SEVERE MACROCYTIC ANAEMIA IN PREGNANT WOMEN IN THE GOLD COAST WITH RECORDS OF 100 CASES, AND WITH SOME REFERENCE TO HYPOCHROMIC ANAEMIA.

by


Thesis presented for the degree of Doctor of Medicine, Edinburgh University, 1939.
## CONTENTS

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>PREAMBLE</td>
<td>1</td>
</tr>
<tr>
<td>DEFINITION OF MACROCYTIC ANAEMIA</td>
<td>2</td>
</tr>
<tr>
<td>LITERATURE ON THE SUBJECT</td>
<td>2</td>
</tr>
<tr>
<td>GENERAL LOCAL BACKGROUND</td>
<td>12</td>
</tr>
<tr>
<td>Common Diseases of the Pregnant Woman.</td>
<td></td>
</tr>
<tr>
<td>ANAEMIA AMONG PREGNANT WOMEN</td>
<td>22</td>
</tr>
<tr>
<td>MACROCYTIC ANAEMIA AS OBSERVED IN THE MATERNITY HOSPITAL, ACCRA.</td>
<td>24</td>
</tr>
<tr>
<td>BLOOD PICTURE</td>
<td>30</td>
</tr>
<tr>
<td>COURSE OF THE DISEASE</td>
<td>31</td>
</tr>
<tr>
<td>1. Untreated</td>
<td></td>
</tr>
<tr>
<td>2. Treated in Hospital</td>
<td></td>
</tr>
<tr>
<td>3. Occurring for the first time in the puerperium.</td>
<td></td>
</tr>
<tr>
<td>PROGNOSIS/</td>
<td></td>
</tr>
<tr>
<td>Section</td>
<td>Page</td>
</tr>
<tr>
<td>-------------------------------</td>
<td>------</td>
</tr>
<tr>
<td>PROGNOSIS</td>
<td>35</td>
</tr>
<tr>
<td>AFTER HISTORY</td>
<td>35</td>
</tr>
<tr>
<td>TREATMENT</td>
<td>36</td>
</tr>
<tr>
<td>DIFFERENTIAL DIAGNOSIS</td>
<td>40</td>
</tr>
<tr>
<td>AETIOLOGY</td>
<td>44</td>
</tr>
<tr>
<td>SYNOPSIS OF CASES</td>
<td>48</td>
</tr>
<tr>
<td>MORTALITY RATE</td>
<td>52</td>
</tr>
<tr>
<td>COMMENT ON SPECIAL CASES</td>
<td>52</td>
</tr>
<tr>
<td>FATAL CASES</td>
<td>56</td>
</tr>
<tr>
<td>SUMMARY OF WRITER'S CONCLUSIONS</td>
<td>58</td>
</tr>
<tr>
<td>SUMMARISED CASE RECORDS</td>
<td>59</td>
</tr>
<tr>
<td>TABLES</td>
<td>201</td>
</tr>
<tr>
<td>REFERENCES</td>
<td>226</td>
</tr>
</tbody>
</table>
SEVERE MACROCYTIC ANAEMIA IN PREGNANT WOMEN IN THE
GOLD COAST, WITH RECORDS OF CASES AND WITH SOME
REFERENCE TO HYPOCHROMIC ANAEMIA.

PREAMBLE.

There are many differences between the practice of obstetrics in England and on the Gold Coast. One of the more dramatic is the occurrence of severe macrocytic anaemia associated with pregnancy, a condition rarely seen at home, but by no means uncommon in West Africa and some other tropical countries. After fourteen years' experience of obstetrics in Accra and other parts of the Gold Coast, the writer has become familiar with the disease and presents herewith notes on the condition, together with records of illustrative cases.
DEFINITION.

Macrocytic anaemia of pregnancy is a rapidly advancing anaemia found in the pregnant woman, generally in the last three months of pregnancy, which does not respond to treatment with iron and which is characterised by a high colour index and the occurrence of macrocytes in the blood. Untreated, the patient may die before, or shortly after delivery, or she may go into premature labour and eventually recover. The condition generally responds rapidly to therapy by liver or liver extract. Relapse does not occur except sometimes in subsequent pregnancy.

The condition may be first observed in the puerperium, when response to treatment is not always satisfactory.

LITERATURE ON THE SUBJECT.

Literature on the subject of anaemia in pregnancy is scanty before the middle of the nineteenth century, though BARNES (1876) states that Hippocrates and Galen saw in the blood of gravid women such impoverishment as to regard it as approaching a pathological condition, and it is only within recent years that our knowledge of the condition has advanced.

CHANNING (1842) published "Notes on Anaemia Principally/
Principally in Connection with the Pregnant State". He collected 17 cases, and all those occurring in pregnant women ended fatally. I have not been able to read Channing's paper, but it is referred to by several later writers and appears to have attracted a good deal of attention.

GUSSEROW (1871) stating that he could find no literature on the subject of pregnancy associated with anaemia, reported on five cases of severe anaemia in the later months of pregnancy, all of which were fatal. The description of his cases suggests that he was dealing with the condition under consideration here. Four of his cases were multiparae, and one a primipara. The only common aetiological factor which he could find in the series of five, was the fact of advanced pregnancy, and his conclusion was that this anaemia was a special disease of pregnancy. On post mortem examination no pathological change was found sufficient to account for death except the high-grade anaemia. This type of anaemia was said to be not uncommon in the Canton of Zurich. As regards treatment he stated that should similar cases again come under his care, he would adopt as treatment termination of the pregnancy, possibly combined with blood transfusion.

DELAFIELD/
DELAFIELD (1882) published a fatal case of puerperal anaemia which is of interest because he mentions cough, oedema of legs and feet, and a temperature which varied between 99° F. and 103° F. and in which the diagnosis from septic endocarditis had to be made.

OSLER (1888) reviewed the literature, and gave priority to Channing. In his paper "Puerperal Anaemia and its Treatment with Arsenic" he reported five post-partum cases three of which recovered and he strongly advocated the use of large doses of arsenic in cases of anaemia which were not responding to iron. Gastro-intestinal disturbance, although usually present, was no contra-indication to its use. This is interesting, because even to-day, after the introduction of liver therapy, one considers there is a definite place for arsenic in the treatment of macrocytic anaemia.

DAVIS (1891) reviewed the literature, and reported on one case with favourable issue, where an underlying chlorotic condition was suspected of having some effect on causation.

OLIVER (1894) considered that a pre-existing anaemia was of great importance in the development of grave anaemia occurring during the later months of pregnancy, particularly if the appetite failed. He believed/
believed that the anaemia was not due to destruction of red cells but to the undue strain placed on the already depleted reserves of the blood-forming organs. He noted the occurrence of bloodless labour, with death rapidly supervening.

CABOT (1908) in Osler's System of Medicine reported 35 cases associated with pregnancy, from among 1200 cases of pernicious anaemia reviewed. He considered these cases presumably to be explained as the result of haemolysis, and a manifestation of the auto-intoxication brought about by the pregnant state which also manifested itself in the occurrence of nephritis, eclampsia, and obstinate vomiting. He separated the condition from true pernicious anaemia by the age and sex incidence, the progress without remissions, and by the fact that occasional cure resulted when pregnancy came to an end. He suggested that eventually a different causal factor would be found in these cases, from that in true pernicious anaemia.

ESCH (1917) published six cases of pernicious anaemia of pregnancy, of which three died and three survived. He subscribed to the haemolytic theory of causation.

FINDLAY (1918) held the view that where pernicious anaemia complicated pregnancy the position of the mother was hopeless.

OSLER/
OSLER (1919) described the condition, as it is now known, both in pregnancy and in the puerperium. He noted that text-books of obstetrics had little to say, and that the gloomy prognosis had the echo of the unfortunate experience of earlier writers. He also stated that "to the nature of the haemolytic agent there is as yet no clue," and that "to use the word toxin almost connotes ignorance." He noted that pernicious anaemia of pregnancy differed in one respect from that of true Addisonian anaemia in that where recovery took place it was permanent.

ADLER (1924) reported on three cases in all of which he found some evidence of pre-existing hypochromic anaemia. He stated "I conceive therefore that this pregnancy anaemia is not a true pernicious anaemia caused by a pregnancy toxin but see in it only a form of reaction of a functionally damaged bone marrow to the pathological or even physiological influence of pregnancy."

LARRABEE (1925) believed true pernicious anaemia to be rare in pregnant women, that it occurred in women already predisposed to other forms of anaemia, and that the cause had to do with the additional strain on the blood-forming organs. He stressed the danger of confusing the condition with septic/
septic and subacute endocarditis, or with puerperal sepsis. As treatment he recommended blood transfusion. He reported on four cases seen between 1906 and 1914, in pre-transfusion days, three of which died, and a further series of four cases which were treated by blood transfusion between 1919 and 1925, all of which recovered.

MINOT and MURPHY (1926) reported on the treatment of pernicious anaemia by a special diet especially rich in liver, proteins, iron, fresh fruit and vegetables.

BALFOUR (1927) reported on the occurrence of 43 cases of pernicious anaemia of pregnancy in Calcutta in a period of 2½ years. She suggested an incidence of 1.69% among the cases of pregnancy examined, with a maternal mortality of 34.88% and a foetal mortality rate of 55.81%. The treatment adopted was intramuscular injection of whole blood. She postulated the possibility of nutritional defect as being of importance in causation.

CASTLE and TOWNSEND (1928) demonstrated the absence of intrinsic factor in pernicious anaemia, and the need of the intrinsic and extrinsic factors in the production of the haemopoietic factor.

WILLS and STEWART (1930) described 50 cases of 'idiopathic' pernicious anaemia of pregnancy in/
in India, with five non-pregnant cases with identical blood findings.

WILLS and MEHTA (1930) produced pernicious anaemia experimentally in albino rats by deficient feeding.

PETESEN, FIELD and MORGAN (1930) reported three cases of pernicious anaemia of pregnancy successfully treated by a diet rich in liver.

WILLS (1931) found that marmite was as effective as liver in the treatment of pernicious anaemia of pregnancy.

MITRA (1931) reported on 86 cases in India.

GÄNSSLEN (1931) reported on the effective use of injections of liver extract, in the form of Campolon, in pernicious anaemia.

STRAUSS and CASTLE (1932) in a study of anaemia in pregnancy, stressed the importance of good intake and of good assimilation. They emphasised the relationship of diet and gastric secretion to blood-formation during the last trimester of pregnancy, when the foetus draws upon the maternal organism for blood and muscle building, and considered that anaemia was only found in those patients who had had for a considerable time a defective diet, or deficiencies in diet conditioned by gastric anacidity or related gastro-intestinal disturbance. They stressed the importance of optimal diet, especially rich in proteins and/
and iron-containing foods, for the prevention of anaemia. They further pointed out that the diet deficiency of macrocytic anaemia is the same as that found in hypochromic anaemia, where the influence of the lack of gastric acidity was well recognised.

STRAUSS and CASTLE (1933) presumed temporary lack of intrinsic factor in macrocytic anaemia of pregnancy, the condition being completely relieved by administration of liver extract.

WILLS (1933) demonstrated that the haemopoietic factor in marmite was not vitamin B1, B3, or B4.

WILLS (1934) stressed the difference between macrocytic anaemia of pregnancy and true pernicious anaemia, and considered that the response to liver therapy suggested a nutritional origin, which was borne out by rat experiment.

JONES and TOCANTINS (1934) in Adair and Stieglitz's "Obstetric Medicine" stated that severe anaemia of pernicious type was rarely reported, but that probably many cases were not recognised. They estimated the death rate in inadequately treated cases at 60-70%.

CASTLE (1934) considered that tropical macrocytic anaemia was a deficiency dyshaemopoietic anaemia due to lack of the haemopoietic factor in the liver.

WILLS/
WILLS and STEWART (1935) separated two fractions from Campolon. One, which presumably contained anahaemin, was active in human pernicious anaemia, and inactive in nutritional macrocytic anaemia of monkeys, while the other was active in the monkey anaemia, which was considered to be similar to the anaemia found in pregnant women.

STRAUSS and CASTLE (1935) considered the condition to be due to a combined deficiency of iron and of haemopoietic factor, also that a temporary lack of intrinsic factor in the gastric juice, or lack of vitamin B2, or extrinsic factor, might produce similar effects.

NAPIER (1936) reported 11 non-pregnant cases of a haemolytic anaemia usually following malaria. He postulated that in addition to vitamin B. complex deficiency there must be other causes of the condition. He suggested that excessive haemolysis of red cells, as occurs in malaria, might be an associated factor.

VAUGHAN (1936) described the disease, and grouped it with the anaemias of defective production, rather than with those of excessive destruction.

WHITBY and BRITTON (1937) considered that it was relatively rare for a dyshaemopoietic anaemia to be due to a single deficiency factor, and that/
that in pregnancy intrinsic factor was temporarily absent, was restored after parturition, and was not necessarily associated with achlorhydria.

