differential Leucocyte count per cu.mm.					
			N.	E.	B.	L.L.	S.L.	M.	N.	E.	B.	L.L.	S.L.	M.
1	1. 2.50	9,200	78	-	-	6	16	-	7,176	-	-	552	1,472	-
2	16. 3.50	4,400	67	-	-	14	17	2	2,948	-	-	616	748	88
3	9. 2.50	4,200	80	-	-	6	11	3	3,360	-	-	252	462	126
4	13. 3.50	9,200	79	-	-	2	16	3	7,268	-	-	184	1,472	276
5	23. 2.50	7,600	77	-	-	13	10	-	5,852	-	-	988	760	-
6	5. 4.50	4,000	55	-	-	30	12	3	2,200	-	-	1,200	480	120
7	13. 4.50	4,000	53	-	-	24	19	4	2,120	-	-	960	760	160
8	7. 4.50	5,000	54	-	-	16	27	3	2,700	-	-	800	1,350	150
9	18. 4.50	4,400												
10	22. 4.50	4,600	69	-	-	6	20	5	3,174	-	-	276	920	230
11	24. 4.50	6,000	58	-	1	21	20	-	3,480	-	60	1,260	1,200	-
12	18. 5.50	6,200	60	-	-	3	35	2	3,720	-	-	186	2,170	124
13	15. 5.50	5,200	57	-	-	8	34	1	2,964	-	-	416	1,768	52
14	20. 5.50	9,200	81	-	-	5	13	1	7,452	-	-	460	1,196	92
15	22. 5.50	5,000	70	1	-	10	16	3	3,500	50	-	500	800	150
16	26. 5.50	4,600	61	-	-	19	19	1	4,392	-	-	1,368	1,368	72
17	3. 6.50	6,200	67	-	-	15	18	-	4,154	-	-	930	1,116	-
18	20. 7.50	5,200	63	1	-	11	23	2	3,276	52	-	572	1,196	104
19	26. 7.50	12,400	75	1	-	5	17	2	9,300	124	-	620	2,108	248
20	21. 8.50	4,800	65	-	-	12	23	-	3,120	-	-	576	1,104	-
21	22. 8.50	4,000	68	3	-	7	21	1	2,720	120	-	280	840	40
22	29. 8.50	11,200	56	3	-	10	26	5	6,272	336	-	1,120	2,912	560
23	31. 8.50	4,400	66	-	-	11	23	-	2,904	-	-	484	1,012	-
24	3. 9.50	7,800	63	-	-	11	24	2	4,914	-	-	858	1,872	156
25	11. 9.50	5,600	57	-	-	12	31	-	3,192	-	-	672	1,736	-
26	16. 9.50	11,400	80	1	-	4	15	-	9,120	114	-	456	1,710	-

TABLE 4 (Continued)

Case No.	Date.	Leucocytes per cu.mm.	Differential Leucocyte count per cent.						Differential Leucocyte count per cu.mm.					
			N.	E.	B.	L.L.	S.L.	M.	N.	E.	B.	L.L.	S.L.	M.
27	21. 9.50	6,400	55	-	-	18	24	3	3,520	-	-	1,152	1,536	192
28	1.10.50	5,200	63	-	-	15	19	3	3,276	-	-	780	988	156
29	11.10.50	2,800	32	-	-	13	54	1	896	-	-	364	1,512	28
30	28.10.50	5,600	74	-	-	4	16	6	4,144	-	-	224	896	336
31	8.12.50	4,000	47	-	1	14	37	1	1,880	-	40	560	1,480	40
32	22. 1.51	3,000	62	-	1	8	27	2	1,860	-	30	240	810	60
33	2. 2.51	6,800	78	-	-	6	14	2	5,304	-	-	408	952	136
34	3. 2.51	8,200	80	-	-	9	6	5	6,560	-	-	720	492	410
35	3. 2.51	5,600	65	-	-	10	23	2	3,640	-	-	650	1,288	112
36	14. 3.51	4,000	68	-	-	6	23	3	2,720	-	-	240	920	120
37	1. 4.51	1,200	72	-	-	11	16	1	864	-	-	132	192	12
38	4. 4.51	10,000	76	-	-	5	16	3	7,600	-	-	500	1,600	300
39	11. 4.51	7,800	66	-	-	8	25	1	5,148	-	-	624	1,950	78
40	13. 4.51	5,400	64	6	-	2	25	3	3,456	324	-	108	1,350	162
41	20. 4.51	3,600	54	5	-	6	11	5	1,944	180	-	216	396	180
42	28. 4.51	5,800	46	10	1	11	22	10	2,668	580	58	638	1,276	580
43	1. 5.51	5,200	48	4	-	9	38	1	2,496	208	-	468	1,976	52
44	2. 5.51	3,600	60	2	-	8	29	1	2,160	72	-	288	1,044	36
45	2. 5.51	3,600	44	-	-	13	41	2	1,584	-	-	468	1,476	72
46	5. 5.51	7,400	71	-	-	12	16	1	5,254	-	-	888	1,184	74
47	8. 5.51	9,600	60	2	-	5	30	3	5,760	192	-	480	2,880	288
48	10. 5.51	5,000	73	2	-	3	21	1	3,650	100	-	150	1,050	50
49	25. 5.51	5,200	76	-	-	6	16	2	3,952	-	-	312	832	104
50	31. 5.51	6,800	63	17	-	5	15	-	3,084	1,156	-	340	1,020	-
51	1. 6.51	3,000	55	-	-	10	32	3	1,650	-	-	300	960	90
52	13. 6.51	6,000	51	14	-	10	22	3	3,060	840	-	600	1,320	180

TABLE 4 (Continued)

Control No.	Leucocytes per cu.mm.	Differential Leucocyte count per cent.						Differential Leucocyte count per cu.mm.					
		N.	E.	B.	L.L.	S.L.	M.	N.	E.	B.	L.L.	S.L.	M.
1	6,200	57	-	-	14	27	2	3,534			868	1,674	124
2	6,000	74	1	-	8	15	2	4,440	60	-	480	900	120
3	7,800	62	-	-	11	24	3	4,836	-	-	858	1,872	234
4	7,800	72	-	-	9	17	2	5,616	-	-	702	1,326	156
5	6,000	63	2	-	3	28	4	3,780	120	-	180	1,680	240
6	7,000	83	-	-	4	11	2	5,810	-	-	280	770	140
7	11,600	66	-	-	1	32	1	7,656	-	-	116	3,712	116
8	9,200	69	-	-	14	16	1	6,348	-	-	1,288	1,472	92
9	6,000	62	-	-	17	18	3	3,720	-	-	1,020	1,080	180
10	5,800	81	-	-	10	7	2	4,698	-	-	580	406	116

TABLE 5

LIST OF ABBREVIATIONS.

E.E.L.	=	PHOTOELECTRIC COLORIMETER.
T.Pr.	=	Total Serum Protein.
Alb.	=	Serum Albumin.

TABLE 5

E.E.L. READING.

Control No.	Duplicate T.Pr. Gm.%	Duplicate Alb. Gm.%	Average. T.Pr. Gm.%	Average. Alb. Gm.%	T.Pr. Gm.%	Alb. Gm.%
1	(25.0 (25.2	(16.5 (16.5	25.1	16.5	6.75	4.45
2	(23.2 (23.0	(15.8 (16.0	23.1	15.9	6.25	4.3
3	(23.0 (22.8	(15.2 (15.2	22.9	15.2	6.2	4.1
4	(24.5 (24.6	(15.8 (15.6	24.55	15.7	6.6	4.25
5	(23.8 (24.0	(16.5 (16.5	23.9	16.5	6.35	4.45
6	(24.2 (24.2	(14.4 (14.4	24.2	14.4	6.55	3.9
7	(26.8 (26.6	(14.8 (14.6	26.7	14.7	7.2	4.0
8	(24.8 (25.0	(15.2 (15.2	24.9	15.2	6.7	4.1
9	(25.0 (25.0	(16.6 (16.4	25.0	16.5	6.75	4.45
10	(23.2 (23.4	(14.6 (14.6	23.3	14.6	6.3	3.95

TABLE 5

E.E.L. READING.

Case No.	Date.	Duplicate. T.Pr. Gm.%	Duplicate. Alb. Gm.%	Average. T.Pr. Gm.%	Average. Alb. Gm.%	T.Pr. Gm.%	Alb. Gm.%
1	8. 3.50	(29.2 (29.2	(12.8 (12.8	29.2	12.8	7.86	3.45
"	7. 4.50	(23.8 (24.2	(13.8 (13.8	24.0	13.8	6.5	3.75
"	27. 4.50	(24.5 (24.0	(13.5 (13.5	24.25	13.5	6.55	3.65
"	2. 5.50	(23.8 (24.0	(13.6 (13.6	23.9	13.6	6.45	3.67
"	17. 5.50	(29.2 (29.2	(16.4 (16.6	29.2	16.5	7.85	4.45
"	8. 6.50	(28.0 (28.2	(16.4 (16.4	28.1	16.4	7.55	4.4
2	16. 3.50	(22.8 (22.7	(13.2 (13.2	22.75	13.2	6.15	3.55
"	3. 5.50	(27.8 (28.2	(18.0 (18.0	28.0	18.0	7.55	4.85
"	24. 5.50	(26.2 (26.2	(17.0 (17.0	26.2	17.0	7.05	4.58
3	4. 3.50	(22.8 (22.8	(12.2 (12.1	22.8	12.15	6.15	3.3
"	3. 4.50	(33.1 (33.1	(21.2 (21.2	33.1	21.2	8.9	5.7
4	13. 3.50	(23.8 (23.7	(14.6 (14.6	23.75	14.6	6.4	3.95
"	30. 3.50	(22.8 (22.8	(16.4 (16.4	22.8	16.4	6.15	4.4

TABLE 5 (Continued)

E.E.L. READING.

Case No.	Date.	Duplicate. T.Pr. Gm.%	Duplicate. Alb. Gm.%	Average. T.Pr. Gm.%	Average. Alb. Gm.%	T.Pr. Gm.%	Alb. Gm.%
5	23. 2.50	(22.2 (22.4	(14.6 (14.8	22.3	14.7	6.0	3.95
"	18. 4.50	(23.4 (23.6	(14.8 (15.0	23.5	14.9	6.35	4.02
6	5. 4.50	(26.6 (26.4	(17.4 (17.2	26.5	17.3	7.15	4.65
"	25. 4.50	(26.8 (26.8	(17.6 (17.6	26.8	17.6	7.22	4.72
7	13. 4.50	(23.2 (23.6	(15.4 (15.8	23.4	15.6	6.3	4.2
"	4. 5.50	(28.2 (28.2	(18.6 (18.6	28.2	18.6	7.6	5.0
"	5. 6.50	(28.4 (28.2	(18.6 (18.4	28.3	18.5	7.65	5.0
8	7. 4.50	(25.2 (25.6	(15.2 (15.2	25.4	15.2	6.85	4.1
"	28. 4.50	(24.5 (24.5	(14.8 (14.8	24.5	14.8	6.6	4.0
9	18. 4.50	(27.8 (27.6	(18.0 (18.2	27.7	18.1	7.45	4.86

TABLE 5 (Continued)

E.E.L. READING.

Case No.	Date.	Duplicate. T.Pr. Gm.%	Duplicate. Alb. Gm.%	Average. T.Pr. Gm.%	Average. Alb. Gm.%	T.Pr. Gm.%	Alb. Gm.%
10	22. 4.50	(29.2 (29.0	(17.4 (17.2	29.1	17.3	7.85	4.65
"	6. 5.50 1.30p.m.	(26.4 (26.4	(15.5 (15.5	26.4	15.5	7.1	4.18
"	6. 5.50 8.0.p.m.	(26.2 (26.5	(15.8 (15.8	26.35	15.8	7.1	4.28
"	29. 5.50	(26.2 (26.2	(15.2 (15.6	26.2	15.4	7.05	4.15
11	24. 4.50	(26.0 (26.2	(15.4 (15.2	26.1	15.3	7.02	4.15
"	11. 6.50	(34.0 (34.2	(20.2 (20.0	34.1	20.1	9.2	5.43
12	18. 5.50	(26.2 (26.2	(16.6 (16.6	26.2	16.6	7.05	4.47
13	15. 5.50	(24.4 (24.6.	(15.4 (15.6	24.5	15.5	6.6	4.18
"	7. 6.50	(24.2 (24.2	(15.8 (15.5	24.2	15.65	6.53	4.25
14	20. 5.50	(23.6 (23.4	(14.6 (14.6	23.5	14.6	6.35	3.95
"	12. 6.50	(25.2 (24.8	(15.2 (15.2	25.0	15.2	6.75	4.1
"	16. 7.50	(24.0 (24.2	(15.4 (15.4	24.1	15.4	6.5	4.15

TABLE 5 (Continued)

E.E.L. READING.