HAMILTON FAIRLY (1938) described macrocytic anaemia in Macedonia, and preferred the term nutritional to tropical anaemia. He mentioned having observed the occurrence of haemorrhages, also the occurrence of the condition in patients who showed evidence of good nutrition. The latter observation has also been made by others.

VAUGHAN (1938) considered macrocytic anaemia due to a conditioned deficiency, and speaks of a megalocytic hyperchromic anaemia occurring during pregnancy or the puerperium not necessarily associated with achlorhydria and due to temporary deficiency of the pernicious anaemia factor. She states that further work must decide whether tropical megalocytic anaemia, not necessarily associated with pregnancy, is the same as pernicious anaemia of pregnancy in temperate zones.

STEVENSON (1938) was of opinion that macrocytic anaemia was commoner than was generally supposed, and was convinced that the anaemias could not be placed in watertight compartments.
GENERAL LOCAL BACKGROUND.

Before proceeding to describe macrocytic anaemia of pregnancy as seen on the Gold Coast, it may be of interest and value to outline briefly some points relating to climate, customs, and common diseases which may have bearing upon the development of the disease.

I. The Country and Climate.

The Gold Coast is a British tropical dependency in West Africa. The Colony is a small but prosperous one. Accra, the seat of government, is situated on the coast, about five degrees north of the equator. The climate is tropical, the temperature averaging between 80° and 90° Fahrenheit, for most of the year. Humidity is high and the rainfall moderate. There is little variation in temperature either throughout the year, or throughout the twenty-four hours of each day. The wet seasons extend from June to July, and from the end of September to the beginning of November.

The country, for various reasons, is not considered suitable for white colonisation. The people are negro, and are primitive agriculturalists in habit. They are not allowed to alienate their land, and are, as a result, comparatively well-to-do as/
as peasant landowners.

The main crop is cocoa, grown for export. At one time two-thirds of the world supply of cocoa was derived from the Gold Coast. In boom years large sums are handled by the people, and are spent on education, houses, food, and clothes. In religion the country is pagan, but as education for the most part is carried on by missionary societies, the educated people are as a rule at least nominally Christian. Polygamy is controlled by a man's purse. If he can afford more than one wife, he usually has two or more. The people as a whole are active and enterprising, and willing to try out new ideas to see if they are good. They are well grown, and of good physique, but are smaller somewhat than the average British. There is no seclusion of women, who indeed do most of the work of planting and gathering, and of preparing food. The women tend to be smaller than the men, and the external pelvic measurements are about an inch less than the average given for the European. Fortunately the infants also are smaller, averaging not more than 6 lb. 5 oz. at birth. Clothing is very simple, and in women consists principally of one or two cloths wound round the body. Men tend to adopt the European shirt and shorts, and all go barefoot. The women take their babies/
babies with them everywhere, tied on their backs.

The houses are of mud, with, where money permits, galvanised iron roofs. Otherwise thatch is used. Sanitation in the towns is good, and some supervision is exercised in the larger villages. On the farms and in the smaller villages sanitation is non-existent, and conditions are completely primitive. There is no general recognition as yet among the people of the laws of health and of the importance of hygiene.

The population moves freely about the country, on foot and by motor lorry, and there are hardly any pure town dwellers. All families, even town dwellers, own land, and all go off for varying periods to cultivate it, or to visit country relations. On the other hand, country people pour into the towns for certain festivals and anniversaries. The result is that there is not much difference in the health of town and country, except in so far as the town people, being in more profitable employ, are better fed, and can get more variety in the way of food.

II. Diet.

Meals are few, not more than two, and sometimes only one, being taken in the day. The bulk of the food is made up of starch, procured from cocoyam/
yam, cassava or plantain. When available, yam is preferred, but it is dearer and not always easy to get in the coastal districts. The preparation of this food is a somewhat lengthy process. The raw materials are boiled, or more often parboiled, and beaten into a mass of dough-like consistence called fu-fu. With this is eaten a relish, or soup containing scraps of meat or sun-dried fish, vegetables, peppers, palm-oil or ground nuts. Maize is grown and eaten fresh in season, and also stored and ground, and eaten as various kinds of porridge. Storing methods are, however, extremely primitive, and before the next harvest very little may be left of the maize except the husk, the rest having been eaten out by weevils. Of recent years white bread has become a favourite article of diet. It is raised with palm wine and sugar, and eaten dry.

Scraps of sun-dried fish are much used as flavouring, and snails, or sometimes a little meat, may be added according to the means of the household. When in funds the people buy freely of corned beef, sardines, etc., but generally there is a marked lack in the diet of animal fat, of good class protein, and of fresh fish. Small domestic goats, pigs and sheep are bred, but not in great numbers, and they are only killed on special occasions. Small native hens are everywhere/
everywhere, but the eggs are never eaten, and the fowls only on certain occasions. There are no cattle, on account of the tse-tse fly, and therefore no milk whatever. No attempt is made to milk the small native goats. This may in part account for the lengthy suckling of infants, which goes on for at least eighteen months.

As a whole the diet is undoubtedly overloaded with bulky and rather poor-class starch. CLARK (1936) is of opinion that cassava and cocoyam are dangerous cyanogenic substances in the absence from the diet of adequate organic sulphur, the main source of which is animal protein. Maize may also at times be faulty in the same way.

The vegetable oil content of the diet is high, and palm oil especially is rich in vitamin A.

Some part of the vitamin B. complex would appear to be scanty, and C. may be only barely adequate. Fruits are not very plentiful, and where they are available they are considered only food for children, not adults, or they are sold to Europeans.

The adult population as a whole has the appearance of being adequately nourished, and it is only in times of physical stress that an underlying dietetic deficiency becomes apparent. The majority of children between the ages of 8 months and 4 years look under-nourished, but how much of this is due to inadequate diet, and how much to the diseases which are/
are rife at that age, it is very hard to decide.

The heavy starchy meals tend to cause constipation, which is only in part counteracted by the large quantity of peppers consumed with the food and which act as an intestinal irritant.

III. Marriage and Childbearing.

Marriage is a family affair for the begetting of children. Girls are married shortly after puberty, but among the educated there is a growing tendency to delay marriage until the twenties. Once married a woman expects and desires to have many children. All women marry, and it is disastrous to the marriage if it is sterile.

Infants are breast-fed until the next one is on the way, and to avoid too frequent pregnancies the women as a rule go home to the maternal roof until the infant is several months old.

The infant mortality rate is very high, and is but little regarded by the people, provided some children of the family survive. A woman is really more concerned to be constantly fertile than to keep alive the children she already has.

Delivery, when all is normal, is usually more rapid than in Europeans, but ignorant and meddlesome interference often enough turns the normal into the abnormal, and hastens disaster in the abnormal/
abnormal. If left to herself, a woman adopts a crouching attitude and bears down hard from the beginning of labour. In primiparae the perineum is nearly always well torn. The native attendant separates the cord with a knife, on the floor, after, never before, the placenta is born. The woman then gets up and washes herself, but does not go about for several days.

Rarely, even in the primipara, is the head engaged before labour starts. It moulds easily and quickly, however, and descends rapidly. The posterior position is one of the most fruitful causes of disaster, as the necessary delay for rotation is not understood, and violent ecbolics are resorted to. Ginger and pepper are favourite remedies, administered by mouth, *per vaginam*, or applied to the umbilicus of the new-born.

The maternal mortality rate is undoubtedly high, but as yet no reliable figures are available. Where interference has taken place severe sepsis frequently follows, and on occasion tetanus. Umbilical sepsis and tetanus neonatorum are not uncommon.

IV. The Maternity Hospital was opened in 1928 to do maternity work among the natives, and to train native midwives. Most of the material for this study of macrocytic anaemia has been derived from this hospital/
hospital where I have at different times held the post of Medical Officer in Charge since 1929.

V. The Common Diseases of the Country.

The diseases of the country naturally take their heaviest toll of the children, but the adult population is by no means as immune as would at times appear to be believed.

Malaria.

The most important of those diseases is malaria. The subtertian variety is the most usual and the most deadly. During infancy and early childhood most of the children suffer from it more or less severely (RUSSELL, 1938) and the death rate directly or indirectly due to malaria is considerable. As the children grow they acquire some degree of immunity, but it is by no means complete among adolescents and young adults.

Yaws.

Yaws, an acute exanthematous disease of childhood, is very prevalent away from the larger towns. It causes much chronic ill-health. Owing mainly to this disease, the positive Wassermann rate for the adult population as a whole is about 32% (Research Institute, Accra). The presence of yaws makes it difficult, and indeed almost impossible to detect the presence of syphilis, if these are indeed different/
different diseases. In the child-bearing population yaws is responsible for ulcers, crab yaws on the feet, bony deformities of the long bones, and possibly for some cases of still birth.

Helminthiasis.

Helminthiasis is almost universal among children, but heavy infestation is rarely seen among the adult women. Round worm is common, and also ankylostomiasis. In some parts schistomiasis is found. It causes albuminuria and sometimes haematuria.

Gonorrhoea.

Gonococcal infection is very prevalent, and but little regarded by the people. Possibly because of native methods of cure I have rarely seen the acute phase. Possibly again the local manifestations are not as a rule severe, although when the infection spreads up to the Fallopian tubes, it can be as damaging as in other countries. The effect of the infection on the infant eye may be severe, but as a rule it does not cause total blindness.

Ulcers.

Ulcers of all kinds, including tropical ulcer, are common in childhood and among the poorer sections of the adult community.

To obtain information with regard to the prevalence of some of the above-mentioned diseases, routine blood tests and examination of faeces were carried/
carried out by myself and others over a period of some years on the in-patients at the Maternity Hospital. The results are recorded later (page 225a).

VI. **Common Diseases found in the Pregnant Woman.**

**Malaria.**

Of the diseases to which the pregnant woman is liable malaria is one of the most important. In primiparae especially it may predispose to severe anaemia, and is a fruitful cause of miscarriage and premature delivery.

**Toxaemia.**

Toxaemia occurs as in temperate climates. Toxic albuminuria has, however, to be carefully distinguished from other prevalent causes of albuminuria.

**Constipation.**

Complaint of constipation is very common, and is combatted among the people by purgatives, rather more than less drastic, of both European and local origin, and also by enemata, both of which measures appear not infrequently to precipitate labour.

**Sore Tongue.**

Sore tongue is common and is the most definite sign of dietetic deficiency which I have been able to discover. The tongue becomes glazed and red/
red, especially at the tip and round the edge; small ulcers may be present, and the angles of the mouth may also be sore. In going over the out-patient records of the Maternity Hospital for one year, I found sore tongue recorded in 10.7% cases. The condition usually clears up after delivery, and it also clears up on the administration of marmite.

**Rheumatism.**

Complaints of 'rheumatic pains', more or less vague and fleeting, are frequent, and are often difficult to attribute to any definite cause apart from malaria or yaws. Valvular disease of the heart is very unusual in the child-bearing population, and rheumatic fever is practically unknown in the country.

**Premature Delivery.**

Premature delivery is common in primiparae, and is probably due to a combination of factors, of which malaria is one of the most important.

**Abortion.**

Induced and spontaneous abortion are common, and lead to much pelvic damage and resultant sterility.

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**ANAEMIA AMONG PREGNANT WOMEN.**

Anaemia is difficult to detect in the Out-Patient Department among negroes. One has to judge by the appearance of the mucous membranes of the mouth and by the conjunctivae, and simple examination of these
may cause flushing which may give one a wholly erroneous impression. The gums are often more reliable than the conjunctivae, but where much pigment is present difficulty again arises.

To gain some idea of the prevalence of anaemia among the pregnant women I made two series of examinations, one in Accra, and one in Kumasi which is some 260 miles inland, but where conditions are very similar. I undertook these investigations in order to find out whether they would throw any light on the occurrence of the severe macrocytic anaemia which is the subject of this paper.

1. In Accra I made routine haemoglobin examinations on 381 out-patients with a Sahli haemoglobinometer and at the same time had the blood films examined for malaria parasites. The results are recorded in Table 4, p. 212.

2. In Kumasi I made further routine examinations. This time I carried out red cell counts, haemoglobin estimations, and examined thick films for malaria parasites in 100 cases. The results are recorded in Table 3, p.208. From these Tables it will be seen that some anaemia and even an anaemia of high colour index is by no means uncommon among the pregnant women. None of these women were selected as being especially anaemic from their appearance. I simply/
simply took blood from the first two or three patients who arrived each day.

**Macrocytic Anaemia.**

Of the less common and more interesting diseases found in pregnant women is the severe macrocytic anaemia to which reference has already been made, and which is the subject of this study.

**MACROCYTIC ANAEMIA AS OBSERVED IN THE MATERNITY HOSPITAL, ACCRA.**

**Selection of Cases.**

Owing to the prevalence of anaemia (Tables 3 and 4, pages 208 & 212) slight cases of macrocytic anaemia tend to escape notice, and these records are, therefore, mainly concerned with severe cases.

The selection of cases has been found to be no easy matter, especially as I came to realise to what extent anaemia is present among pregnant women, and how difficult it is to find a case of macrocytic anaemia in which the picture is not more or less blurred by the occurrence of a concomitant iron-deficiency anaemia. Further, as the two conditions so often occur together, and as clinically the results of treating all cases with both iron and liver are so much more satisfactory than the treatment with iron or liver alone, the selection of cases of what I believe to be essentially macrocytic anaemia has not been easy.
All cases of haemorrhage have been excluded. It has not been the writer's experience that cases of macrocytic anaemia show any tendency to undue loss of blood; and labour is, if anything, unusually bloodless. No cases have been included except those in which investigation has been sufficiently thorough, or the response to treatment sufficiently definite to justify the diagnosis.

With regard to cases complicated by intercurrent or concurrent conditions, I have followed the line taken by WILLS and MEHTA. At first DR. WILLS (1930) discarded all cases that were not 'idiopathic'. Later she included cases with concurrent malaria, hookworm, and sprue (WILLS and MEHTA, 1931). I have adopted the same course and have also included cases with concurrent urinary infection. In all of these cases the anaemia was so striking in contrast with cases of similar infection but with no anaemia, that I had no hesitation in considering them to be cases of macrocytic anaemia. The concurrent condition may be accidental, or the last straw on the camel's back in precipitating severe anaemia.

**Difficulty of Detection.**

The detection of anaemia has to be arrived at/
at in the Out-patient Department. The appearance of the complexion even in the white-skinned person may be very deceptive; in the negro, where only the mucous membranes can act as guide, diagnosis is equally, if not more, difficult, and the results of routine haemoglobin examination undertaken by myself (Tables 3 and 4) came as a considerable surprise. The history as given by the patient is of little assistance, and the doctor has to rely upon his own observation. Special difficulties are:

(1) that one has to work through an interpreter in most cases, and
(2) that the patient has no sense of time, and very little capacity for observing the earlier symptoms of disease.

This is possibly due to the fact that the people are so much accustomed to chronic ill-health in childhood, and further that their ideas of illness do not go beyond the results of witchcraft and poison. Consequently I cannot claim to have recorded all cases of macrocytic anaemia occurring during the period dealt with. The 100 cases presented represent 1% of the total number of in-patients, but only the more definite and severe cases were collected.

Clinical History.

Macrocytic anaemia as observed in my cases is a disease of apparently rapid onset, and is by no means/
means rarely met with in the Gold Coast, having been found in 1% of in-patients since the Maternity Hospital was opened. So far as it can be obtained, the history is seldom one of more than two weeks' duration, and at times the condition appears to develop almost over-night, as in one or two cases admitted to hospital on account of some other condition, and suddenly observed to be severely anaemic after several days in hospital. The onset, however, is probably not as rapid as appears from the histories obtainable, and this is a question which could be further investigated with profit. It was in order to obtain light upon this point that the writer made the blood examinations which are summarised in Tables 3 and 4 already referred to.

**Signs and Symptoms in Severe Cases of Macrocytic Anaemia.**

When the patient is first seen, anaemia is usually well advanced, and she is complaining of one or more of the symptoms of anaemia, - breathlessness, palpitation, weakness, or giddiness. Other common complaints are of swelling of the feet and of cough. Sometimes there is no complaint of ill-health at all, and at other times the complaint may be very misleading, as, for example, when fever is complained of, or diarrhoea, dysentery, cough, or the passage of small/
small quantities of urine.

On examination the patients appear to be, as a rule, well-nourished. The common findings are, in the first place, severe anaemia. In severe cases there is profound pallor of the conjunctivae, but in the less severe, temporary congestion may be misleading, and the mucous membrane of the gums may be a more reliable index. At the same time there is to be noted a slight blurring of the features due to an almost imperceptible oedema, which is nevertheless very characteristic. Frank oedema of the feet is common, and may extend up the legs to the abdominal wall. Faint icteric tingeing of the conjunctiva may be present, but this perhaps is usually associated with concurrent malaria. The tongue is moist and white, and indented by the teeth. In some cases it may be glazed and red, but sore mouth does not appear to be more common in macrocytic anaemia than it is in the pregnant population as a whole.

The temperature is, as a rule, raised, and sometimes so considerably as to make one think of typhoid, or some septicaemic condition.

**Circulatory System.**

Marked breathlessness on exertion is a common symptom, and it is amazing how some of these patients walk into hospital as they do. The pulse is rapid, and systolic murmurs of a soft blowing character are heard all over the praecordium.

**Respiratory/**
Respiratory System.

The respiratory rate at rest in severe cases is raised, and a constant loose cough may be present and be very exhausting. On examination of the chest, moist sounds are apparent, which suggest that the cough is due to an extension to the lungs of the oedematous process so obvious in the subcutaneous tissues, or perhaps to a failing myocardium.

Digestive System.

Enteritis may be severe and misleading. The stools may contain mucus, and are often most offensive. Repeated attempts have failed to convict any of the known pathogenic organisms as a cause of the enteritis and the condition subsides as health improves under treatment of the anaemia. In this connection it may be of interest to note in passing that apart from cases of anaemia, patients who have been much exhausted by a long and difficult labour frequently suffer from exhausting diarrhoea for some time after delivery, even when there is no true pelvic peritonitis. Debilitated patients appear to be abnormally liable to enteritis.

Upon further physical examination, liver and spleen may or may not be palpable. It is as well to remember that in a malarious and worm-infested population/
population the spleen may be permanently much enlarged and fibrosed. The patient in the majority of cases is a primipara, and in the later months of pregnancy. (See p. 48).

Nervous System.

No sign of involvement of the nervous system has been noted. I have not seen polyneuritis nor beri-beri in pregnant women.

Urinary System.

Albumen and casts are fairly common findings in the urine, and on occasion have led to a wrong diagnosis. Organisms and pus cells may be present, particularly in cases seen after delivery. Of the organisms, B. coli is the most frequently observed. It is possible, and indeed probable, that pyelitis or cystitis may in some cases be a predisposing factor.

BLOOD PICTURE.

The blood examination shows a severe reduction in the number of red cells. A very common count, when a case is first seen, is one of about one million red cells per c. mm. The number may be below this and yet the patient recover. With such a count haemoglobin may be about 24% or higher. The colour index is as a rule unity or more, and leucocytes are not/
not as a rule increased. In the series examined, no constant variation from the normal was found, and the inversion of polymorphs and lymphocytes mentioned in literature was only observed in one fatal case.

Blood films very constantly show considerable anisocytosis, and large cells (macrocytes) are generally present. Polychromasia may be found. Poikilocytosis is not a marked feature in these cases, as a rule, and is the one point in which the blood picture may differ from that seen in true pernicious anaemia.

Nucleated red cells, normoblasts and megaloblasts, may be present. As a rule one or two can be found, and occasionally they are numerous. In one or two cases primitive white cells were also observed. The red cell count, percentage of haemoglobin, and colour index in the series of 100 cases are shown in Table 1, p. 201. The results in the cases in which full differential counts were made are shown in Table 2, p. 206.

COURSE OF THE DISEASE.

Untreated Cases.

Without treatment the condition progresses with great rapidity, and in the course of a few days the patient may become comatose and die from lack of blood. On the other hand many cases tend to be cut short/
short by the onset of premature labour. The woman may, of course, die during labour or in the first day or two of the puerperium; if, however, she survives this period, recovery often sets in and may be as rapid as the progress of the illness in the first place. Labour, in these cases, is as a rule premature, rapid and singularly bloodless. The uterine muscle does not appear to be at all weakened, and provided the cardiac muscle does not give way, birth is swift. I have never observed any tendency to haemorrhage in such cases. Were it otherwise the outlook would be grave indeed. Some writers have mentioned the occurrence of haemorrhage, but it has not been my own experience. Indeed the reverse is true, and the loss during parturition is less than normal.

The infant mortality and still-birth rates are high. This, I am inclined to think, is only partly accounted for by prematurity. After delivery, as mentioned above, recovery may or may not set in. The puerperium, as might be expected, tends to be febrile, and sepsis added to the anaemia seriously affects the chances of survival. Balfour (1927) writing of India estimated the maternal death-rate in untreated cases at 42%.
Cases Treated in Hospital.

In great contrast to the above is the history of cases treated in hospital. Response to treatment is usually rapid and gratifying in the extreme. Twenty-four hours after admission the patient may look, if anything, more bloodless than when first seen. In forty-eight hours, however, one can say quite definitely that improvement has begun, and this without recourse to blood examination. There is a faint, very faint, flushing of the conjunctivae which the clinician comes to look for and which when once seen causes one to feel satisfied about the patient for the time being. Every day thereafter improvement is apparent. Blood examination a week after admission shows a gain of at least half a million in red cells, and of 5-10% in haemoglobin. The following week should show the same advance, especially if iron has been added to the liver treatment. At the end of a week, oedema, if any, should have largely disappeared, as also the cough. The temperature settles down in ten days or so, and intercurrent gastrointestinal and urinary complications usually recover as the anaemia improves. At the end of a week's treatment the patient feels much better, and in two weeks she becomes restive. Probably she may be prevailed upon to stay another week in hospital, and/
and a few very severe cases have remained even longer. By the end of three weeks improvement is so pronounced that the patient is allowed to go, usually with instructions to continue to take liver. This advice is very rarely followed, but in those cases where a follow-up was at all possible, it was found that the anaemia did not return in that pregnancy.

Nothing is more satisfactory than the treatment of a severe case of macrocytic anaemia, admitted seriously ill, and discharged in two or three weeks looking and feeling well. No doubt a critical blood examination tells a different tale, but apparently, in the short period of treatment, enough of the lacking haemopoietic substance has been supplied to last over the special strain of pregnancy. Once response to treatment is definitely established labour rarely gives rise to anxiety.

Acute Macrocytic Anaemia appearing for the first time in the Puerperium.

When acute anaemia appears for the first time in the puerperium, it may cause anxiety. Oedema is not a marked feature, and the question of sepsis has to be considered. Even a low-grade infection makes the response to treatment less satisfactory. Some of these cases tend in the end to become aplastic in type.

Prognosis/
Prognosis.

With regard to prognosis, if the patient has no treatment, and unless she early goes into labour, the outlook is bad. The prognosis is good under treatment provided the patient is not in extremis when she is first seen and if she is not in labour. If the haemoglobin is not less than 20%, and the red cells not below 1,000,000, recovery is the rule. One or two patients under the writer's care had a much lower haemoglobin value and recovered, but such cases give cause for anxiety for some time, and must be considered in a critical condition, especially should they go into labour.

The prognosis has greatly improved since liver extract for parenteral administration has been available. As is to be expected in patients so ill, their ability to take, retain, and absorb liver by the mouth is uncertain and variable.

After History.

Some time after discharge from hospital these women appear to recover their normal health, and relapse does not occur except during the course of another pregnancy when the anaemia may again be severe. Relapse in the same pregnancy has not been seen, but of the ten cases which were traced through subsequent/
subsequent pregnancies 9 again suffered from severe anaemia. As before mentioned, cases which develop after delivery may not do well and tend sometimes to become aplastic in type.

**Treatment.**

Treatment was in practically all cases by rest in bed, full diet, and raw liver or liver extract by mouth or by injection. The patients preferred to take liver rather than marmite, and there was some evidence that liver was more quickly beneficial. If the patient could take it, \( \frac{1}{2} \) lb. of raw liver was given daily, finely minced, with salt and pepper, and eaten with orange juice.

In India marmite is the sheet anchor of therapy. It was used also in this series of cases, but only in small doses, which probably had little effect. The patients did not care for it and there was always difficulty in persuading them to take it. On the Gold Coast there is no prejudice against meat eating, while the price of marmite made it an expensive remedy.

Campolon injections were used in all severe cases from the middle of 1935. The dose at first was 2 cc. injected into the buttock every second day for five to seven injections. Later it was found that this was often inadequate and that a satisfactory result/
result did not obtain until 4 cc. were given daily.

As the macrocytic anaemia responds to treatment, the underlying and concurrent hypochromic anaemia is unmasked, and useful response to iron then takes place. Usually iron therapy was started as soon as the initial response to liver therapy was well established, that is, in five to seven days. Iron was given either as one of the scale preparations in thirty grain doses thrice daily, or in conjunction with arsenic. Injections of iron and arsenic (FRAISSE) were thought to be useful in promoting further improvement. The benefit thought to be derived from the use of arsenic is of interest in the light of Osler's advocacy, even as far back as 1888, of the use of arsenic in severe puerperal anaemia which did not respond to iron.

It was not possible always to have blood counts made in support of treatment, and one was guided, as a rule, by one's clinical experience, as to whether improvement was or was not progressing satisfactorily.