Case No.	Date.	Duplicate. T.Pr. Gm.%	Duplicate. Alb. Gm.%	Average. T.Pr. Gm.%	Average. Alb. Gm.%	T.Pr. Gm.%	Alb. Gm.%
15	22. 5.50	(25.5 (25.8	(17.5 (17.8	25.65	17.65	6.9	4.75
"	9. 6.50	(24.4 (24.2	(17.0 (17.0	24.3	17.0	6.55	4.6
16	26. 5.50	(19.6 (19.4	(12.4 (12.6	19.5	12.5	5.27	3.4
"	15. 6.50	(23.8 (24.2	(15.8 (16.2	24.0	16.0	6.5	4.3
"	17. 8.50	(25.0 (25.0	(16.8 (17.0	25.0	16.9	6.75	4.55
17	3. 6.50	(21.4 (21.6	(14.4 (14.2	21.5	14.3	5.8	3.85
"	13. 6.50	(23.6 (23.4	(15.0 (15.0	23.5	15	6.35	4.05
"	13. 7.50	(24.8 (25.0	(16.0 (16.2	24.9	16.1	6.7	4.33
18	20. 7.50	(17.6 (17.6	(11.5 (11.5	17.6	11.5	4.75	3.1
"	18. 8.50	(23.4 (23.4	(14.8 (14.8	23.4	14.8	6.3	4.0
"	25.10.50	(26.8 (27.0	(17.8 (17.6	26.9	17.7	7.25	4.75

TABLE 5 (Continued)

E.E.L. READING.

Case No.	Date.	Duplicate. T.Pr. Gm.%	Duplicate. Alb. Gm.%	Average. T.Pr. Gm.%	Average. Alb. Gm.%	T.Pr. Gm.%	Alb. Gm.%
19	26. 7.50	(29.2 (29.5	(16.6 (17.0	29.35	16.8	7.9	4.5
"	27. 7.50	(27.5 (27.5	(15.6 (15.6	27.5	15.6	7.4	4.2
"	28. 7.50	(27.0 (27.2	(15.0 (15.0	27.1	15.0	7.3	4.05
"	30. 7.50	(28.0 (28.2	(15.0 (15.2	28.1	15.1	7.56	4.07
"	3. 8.50	(32.0 (32.0	(17.2 (17.2	32.0	17.2	8.6	4.6
"	10. 8.50	(29.8 (30.2	(16.2 (16.4	30.0	16.3	8.1	4.4
"	11. 8.50 (Upright	(33.2 (33.4	(17.2 (17.2	33.3	17.2	8.95	4.65
20	21. 8.50	(29.0 (29.0	(15.8 (15.8	29.0	15.8	7.8	4.25
"	2.10.50	(28.4 (28.2	(16.0 (16.0	28.3	16.0	7.65	4.3
21	22. 8.50	(26.4 (26.2	(15.8 (15.8	26.3	15.8	7.1	4.27
"	16.10.50	(22.4 (22.2	(12.8 (12.8	22.3	12.8	6.02	3.5
22	29. 8.50	(20.8 (20.8	(10.8 (11.0	20.8	10.9	5.6	2.95
"	14.10.50	(19.2 (19.4	(9.2 (9.4	19.3	9.3	5.2	2.52

TABLE 5 (Continued)

E.E.L. READING.

Case No.	Date.	Duplicate. T.Pr. Gm.%	Duplicate. Alb. Gm.%	Average. T.Pr. Gm.%	Average. Alb. Gm.%	T.Pr. Gm.%	Alb. Gm.%
23	31. 8.50	(29.2 (29.2	(19.6 (19.6	29.2	19.6	7.85	5.3
"	1. 9.50	(28.4 (28.2	(18.8 (18.8	28.3	18.8	7.62	5.1
"	2. 9.50	(30.0 (30.2	(19.8 (19.8	30.1	19.8	8.1	5.35
"	14. 9.50	(26.4 (26.8	(17.6 (17.6	26.6	17.6	7.18	4.72
24	3. 9.50 8.0.a.m.	(26.2 (26.4	(16.2 (16.4	26.3	16.3	7.05	
"	3. 9.50 9.0.a.m.	(29.4 (29.2	(18.8 (18.8	29.3	18.8	7.9	5.07
25	11. 9.50 8.0.a.m.	(27.2 (27.2	(13.4 (13.4	27.2	13.4	7.34	3.62
"	11. 9.50 9.0.a.m.	(29.4 (29.6	(15.0 (15.0	29.5	15.0	7.95	4.05
26	16. 9.50 8.0.a.m.	(22.0 (22.0	(14.0 (14.0	22.0	14.0	5.95	3.8
"	16. 9.50 9.0.a.m.	(23.4 (23.4	(14.6 (14.8	23.4	14.7	6.3	3.95
"	10.10.50 8.0.a.m.	(28.6 (28.6	(13.4 (13.2	28.6	13.3	7.7	3.6

TABLE 5 (Continued)

E.E.L. READING.

Case No.	Date.	Duplicate. T.Pr. Gm.%	Duplicate. Alb. Gm.%	Average. T.Pr. Gm.%	Average. Alb. Gm.%	T.Pr. Gm.%	Alb. Gm.%
27	21. 9.50 8.0.a.m.	(29.2 (29.2	(18.6 (18.8	29.2	18.7	7.85	5.02
"	21. 9.50 9.0.a.m.	(31.2 (31.4	(20.1 (20.0	31.3	20.05	8.45	5.4
28	1.10.50	(24.2 (24.2	(15.6 (15.6	24.2	15.6	6.52	4.2
"	6.11.50	(24.6 (24.6	(16.2 (16.2	24.6	16.2	6.62	4.35
29	11.10.50 8.0.a.m.	(26.8 (26.6	(16.6 (16.8	26.7	16.7	7.2	4.5
"	11.10.50 9.0.a.m.	(27.8 (27.6	(17.6 (17.6	27.7	17.6	7.45	4.71
30	28.10.50 9.0.a.m.	(25.0 (25.0	(16.8 (16.8	25.0	16.8	6.75	4.55
"	25.11.50 8.0.a.m.	(24.8 (25.2	(17.4 (17.2	25.0	17.3	6.75	4.65
"	25.11.50 9.0.a.m.	(26.6 (26.8	(17.2 (17.2	26.7	17.2	7.2	4.62
31	8.12.50	(19.0 (19.0	(14.5 (14.5	19.0	14.5	5.1	3.9
"	2. 1.51	(24.4 (24.8	(15.6 (15.8	24.6	15.7	6.65	4.25

TABLE 5 (Continued)

E.E.L. READING.

Case No.	Date.	Duplicate. T.Pr. Gm.%	Duplicate. Alb. Gm.%	Average. T.Pr. Gm.%	Average. Alb. Gm.%	T.Pr. Gm.%	Alb. Gm.%
32	22. 1.51	(20.4 (20.4	(12.4 (12.2	20.4	12.3	5.5	3.35
"	30. 1.51	(22.8 (23.0	(14.2 (14.0	22.9	14.1	6.2	3.8
"	13. 3.51	(23.6 (23.6	(14.0 (14.0	23.6	14.0	6.37	3.8
33	2. 2.51	(26.4 (26.6	(14.0 (14.0	26.5	14.0	7.15	3.8
"	28. 2.51	(24.2 (24.4	(13.2 (13.2	24.3	13.2	6.55	3.55
34	3. 2.51	(22.6 (22.8	(14.6 (14.8	22.7	14.7	6.15	4.0
"	12. 3.51	(22.5 (22.8	(15.0 (15.0	22.65	15.0	6.1	4.05
35	3. 2.51	(22.2 (22.2	(11.8 (12.0	22.2	11.9	6.0	3.25
"	27. 3.51	(24.6 (24.4	(14.8 (14.8	24.5	14.8	6.6	4.0
36	14. 3.51	(21.8 (21.8	(13.4 (13.4	21.8	13.4	5.9	3.6
"	10. 4.51	(23.4 (23.0	(14.4 (14.2	23.2	14.3	6.25	3.85

TABLE 5 (Continued)

E.E.L. READING.

Case No.	Date.	Duplicate. T.Pr. Gm.%	Duplicate. Alb. Gm.%	Average. T.Pr. Gm.%	Average. Alb. Gm.%	T.Pr. Gm.%	Alb. Gm.%
37	1. 4.51	(20.8 (21.0	(13.2 (13.2	20.9	13.2	5.65	3.6
"	21. 4.51	(21.0 (20.8	(13.0 (13.0	20.9	13.0	5.65	3.5
"	16. 5.51	(24.8 (24.8	(15.6 (15.6	24.8	15.6	6.7	4.2
38	4. 4.51	(27.2 (27.0	(16.8 (16.8	27.1	16.8	7.3	4.5
"	5. 4.51	(24.0 (24.0	(14.0 (14.0	24.0	14.0	6.5	3.8
"	6. 4.51	(25.0 (24.8	(15.4 (15.6	24.9	15.5	6.7	4.2
"	7. 4.51	(27.4 (27.2	(16.8 (16.8	27.3	16.8	7.35	4.5
"	28. 5.51	(25.2 (25.2	(15.5 (15.5	25.2	15.5	6.8	4.2
39	11. 4.51	(25.4 (25.4	(15.2 (15.4	25.4	15.3	6.85	4.15
"	12. 4.51	(22.0 (22.0	(14.0 (14.0	22.0	14.0	5.95	3.8
"	13. 4.51	(22.0 (22.0	(13.8 (13.6	22.0	13.7	5.95	3.7
"	14. 4.51	(23.8 (23.4	(14.0 (14.0	23.6	14.0	6.35	3.8
"	16. 4.51	(23.2 (23.2	(14.4 (14.4	23.2	14.4	6.25	3.9
"	23. 4.51	(23.6 (23.8	(15.4 (15.5	23.7	15.45	6.4	4.2

TABLE 5 (Continued)

E.E.L. READINGS.

Case No.	Date.	Duplicate. T.Pr. Gm.%	Duplicate. Alb. Gm.%	Average. T.Pr. Gm.%	Average. Alb. Gm.%	T.Pr. Gm.%	Alb. Gm.%
40	13. 4.51	(22.4 (22.6	(14.0 (14.0	22.5	14.0	6.1	3.8
"	27. 4.51	(24.2 (24.0	(15.0 (15.0	24.1	15.0	6.5	4.05
41	20. 4.51	(18.6 (18.4	(12.4 (12.4	18.5	12.4	5.0	3.35
"	22. 4.51	(19.4 (19.4	(13.4 (13.2	19.4	13.3	5.25	3.6
"	5. 6.51	(27.0 (27.2	(17.0 (17.0	27.1	17.0	7.3	4.6
42	28. 4.51	(24.8 (25.2	(15.6 (15.8	24.6	15.7	6.75	4.25
"	17. 5.51	(27.0 (27.0	(16.5 (16.5	27.0	16.5	7.3	4.45
43	1. 5.51	(27.0 (27.0	(18.2 (18.0	27.0	18.1	7.3	5.15
"	22. 5.51	(25.0 (25.0	(17.5 (17.5	25.0	17.5	6.75	4.7
44	2. 5.51	(21.6 (21.6	(14.8 (14.8	21.6	14.8	5.8	4.0
"	18. 5.51	(23.0 (22.8	(14.4 (14.6	22.9	14.5	6.2	3.9

TABLE 5 (Continued)

E.E.L. READING.

Case No.	Date.	Duplicate. T.Pr. Gm.%	Duplicate. Alb. Gm.%	Average. T.Pr. Gm.%	Average. Alb. Gm.%	T.Pr. Gm.%	Alb. Gm.%
45	2. 5.51	(23.0 (23.0	(16.6 (16.6	23.0	16.6	6.2	4.45
"	18. 5.51	(27.5 (27.8	(18.2 (18.2	27.65	18.2	7.45	4.9
46	5. 5.51	(20.6 (20.8	(12.4 (12.4	20.7	12.4	5.6	3.35
47	8. 5.51	(22.0 (22.4	(15.0 (15.0	22.2	15.0	6.0	4.05
"	4. 6.51	(26.0 (26.0	(14.8 (15.0	26.0	14.9	7.0	4.0
48	10. 5.51	(22.4 (22.4	(15.0 (15.0	22.4	15.0	6.05	4.05
"	21. 5.51	(22.8 (23.0	(15.4 (15.4	22.9	15.4	6.2	4.15
49	25. 5.51	(29.0 (29.0	(17.8 (17.6	29.0	17.7	7.95	4.8
"	26. 5.51	(26.4 (26.6	(15.5 (15.5	26.5	15.5	7.15	4.2
"	12. 6.51	(26.8 (26.8	(16.0 (16.0	26.8	16.0	7.25	4.3
50	31. 5.51	(18.2 (18.4	(12.0 (12.0	18.3	12.0	4.95	3.25
	7. 6.51	(21.6 (21.6	(13.0 (13.0	21.6	13.0	5.8	3.5

TABLE 5 (Continued)

E.E.L. READINGS.