Progress was, as a rule, so rapid and satisfactory, and the patients quickly felt so much better, that there was always difficulty in persuading them to remain in hospital for a reasonable time/
time. The best results were undoubtedly obtained in those who remained until the red count was over 3,000,000 but generally one had to be content when a rapidly advancing anaemia was changed to a rapidly improving one. Quinine was always administered at first if the patient had a temperature, whether parasites were found or not.

Once the acute progressive anaemia has been controlled, one has to consider any concurrent or predisposing disease which may be present. Sore mouth is treated locally by glycerine and borax, but reliance is put on the improved diet to clear up the condition in a short time. Enteritis also improves with the general condition.

Care, of course, must be taken to eliminate dysentery and typhoid, which the symptoms may lead one strongly to suspect, but it is highly important also to bear in mind that to treat an anaemia on the diet considered suitable for one of these diseases leads rapidly to worsening of the whole condition.

In a considerable proportion of cases there is albumen, even casts, pus cells, and organisms, in the urine. Apart from rest in bed and the administration of plenty of fluids, it is sufficient to concentrate/
concentrate on the treatment of the anaemia. In nearly all cases the urine clears as the anaemia improves. Casts disappear, but sometimes a trace of albumen remains. Here again, to concentrate on the urinary condition in the mistaken belief that it is primary, leads rapidly to disaster.

**Blood Transfusion.**

Only seldom has a blood transfusion been necessary. Occasionally, when response is unsatisfactory, a small transfusion, intravenously or intramuscularly (BALFOUR, 1927) will give the necessary stimulus towards recovery. In the case of a double plus Wassermann, it is as well to give a course of N.A.B. This drug has a good tonic effect, besides serving as treatment for any specific infection which may be present.

Induction of labour is seldom called for, but cannot be dismissed as never indicated. In a few cases which respond well at first, and then remain stationary with haemoglobin and red cells still at very low figures, it is at times desirable to induce labour in the interest of the mother. One has to remember, however, that it is by no means a measure devoid of risk, particularly because of the possibility of sepsis in the puerperium. The procedure is worth/
worth trying in cases where the marrow will not respond beyond a certain point, even to large doses of Campolon.

**Cases Discharged before Delivery.**

In cases discharged from hospital before delivery it is impossible to insist upon continuance of treatment or even to keep track of patients. Those of whose after history anything is known seem to have gone through the approaching labour safely, even without further treatment. The danger of the recurrence of anaemia in future pregnancies can probably be reduced by careful antenatal supervision, and by the treatment of all cases of pregnancy showing an unduly low haemoglobin level.

**Differential Diagnosis.**

Differential diagnosis may have to be made from the following: true pernicious anaemia, other blood conditions, typhoid or enteritis, bronchitis or pneumonia, toxic albuminuria, pyelitis, malaria, internal haemorrhage, puerperal sepsis, septic endocarditis and sprue.

The points which aid in distinguishing between macrocytic anaemia of pregnancy and true pernicious anaemia are as follows:

(a)/
(a) The patients in the former are younger than in pernicious anaemia;

(b) the anaemia is associated with pregnancy, and tends to cure itself after delivery;

(c) there are no remissions as in pernicious anaemia;

(d) the condition of the gastric secretion. Free hydrochloric acid is present in most cases of macrocytic anaemia;

(e) the absence of nervous involvement;

(f) the blood picture, which though almost the same, shows as a rule considerably less anisocytosis than in true pernicious anaemia.

Other blood conditions are eliminated by the characteristic blood picture. Perhaps some of the more obscure of the **aplastic anaemias** are the most likely to be mistaken for macrocytic anaemia of pregnancy, as also **achrestic anaemia**. The lack of adequate response to treatment, by inviting a more careful review of the blood picture, soon leads to their elimination.

Clinically it is more important not to mistake a case for one of **typhoid** or **enteritis**, because of offensive loose stools containing mucus, and temperature; for **bronchitis** or even **pneumonia** on account of the rising respiration rate, and constant irritating cough, with râles in the chest; for **toxic albuminuria** on account of albumen and casts in the urine; for **pyelitis** because of temperature and pyuria; and/
and for malaria because of the presence of malaria parasites and temperature.

In differentiating from the above, the extreme pallor of the mucous membranes should at once point to the condition most urgently requiring attention. Where the diagnosis is most likely to be missed is perhaps in the patient who has been some days in hospital for the treatment of a straightforward attack of malaria or pyelitis, where the temperature comes down with appropriate therapy, and a severe anaemia develops almost, as it were, under the eye of the Medical Officer. One has to be on guard constantly against such an occurrence.

In my own experience the need to make a differential diagnosis from internal haemorrhage has hardly arisen. There is usually some history available, and the peculiar puffy appearance of the sufferer is very characteristic. In one case where the patient was already dead on admission the diagnosis had to be made from toxaemia with concealed accidental haemorrhage. In life, however, the tense tender condition of the uterus would make the differential diagnosis easy. The condition of the urine in both may be similar.

With regard to differential diagnosis from puerperal sepsis, the writer has found it a sound rule...
to treat all cases with a temperature in the puerperium as potentially cases of macrocytic anaemia. Occasionally it is possible to say that a particular case is one of puerperal fever only, but more often the patient's general state presents a confused picture of sepsis and malnutrition with its accompanying anaemia, inextricably mixed.

Anaemia must be distinguished from septic endocarditis. The signs of enlargement of the heart, with soft murmurs, breathlessness, and irregular temperature, may be the same in both diseases, and the anaemia of endocarditis may be severe. In the writer's experience, however, there are never any petechiae, nor is there any haemorrhagic tendency in macrocytic anaemia.

The response to suitable liver therapy, moreover, is different, for in macrocytic anaemia the cardiac signs and symptoms improve pari passu with the anaemia. Valvular disease is unusual in the pregnant native woman and rheumatic heart disease is almost unknown.

The writer has not seen a case of sprue, and it is very rare, indeed practically unknown, in European or Africans, on the Gold Coast.

AETIOLOGY/
AETIOLOGY.

The present position as regards etiology, as gleaned from perusal of the more recent literature, is that macrocytic anaemia is a deficiency disease due to lack of the haemopoietic factor necessary to normal blood formation. This lack may be due to several factors of which the three most important are:

1. Conditional lack due to temporary lack of intrinsic factor in the gastric secretion or to temporary failure of power of assimilation.

2. The lack of extrinsic factor or of vitamin B2 in the diet.

3. A predisposing iron deficiency anaemia believed by some writers always to be present before macrocytic anaemia can develop and due mainly to unsatisfactory diet.

I believe that all of these factors may have some bearing on the prevalence of this form of anaemia in the Gold Coast.

In the tropics, disease is much more rife than in temperate zones, and whereas in Britain one expects to find a patient suffering from one disease at a time the opposite is the case in warmer and more primitive countries. The sick man is probably afflicted with two, three or four different pathological conditions/
conditions, and as in any condition of ill-health the gastric secretion is seriously concerned, it is not unreasonable to presume lack of power of assimilation and perhaps temporary lack of intrinsic factor, although this has not as yet been worked out.

The diet of the people on the Gold Coast is demonstrably poor in vitamin B2, or extrinsic factor, so that even in health there may be little margin.

The amount and degree of anaemia present is demonstrated in Tables 3 and 4. WILLS (1939) stated that all anaemia under 90% of haemoglobin in pregnant women should be treated. If this is taken as the standard of normality, then the amount of anaemia on the Gold Coast is grave indeed.

Another factor which may have some bearing on the prevalence of macrocytic anaemia is the question of liver disease and its influence on the ability of the body to store or to utilize haemopoietic substance.

GOLDHAMER, ISAACS, and STURGES (1934) demonstrated that the liver stores haemopoietic substance, but that if diseased it fails to do so or is unable to release it.

CLARK (1936) reported that disease of the liver appears to be more frequent in Nigeria, where dietetic conditions are similar to those on the Gold Coast/
Coast, than it is at home. He held, as a result of a rat experiment that this is largely due to the poor-class carbohydrates, especially cassava and coco-yam, which are largely consumed. He considered these roots to be dangerous cyanogenic substances in the absence of a sufficient supply of good-class protein. Under some circumstances maize may act in the same way.

FAIRLEY (1938) found that malaria caused liver damage in Macedonia, and malaria is very prevalent on the Gold Coast.

It would appear, then, that the factors predisposing to macrocytic anaemia may be various but the most important are, I believe, the three mentioned on p. 44.

To what extent tropical macrocytic anaemia of pregnancy and macrocytic nutritional anaemia occurring in the non-pregnant are one and the same disease has not yet been finally agreed. BALFOUR (1927) reported cases which appeared to be identical except that pregnancy did not co-exist.

FAIRLEY (1938) suggested that the term "nutritional" rather than "tropical" macrocytic anaemia was a more suitable term, as he was reporting cases in Macedonia.

DAVIDSON/
DAVIDSON and FULLERTON (1939) in Dunlop, Davidson and MacNee’s "Text Book of Medical Treatment", refer to nutritional macrocytic anaemia and to macrocytic anaemia of pregnancy under different headings.

My own experience of anaemia among the non-pregnant is too limited to be of value, but I believe the problem to be essentially one of nutritional defect. From observation of the disease and especially from seeing the prompt relief which follows the administration of liver or liver extract, I do not consider it a disease of, but rather a disease associated with, pregnancy. Pregnancy is the most common and prolonged strain to which the young are subjected. Add an unsatisfactory diet to that, with the disorder of gastric secretion which is common in pregnancy, and the stage is set for breakdown in the blood forming system of the young person. Without pregnancy the breakdown would probably not occur or at least not occur until a more advanced age. The resemblance to toxic albuminuria is in some ways very close and it is of great importance not to confuse the two conditions. Both may have oedema and albuminuria, and even casts in the urine. In ADAIR and STIEGLITZ’S "Obstetric Medicine", 1934, Jones and Tocantins suggest/
suggest that this is probably of frequent occurrence and that cases of macrocytic anaemia may be returned as cases of "cardiorenal vascular disease".

SYNOPSIS OF CASES.

The hundred cases of macrocytic anaemia of which the reports are appended, p. 59, have been collected from the Maternity Hospital, Accra, from the time it was opened in 1928 until October 1938.

Thirty-eight of these were under the writer's own care, when from time to time she acted as medical officer in charge of the hospital, and the others were under various other medical officers. Follow-up of cases was carried out for the purposes of this paper as far as possible, but peculiar difficulties are met with when dealing with an illiterate population who may not give their correct names to the hospital authorities, and may not have any idea of their house address.

Age and Maturity.

Ages are not stated, as patients do not know them. Of the hundred cases sixty-four occurred in primiparae and only thirty-six in multiparae, so that multiparity would not appear to be an important factor in aetiology on the Gold Coast. Twenty-nine cases/
cases were in women of less than twenty-eight weeks maturity, so that it is not the case, in this series, that macrocytic anaemia is a disease of the last trimester only; it may occur with considerable severity at a much earlier date in pregnancy.

**Concurrent Infections.**

**Yaws.**

Positive or negative history of yaws is recorded in each case, but as it depends entirely on the patient's statement, it is not necessarily or even probably correct.

The Wassermann reaction was double plus in eleven of the fifty-three upon whom the test was carried out, and seven had a single plus or plus minus reaction, a result not differing greatly from the rate - 32% - for the population as a whole (Medical Research Institute, Accra).

**Malaria.**

Malaria parasites were present in twenty-nine.

**Urine**/
Urine.

Micro-organisms were found in nine of the twenty-five cases in which the urine was examined bacteriologically. Schistosome ova were present in two cases.

Faeces.

The faeces were examined in eighty-five cases. Ascaris ova were found in seventeen, hookworm ova in sixteen, entamoeba histolytica in two, trichuris ova in two and strongyloides larvae in two. Entamoeba coli was present in seven cases, and flagellates in fifteen. With regard to the latter, they are generally in evidence in patients who are debilitated, but there seems little reason to consider them as the cause of the enteritis which is so frequently present.

Other Findings.

Sickling of red cells was found to be present in two of the nine cases examined. The rate for the pregnant women as a whole is 13% (Medical Research)
Research Institute, Accra), so that the result does not appear to be out of the way.

The Van den Bergh reaction was only carried out in three cases, numbers 4, 6 and 10. The direct reaction was negative in all three and the indirect positive, which conforms with the general findings in this disease.

**Signs and Symptoms.**

All patients had a raised pulse rate and in eighty-two there was some degree of pyrexia. Cough was present in twenty-four, oedema in thirty-eight, some evidence of enteritis in twenty-three, and slight jaundice in six. Sore mouth was found in four cases. Albuminuria was present in fifty-four. Splenic enlargement was recorded in eleven cases.

In most of the cases anaemia was of considerable severity, with haemoglobin below 40% and with a red cell count below 2,000,000. In Table 1 parity, maturity, the red cell counts, haemoglobin percentages, and colour indices, are tabulated.

**Progress in Hospital.**

A case was considered to be in a critical condition if the haemoglobin was below 20%, but even then good response to treatment was the rule. The death rate of five is low compared to the figure for untreated cases in India.