Case No.	Date.	Duplicate. T.Pr. Gm.%	Duplicate. Alb. Gm.%	Average. T.Pr. Gm.%	Average. Alb. Gm.%	T.Pr. Gm.%	Alb. Gm.%
51	1. 6.51	(23.0 (23.0	(16.0 (16.0	23.0	16.0	6.2	4.3
"	11. 6.51	(23.4 (23.6	(16.2 (16.0	23.5	16.1	6.35	4.3
52	13. 6.51	(24.0 (24.0	(15.4 (15.2	24.0	15.3	6.5	4.15

TABLE 6

Case No.	Date.	Observed Specific Conductance 25°	Calculated Serum Total Base m.Eq./l.
18	20. 7.50	13.5	
"	18. 8.50	12.8	155.0
"	25.10.50	12.0	148.0
19	10. 8.50	11.6	145.0
"	11. 8.50	11.3	142.5
20	21. 8.50	11.9	147.5
"	2.10.50	12.48	
21	22. 8.50	12.0	147.5
"	16.10.50	12.3	149.0
22	29. 8.50	12.05	145.5
"	14.10.50	12.42	148.5
23	31. 9.50	11.9	147.5
"	1.10.50	12.0	148.2
"	2. 9.50	11.8	147.0
"	14. 9.50	12.18	149.5
24	3. 9.50	12.1	148.5
"	3. 9.50	11.9	147.5
25	11. 9.50	12.14	149.5
"	11. 9.50	11.9	147.8
26	16. 9.50	12.56	152.0
"	16. 9.50	12.44	151.0
"	10.10.50	11.75	
27	21. 9.50	12.05	149.5
"	21. 9.50	12.0	149.0
28	1.10.50	12.25	148.2
"	6.11.50	12.1	148.0
29	11.10.50	12.1	148.7
"	11.10.50	11.9	147.0

TABLE 6 (Continued)

Case No.	Date.	Observed Specific Conductance 25°	Calculated Serum Total Base m.Eq./l.
30	28.10.50	11.9	
"	25.11.50	11.92	146.0
"	25.11.50	11.0	137.0
31	8.12.50	12.3	147.0
32	22. 1.51	12.65	151.3
"	30. 1.51	12.35	149.8
"	13. 3.51	12.5	151.7
33	2. 2.51	12.4	151.7
"	28. 2.51	12.5	152.0
34	3. 2.51	12.9	148.0
"	12. 3.51	12.28	148.9
35	3. 2.51	12.12	147.0
"	27. 3.51	12.24	149.4
36	14. 3.51	12.4	149.7
"	10. 4.51	12.5	151.4
37	1. 4.51	12.5	150.3
"	21. 4.51	12.5	150.3
"	16. 5.51	12.25	149.5
38	4. 4.51	11.85	146.3
"	5. 4.51	12.3	149.8
"	6. 4.51	12.2	149.1
"	7. 4.51	11.99	148.0
"	28. 5.51	12.12	148.3
39	11. 4.51	11.85	145.7
"	12. 4.51	12.4	149.8
"	13. 4.51	12.65	152.3
"	14. 4.51	12.3	149.5
"	16. 4.51	12.25	148.7
"	23. 4.51	12.1	145.5
40	13. 4.51	12.54	151.5
"	27. 4.51	12.64	153.2

TABLE 6 (Continued)

Case No.	Date.	Observed Specific Conductance 25°	Calculated Serum Total Base. m.Eq./l.
41	20. 4.51	12.5	148.7
"	22. 4.51	12.7	151.2
42	28. 4.51	12.2	149.2
"	17. 5.51	12.0	147.9
43	1. 5.51	11.9	146.8
"	22. 5.51	12.05	147.7
44	2. 5.51	12.45	150.0
"	18. 5.51	12.4	150.3
45	2. 5.51	12.5	151.4
"	18. 5.51	12.15	149.5
46	5. 5.51	12.75	152.7
47	8. 5.51	12.5	151.0
48	10. 5.51	12.5	151.1
"	21. 5.51	12.5	151.4
49	25. 5.51	11.65	145.2
"	26. 5.51	11.65	144.1
51	1. 6.51	12.2	148.3

TABLE 6 (Continued)

Control No.	Observed Specific Conductance 25°	Calculated Serum Total Base in Eq./l.
1	11.95	146.6
2	11.9	145.3
3	12.4	150.3
4	12.15	148.3
5	12.28	149.3
6	11.9	145.8
7	11.65	144.2
8	12.5	152.2
9	12.1	148.1
10	11.9	145.3

TABLE 7

Male patients. Oedema present. Before treatment.

Case No.	T.Pr. Gm.%	Alb. Gm.%	Glob. Gm.%	P.C.V.	Hb. Gm.%	R.B.C. millions per cu.mm.	M.C.V. cubic microns.	M.C.H.C. %
1	5.75	3.4	2.35	40	13.0	3.92	102	33
3	6.5	3.6	2.9	31	9.6	3.07	101	31
7	6.3	4.2	2.1	43	14.3	4.08	105	33
10	7.85	4.65	3.2	43	13.0	4.92	87	30
11	7.02	4.15	2.87	44	12.6	4.92	89	29
14	6.35	3.95	2.4	39	12.3	3.92	99	32
16	5.27	3.4	1.87	38	11.7	3.68	103	31
17	5.8	3.85	1.95	42	12.6	4.79	88	30
18	4.75	3.1	1.65	42	12.49	3.88	108	30
19	7.4	4.2	3.2	40	13.0	4.54	88	32
26	5.95	3.8	2.15	48	14.05	5.28	91	29
31	5.1	3.9	1.2	20	5.6	2.07	97	28
32	5.5	3.35	2.15	31	9.2	2.86	108	30
34	6.15	4.0	2.15	43	12.6	3.89	110	29
41	5.0	3.35	1.65	40	12.2	4.29	93	30.5
44	5.8	4.0	1.8	45.5	13.2	4.96	92	29
50	4.95	3.25	1.7	39	11.2	3.79	103	29
Total 17	101.44	64.15	37.29	668	202.6	69.86	1,664	515
Average	5.97	3.77	2.19	39	11.9	4.109	98	30

TABLE 7 (Continued)

Male patients. Oedema diminished or absent, after treatment. Skin healed.

Case No.	T.Pr. Gm.%	Alb. Gm.%	Glob. Gm.%	P.C.V.	Hb. Gm.%	R.B.C. millions per cu.mm.	M.C.V. cubic microns.	M.C.H.C. %
1	7.55	4.4	3.15	43	13.6	4.38	98	32
3	8.9	5.7	3.2	50	16.0	5.71	88	32
7	7.65	5.0	2.65	45	13.4	4.56	99	30
10	7.05	4.15	2.9	43	13.2	4.45	97	31
11	9.2	5.43	3.77	44	13.0	5.22	84	30
14	6.5	4.15	2.35	43	13.4	5.05	85	31
16	6.75	4.55	2.2	43	12.9	4.22	102	30
17	6.7	4.33	2.37	46	14.15	5.02	92	31
18	7.25	4.75	2.5	46	14.0	4.55	101	30
19	8.1	4.4	3.7	43	14.35	4.8	90	33
26	7.7	3.6	4.1	43	13.4	4.38	98	31
31	6.65	4.25	2.4	41	12.2	4.014	102	30
32	6.37	3.8	2.57	45	13.6	4.32	104	30
34	6.1	4.05	2.05	48	14.15	4.98	96	29
41	7.3	4.6	2.7	46	13.8	4.92	93	30
44	6.2	3.9	2.3	46	13.4	4.56	101	29
50	5.8	3.5	2.3	41	12.2	4.44	92	30
Total 17	121.77	74.56	47.21	756	231.1	79.54	1,622	519
Average	7.16	4.39	2.78	44	13.6	4.68	95	31

TABLE 7 (Continued)

Male patients. No oedema. On admission.

Case No.	T.Pr. Gm.%	Alb. Gm.%	Glob. Gm.%	Hb. Gm.%	R.B.C. mill-:ions per cu.mm.	P.C.V. ml.%	M.C.V. cubic mic-:rons	M.C.H.C. %
2	6.15	3.55	2.6	11.0	3.1	30	97	37
8	6.85	4.1	2.75	11.0	5.06	38	75	29
9	7.45	4.86	2.59	14.2	4.37	47	108	30
20	7.8	4.25	3.55	13.4	4.7	44	90	30
21	7.1	4.27	2.83	14.35	5.06	48	95	30
24	7.05	4.4	2.65	13.9	5.07	46	91	30
27	7.85	5.02	2.83	14.55	5.29	47	89	31
36	5.9	3.6	2.3	13.2	4.49	45	100	29
37	5.65	3.6	2.05	7.4	2.23	24	108	31
40	6.1	3,8	2.3	12.6	4.21	43	102	29
45	6.2	4.45	1.75	13.6	4.96	44.5	90	31
47	6.0	4.05	1.95	13.6	5.02	46.5	93	29
48	6.05	4.05	2.0	12.8	4.06	42	103	30
49	7.15	4.2	2.95	13.55	4.33	44.5	103	30
51	6.2	4.3	1.9	10.7	3.59	34	95	31
Total 15	99.5	62.5	37.0	189.8	65.54	623.5	1,439	457
Average	6.63	4.17	2.47	12.7	4.37	42	96	30

TABLE 7 (Continued)

Male patients. No oedema. Skin healed.

Case No.	T.Pr. Gm.%	Alb. Gm.%	Glob. Gm.%	Hb. Gm.%	R.B.C. millions per cu.mm.	P.C.V. ml.%	M.C.V. cubic microns	M.C.H.C. %
2	7.05	4.58	2.47	11.9	4.09	35	86	34
8	6.6	4.0	2.6	11.0	4.27	38	89	29
9	Not done.							
20	7.65	4.3	3.35	12.4	4.45	40	90	31
21	6.02	3.5	2.52	13.6	4.91	44	90	31
24	Not done.							
27	Not done.							
36	6.25	3.85	2.4	13.6	4.68	48	103	28
37	6.7	4.2	2.5	12.1	3.79	40	106	30
40	6.5	4.05	2.45	12.6	3.96	42	106	30
45	7.45	4.9	2.55	14.3	5.25	48.5	92	29
47	7.0	4.0	3.0	12.8	4.49	44	98	29
48	6.2	4.15	2.05	13.6	4.48	44	98	31
49	7.25	4.3	2.95	13.6	4.59	43.5	95	31
51	6.35	4.3	2.05	11.85	3.76	38	101	31
Total 12	81.02	50.13	30.89	153.3	52.72	505.	1,154	364
Average	6.75	4.18	2.57	12.8	4.393	42	96	30

TABLE 7 (Continued)

Female patients. Oedema present. Before treatment.

Case No.	T.Pr. Gm.%	Alb. Gm.%	Glob. Gm.%	P.C.V.	Hb. Gm.%	R.B.C. mill-:ions per cu.mm.	M.C.V. cubic mic-:rons.	M.C.H.C. %
5	6.0	3.95	2.05	44	13.7	3.72	118	31
6	7.15	4.65	2.5	40	13.5	4.36	92	34
13	6.6	4.18	2.42	42	12.8	4.3	98	30
15	6.9	4.75	2.15	43	13.6	4.3	100	32
23	7.62	5.1	2.52	44	13.7	4.4	100	31
28	6.52	4.2	2.32	40	12.3	4.0	100	31
30	6.75	4.55	2.2	46	13.8	5.48	84	30
33	7.15	3.8	3.35	34	10.3	3.19	107	30
35	6.0	3.25	2.75	36	10.85	3.41	106	30
38	6.5	3.8	2.7	41	12.0	4.77	86	29
Total 10	67.19	42.23	24.96	410	126.5	41.9	991	308
Average.	6.72	4.22	2.5	41	12.65	4.19	99	31

Female patients. Oedema diminished or absent after treatment. Skin healed.

Case No.	T.Pr. Gm.%	Alb. Gm.%	Glob. Gm.%	P.C.V.	Hb. Gm.%	R.B.C. mill-:ions per cu.mm.	M.C.V. cubic mic-:rons.	M.C.H.C. %
5	6.35	4.02	2.33	43	13.9	4.09	105	32
6	7.22	4.72	2.5	40	12.2	4.46	90	31
13	6.53	4.25	2.28	40	12.3	4.46	90	31
15	6.55	4.6	1.95	42	13.0	4.6	91	31
23	7.18	4.72	2.46	41	13.2	3.87	106	32
28	6.62	4.35	2.27	39.5	12.2	3.96	100	31
30	6.75	4.65	2.1	47	13.6	4.63	102	29
33	6.55	3.55	3.0	33	10.1	2.72	110	31
35	6.6	4.0	2.6	36	10.9	3.69	98	30
38	6.8	4.2	2.6	42.5	12.5	5.33	80	29
Total 10	67.15	43.06	24.09	404	123.9	41.81	972	307
Average	6.715	4.31	2.41	40	12.4	4.181	97	31

TABLE 7 (Continued)

Female Patients. No oedema. On admission.

Case No.	T.Pr. Gm.%	Alb. Gm.%	Glob. Gm.%	Hb. Gm.%	R.B.C. mill-:ions per cu.mm.	P.C.V. ml.%	M.C.V. cubic mic-:rons.	M.C.H.C. %
4	6.4	3.95	2.45	15.8	5.35	49	92	32
22	5.6	2.95	2.65	10.35	3.61	35	97	30
29	7.2	4.5	2.7	11.4	4.05	36.5	90	31
39	5.95	3.8	2.15	13.6	4.8	45	94	30
42	6.75	4.25	2.5	11.75	4.46	39	87	30
43	7.3	5.15	2.15	13.6	4.75	46	97	30
52	6.5	4.15	2.35	11.85	3.91	41.5	106	29
Total 7	45.7	28.75	16.95	88.35	30.93	292	663	212
Average	6.53	4.12	2.42	12.6	4.418	42	95	30

Female Patients. No oedema. Skin healed.

Case No.	T.Pr. Gm.%	Alb. Gm.%	Glob. Gm.%	Hb. Gm.%	R.B.C. mill-:ions per cu.mm.	P.C.V. ml.%	M.C.V. cubic mic-:rons.	M.C.H.C. %
4	6.15	4.4	1.75	15.1	4.53	44	97	34
22	5.2	2.52	2.68	9.1	2.97	29	98	31
29	Not done.							
39	6.4	4.2	2.2	13.2	4.89	45	92	29
42	7.3	4.45	2.85	12.1	4.32	41	95	30
43	6.75	4.7	2.05	12.1	3.94	40	101.5	30
52	Not done.							
Total 5	31.8	20.27	11.53	61.6	20.65	199	483.5	154
Average	6.36	4.05	2.31	12.3	4.13	40	97	31

TABLE 8

ERYTHROCYTE SEDIMENTATION RATES CORRECTED ACCORDING TO THE METHOD OF HYNES AND WHITBY (1938)

Case No.	Corrected E.S.R. mm. in 1st hour	Case No.	Corrected E.S.R. mm. in 1st hour.
1	22	28	31
2	10	29	15
5	31	30	3
6	31	31	15
7	22	32	10
8	31	33	31
9	31	34	9
10	31	35	22
11	22	36	8
12	22	37	16
13	15	38	10
14	22	39	10
15	15	40	31
16	5	41	15
17	22	42	15
18	10	43	25
19	31	44	10
20	31	45	15
21	31	46	10
22	31	47	15
23	21	48	10
24	21	49	22
25	31	50	22
26	2	51	15
27	2	52	30

TABLE 9

T.O.P. = Total osmotic pressure.

From formula $P = C(21.4 + 5.9A)$ after Wells.

where:-

C = total protein concentration gm./100c.c.

P = total osmotic pressure in mm.H_2O.

A = Albumin concentration gm./100c.c.

Case No.	Date.	T.O.P. mm.H_2O	Oedema
1	1. 2.50	240	+ +
"	18. 2.50	255	+ +
"	8. 3.50	330	+
"	7. 4.50	285	+
"	27. 4.50	280	+
"	2. 5.50	280	+
"	17. 5.50	380	+
"	8. 6.50	360	+
2	16. 3.50	260	NIL.
"	3. 5.50	380	NIL.
"	24. 5.50	340	NIL.
3	9. 2.50	280	+
"	4. 3.50	250	NIL.
"	3. 4.50	490	NIL.
4	13. 3.50	290	NIL.
"	30. 3.50	290	NIL.
5	23. 3.50	270	+
"	18. 4.50	290	+
6	5. 4.50	350	+
"	25. 4.50	360	NIL.
7	13. 4.50	290	+
"	4. 5.50	390	NIL.
"	5. 6.50	390	NIL.

TABLE 9 (Continued)

Case No.	Date.	T.O.P mm.H_2O	Oedema
8	7. 4.50	310	NIL.
"	28. 4.50	300	NIL.
9	18. 4.50	385	NIL.
10	22. 4.50	385	+
"	6. 5.50	330	+
"	6. 5.50	330	+
"	29. 5.50	325	NIL.
11	24. 4.50	325	+
"	11. 6.50	490	NIL.
12	18. 5.50	390	+
13	15. 5.50	300	+
"	7. 6.50	305	NIL.
14	20. 5.50	285	+ +
"	12. 6.50	310	+
"	16. 7.50	300	NIL.
15	22. 5.50	340	+
"	9. 6.50	320	NIL.
16	26. 5.50	220	+
"	15. 6.50	305	NIL.
"	17. 8.50	330	NIL.
17	3. 6.50	255	+ +
"	13. 6.50	290	+
"	13. 7.50	320	+
18	20. 7.50	190	+ +
"	18. 8.50	285	NIL.
"	25.10.50	360	NIL.
19	26. 7.50	380	+
"	27. 7.50	345	+
"	28. 7.50	330	+
"	30. 7.50	345	NIL.
"	3. 8.50	420	NIL.
"	10. 8.50	390	NIL.
"	11. 8.50	440	NIL.

TABLE 9 (Continued)

Case No.	Date.	T.O.P. mm.H_2O	Oedema
20	21. 8.50	360	NIL.
"	2.10.50	360	NIL.
21	22. 8.50	330	NIL.
"	16.10.50	255	NIL.
22	29. 8.50	220	NIL.
"	14.10.50	190	NIL.
23	31. 8.50	415	+
"	1. 9.50	390	+
"	2. 9.50	430	+
"	14. 9.50	350	NIL.
24	3. 9.50	335	NIL.
"	3. 9.50	405	NIL.
25	11. 9.50	320	+
"	11. 9.50	360	+
26	16. 9.50	260	+ +
"	16. 9.50	280	+ +
"	10.10.50	330	NIL.
27	21. 9.50	400	NIL.
"	21. 9.50	450	NIL.
28	1.10.50	300	+
"	6.11.50	310	NIL.
29	11.10.50	350	NIL.
"	11.10.50	370	NIL.
30	28.10.50	325	
"	25.11.50	330	NIL.
"	25.11.50	350	NIL.
31	8.12.50	230	+ +
"	2. 1.51	300	NIL.
32	22. 1.51	225	+ +
"	30. 1.51	270	NIL.
"	13. 3.51	280	NIL.

TABLE 9 (Continued)

Case No.	Date.	T.O.P. mm.H_2O.	Oedema.
33	2. 2.51	315	+
"	28. 2.51	280	NIL.
34	3. 2.51	280	+
"	13. 3.51	280	NIL.
35	3. 2.51	245	+
"	27. 3.51	300	NIL.
36	14. 3.51	250	NIL.
"	10. 4.51	275	NIL.
37	1. 4.51	240	NIL.
"	21. 4.51	240	NIL.
"	16. 5.51	310	NIL.
38	4. 4.51	350	+++
"	5. 4.51	285	++
"	6. 4.51	310	+
"	7. 4.51	350	NIL.
"	28. 5.51	315	NIL.
39	11. 4.51	315	NIL.
"	12. 4.51	260	NIL.
"	13. 4.51	255	NIL.
"	14. 4.51	280	NIL.
"	16. 4.51	280	NIL.
"	23. 4.51	295	NIL.
40	13. 4.51	270	NIL.
"	27. 4.51	295	NIL.
41	20. 4.51	205	+
"	22. 4.51	225	NIL.
"	5. 6.51	360	NIL.
42	28. 4.51	315	NIL.
"	17. 5.51	350	NIL.

TABLE 9 (Continued)

Case No.	Date.	T.O.P. mm.H_2O	Oedema.
43	1. 5.51	380	NIL.
"	22. 5.51	330	NIL.
44	2. 5.51	260	+
"	18. 5.51	275	NIL.
45	2. 5.51	295	NIL.
"	18. 5.51	375	NIL.
46	5. 5.51	230	+
47	8. 5.51	270	NIL.
"	4. 6.51	315	NIL.
48	10. 5.51	275	NIL.
"	21. 5.51	285	NIL.
49	25. 5.51	400	NIL.
"	26. 5.51	330	NIL.
"	12. 6.51	340	NIL.
50	31. 5.51	200	++
"	7. 6.51	245	NIL.
51	1. 6.51	290	NIL.
"	11. 6.51	300	NIL.
52	13. 6.51	300	NIL.

TABLE 9 (Continued)

Control No.	T.O.P. mm.H_2O
1	320
2	290
3	285
4	310
5	300
6	290
7	325
8	305
9	320
10	280

TABLES 10 to 17

LIST OF ABBREVIATIONS.

T.Pr.	=	Total serum protein.	(gm.%)
Alb.	=	Serum albumin.	(gm.%)
Glob.	=	Serum globulin.	(gm.%)
Hb.	=	Haemoglobin.	(gm.%)
R.B.C.	=	Red blood corpuscles.	(millions per mm. mills./c.mm.)
P.C.V.	=	Packed cell volume or corpuscular volume.	(ml.)
M.C.V.	=	Mean corpuscular volume	(cubic microns.)
M.C.H.	=	Mean corpuscular haemoglobin	(micromicrograms)
M.C.H.C.	=	Mean corpuscular haemoglobin concentration.	(per cent.)

TABLE 10

Male patients with Free HCL. (Gruel meal). On admission.

Case No.	T.Pr. Gm.%	Alb. Gm.%	Glob. Gm.%	Hb. Gm.%	R.B.C. mill-:ions per cu.mm.	P.C.V. ml.%	M.C.V. cubic mic-:rons.	M.C.H.C. %
1	5.75	3.4	2.35	13.0	3.92	40	102	33
2	6.15	3.55	2.6	11.0	3.1	30	97	37
32	5.5	3.35	2.15	9.2	2.86	31	108	30
34	6.15	4.0	2.15	12.6	3.89	43	110.5	29
36	5.9	3.6	2.3	13.2	4.49	45	100	29
37	5.65	3.6	2.05	7.4	2.23	24	108	31
45	6.2	4.45	1.75	13.6	4.96	44.5	90	31
47	6.0	4.05	1.95	13.6	5.02	46.5	93	29
49	7.15	4.2	2.95	13.55	4.33	44.5	103	30
Total 9	54.45	34.2	20.25	107.15	34.8	348.5	911.5	279
Average	6.05	3.8	2.25	11.9	3.9	39	101	31

Male patients with Achlorhydria (Gruel meal). On admission.

Case No.	T.Pr. Gm.%	Alb. Gm.%	Glob. Gm.%	Hb. Gm.%	R.B.C. mill-:ions per cu.mm.	P.C.V. ml.%	M.C.V. cubic mic-:rons.	M.C.H.C. %
3	6.5	3.6	2.9	9.6	3.07	31	101	31
7	6.3	4.2	2.1	14.3	4.08	43	105	33
8	6.85	4.1	2.75	11.0	5.06	38	75	29
11	7.02	4.15	2.87	12.6	4.92	44	89	29
14	6.35	3.95	2.4	12.3	3.92	39	99	32
17	5.8	3.85	1.95	12.6	4.79	42	88	30
19	7.4	4.2	3.2	13.0	4.54	40	88	32
20	7.8	4.25	3.55	13.4	4.7	44	90	30
31	5.1	3.9	1.2	5.6	2.07	20	97	28
51	6.2	4.3	1.9	10.7	3.59	34	95	31
Total 10	65.32	40.5	24.82	115.1	407.4	375	927	305
Average	6.53	4.05	2.48	11.5	4.074	37.5	93	30

TABLE 10 (Continued)

Female patients with Free HCl. (Gruel meal). On admission.

Case No.	T.Pr. Gm.%	Alb. Gm.%	Glob. Gm.%	Hb. Gm.%	R.B.C. mill-:ions per cu.mm.	P.C.V. ml.%	M.C.V. cubic mic-:rons.	M.C.H.C. %
4	6.4	3.95	2.45	15.8	5.35	49	92	32
5	6.0	3.95	2.05	13.7	3.72	44	118	31
6	7.15	4.65	2.5	13.5	4.36	40	92	34
23	7.62	5.1	2.52	13.7	4.4	44	100	31
35	6.0	3.25	2.75	10.85	3.41	36	106	30
38	6.5	3.8	2.7	12.0	4.77	41	86	29
42	6.75	4.25	2.5	11.75	4.46	39	87	30
43	7.3	5.15	2.15	13.6	4.75	46	97	30
Total 8	53.72	34.1	19.62	104.9	35.22	339	778	247
Average	6.71	4.26	2.45	13.1	4.402	42	97	31

Female patients with Achlorhydria (Gruel meal). On admission.

Case No.	T.Pr. Gm.%	Alb. Gm.%	Gloh. Gm.%	Hb. Gm.%	R.B.C. mill-:ions per cu.mm.	P.C.V. ml.%	M.C.V. cubic mic-:rons.	M.C.H.C. %
28	6.52	4.2	2.32	12.3	4.0	40	100	31
29	7.2	4.5	2.7	11.4	4.05	36.5	90	31
30	6.75	4.55	2.2	13.8	5.48	46	84	30
33	7.15	3.8	3.35	10.3	3.19	34	107	30
Total 4	27.62	17.05	10.57	47.8	16.72	156.5	381	122
Average	6.9	4.2	2.64	11.9	4.18	39	95	30

TABLE 11

Undernourished male patients. On admission.