Out of fifty-three deliveries in hospital the/
the prematurity rate was 64%. Still-births, and deaths within the first week of life, were at the rate of 40% and the morbidity rate for the puerperium was 34%, all rather formidable figures.

**Mortality Rate.**

There were five deaths. They are examined on p. 56.

Table 2 shows the results of more detailed blood examinations in twenty-five cases.

**COMMENT ON SPECIAL CASES.**

**Case 1.**

The first case of macrocytic anaemia to be treated at the Maternity Hospital, Accra, spent a very long time in hospital, and it is possible that liver therapy was not carried out sufficiently energetically. The poikilocytosis noted here is not as a rule a characteristic feature of these cases. Slight jaundice, although not common, does occur and is perhaps generally associated with malaria. Follow-through in the patient's next pregnancy is interesting in the fact that improvement was so much more rapid. By 1931 when she was admitted for the second time treatment was more systematised.

**Case 9.**

In this case toxemia appeared to be supervening on the anaemia, and it was deemed wiser to induce labour. Albuminuria and oedema are common findings in severe anaemia, but increasing oedema and a rising/
rising blood pressure while under liver treatment are danger signals.

**Case 10.**

This case is interesting because, although by local standards the anaemia was not very severe, the blood picture is typical. It gives some indication of how a less severe case of macrocytic anaemia may easily pass unnoticed among a population in which so much anaemia occurs as in the Gold Coast. Treatment did not result in marked, or indeed in any, improvement in the blood picture, although the patient surmounted labour successfully. In the light of later experience one would say that half a pound of raw liver was either not enough or was not being absorbed well. In other words, that the factor in this case was probably failure of gastric secretion or of powers of absorption, and not lack of extrinsic factor.

**Case 14.**

This patient was the only one in the series suspected of being a case of infective endocarditis. General improvement under liver therapy was rapid once it was started, but it was only the complete absence of all cardiac signs and symptoms when the patient was re-admitted for labour that finally ruled endocarditis out of court.

**Case 18.**

This patient in her first pregnancy was tided over a very severe attack of anaemia in hospital but/
but I received news of her death from anaemia in a subsequent untreated pregnancy two years later. On admission in the first pregnancy she was in labour with a red cell count of 890,000 and haemoglobin 12%. She was given a small blood transfusion and was safely delivered of a still-born infant the same day. From the history available for the following pregnancy the anaemia, which was untreated, was very rapidly progressive to a fatal issue.

**Case 25.**

In this case a severe secondary anaemia was unmasked under liver therapy. It is held in some quarters that there is always hypochromic anaemia as a predisposing factor in cases of macrocytic anaemia.

**Case 43.**

In the light of later experience one believes that this case suffered from inadequate treatment. Parenteral injection of liver extract would have been more satisfactory, but the extract was not available for use in the hospital until a slightly later date.

**Case 47 - 28.8.35.**

The first case to be treated by Campolon, and with gratifying results.

**Case 53.**

This case was at first treated as an ordinary attack of malaria and it was only after one week/
week in hospital that severe anaemia was observed, with a haemoglobin of 38% (Sahli), dropped from 55% (Talquist) on admission. As a general rule one finds that the Talquist reading is 5% greater than the Sahli. The Talquist method has the advantage of extreme simplicity and can be used by a nurse, but it is unreliable where the anaemia is severe, and is at no time very exact.

Case 60.

Although Campolon injections were started two days after admission in this case, there was deterioration in the general condition the following week. This is an unusual occurrence, but again on examination of the treatment chart it is believed that the dosage of Campolon was inadequate. It was given on alternate days and only 2 cc. at a time. In some refractory cases it is necessary to give Campolon daily and in 4 cc. doses at least. WILLS (1939) mentions 5-10 cc. daily.

Case 97.

Although anaemic on admission this patient was first treated as a case of albuminuria. After labour a more severe anaemia developed rapidly, but responded quickly to specific therapy. This was not a true case of late development in the puerperium, for before delivery the haemoglobin was only 35% (Talquist)/
(Talquist), but rather one of late diagnosis. And, as so often happens, the puerperium was pyrexial.

Case 98.

This case also was only diagnosed in the puerperium, but undoubtedly anaemia was present before delivery. In such cases, if not too severe, improvement may set in immediately after delivery. Otherwise, the anaemia tends rapidly to advance.

FATAL CASES.

There were five fatal cases.

Case 3.

On admission this woman had 680,000 red cells and 10% haemoglobin. Two days after admission she had a very sudden precipitate labour. There was practically no loss of blood, but air hunger set in and the patient died very shortly after delivery.

Case 15.

This case was brought in to hospital in extremis and died within a few hours. She had a haemoglobin of 14%.

Case 23.

This patient died five days after admission to hospital. She could not take liver, and the use of Campolon had not yet been started.

Case 44.

This case was admitted in extremis, after a premature labour. She had been successfully treated in/
in her first pregnancy for a severe attack of anaemia, but did not come to hospital in the next pregnancy until brought in by a midwife after delivery.

Case 76.

This patient was in hospital for a week before labour set in, and as a rule seven to ten days is sufficient to tide a case over labour. On this occasion, however, signs of cardiac distress developed early, and the patient died of heart failure in the third stage. The infant, weighing 5 lb. 2 oz., was still-born.

SUBSEQUENT PREGNANCIES.

Ten cases were traced through subsequent pregnancies, viz. Nos. 1, 4, 5, 6, 10, 18, 19, 44, 45, 49, of which six underlined were again treated in hospital. The last is the only one of the ten in which anaemia in a severe form did not recur. Of the other nine cases severe anaemia recurred in eight, and in one, case 19, although the subsequent pregnancy was apparently normal, the patient was re-admitted with severe anaemia in the third.

In the cases of recurrence no special points were made out as to severity, etc., but the result of blood examination of the six cases treated again, viz. Nos. 1, 4, 5, 6, 19 and 45 are to be found in Table 1.

SUMMARY/
SUMMARISED CASE RECORDS OF 100 IN-PATIENTS SUFFERING FROM MACROCYTIC ANAEMIA.
SUMMARY OF WRITER'S CONCLUSIONS.

Macrocytic anaemia of a severe kind is not uncommonly met with in practice among pregnant women on the Gold Coast.

In both treated and untreated cases there is marked tendency to premature labour with stillbirth or death of the infant in the first week of life. The maternal mortality rate also is undoubtedly high in untreated cases. After delivery recovery may be rapid if the anaemia is not too far advanced. If it is severe, then death rapidly supervenes.

Macrocytic anaemia is a disease that responds rapidly and satisfactorily to treatment with liver or liver extract, preferably administered parenterally and in adequate doses.

The more important predisposing factors are unsatisfactory diet, intercurrent disease, especially malaria, and hypochromic anaemia.

From her own observations the writer has found some degree of anaemia to be very common among pregnant women. See Tables 3 and 4, pages 208 & 212.

Prevention of the condition is bound up with improvement in the general standard of living of the people. In the meantime the obstetrician must be constantly on the alert to detect and treat all patients with low haemoglobin values, in the expectation that by so doing severe anaemia may be successfully prevented from appearing.
CASE 1. (18).

ADMITTED: 20.7.28.

Primipara, 32 weeks maturity. Complaining of breathlessness and swelling of the legs. History of yaws positive.

SIGNS AND SYMPTOMS.

Anaemia. Oedema of legs and slight jaundice. Heart enlarged, with systolic murmurs. Much cough with a lot of sputum. Scattered râles in chest. Temperature 99.4; pulse 104; respirations 34.

Urine.

Albumin present. Gram positive cocci and diplococci present.

Faeces.

Occasional flagellates present, no ova.

BLOOD EXAMINATION.

Red cells: 400,000
White cells: 7,000
Haemoglobin: 20%
Colour Index: 2.5.
Irregularity in size and shape of red cells.
Malaria parasites: negative.
Wassermann: double plus.

TREATMENT.

Rest in bed, light diet including eggs and raw liver. Weekly injections of N.A.B. and a course of injections of iron and arsenic; also iron by mouth.

PROGRESS.

Patient ran an irregular temperature for three weeks, which gradually settled down, and at the same time the oedema and cough improved.

Confinement/
CASE 1. (Continued).

Confinement.

Normal delivery three months after admission to hospital.

2.11.28.

Full term living child, weight 6 lbs. 14 ozs.

Puerperium.

Non-morbid.

Blood examination after ten weeks in hospital.

Red cells: 2,800,000.
Haemoglobin: 55%
Colour Index: 1

Urine.

After delivery the albumin disappeared.

DISCHARGED: 22.11.28.

AFTER HISTORY.

READMITTED: 27.8.31.

Readmitted in the next pregnancy. (458).
32 weeks pregnant, suffering from anaemia.

SIGNS AND SYMPTOMS.

Anaemic.

Urine.

Trace of albumin.

Faeces.

No ova.

BLOOD EXAMINATION/
CASE 1. (Continued)

BLOOD EXAMINATION.

Red cells: 1,000,000.
White cells: 4,150
Haemoglobin: 27%
Colour Index: 1.3.
Irregularity in size and shape of red cells.
Polychromatophilia. Punctate basophilia.
Macrocytes present.
Malaria parasites: negative.

TREATMENT.

Raw liver and marmite by mouth; iron and arsenic injections.

PROGRESS.

Marked improvement during two weeks in hospital.

4.9.31:

Haemoglobin: 50%.
Still a trace of albumin in the urine on discharge.

DISCHARGED: 12.9.31.

AFTER HISTORY.

She delivered as an out-patient; was seen afterwards looking well.

COMMENTS.

This was the first acute anaemia treated in hospital, which may account for the very long stay. The case is of interest as it was followed through another pregnancy in which anaemia recurred.
CASE 2. (150).

ADMITTED: 27.2.29.

Primipara; 22 weeks maturity. Complaining of cough for two weeks and swelling of feet for one.

SIGNS AND SYMPTOMS.


Urine.

Trace of albumin present.

Faeces.

Strongyloides larvae and flagellates present.

BLOOD EXAMINATION.

Red cells: 1,750,000
White cells: 7,500
Haemoglobin: 35%
Colour Index: 1
Irregularity in size and shape of red cells. Polychromatophilia.
Malaria parasites: plus.

TREATMENT.

½ lb. of raw liver by mouth daily. Cod liver oil and iron by mouth.

PROGRESS.

Temperature 99.4; was slightly irregular for first week in hospital. Patient much improved after one month in hospital.
CASE 2. (Continued)

Urine.

No albumin after the first ten days.

Blood examination after three weeks in hospital.

Red cells: 2,800,000
White cells: 7,500
Haemoglobin: 48%
Colour Index: .85.

DISCHARGED: 30.3.29.

AFTER HISTORY: 14.4.29.

Re-admitted in labour. Still anaemic though much better.

Premature delivery of living infant which died next day.

Puerperium.

Temperature for one day, thereafter normal: patient discharged 9 days after delivery.

BLOOD EXAMINATION.

At the time of delivery malaria parasites were present in the peripheral blood and in the placenta.
CASE 3. (201).

ADMITTED: 14.4.29.

Primipara; 36 weeks pregnant. Complaining of breathlessness.

SIGNS AND SYMPTOMS.


Urine.

Trace of albumin.

BLOOD EXAMINATION.

Red cells: 680,000
Leucocytes: 12,000
Haemoglobin: 10%
Colour Index: .8
Irregularity in size and shape of red cells. Nucleated red cells present.

TREATMENT.

½ lb. of raw liver by mouth. Salines per rectum.

PROGRESS.

Patient had a sudden precipitate labour two days after admission. Exhaustion and air hunger developed rapidly and the patient died four hours after delivery. The infant was still-born.

Blood examination.

Showed the reversal of numbers of polymorphs and lymphocytes mentioned in literature but rarely seen in this series.
POST MORTEM REPORT.

Liver.

Round celled infiltration of portal canal; columns of hepatic cells are not broken, but cells are shrunken and intervening blood capillaries are dilated. There is considerable fatty infiltration of hepatic cells nearest to the central veins. The Kupfer cells are very conspicuous; they seem to be enlarged and project into the capillaries. They contain much black pigment.

Spleen.

The pulp appears to contain an excess of red blood corpuscles. There are numerous tissue cells containing pigment scattered throughout the organ.

Kidney.

No marked changes.

Small Intestine.

Shows loss of epithelium except at the bottom of the crypts; no inflammatory changes.
CASE 4. (402)

ADMITTED: 8.11.29.

Primipara about 32 weeks pregnant. Complaining of dizziness and swelling of face.

Yaws.

Positive history.

SIGNS AND SYMPTOMS.

Oedema of legs and face. Temperature 99.4; pulse 120; respirations 32.

Urine.

Albumin present; deposit shows a few leucocytes.

Faeces.

Stools frequent, not examined.

BLOOD EXAMINATION.