Case No.	T.Pr. Gm.%	Alb. Gm.%	Glob. Gm.%	Hb. Gm.%	R.B.C. millions per cu.mm.	P.C.V. ml%	M.C.V. cubic microns	M.C.H.C. %
1	5.75	3.4	2.35	13.0	3.92	40	102	33
2	6.15	3.55	2.6	11.0	3.1	30	97	37
3	6.5	3.6	2.9	9.6	3.07	31	101	31
7	6.3	4.2	2.1	14.3	4.08	43	105	33
14	6.35	3.95	2.4	12.3	3.92	39	99	32
16	5.27	3.4	1.87	11.7	3.68	38	103	31
18	4.75	3.1	1.65	12.49	3.88	42	108	30
19	7.4	4.2	3.2	13.0	4.54	40	88	32
26	5.95	3.8	2.15	14.05	5.28	48	91	29
31	5.1	3.9	1.2	5.6	2.07	20	97	28
36	5.9	3.6	2.3	13.2	4.49	45	100	29
48	6.05	4.05	2.0	12.8	4.06	42	103	30
51	6.2	4.3	1.9	10.7	3.59	34	95	31
Total 13	77.67	49.05	28.62	153.7	49.68	492	1,289	406
Average	5.97	3.77	2.2	11.8	3.821	38	99	31

Undernourished male patients. Skin healed.

Case No.	T.Pr. Gm.%	Alb. Gm.%	Glob. Gm.%	Hb. Gm.%	R.B.C. millions per cu.mm.	P.C.V. ml%	M.C.V. cubic microns.	M.C.H.C. %
1	7.55	4.4	3.15	13.6	4.38	43	98	32
2	7.05	4.58	2.47	11.9	4.08	35	86	34
3	8.9	5.7	3.2	16.0	5.71	50	88	32
7	7.65	5.0	2.65	13.4	4.56	45	99	30
14	6.5	4.15	2.35	13.4	5.05	43	85	31
16	6.75	4.55	2.2	12.9	4.22	43	102	30
18	7.25	4.75	2.5	14.0	4.55	46	101	30
19	8.1	4.4	3.7	14.35	4.8	43	90	33
26	7.7	3.6	4.1	13.4	4.38	43	98	31
31	6.65	4.25	2.4	12.2	4.014	41	102	30
36	6.25	3.85	2.4	13.6	4.68	48	103	28
48	6.2	4.15	2.05	13.6	4.48	44	98	31
51	6.35	4.3	2.05	11.85	3.76	38	101	31
Total 13	92.9	57.68	35.22	174.2	58.66	562	1,251	403
Average	7.15	4.44	2.71	13.4	4.512	43	96	31

TABLE 11 (Continued)

Well-nourished male patients. On admission.

Case No.	T.Pr. Gm.%	Alb. Gm.%	Glob. Gm.%	Hb. Gm.%	R.B.C. millions per cu.mm.	P.C.V. ml.%	M.C.V. cubic microns	M.C.H.C. %
8	6.85	4.1	2.75	11.0	5.06	38	75	29
9	7.45	4.86	2.59	14.2	4.37	47	108	30
10	7.85	4.65	3.2	13.0	4.92	43	87	30
11	7.02	4.15	2.87	12.6	4.92	44	89	29
17	5.8	3.85	1.95	12.6	4.79	42	88	30
20	7.8	4.25	3.55	13.4	4.7	44	90	30
21	7.1	4.27	2.83	14.35	5.06	48	95	30
24	7.05	4.4	2.65	13.9	5.07	46	91	30
25	7.34	3.62	3.72	11.7	4.21	37	88	32
27	7.85	5.02	2.83	14.55	5.29	47	89	31
32	5.5	3.35	2.15	9.2	2.86	31	108	30
34	6.15	4.0	2.15	12.6	3.89	43	110.5	29
37	5.65	3.6	2.05	7.4	2.23	24	108	31
40	6.1	3.8	2.3	12.6	4.21	43	102	29
41	5.0	3.35	1.65	12.2	4.29	40	93	30.5
44	5.8	4.0	1.8	13.2	4.96	45.5	92	29
45	6.2	4.45	1.75	13.6	4.96	44.5	90	31
47	6.0	4.05	1.95	13.6	5.02	46.5	93	29
49	7.15	4.2	2.95	13.55	4.33	44.5	103	30
50	4.95	3.25	1.7	11.2	3.79	39	103	29
Total 20	130.6	81.22	49.39	250.4	88.93	837	1902	598
Average	6.53	4.06	2.47	12.5	4.446	42	95	30

TABLE 11 (Continued)

Well-nourished male patients. Skin healed.

Case No.	T.Pr. Gm.%	Alb. Gm.%	Glob. Gm.%	Hb. Gm.%	R.B.C. mill-:ions per cu.mm.	P.C.V. ml.%	M.C.V. cubic mic-:rons.	M.C.H.C. %
8	6.6	4.0	2.6	11.0	4.27	38	89	29
9	Not done.							
10	7.05	4.15	2.9	13.2	4.45	43	97	31
11	9.2	5.43	3.77	13.0	5.22	44	84	30
17	6.7	4.33	2.37	14.15	5.02	46	92	31
20	7.65	4.3	3.35	12.4	4.45	40	90	31
21	6.02	3.5	2.52	13.6	4.91	44	90	31
24	Not done.							
25	Not done.							
27	Not done.							
32	6.37	3.8	2.57	13.6	4.32	45	104	30
34	6.1	4.05	2.05	14.15	4.98	48	96	29
37	6.7	4.2	2.5	12.1	3.79	40	106	30
40	6.5	4.05	2.45	12.6	3.96	42	106	30
41	7.3	4.6	2.7	13.8	4.92	46	93	30
44	6.2	3.9	2.3	13.4	4.56	46	101	29
45	7.45	4.9	2.55	14.3	5.25	48.5	92	29
47	7.0	4.0	3.0	12.8	4.49	44	98	29
49	7.25	4.3	2.95	13.6	4.59	43.5	95	31
50	5.8	3.5	2.3	12.2	4.44	41	92	30
Total 16	109.89	67.01	42.88	209.9	73.62	699	1,525	480
Average	6.87	4.19	2.68	13.1	4.601	44	95	30

TABLE 11 (Continued)

Undernourished female patients. On admission.

Case No.	T.Pr. Gm.%	Alb. Gm.%	Glob. Gm.%	Hb. Gm.%	R.B.C. mill-:ions per cu.mm.	P.C.V. ml.%	M.C.V. cubic mic-:rons.	M.C.H.C. %
4	6.4	3.95	2.45	15.8	5.35	49	92	32
5	6.0	3.95	2.05	13.7	3.72	44	118	31
13	6.6	4.18	2.42	12.8	4.3	42	98	30
22	5.6	2.95	2.65	10.35	3.61	35	97	30
33	7.15	3.8	3.35	10.3	3.19	34	107	30
35	6.0	3.25	2.75	10.85	3.41	36	106	30
39	5.95	3.8	2.15	13.6	4.8	45	94	30
42	6.75	4.25	2.5	11.75	4.46	39	87	30
Total 8	50.45	30.13	20.32	99.15	32.84	324	799	243
Average	6.31	3.77	2.54	12.4	4.105	40	100	30

Undernourished female patients. Skin healed.

Case No.	T.Pr. Gm.%	Alb. Gm.%	Glob. Gm.%	Hb. Gm.%	R.B.C. mill-:ions per cu.mm.	P.C.V. ml.%	M.C.V. cubic mic-:rons.	M.C.H.C. %
4	6.15	4.4	1.75	15.1	4.53	44	97	34
5	6.35	4.02	2.33	13.9	4.09	43	105	32
13	6.53	4.25	2.28	12.3	4.46	40	90	31
22	5.2	2.52	2.68	9.1	2.97	29	98	31
33	6.55	3.55	3.0	10.1	2.72	33	110	31
35	6.6	4.0	2.6	10.9	3.69	36	98	30
39	6.4	4.2	2.2	13.2	4.89	45	92	29
42	7.3	4.45	2.85	12.1	4.32	41	95	30
Total 8	51.08	31.39	19.69	96.7	31.67	311	785	248
Average	6.38	3.92	2.46	12.1	3.958	39	98	31

TABLE 11 (Continued)

Well-nourished female patients. On admission.

Case No.	T.Pr. Gm.%	Alb. Gm.%	Glob. Gm.%	Hb. Gm.%	R.B.C. mill-:ions per cu.mm.	P.C.V. ml.%	M.C.V. cubic mic-:rons.	M.C.H.C. %
6	7.15	4.65	2.5	13.5	4.36	40	92	34
12	7.05	4.47	2.58	13.6	4.26	44	103	31
15	6.9	4.75	2.15	13.6	4.3	43	100	32
23	7.62	5.1	2.52	13.7	4.4	44	100	31
28	6.52	4.2	2.32	12.3	4.0	40	100	31
29	7.2	4.5	2.7	11.4	4.05	36.5	90	31
30	6.75	4.55	2.2	13.8	5.48	46	84	30
38	6.5	3.8	2.7	12.0	4.77	41	86	29
43	7.3	5.15	2.15	13.6	4.75	46	97	30
46	5.6	3.35	2.25	7.2	2.31	27	117	27
52	6.5	4.15	2.35	11.85	3.91	41.5	106	29
Total 11	75.09	48.67	26.37	136.5	46.59	449	1,075	335
Average	6.83	4.42	2.4	12.4	4.235	41	98	30

Well-nourished female patients. Skin healed.

Case No.	T.Pr. Gm.%	Alb. Gm.%	Glob. Gm.%	Hb. Gm.%	R.B.C. mill-:ions per cu.mm.	P.C.V. ml.%	M.C.V. cubic mic-:rons.	M.C.H.C. %
6	7.22	4.72	2.5	12.2	4.46	40	90	31
12	Not done.							
15	6.55	4.6	1.95	13.0	4.6	42	91	31
23	7.18	4.72	2.46	13.2	3.87	41	106	32
28	6.62	4.35	2.27	12.2	3.96	39.5	100	31
29	Not done.							
30	6.75	4.65	2.1	13.6	4.63	47	102	29
38	6.8	4.2	2.6	12.5	5.33	42.5	80	29
43	6.75	4.7	2.05	12.1	3.94	40	101	30
46	Not done.							
52	Not done.							
Total 7	47.87	31.94	15.93	88.8	30.79	292	670	213
Average	6.84	4.56	2.27	12.7	4.398	42	96	30

TABLE 12

Infectious eczematoid dermatitis. On admission.

Case No.	T.Pr. Gm.%	Alb. Gm.%	Glob. Gm.%	Hb. Gm.%	R.B.C. mill-:ions per cu.mm.	P.C.V. ml.%	M.C.V. cubic mic-:rons.	M.C.H.C. %
1	5.75	3.4	2.35	13.0	3.92	40	102	33
2	6.15	3.55	2.6	11.0	3.1	30	97	37
3	6.5	3.6	2.9	9.6	3.07	31	101	31
5	6.0	3.95	2.05	13.7	3.72	44	118	31
18	4.75	3.1	1.65	12.49	3.88	42	108	30
Total 5	29.15	17.6	11.55	59.79	17.69	187	526	162
Average	5.83	3.52	2.31	12.0	3.538	37	105	32

Infectious eczematoid dermatitis. Skin healed. After Protein treatment.

Case No.	T.Pr. Gm.%	Alb. Gm.%	Glob. Gm.%	Hb. Gm.%	R.B.C. mill-:ions per cu.mm.	P.C.V. ml.%	M.C.V. cubic mic-:rons.	M.C.H.C. %
1	7.85	4.45	3.4	13.6	5.06	44	87	31
2	7.55	4.85	2.7	12.9	4.35	37	85	35
3	8.9	5.7	3.2	16.0	5.71	50	88	32
5	6.35	4.02	2.33	13.9	4.09	43	105	32
18	6.3	4.0	2.3	13.3	4.59	43	94	31
Total 5	36.95	23.02	13.93	69.7	23.8	217	459	161
Average	7.39	4.6	2.79	13.9	4.76	43	92	32

TABLE 13

Infectious eczematoid dermatitis. On admission.

Case No.	T.Pr. Gm.%	Alb. Gm.%	Glob. Gm.%	Hb. Gm.%	R.B.C. millions per cu.mm.	P.C.V. ml.%	M.C.V. cubic microns.	M.C.H.C. %
4	6.4	3.95	2.45	15.8	5.35	49	92	32
8	6.85	4.1	2.75	11.0	5.06	38	75	29
28	6.52	4.2	2.32	12.3	4.0	40	100	31
30	6.75	4.55	2.2	13.8	5.48	46	84	30
33	7.15	3.8	3.35	10.3	3.19	34	107	30
34	6.15	4.0	2.15	12.6	3.89	43	110	29
39	5.95	3.8	2.15	13.6	4.8	45	94	30
42	6.75	4.25	2.5	11.75	4.46	39	87	30
47	6.0	4.05	1.95	13.6	5.02	46.5	93	29
Total 9	58.52	36.7	21.82	114.7	41.25	380.5	842	270
Average	6.50	4.08	2.42	12.7	4.583	42	94	30

Infectious eczematoid dermatitis. Skin healed. No Protein treatment.

Case No.	T.Pr. Gm.%	Alb. Gm.%	Glob. Gm.%	Hb. Gm.%	R.B.C. millions per cu.mm.	P.C.V. ml.%	M.C.V. cubic microns.	M.C.H.C. %
4	6.15	4.4	1.75	15.1	4.53	44	97	34
8	6.6	4.0	2.6	11.0	4.27	38	89	29
28	6.62	4.35	2.27	12.2	3.96	39.5	100	31
30	6.75	4.65	2.1	13.6	4.63	47	102	29
33	6.55	3.55	3.0	10.1	2.72	33	110	31
34	6.1	4.05	2.05	14.15	4.98	48	96	29
39	6.4	4.2	2.2	13.2	4.89	45	92	29
42	7.3	4.45	2.85	12.1	4.32	41	95	30
47	7.0	4.0	3.0	12.8	4.49	44	98	29
Total 9	59.47	37.65	21.82	114.25	38.79	379.5	879	271
Average	6.61	4.18	2.42	12.7	4.31	42	98	30

TABLE 14

Male patients. Infectious eczematoid dermatitis. On admission.

Case No.	T.Pr. Gm.%	Alb. Gm.%	Glob. Gm.%	Hb. Gm.%	R.B.C. mill-:ions per cu.mm.	P.C.V. ml.%	M.C.V. cubic mic-:rons.	M.C.H.C. %
1	5.75	3.4	2.35	13.0	3.92	40	102	33
2	6.15	3.55	2.6	11.0	3.1	30	97	37
3	6.5	3.6	2.9	9.6	3.07	31	101	31
8	6.85	4.1	2.75	11.0	5.06	38	75	29
25	7.34	3.62	3.72	11.7	4.21	37	88	32
34	6.15	4.0	2.15	12.6	3.89	43	110	29
47	6.0	4.05	1.95	13.6	5.02	46.5	93	29
Total 7	44.74	26.32	18.42	82.5	28.27	265.5	666	220
Average	6.39	3.76	2.63	11.8	4.038	38	95	31
Excluding Case 25								
Total 6	37.4	22.7	14.7	70.8	24.06	228.5	578	188
Average	6.23	3.78	2.45	11.8	4.01	38	96	31

Male patients. Infectious eczematoid dermatitis. Skin healed.

Case No.	T.Pr. Gm.%	Alb. Gm.%	Glob. Gm.%	Hb. Gm.%	R.B.C. mill-:ions per cu.mm.	P.C.V. ml.%	M.C.V. cubic mic-:rons.	M.C.H.C. %
1	7.55	4.4	3.15	13.6	4.38	43	98	32
2	7.05	4.58	2.47	11.9	4.09	35	86	34
3	8.9	5.7	3.2	16.0	5.71	50	88	32
8	6.6	4.0	2.6	11.0	4.27	38	89	29
34	6.1	4.05	2.05	14.15	4.98	48	96	29
47	7.0	4.0	3.0	12.8	4.49	44	98	29
Total 6	43.2	26.73	16.47	79.45	27.92	258	555	185
Average	7.2	4.45	2.74	13.2	4.653	43	92	31

TABLE 14 (Continued)

Female patients. Infectious eczematoid dermatitis. On admission.

Case No.	T.Pr. Gm.%	Alb. Gm.%	Glob. Gm.%	Hb. Gm.%	R.B.C. millions per cu.mm.	P.C.V. ml.%	M.C.V. cubic microns.	M.C.H.C. %
4	6.4	3.95	2.45	15.8	5.35	49	92	32
5	6.0	3.95	2.05	13.7	3.72	44	118	31
28	6.52	4.2	2.32	12.3	4.0	40	100	31
30	6.75	4.55	2.2	13.8	5.48	46	84	30
33	7.15	3.8	3.35	10.3	3.19	34	107	30
39	5.95	3.8	2.15	13.6	4.8	45	94	30
42	6.75	4.25	2.5	11.75	4.46	39	87	30
Total 7	45.52	28.5	17.02	91.25	31	297	682	214
Average	6.5	4.07	2.43	13.04	4.429	42	97	31

Female patients. Infectious eczematoid dermatitis. Skin healed.

Case No.	T.Pr. Gm.%	Alb. Gm.%	Glob. Gm.%	Hb. Gm.%	R.B.C. millions per cu.mm.	P.C.V. ml.%	M.C.V. cubic microns.	M.C.H.C. %
4	6.15	4.4	1.75	15.1	4.53	44	97	34
5	6.35	4.02	2.33	13.9	4.09	43	105	32
28	6.62	4.35	2.27	12.2	3.96	39.5	100	31
30	6.75	4.65	2.1	13.6	4.63	47	102	29
33	6.55	3.55	3.0	10.1	2.72	33	110	31
39	6.4	4.2	2.2	13.2	4.89	45	92	29
42	7.3	4.45	2.85	12.1	4.32	41	95	30
Total 7	46.12	29.62	16.5	90.2	29.14	292.5	701	216
Average	6.59	4.23	2.36	12.9	4.162	42	100	31

TABLE 15

Scurvy. Before treatment.

Case No.	T.Pr. Gm.%	Alb. Gm.%	Glob. Gm.%	Hb. Gm.%	R.B.C. mill-:ions per cu.mm.	P.C.V. ml.%	M.C.V. cubic mic-:rons.	M.C.H.C. %
14	6.35	3.95	2.4	12.3	3.92	39	99	32
16	5.27	3.4	1.87	11.7	3.68	38	103	31
17	5.8	3.85	1.95	12.6	4.79	42	88	30
31	5.1	3.9	1.2	5.6	2.07	20	97	28
32	5.5	3.35	2.15	9.2	2.86	31	108	30
35	6.0	3.25	2.75	10.85	3.41	36	106	30
37	5.65	3.6	2.05	7.4	2.23	24	108	31
40	6.1	3.8	2.3	12.6	4.21	43	102	29
51	6.2	4.3	1.9	10.7	3.59	34	95	31
Total 9	51.97	33.4	18.57	92.95	30.76	307	906	272
Average	5.77	3.71	2.06	10.3	3.41	34	101	30

Scurvy. After treatment.

Case No.	T.Pr. Gm.%	Alb. Gm.%	Glob. Gm.%	Hb. Gm.%	R.B.C. mill-:ions per cu.mm.	P.C.V. ml.%	M.C.V. cubic mic-:rons.	M.C.H.C. %
14	6.5	4.15	2.35	13.4	5.05	43	85	31
16	6.75	4.55	2.2	12.9	4.22	43	102	30
17	6.7	4.33	2.37	14.15	5.02	46	92	31
31	6.65	4.25	2.4	12.2	4.014	41	102	30
32	6.37	3.8	2.57	13.6	4.32	45	104	30
35	6.6	4.0	2.6	10.9	3.69	36	98	30
37	6.7	4.2	2.5	12.1	3.79	40	106	30
40	6.5	4.05	2.45	12.6	3.96	42	106	30
51	6.35	4.3	2.05	11.85	3.76	38	101	31
Total 9	59.12	37.63	21.49	113.7	37.82	374	896	273
Average	6.57	4.18	2.39	12.6	4.203	41.5	100	30

TABLE 16

Scurvy. Before receiving Protein, Iron and Vitamin Treatment.

Case No.	T.Pr. Gm.%	Alb. Gm.%	Glob. Gm.%	Hb. Gm.%	R.B.C. mill-:ions per cu.mm.	P.C.V. ml.%	M.C.V. cubic mic-:rons.	M.C.H.C. %
14	6.35	3.95	2.4	12.3	3.92	39	99	32
16	5.27	3.4	1.87	11.7	3.68	38	103	31
31	5.1	3.9	1.2	5.6	2.07	20	97	28
37	5.65	3.5	2.15	10.3	2.93	33.5	113	31
Total 4	22.37	14.75	7.62	39.9	12.6	130.5	412	122
Average	5.59	3.69	1.9	10.0	3.15	33	103	30.5

Scurvy. After receiving Protein, Iron and Vitamin Treatment.

Case No.	T.Pr. Gm.%	Alb. Gm.%	Glob. Gm.%	Hb. Gm.%	R.B.C. mill-:ions per cu.mm.	P.C.V. ml.%	M.C.V. cubic mic-:rons.	M.C.H.C. %
14	6.75	4.1	2.65	12.6	4.28	41	96	31
16	6.5	4.3	2.2	12.7	4.39	43	98	30
31	6.65	4.25	2.4	12.2	4.014	41	102	30
37	6.7	4.2	2.5	12.1	3.79	40	106	30
Total 4	26.6	16.85	9.75	49.6	16.47	165	402	121
Average	6.65	4.21	2.44	12.4	4.118	41	100	30

TABLE 16 (Continued)

Scurvy. Before receiving Iron and Vitamin Treatment.

Case No.	T.Pr. Gm.%	Alb. Gm.%	Glob. Gm.%	Hb. Gm.%	R.B.C. mill-:ions per cu.mm.	P.C.V. ml.%	M.C.V. cubic mic-:rons.	M.C.H.C. %
32	5.5	3.35	2.15	9.2	2.86	31	108	30
35	6.0	3.25	2.75	10.85	3.41	36	106	30
37	5.65	3.6	2.05	7.4	2.23	24	108	31
40	6.1	3.8	2.3	12.6	4.21	43	102	29
51	6.2	4.3	1.9	10.7	3.59	34	95	31
Total 5	29.45	18.3	11.15	50.75	16.3	168	519	151
Average	5.89	3.66	2.23	10.15	3.26	34	104	30

Scurvy. After receiving Iron and Vitamin Treatment.

Case No.	T.Pr. Gm.%	Alb. Gm.%	Glob. Gm.%	Hb. Gm.%	R.B.C. mill-:ions per cu.mm.	P.C.V. ml.%	M.C.V. cubic mic-:rons.	M.C.H.C. %
32	6.37	3.8	2.57	13.6	4.32	45	104	30
35	6.6	4.0	2.6	10.9	3.69	36	98	30
37	5.65	3.5	2.15	10.3	2.93	33.5	113	31
40	6.5	4.05	2.45	12.6	3.96	42	106	30
51	6.35	4.3	2.05	11.85	3.76	38	101	31
Total 5	31.47	19.65	11.82	59.25	18.66	194.5	522	152
Average	6.29	3.93	2.36	11.85	3.73	39	104	30

TABLE 17

Male patients. Varicose conditions. On admission.

Case No.	T.Pr. Gm.%	Alb. Gm.%	Glob. Gm.%	Hb. Gm.%	R.B.C. millions per cu.mm.	P.C.V. ml.%	M.C.V. cubic microns.	M.C.H.C. %
7	6.3	4.2	2.1	14.3	4.08	43	105	33
9	7.45	4.86	2.59	14.2	4.37	47	108	30
11	7.02	4.15	2.87	12.6	4.92	44	89	29
19	7.4	4.2	3.2	13.0	4.54	40	88	32
20	7.8	4.25	3.55	13.4	4.7	44	90	30
24	7.05	4.4	2.65	13.9	5.07	46	91	30
27	7.85	5.02	2.83	14.55	5.29	47	89	31
36	5.9	3.6	2.3	13.2	4.49	45	100	29
45	6.2	4.45	1.75	13.6	4.96	44.5	90	31
48	6.05	4.05	2.0	12.8	4.06	42	103	30
49	7.15	4.2	2.95	13.55	4.33	44.5	103	30
Total 11	76.17	47.38	28.79	149.1	50.81	487.	105.6	335
Average	6.92	4.31	2.62	13.5	4.619	44	96	30

Male patients. Varicose conditions. Skin healed.

Case No.	T.Pr. Gm.%	Alb. Gm.%	Glob. Gm.%	Hb. Gm.%	R.B.C. millions per cu.mm.	P.C.V. ml.%	M.C.V. cubic microns.	M.C.H.C. %
7	7.65	5.0	2.65	13.4	4.56	45	99	30
9	Not done.							
11	9.2	5.43	3.77	13.0	5.22	44	84	30
19	8.1	4.4	3.7	14.35	4.8	43	90	33
20	7.65	4.3	3.35	12.4	4.45	40	90	31
24	Not done.							
27	Not done.							
36	6.25	3.85	2.4	13.6	4.68	48	103	28
45	7.45	4.9	2.55	14.3	5.25	48.5	92	29
48	6.2	4.15	2.05	13.6	4.48	44	98	31
49	7.25	4.3	2.95	13.6	4.59	43.5	95	31
Total 8	59.75	36.33	23.42	108.2	38.03	356	751	243
Average.	7.47	4.54	2.93	13.5	4.753	44.5	94	30

TABLE 17 (Continued)

Female patients. Varicose conditions. On admission.