Red cells: 1,300,000
Haemoglobin: 30%
Colour Index: 1.1
Nucleated red cells present.
No malaria parasites.

TREATMENT.

Raw liver ½ lb. daily.

PROGRESS.

Went into labour two days after admission.

Confinement.

Delivered of a still-born child weighing 5 lb. 10 ozs. on 12.11.29.

Puerperium.

Morbid. Total stay in hospital, 1 month.

DISCHARGED: 5.12.29.

Condition/
CASE 4. (Continued).

Condition:

Very much improved, anaemia less.

AFTER HISTORY.

Readmitted in July for evacuation of abscess of groin.

READMITTED: 8.5.31 (84).

In next pregnancy, 26-28 weeks maturity. Com- plaining of swelling of feet and diarrhoea of two months duration.

SIGNS AND SYMPTOMS.

Anaemia, swelling of feet. Temperature 98.6; pulse 80; respirations 84.

Haemic murmurs heard over heart, spleen enlarged to umbilicus, liver not palpable.

Urine.

A trace of albumin present and a few leucocytes. Sterile on culture.

Faeces.

Hookworm ova present.

BLOOD EXAMINATION.

Red cells: 1,450,000
Haemoglobin: 30%
Colour Index: 1
Irregularity in size and shape of red cells. Polychromatophilia; punctate basophilia; macrocytes present; nucleated red cells present.
Malaria parasites: negative.
Wassermann: plus.

TREATMENT/
CASE 4. (Continued)

TREATMENT.

Liver, $\frac{1}{2}$ lb. raw by mouth daily. Iron and arsenic injections; Carbon Tetrachloride by mouth and, later, N.A.B. injections.

PROGRESS.

Patient was treated in hospital for one month and was improving on discharge. Ran some temperature for the first ten days, as high as 101°F.

Blood examination: 28.5.31.

Red cells: 2,160,000
Haemoglobin: 52%
Colour Index: 1.2.

DISCHARGED: 4.6.31.

AFTER HISTORY.

Attended Out-Patients for treatment with N.A.B.

READMITTED: 18.7.31 in labour.

Confinement.

Delivered of twins prematurely, 19.7.31. First macerated, second weighing 4 lbs., living, but died in four days.

Puerperium.

Temperature raised once after delivery to 100°F.

DISCHARGED: 28.7.31 after a stay of ten days.
CASE 5.  (456)

ADMITTED:  30.12.29.

Primipara of 38 weeks maturity, complaining of cough and lower abdominal pain.

Yaws:

Negative history.

SIGNS AND SYMPTOMS.

Anaemia, with haemic murmurs over the heart; no temperature; pulse 116; respirations 24.

Urine:

No albumin. Heavy deposit of squamous epithelial cells.

Faeces:

No ova found.

BLOOD EXAMINATIONS.

Red cells: 825,000
White cells: 2,500
Haemoglobin: 31%
Colour Index: 1.8
Irregularity in size and shape of red cells. Polychromatophilia; nucleated red cells present.
Malaria parasites present.
Wassermann: negative.

TREATMENT.

Raw liver ½ lb. daily, and quinine. Small injections of cord blood given intramuscularly twice.

PROGRESS.

Ran a slight temperature up to 99.2° for first few days in hospital. Improved steadily.

Blood examination/
CASE 5. (Continued)

**Blood examination:** 9.1.30.
- Red cells: 2,185,000
- Haemoglobin: 48%
- Colour Index: 1.1

**Blood examination:** 2.2.30.
- Red cells: 3,700,000
- Haemoglobin: 55%
- Colour Index: 74

**Confinement:**
Delivered normally 14.2.30 of living female child weighing 5 lbs. 14 ozs.

**Puerperium:**
Non-morbid.

**DISCHARGED:** 24.2.30 after eight weeks in hospital.

**READMITTED:** 3.10.32 (668).
In following pregnancy, 34 weeks maturity, complaining of pains in stomach.

**SIGNS AND SYMPTOMS.**
- Anaemia; no temperature.
- Urine:
  - No albumin.
- Faeces:
  - Ascaris ova present.

**BLOOD EXAMINATION.**
- Red cells: 1,550,000
- Haemoglobin: 27%
- Colour Index: 1.9
- Malaria parasites absent.
- Wassermann: plus.

**TREATMENT.**
CASE 5. (Continued)

TREATMENT.

Raw liver 6 ozs. daily; iron and arsenic injections. Santonin.

PROGRESS.

Was an in-patient for three weeks; much improved on discharge.

DISCHARGED: 24.10.34.

BLOOD EXAMINATION.

Red cells: 3,900,000
White cells: 6,600
Haemoglobin: 60%
Colour Index: .73.
CASE 6. (288).

ADMITTED: 7.9.30.

Para 1. 34-35 weeks maturity, complaining of shortness of breath and swelling of face, hands, and feet.

Yaws.

Positive history.

SIGNS AND SYMPTOMS.

Very anaemic; oedema of face, hands and legs. Temperature 98.8; pulse 124; respirations 40. Heart, systolic murmur at apex.

Urine.

Pus and granular casts present.

Faeces.

No ova found.

BLOOD EXAMINATION.

Red cells: 700,000
White cells: 9,400
Haemoglobin: 17%
Colour Index: 1.2
Irregularity in size of red cells; slight irregularity in shape; punctate basophilia; polychromatophilia; nucleated red cells present.
Malaria parasites: negative.
Wassermann: double plus.

TREATMENT.

Liver ½ lb. daily. Iron and arsenic injections.

PROGRESS/
CASE 6. (Continued)

PROGRESS.

Ran a temperature as high as 100° F., which gradually settled in ten days as general condition improved.

Blood examination: 8.10.30.

Red cells: 1,640,000
Haemoglobin: 42%
Colour Index: 1.2.

Confinement: 19.11.30.

Delivered of a full-time living female child weighing 7 lb. 4 ozs. Had some post-partum haemorrhage.

Puerperium.

Non-morbid.


General condition satisfactory.

READMITTED: 3.8.33. (360).

In following pregnancy, 28 weeks maturity, complaining of fever, giddiness and palpitation.

SIGNS AND SYMPTOMS.

Very anaemic; temperature 98.6; pulse 84; respirations 22.

Urine.

Pus and bacilli present. On culture B. Coli present.

BLOOD EXAMINATION/
CASE 6. (Continued)

BLOOD EXAMINATION.

Red cells: 1,000,000
Malaria parasites: negative
Wassermann: double plus.

TREATMENT.

Raw liver by mouth; marmite; N.A.B. and iron and arsenic injections. Had one small blood transfusion.

PROGRESS.

Satisfactory.

Blood examination: 17.8.33.
Red cells: 2,250,000.
Haemoglobin: 44%
Colour Index: 1.

Blood examination: 5.9.33.
Red cells: 2,850,000
Haemoglobin: 56%
Colour Index: 1.

Urine.

Clear on discharge.

DISCHARGED: 11.9.33.

Condition satisfactory.
CASE 7. (653).

ADMITTED: 18.3.31.

Pregnant. Had one previous miscarriage. Complaining of cough.

Yaws.

Positive history.

SIGNS AND SYMPTOMS.

Anaemia and cough. Temperature 98.4; pulse 106; respirations 24.

Urine.

Albumin present. Some leucocytes. Urobilin present.

Faeces.

Liquid, containing non-cellular mucus. A few amoebae present, probably E. Coli.

BLOOD EXAMINATION.

Red cells: 750,000
White cells: 12,400
Haemoglobin: 17%
Colour Index: 1.2
Irregularity in size of red cells well marked; slight irregularity in shape; some poly-chromatophilia; nucleated red cells present.
Malaria: negative.
Wassermann: double plus.

TREATMENT.

Raw liver, ½ lb. daily; iron and arsenic by mouth.

PROGRESS/
CASE 7. (Continued).

PROGRESS.

Ran temperature, up to 99.6, which gradually settled within five days. Pulse rate decreased as general condition improved.

**Blood examination:** 27.3.31.

- Red cells: 1,660,000
- White cells: 8,000
- Haemoglobin: 39%
- Colour Index: 1.2

**DISCHARGED:** 31.3.31.

Two weeks in hospital.

Progressing satisfactorily; not seen again.
Case 8. (5).

Admitted: 2.4.31.

Para 7; 30 weeks maturity. History of difficult labours.

Yaws.

Positive history.

Signs and Symptoms.

Patient anaemic; temperature 98.4; pulse 84; respirations 24.

Urine.

Clear.

Faeces.

Hookworm ova present, E. Coli and flagellates.

Blood Examination.

Red cells: 1,440,000
White cells: 10,500
Haemoglobin: 34%
Colour Index: 1.2
Irregularity in size of red cells; polychromatophilia; nucleated red cells present.
Malaria parasites: negative.
Wassermann: double plus.

Treatment.


Progress.

Patient improved during two weeks in hospital.

Blood examination: 15.4.31

Red cells: 1,900,000
White cells: 7,200
Haemoglobin: 47%
Colour Index: 1.2

Discharged: 18.4.31.

Two weeks in hospital. Improving.
CASE 9. (34).

ADMITTED: 16.4.31.

Primipara. 34 weeks pregnant; not feeling well.

Yaws.

Negative history.

SIGNS AND SYMPTOMS.

Patient anaemic. Temperature 99.8; pulse 120; respirations 32.

Urine.

Clear on admission. Developed albumin 2 days later. Leucocytes and granular casts present.

Faeces.

Liquid. Non-cellular mucus present; no ova nor amoebae seen.

BLOOD EXAMINATION.

Red cells: 900,000
White cells: 9,000
Haemoglobin: 22%
Colour Index: 1.2
Well marked irregularity in size of red cells; polychromatophilia and punctate basophilia; macrocytes present; nucleated red cells present.
Malaria: no parasites.
Wassermann: double plus.

TREATMENT.

½ lb. raw liver; Pot. Iod. Injections of iron and arsenic.

PROGRESS/
CASE 9. (Continued)

PROGRESS.

Patient developed albuminuria which increased in amount and some oedema appeared.

25.4.31.

Blood pressure.
Systolic, 134 mm.
Diastolic, 70 mm.

25.4.31.

Surgical induction of labour carried out by insertion of bougies.

Confinement.

Premature delivery on 1.5.31 of living infant, 4 lbs. in weight.

Puerperium.

Morbid. Morbid for ten days after delivery.

Blood examination: 29.4.31.

Red cells: 1,740,000
Haemoglobin: 37%
Colour Index: 1.

DISCHARGED: 13.5.31.

One month in hospital.

Condition satisfactory. Infant alive.
CASE 10. (83).

ADMITTED: 8.5.31.
Para 3. Not well.

Yaws.
History negative.

SIGNS AND SYMPTOMS.
Anaemia. Temperature 98.2; pulse 80; respirations 24.

Urine.

Faeces.
E. Coli present. No ova.

BLOOD EXAMINATION.
Red cells: 2,080,000
Haemoglobin: 56%
Colour Index: 1.4
Irregularity in size of red cells; polychromatophilia; macrocytes and nucleated red cells present.
Leucocytes: 7,350
Malaria parasites: negative.
Wassermann: Plus.

TREATMENT.
Liver and iron by mouth.

PROGRESS.
Satisfactory.

15.5.31/
CASE 10. (Continued)

15.5.31.

Confinement. Normal delivery; living child, birth weight 5 lb. 13 ozs.

Puerperium.

Non-morbid.

22.5.31.

Red cells: 3,200,000
Haemoglobin: 49%
Colour Index: .7

DISCHARGED: 22.5.31.
Two weeks in hospital.
Still anaemic. Haemoglobin 49%.

AFTER HISTORY: 7.11.32.
Again pregnant and very anaemic.
CASE 11. (173).

**ADMITTED:** 15.5.31.

Para 3; 36 weeks maturity. Complaining of vaginal discharge and weakness.

**Yaws.**

History negative.

**SIGNS AND SYMPTOMS.**

Patient very anaemic. Temperature 99.4; pulse 104; respirations 24. Spleen enlarged; external haemorrhoids present; slight cervicitis present; no evidence of nervous involvement.

**Urine.**

No albumin.

**Faeces.**

No ova; much of muco-pus present. Abundant rather disintegrated pus cells and a few red cells. No parasites seen.

**BLOOD EXAMINATION.**

Red cells: 710,000
White cells: 6,000
Haemoglobin: 23%
Colour Index: 1.6
Irregularity in size of red cells well marked; polychromatophilia; macrocytes present; nucleated red cells present.
Malaria parasites: negative.
Wassermann: negative.

**TREATMENT.**

Raw liver by mouth.

**PROGRESS.**

Improved rapidly.

Blood examination/
CASE 11. (Continued)

   Red cells: 1,040,000
   Haemoglobin: 21%
   Colour Index: 1

11.6.31.
   Premature labour.
   Confinement.
       Living premature female child, 3½ lbs.
       delivered 11.6.31.

Puerperium.
   Temperature rose to 100.8 once.