Case No.	T.Pr. Gm.%	Alb. Gm.%	Glob. Gm.%	Hb. Gm.%	R.B.C. mill-:ions per cu.mm.	P.C.V. ml.%	M.C.V. cubic mic-:rons.	M.C.H.C. %
6	7.15	4.65	2.5	13.5	4.36	40	92	34
12	7.05	4.47	2.58	13.6	4.26	44	103	31
23	7.62	5.1	2.52	13.7	4.4	44	100	31
29	7.2	4.5	2.7	11.4	4.05	36.5	90	31
38	6.5	3.8	2.7	12.0	4.77	41	86	29
43	7.3	5.15	2.15	13.6	4.75	46	97	30
Total 6	42.82	27.67	15.15	77.8	26.59	251.5	568	186
Average	7.14	4.61	2.52	13.0	4.43	42	95	31

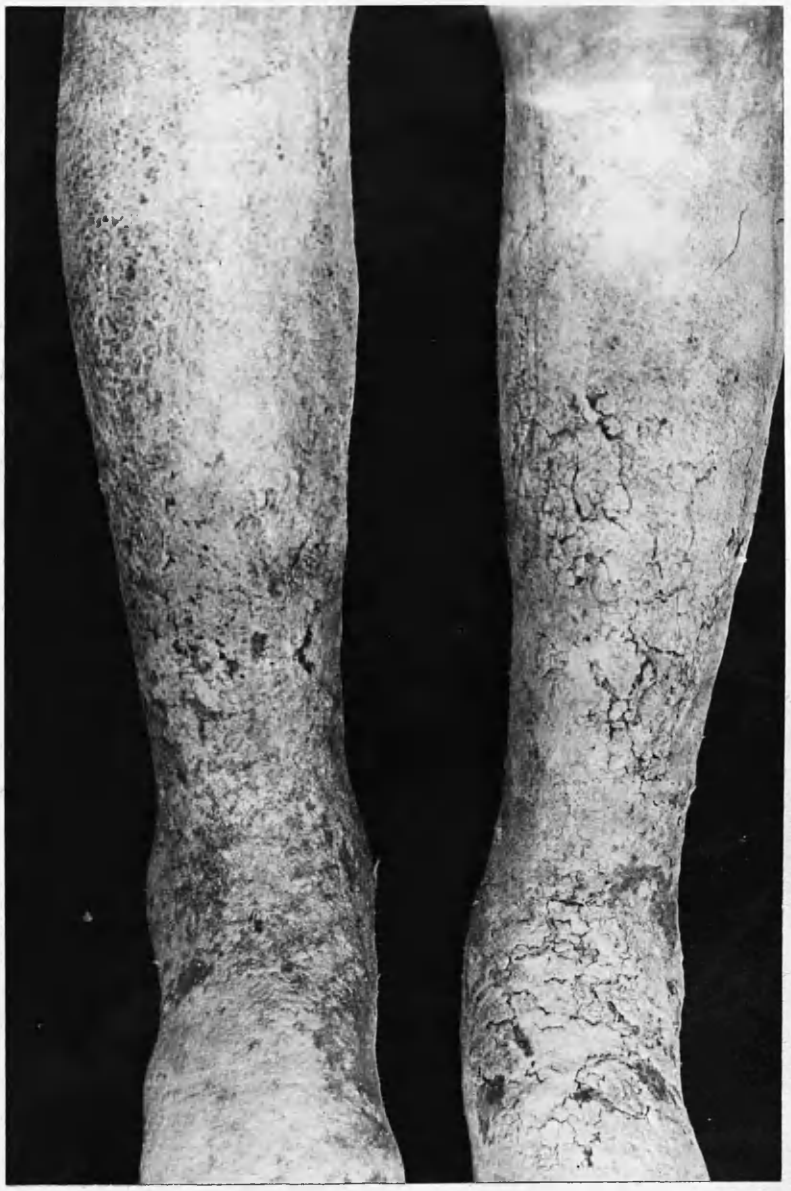
Female patients. Varicose conditions. Skin healed.

Case No.	T.Pr. Gm.%	Alb. Gm.%	Glob. Gm.%	Hb. Gm.%	R.B.C. mill-:ions per cu.mm.	P.C.V. ml.%	M.C.V. cubic mic-:rons.	M.C.H.C. %
6	7.22	4.72	2.5	12.2	4.46	40	90	31
12	Not done.							
23	7.18	4.72	2.46	13.2	3.87	41	106	32
29	Not done.							
38	6.8	4.2	2.6	12.5	5.33	42.5	80	29
43	6.75	4.7	2.05	12.1	3.94	40	101.5	30
Total 4	27.95	18.34	9.61	50	17.6	163.5	377.5	122
Average	6.99	4.58	2.4	12.5	4.4	41	94	30

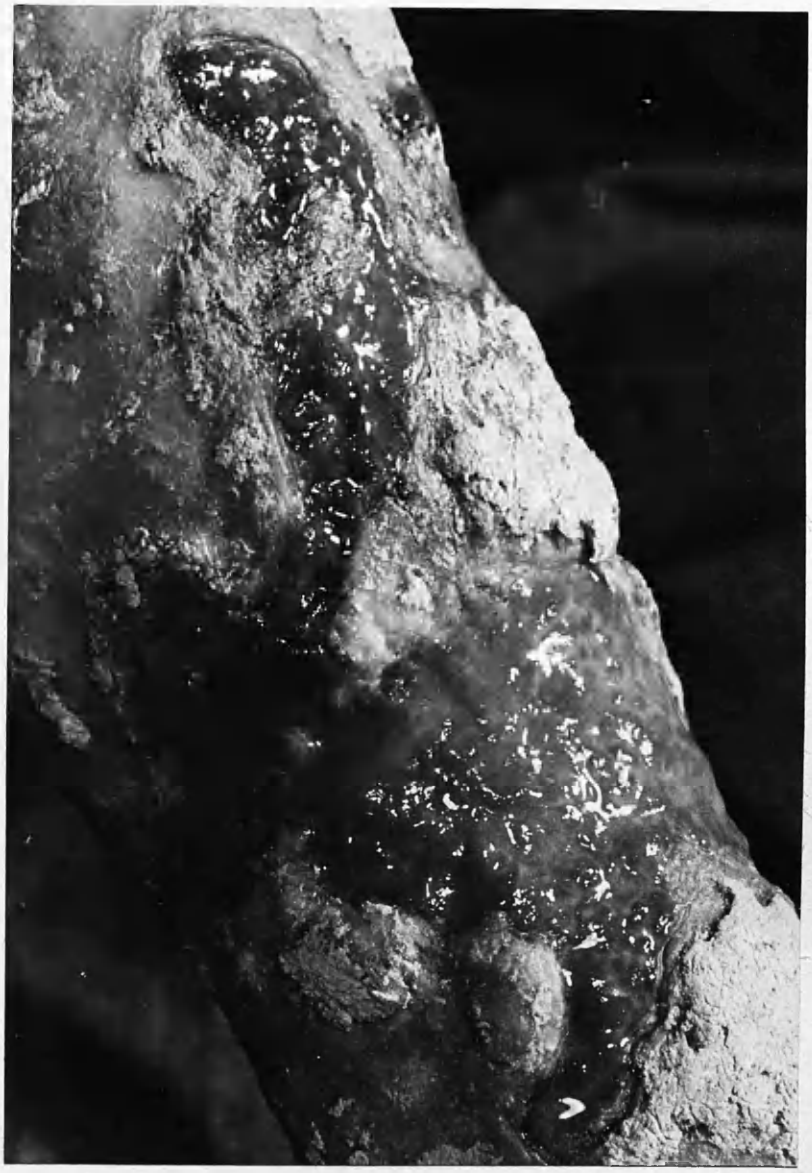
TABLE 18

Cases of Pemphigus. On admission.

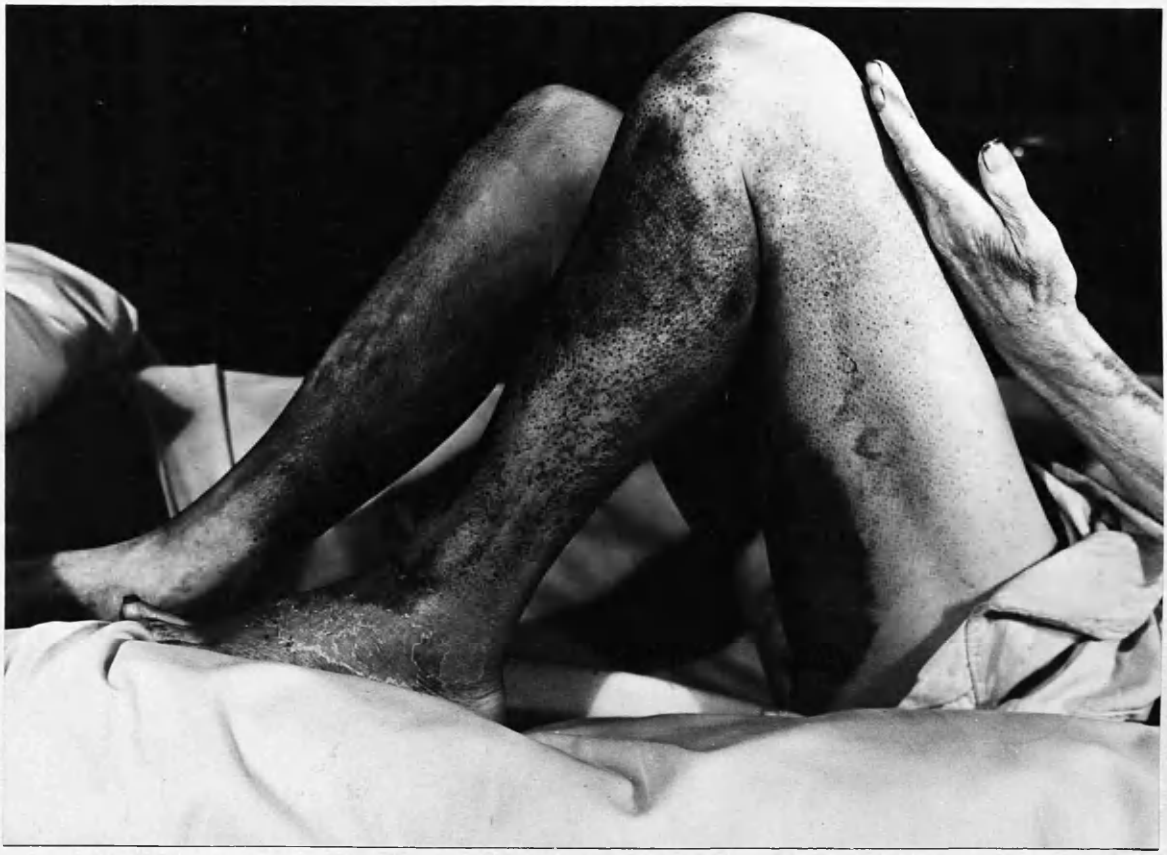
Case No.	T.Pr. Gm.%	Alb. Gm.%	Glob. Gm.%	Hb. Gm.%	R.B.C. millions per cu.mm.	P.C.V. ml.%	M.C.V. cubic microns.	M.C.H.C. %
21	7.1	4.27	2.83	14.35	5.06	48	95	30
22	5.6	2.95	2.65	10.35	3.61	35	97	30
26	5.95	3.8	2.15	14.05	5.28	48	91	29
50	4.95	3.25	1.7	11.2	3.79	39	103	29
52	6.5	4.15	2.35	11.85	3.91	41.5	106	29



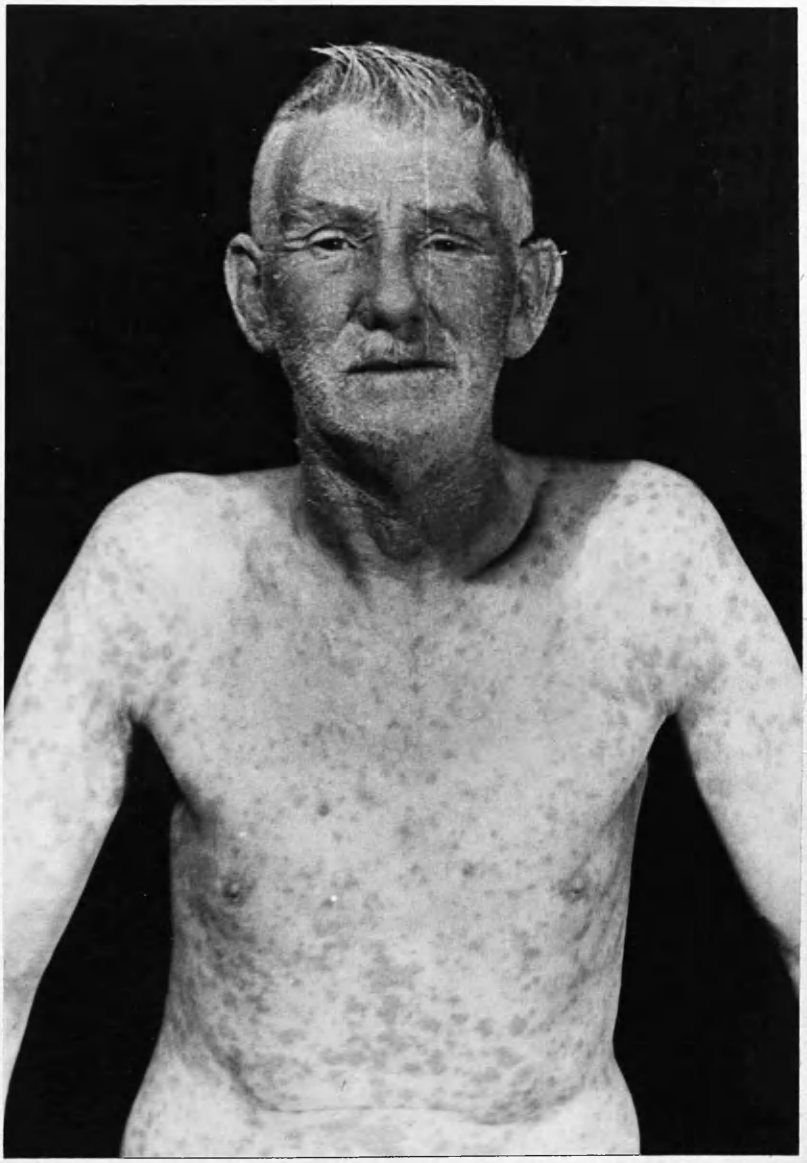
Case 1. Infectious eczematoid dermatitis. The heavy crusting is due partly to local applications employed before admission.



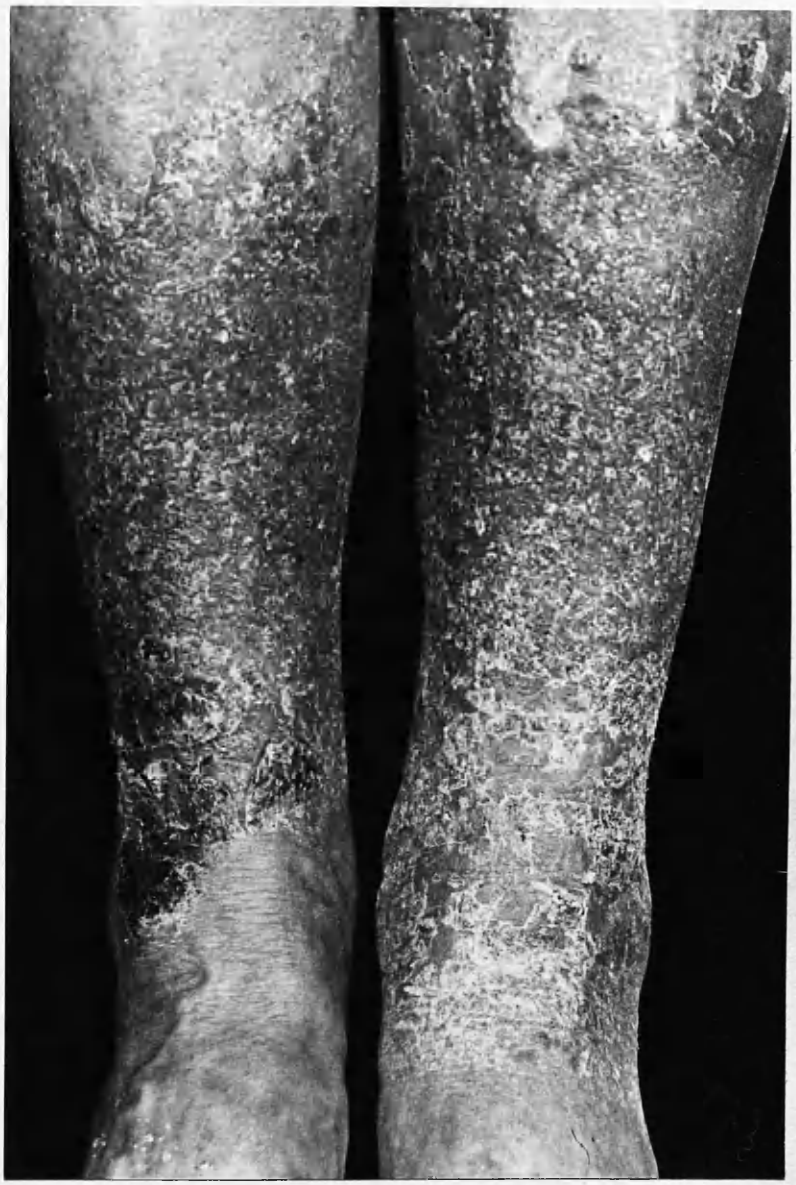
Case 24. Indolent varicose ulcer, shewing surrounding sclerotic tissue.



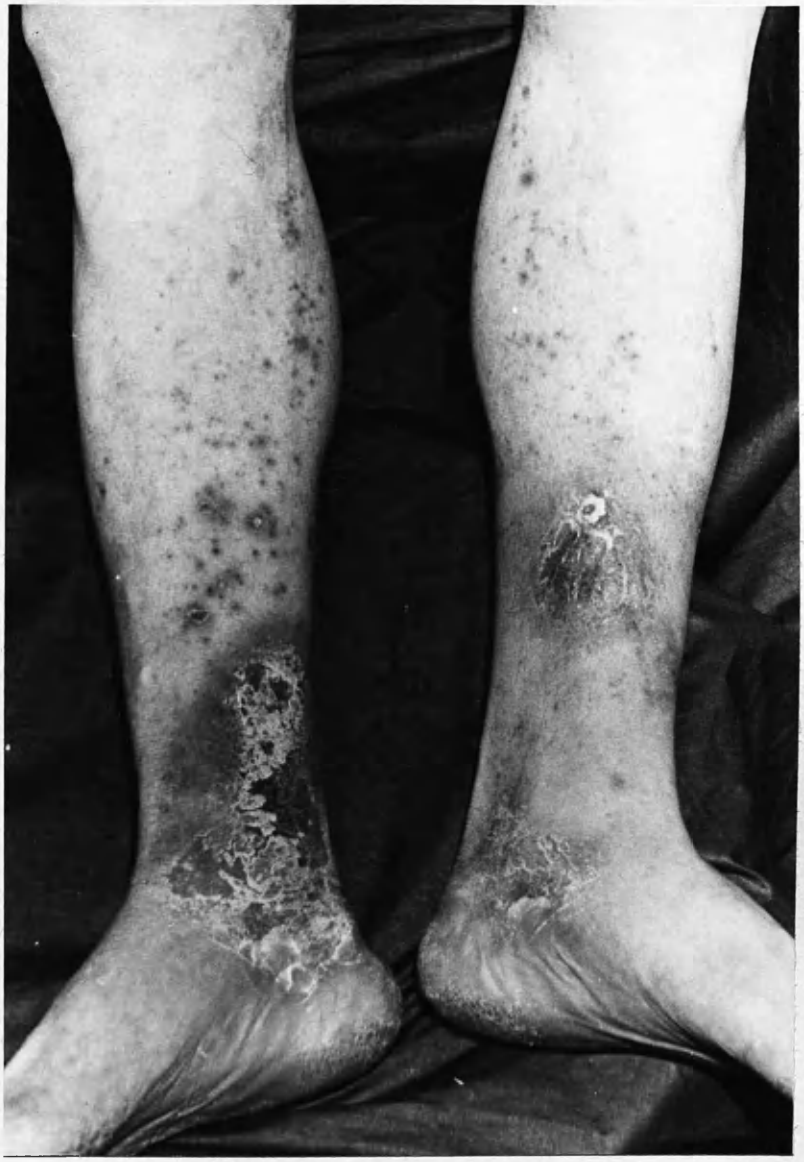
Case 31. Scurvy. Shewing follicular petechial haemorrhages and larger areas of ecchymoses.



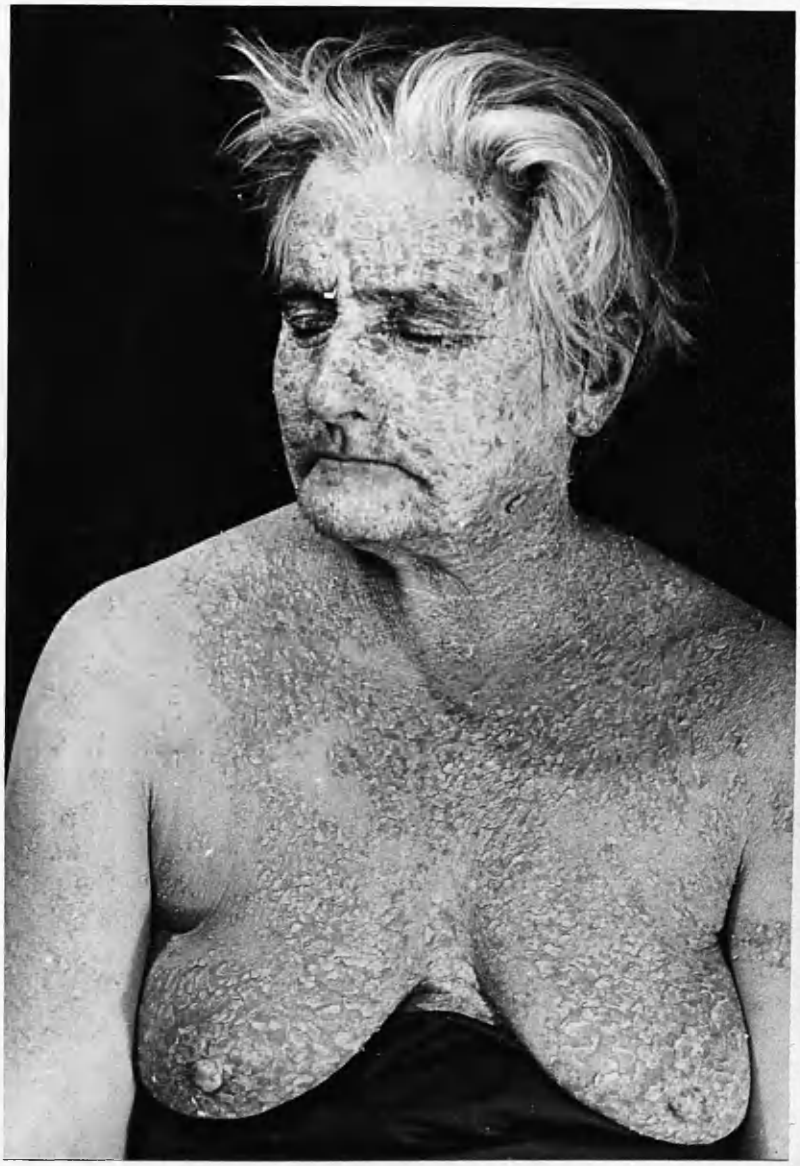
Case 41. Multiform sulphonamide eruption. Healing stage.



Case 45. Varicose eczema. The veins were more prominent in the standing posture.



Case 51. Scurvy. Shewing follicular petechial haemorrhages, ecchymoses and scaling.



Case 52. Pemphigus foliaceus. Shewing centrally attached scales.

Case 21. Histological appearances.

Parakeratosis, acanthosis and elongation of the rete pegs are present. The rete malpighii shows intercellular and intracellular oedema and a few inflammatory cells are interspersed between the epidermal cells. The upper corium is oedematous and is infiltrated by chronic inflammatory cells. The picture is consistent with a diagnosis of pemphigus foliaceus.

Case 22. Histological appearances.

A sub-epidermal vesicle containing serum and numerous eosinophil leucocytes is present. The surrounding epidermis contains several areas of intra-cellular oedema with evidence of impending vesicle formation. In the floor of the vesicle there is an inflammatory reaction in which eosinophil leucocytes are prominent. The appearances are more suggestive of a diagnosis of dermatitis herpetiformis than of pemphigus vulgaris but are still consistent with the latter diagnosis.

Case 26. Histological appearances.

A sub-epidermal vesicle containing coagulated serum, a few mononuclear cells and an occasional polymorphonuclear leucocyte is present. The floor of the vesicle is formed by the corium which shows a mild inflammatory reaction. The appearances are consistent with a diagnosis of pemphigus vulgaris.

Case 50. Histological appearances.

The section has been cut obliquely. One large vesicle, apparently sub-epidermal in position, is present. A number of small vesicles are seen in the epidermis. A moderate number of polymorphonuclear cells and of round cells are present in the vesicles and in the dermis. The appearances are consistent with a diagnosis of pemphigus vulgaris.

Case 52. Histological appearances.

The horny layer is almost completely denuded. High up in the rete the remains of a vesicle, which is roofed by a parakeratotic scale, can be seen in one area. The corium and especially the papillary layer is very oedematous and is sparsely infiltrated by chronic inflammatory cells, among which are occasional eosinophils. The picture is fairly typical of pemphigus foliaceus.