DISCHARGED: 19.6.31.
   Five weeks in hospital.
   Discharged to general hospital; condition improving.
ADMITTED: 6.7.31.

Primipara; 24-26 weeks maturity. Some swelling of legs.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Anaemia, and some swelling of legs; temperature 98.4; pulse 80; respirations 22.

Urine.

Clear.

Faeces.

Flagellates present.

BLOOD EXAMINATION.

Red cells: 1,220,000
White cells: 4,700
Haemoglobin: 23%
Colour Index: .95
Slight irregularity in size of red cells; macrocytes present; nucleated red cells present.
Malaria: negative.
Wassermann: double plus.

TREATMENT.

Liver, iron and arsenic by mouth; Pot. Iod. and Hydrarg. Perchlor.

PROGRESS.

Patient improved; ran some temperature at first, up to 100, which settled gradually. Oedema improved.

DISCHARGED: 19.7.31.

After two weeks in hospital.
CASE 13. (679).

ADMITTED: 24.11.31.

Para 2. With cough and anaemia.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Anaemia and cough. Temperature 99.8; pulse 88; respirations 22.

Urine.

No albumin. Epithelial cells and leucocytes present.

Faeces.

No abnormality detected.

Sputum.

No tubercle bacilli.

BLOOD EXAMINATION.

Red cells: 1,600,000
White cells: 5,200
Haemoglobin: 30%
Colour Index: .95
Some irregularity in size of red cells; nucleated red cells present.
Malaria parasites: negative.
Wassermann: negative.

TREATMENT.

Raw liver ½ lb. daily. Marmite 1 dr. t.i.d.
Iron and arsenic injections.

PROGRESS.

Improved.

DISCHARGED: 3.12.31.

After ten days in hospital.

ADMITTED: 23.11.31.

Primipara; 24 weeks maturity. Suffering from anaemia.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Temperature 101.6; pulse 120; respirations 40. Haemic murmurs over heart especially at base. A troublesome cough. Liver and spleen enlarged.

Sputum.

No tubercle bacilli found.

Urine.

No albumin. A few epithelial cells and leucocytes.

Faeces.

Nothing abnormal discovered.

BLOOD EXAMINATION.

On admission.

Blood culture, sterile after three days.

One week after admission: 2.12.31.

Red cells: 1,000,000
White cells: 6,900
Haemoglobin: 23%
Colour Index: 1.15
Irregularity in size of red cells marked; slight irregularity in shape of red cells; polychromatophilia; nucleated red cells present.
Malaria parasites: negative.
Wassermann: negative.
Widal: negative.

TREATMENT/
CASE 14. (Continued)

TREATMENT.

For ten days treated with quinine and cough mixture.

4.12.31.

Started liver, ½ lb. daily, marmite, iron and arsenic by mouth.

PROGRESS.

Improved steadily once liver treatment begun.

DISCHARGED: 27.12.31.

After five weeks in hospital, condition much improved.

READMITTED: 7.1.32

For labour. Heart now found to be entirely clear of murmurs.

Confinement.


Puerperium.

Non-morbid.

COMMENT.

This patient was considered as a possible septic endocarditis on first admission. The fact that on readmission the heart appeared to be perfectly healthy ruled that finally out of court. Twenty-four weeks maturity is an early stage at which to find so severe an anaemia.
CASE 15. (88).

ADMITTED: 25.4.32.

Primipara; complaining of abdominal pain and swelling of body.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Sick patient, very anaemic, with generalised oedema. Temperature 99; pulse 112; respirations 42.

Urine.

Albumin plus. Many epithelial and granular casts present.

BLOOD EXAMINATION.

Haemoglobin: 14%

Irregularity in size of red cells; well-marked nucleated red cells present.

Patient died within twenty-four hours of admission.
CASE 16. (669).

ADMITTED: 3.10.32.

Primipara; 28 weeks maturity. Suffering from anaemia.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Anaemic patient. Temperature 99.6; pulse 84; respirations 22.

Urine.

Clear.

Faeces.

Ankylostome ova present.

BLOOD EXAMINATION.

Red cells: 1,100,000
White cells: 5,600
Haemoglobin: 20%
Colour Index: .9
Malaria parasites: negative.
Wassermann: negative.

TREATMENT.

Raw liver, iron and arsenic by mouth, oil of Chenopodium.

PROGRESS.

Improved.

DISCHARGED: 15.10.32.

Insisted on leaving hospital after twelve days.
CASE 17. (1228).

ADMITTED: 16.2.33.

Primipara; 26 weeks maturity. Complaining of swelling of feet.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Anaemia. Oedema of feet. Temperature 100; pulse 112; respirations 40.

Urine.

Albumin plus. Leucocytes and granular casts present. No ova seen.

Faeces.

Offensive specimen. E. Coli and flagellates present.

BLOOD EXAMINATION.

Red cells: 1,420,000
Haemoglobin: 28%
Colour Index: 1
Irregularity in size of red cells well marked; nucleated red cells present.
Malaria parasites: negative.
Wassermann: negative.

TREATMENT.

Liver by mouth; quinine by mouth. Iron and arsenic injections.

PROGRESS.

Satisfactory.

22.2.33. Haemoglobin: 36%
1.3.33. Haemoglobin: 42%

Urine clear on discharge.

DISCHARGED: 4.3.33.

Two weeks in hospital.
CASE 18. (1277).

ADMITTED: 27.2.33.

Primipara; 36 weeks maturity. Very ill and in labour.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Very severe anaemia, and in labour; some general oedema. Temperature 98.8; pulse 128; respirations 28.

Urine.

Albumin, a trace. A few hyaline and granular casts present.

Faeces.

Nothing abnormal discovered.

BLOOD EXAMINATION.

Red cells: 890,000.
Haemoglobin: 12%
Colour Index: .75
Malaria parasites: negative.
Wassermann: negative.

TREATMENT.

Blood transfusion. 250 c.c. of citrated blood on day of admission. Raw liver ½ lb. daily. Iron and arsenic injections.

PROGRESS.

Confinement on day of admission; infant still-born, weighing 5 lb. 2 ozs.

Puerperium.

Morbid. Anaemic condition improved steadily.

4.3.33. Urine: trace of albumin, still a few casts.
Blood: Haemoglobin 30%.

DISCHARGED: 13.3.33. Greatly improved.

AFTER HISTORY.

Two years later received word that patient had died in Kumasi of heart failure and anaemia in following pregnancy.
CASE 19. (102).

ADMITTED: 27.4.33.

Primipara. Admitted in the third stage of labour, having delivered premature living infant, weight 4 lb. 8 ooz., on the way to hospital.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

On admission patient very anaemic; no haemorrhage; temperature 99.4; pulse 128; respirations 32.

Urine.

Albumin present. Catheter specimen crowded with cocci and bacilli. A few leucocytes present.

BLOOD EXAMINATION.

Red cells: 97,000
Haemoglobin: 15%
Colour Index: .8.

TREATMENT.

Liver and marmite by mouth, iron and arsenic injections.

PROGRESS.

Puerperium.

Morbid. Patient suffered from B. Coli septicaemia. High temperature for 3 weeks, thereafter temperature fell to normal and patient made satisfactory recovery.

12.6.33/
CASE 19. (Continued)

12.6.33.

Red cells: 3,180,000
Haemoglobin 58%
Colour Index .9

DISCHARGED: 14.6.33.

Improving, after 7 weeks in hospital.

READMITTED: 1.2.37. (117).

Para 2. 30 weeks maturity. Admitted with fever, general weakness and swelling of feet. Seen in Out-Patients 3 weeks before; no anaemia noted.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Anaemia. Temperature 101.2; pulse 136; respirations 32.

Urine.

High colour; trace of albumin; a few leucocytes and epithelial cells. Sterile on culture.

Faeces.

Nothing abnormal detected. Stools frequent.

BLOOD EXAMINATION.

Red cells: 2,136,000
Haemoglobin: 42%
Colour Index: 1
Malaria parasites: negative.
Wassermann: negative.

TREATMENT/
TREATMENT.

Liver, ½ lb. daily; iron and arsenic injections; Campolon injections started 14.2.37.

PROGRESS.

Ran some temperature, but not over 99, except on day of admission.

9.2.37.

Red cells: 1,712,000
Haemoglobin: 33%
Colour Index: 1

12.2.37.

Haemoglobin 35%. Campolon injections started.

20.2.37.

Red cells: 2,104,000.
Haemoglobin: 53%
Colour Index: 1.2.

DISCHARGED.

Discharged after 3 weeks in hospital. Condition improving.

READMITTED: 7.4.37.

With cough and loose stools. Premature delivery of twins, living, and both weighing 4 lb. 8 ozs.

Puerperium.

Morbid. Patient suffered from an attack of Flexner dysentery while in hospital.
CASE 19. (Continued)

BLOOD EXAMINATION.

On admission.

Red cells: 2,520,000
Haemoglobin: 50%.
Colour Index: 1.

26.4.37.

Red cells: 3,240,000
Haemoglobin: 70%.
Colour Index: 1.

DISCHARGED: 26.4.37.

Nearly three weeks in hospital.
CASE 20. (144)

ADMITTED: 8.5.53.
Primipara; 32 weeks maturity. Admitted with swelling of feet and fever.

Yaws.
History positive.

SIGNS AND SYMPTOMS.
Anaemia and oedema of feet. Temperature 100.4; pulse 116; respirations 38.

Urine.
High coloured; a few pus cells present. Culture, sterile.

BLOOD EXAMINATION.

Red cells: 1,080,000
Haemoglobin: 25%
Colour Index: 1.2
Malaria parasites: negative.
Wassermann: negative.

TREATMENT.
Liver and marmite by mouth. Iron and arsenic injections.

PROGRESS.

Confinement.
Premature delivery 3 days after admission. Living infant, weight 5 lb. 6 ozs. which died in half an hour.

Puerperium.
Morbid.

Blood examination: 5.6.33.
Red cells: 2,200,000
Haemoglobin: 40%
Colour Index: .9.

DISCHARGED after one month - progressing satisfactorily.
CASE 21. (143)

ADMITTED: 8.5.33.

Primipara; 34 weeks maturity. Not well.

Yaws.

History negative.

SIGN AND SYMPTOMS.

Anaemia. Temperature 98.8; pulse 84.

Urine.

Clear.

BLOOD EXAMINATION.

Red cells: 2,320,000
Haemoglobin: 45%
Colour Index: .97
Malaria parasites: negative.

TREATMENT.

Raw liver by mouth. Iron and arsenic by mouth and by injection.

PROGRESS.

Improved.

DISCHARGED: 13.5.33.

Five days in hospital.
CASE 22. (167).

ADMITTED: 15.5.33.

Primipara; 32 weeks pregnant. Admitted with anaemia.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Very anaemic. Temperature 101.4; pulse 140.

Urine.

Clear.

BLOOD EXAMINATION.

Red cells: 790,000
Haemoglobin: 17%
Colour Index: 1.2
Malaria parasites: plus.
Wassermann: plus.

TREATMENT.

Raw liver, marmite and quinine by mouth; iron and arsenic injections.

PROGRESS.

Satisfactory.

Blood examination: 2.6.33.

Red cells: 3,060,000
Haemoglobin: 40%
Colour Index: .66

DISCHARGED: 8.6.33.

Three weeks in hospital.
CASE 23. (185).

ADMITTED: 25.5.33.

Para 2. 36 weeks maturity. Complaining of palpitation. Temperature 100; pulse 96; respirations 24.

Yaws.

History positive.

SIGNS AND SYMPTOMS.

Patient very anaemic. Liver and spleen enlarged.

Urine.

Clear.

BLOOD EXAMINATION.

Red cells: 960,000
Haemoglobin: 18%.
Colour Index: 1
Malaria parasites: negative.

TREATMENT.

Liver and marmite by mouth. Iron and arsenic injections.

PROGRESS.

Could not take liver, and died five days later, before blood transfusion could be arranged.

POST MORTEM.

Profound anaemia of all organs was present, with enlargement of liver and spleen.
CASE 24. (187).

ADMITTED: 25.5.33 -

Para 1; 24 weeks maturity. Admitted with fever.

Yaws.

History positive.

SIGNS AND SYMPTOMS.

Patient anaemic and thin. Temperature 101.2; pulse 128; respirations 34.

Urine.

Clear.

BLOOD EXAMINATION.

Red cells: 1,260,000
Haemoglobin: 19%
Colour Index: .86
Malaria parasites: negative.
Wassermann: plus.

TREATMENT.

Liver, marmite by mouth; full diet; iron and arsenic by the mouth and by injection.

PROGRESS.

Some temperature for over a week, which gradually settled.

DISCHARGED: 19.6.39.

Three weeks in hospital, much improved.
CASE 25. (256).

ADMITTED: 22.6.33

Primipara; 26 weeks maturity. Not well.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Anaemia; temperature 98.6; pulse 100; respirations 28.

Urine.

Trace of albumin.

Faeces.

Not examined for parasites. Stools rather frequent.

BLOOD EXAMINATION.

Red cells: 2,200,000.
Haemoglobin: 38%.
Colour Index: .86
Malaria parasites: negative. Microfilaria: present.
Wassermann: negative.

TREATMENT.

Liver, marmite, iron and arsenic by mouth.

PROGRESS.

No temperature after admission, for one week, then sharp rise, which subsided after quinine injection intramuscularly.

7.7.33:

Red cells: 3,720,000.
Haemoglobin: 42%.
Colour Index: .56

DISCHARGED: 10.7.33

Much improved.
CASE 26. (336).

ADMITTED: 27.7.33.  
Primi para; 33 weeks maturity. Admitted with fever.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Anaemia. Temperature 98.6; pulse 84; respirations 24.

Urine.

Albumin: plus.

Blood pressure: 126 mm.

BLOOD EXAMINATION.

Red cells: 1,309,000.
Haemoglobin: 28%.
Colour Index: 1.
Malaria parasites: negative.
Wassermann: negative.

TREATMENT.

Raw liver, marmite, and iron by mouth.

PROGRESS.

Satisfactory.

Blood Examination: 15.8.33

Red cells: 2,660,000.
Haemoglobin: 43%.
Colour Index: .8

DISCHARGED: 17.8.33.

Three weeks in hospital. Condition improved.
ADMITTED: 10.8.33.

Primipara; 20 weeks maturity. Complaining of fever.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Anaemia. Temperature 98.4; pulse 100; respirations 22.

Urine.

Clear.

Faeces.

Not examined for parasites. Loose stools.

BLOOD EXAMINATION.

Red cells: 2,350,000
Haemoglobin: 47%
Colour Index: 1.
Malaria parasites: a few.
Wassermann: negative.

TREATMENT.

Liver, iron and quinine by mouth.

PROGRESS.

Improved.

DISCHARGED: 23.8.33.

Two weeks in hospital. Advised to continue liver at home.
CASE 28. (646).

ADMITTED: 24.10.33.

Para 2; 16 weeks maturity. Complaining of fever, breathlessness and giddiness.

Yaws.

History positive.

SIGNS AND SYMPTOMS.

Patient anaemic. Temperature 100; pulse 132; respirations 28.

Urine.

High coloured, trace of albumin. On culture B. Coli found present.

Faeces.

Stools loose. No ova nor amoebae seen on culture, no non-lactose fermenters present.

BLOOD EXAMINATION.

Red cells: 1,400,000
Haemoglobin: 24%
Colour Index: 185
Malaria parasites: negative.

TREATMENT.

Liver and marmite by mouth. Iron and arsenic injections, and iron and arsenic by mouth.

PROGRESS.

Satisfactory.

6.11.33.

Red cells: 3,600,000
Haemoglobin 68%
Colour Index: .9

DISCHARGED: 13.11.33.

After three weeks in hospital.
CASE 29. (703).

ADMITTED: 6.11.33.

Para 1; 32 weeks maturity. Complaining of giddiness, palpitation, vomiting and weakness. When seen one month previously, anaemia not observed.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Anaemia. Temperature 99.4; pulse 104; respirations 36.

Urine.

Albumin, plus. A few leucocytes present. On culture, a few B. Coli found.

Faeces.

Ascaris ova present. No non-lactose fermenters present on culture.

BLOOD EXAMINATION.

Red cells: 1,110,000
Haemoglobin: 19%
Colour Index: .8
Malaria parasites: a few.

TREATMENT.

Liver and marmite and quinine by mouth; iron and arsenic injections.

PROGRESS.

Slight temperature for five days; steady improvement.

17.11.33.

Haemoglobin: 48%

28.11.33.

Red cells: 3,000,000
Haemoglobin: 50%
Colour Index: .83.

DISCHARGED: 3.12.33.

Improved. Four weeks in hospital.
ADMITTED: 13.11.33.

Primipara; 28-30 weeks maturity. Suffering from anaemia.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Anaemia. Temperature 99; pulse 88; respirations 24.

Urine.

Clear.

BLOOD EXAMINATION.

Red cells: 1,950,000
Haemoglobin: 38%
Colour Index: 1
Malaria parasites: plus.

TREATMENT.

Raw liver and quinine by mouth; iron and arsenic injections.

PROGRESS.

Improved.

Blood examination: 24.11.33.

Red cells: 2,300,000
Haemoglobin: 42%
Colour Index: .9.

DISCHARGED: 27.11.33.

After two weeks in hospital.
CASE 31. (726).

ADMITTED: 13.11.33.

Primipara; 30 weeks maturity. Not well.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Anaemia. Temperature 98.6; pulse 96; respirations 24.

Urine.

Clear.

Faeces.

Loose stools.

BLOOD EXAMINATION.

Red cells: 2,100,000
Haemoglobin: 40%
Colour Index: .9
Malaria parasites: plus.
Wassermann: negative.

TREATMENT.

Liver, marmite, quinine and iron by mouth.
Iron and arsenic injections.

PROGRESS.

No temperature after the first two days. In-patient for ten days. Much improved when discharged.

DISCHARGED: 23.11.33.

Ten days in hospital.
CASE 12. (1160).

ADMITTED: 1.3.34.

Primipara; 28-30 weeks maturity. Admitted with fever.

Yaws.

History positive.

SIGNS AND SYMPTOMS.

Anaemia. Temperature 99; pulse 96; respirations 24.

Urine.

Bilharzia ova present; red blood cells and leucocytes. Culture, sterile.

Faeces.

Stools frequent - not examined.

BLOOD EXAMINATION.

Red cells: 1,040,000
Haemoglobin: 18%
Colour Index: .9
Malaria parasites: plus.

TREATMENT.

Raw liver and iron by mouth; iron and arsenic injections.

PROGRESS.

Satisfactory.

Blood examination.

12.3.34. Red cells: 1,550,000
Haemoglobin: 20%

22.3.34. Red cells: 2,900,000
Haemoglobin: 40%
Colour Index: .7.

DISCHARGED: 23.3.34, after 3 weeks in hospital.
CASE 33. (99).

ADMITTED: 24.4.34.

Primipara; 30 weeks maturity. First attendance admitted with fever and swelling of legs.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Anaemia and oedema of body. Temperature 99.4; pulse 108; respirations 24.

Urine.

Trace of albumin; culture sterile.

Faeces.

Hookworm ova present and flagellates.

BLOOD EXAMINATION.

Red cells: 870,000
Haemoglobin: 18%
Colour Index: 1.1
Malaria parasites: negative.
Wassermann: negative.

TREATMENT.

Raw liver and marmite by mouth; iron and arsenic injections. Carbon Tetrachloride.

PROGRESS.

Ran some temperature for the first ten days; improved steadily.

Blood examination: 18.5.34.

Red cells: 2,652,000
Haemoglobin: 56%
Colour Index: 1.

DISCHARGED: 24.5.34, after one month in hospital.
CASE 34. (104).

ADMITTED: 25.4.34.

Para 2. 24 weeks maturity. First attendance: not well.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Anaemia. Temperature 100.2; pulse 136; respirations 28.

Urine.

Trace of albumin.

Faeces.

E. Histolytica, trichuris and hookworm ova, white blood cells and flagellates present.

BLOOD EXAMINATION.

Red cells: 750,000
Haemoglobin: 13%
Colour Index: .9
Malaria parasites: negative.
Wassermann: negative.

TREATMENT.

Raw liver and marmite by mouth; iron and arsenic injections. Carbon Tetrachloride and Santonin. Emetim started 12.5.34.

PROGRESS.

Patient steadily improved.

Blood examination: 4.5.34.

Red cells: 1,770,000
Haemoglobin: 40%
Colour Index: 1.1.

18.5.34.

Red cells: 2,820,000
Haemoglobin: 50%
Colour Index: .89.

DISCHARGED: 24.5.34, after 1 month in hospital.
Primipara; 24 weeks maturity. Admitted with fever and swelling of legs.

Yaws.

History negative.

**SIGNS AND SYMPTOMS.**

Anaemia, oedema of legs. Temperature 99.2; pulse 84; respirations 28. Spleen enlarged, vaginal ulceration present.

**Urine.**

High coloured; trace of albumin; culture sterile.

**Faeces.**

Stools rather frequent. On microscopic examination no abnormality discovered. Culture; no non-lactose fermenters present.

**BLOOD EXAMINATION.**

Red cells: 1,720,000.
Haemoglobin: 34%
Colour Index: 1
Malaria parasites: negative.
Wassermann: negative.

**TREATMENT.**

Liver and marmite, iron and quinine by mouth N.A.B. injections.

**PROGRESS.**

Steady improvement.

**Blood examination:** 10.7.34.

Red cells: 2,768,000.
Haemoglobin: 42%
Colour Index: .77

**DISCHARGED:** 18.7.34.

Much improved. Three weeks in hospital.
CASE 36. (466).

ADMITTED: 18.7.34.

Primipara; 26 weeks maturity. Admitted with cough, fever and swelling of legs. First attendance.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Anaemia, oedema and troublesome cough. Temperature 100.4; pulse 112; respiration 28. Haemic murmurs over precordia.

Urine.

Trace of albumin. Scanty leucocytes and epithelial cells present. Sterile on culture.

Faeces.

No ova seen; on culture, no non-lactose fermenters seen.

Cervical Swab.

Short chain streptococci and a few staphylococci seen.

BLOOD EXAMINATION.

Red cells: 1,736,000
Haemoglobin: 30%
Colour Index: .88
Malaria parasites: a few.
Wassermann: negative.

TREATMENT.

Liver, marmite, iron and quinine by mouth.

PROGRESS.

Satisfactory.

Blood examination: 10.8.34.

Haemoglobin: 50%

DISCHARGED: 15.8.34.

Very much improved. One month in hospital.
CASE 37. (753).

ADMITTED: 27.9.34.

Para 3; 32 weeks maturity. Complaining of cough, breathlessness and swelling of feet.

Yaws.

Positive history.

SIGNS AND SYMPTOMS.

Oedema of feet. Some bronchitis. Temperature 99.4; pulse 100; respirations 26.

Urine.

Trace of albumin.

Faeces.

No ova nor cysts. Semi-solid stools, rather frequent.

BLOOD EXAMINATION.

Red cells: 1,532,000.
Haemoglobin: 30%
Colour Index: 1
Nucleated red cells present.
Malaria parasites: scanty.

TREATMENT.

Raw liver and marmite by mouth. Quinine, iron and arsenic by mouth. Iron and arsenic injections.

PROGRESS.

3.10.34. Haemoglobin: 30%
7.10.34. Delivered. A living child, 5 lbs. 4 ozs. Ran some temperature in the puerperium.
10.10.34. Haemoglobin: 33%
15.10.34. Haemoglobin: 41%
20.10.34. Haemoglobin: 44%

DISCHARGED: 20.10.34.

After three weeks in hospital. Progressing satisfactorily.
CASE 38. (760).

ADMITTED: 30.9.34.

Primipara; 30 weeks maturity. Complaining of pains all over.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Admitted for observation. Temperature 98.2; pulse 88; respirations 32.

Urine.

Clear.

Faeces.

No ova nor protozoa.

BLOOD EXAMINATION.

On admission.

Malaria parasites: numerous.
Wassermann: negative.

Blood count one week after admission.

Red cells: 1,064,000.
Haemoglobin 25%
Colour Index: 1.2

TREATMENT.

Treated with quinine, liver and marmite by mouth. N.A.B. injections.

PROGRESS.

This patient was treated as a case of malaria until 6.10.34. when severe anaemia was noted.

Confinement.

8.10.34. Premature macerated infant weighing 3 lb. 4 oz. Patient ran a temperature both before and after delivery.

15.10.34. /
CASE 38. (Continued).

15.10.34.

Haemoglobin: 48%

Developed swelling on the leg, thought to be a gumma, which improved after injection of N.A.B.

DISCHARGED: 19.10.34.

Three weeks in hospital.
Progressing satisfactorily.
CASE 39. (809).

ADMITTED: 11.10.34.

Primipara; 28 weeks maturity. Complaining of giddiness and cough.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Anaemia. Temperature 99; pulse 120; respirations 32; blood pressure 106/54.

Urine.

Trace of albumin; leucocytes and epithelial cells in deposit.

Faeces.

Liquid, hookworm ova present. Stools frequent.

BLOOD EXAMINATION.

Red cells: 1,136,000.
Haemoglobin: 25%
Colour Index: 1.1
Malaria parasites: negative.

TREATMENT.

Raw liver and marmite by mouth; iron and arsenic injections. Oil of Chenopodium.

PROGRESS.

Some temperature for the first six days; general progress satisfactory. Urine clear after five days.

BLOOD EXAMINATION.

22.10.34.

Red cells: 1,464,000.
Haemoglobin: 35%
Colour Index: 1.2

30.10.34.

Red cells: 2,500,000.
Haemoglobin: 40%
Colour Index: .8

DISCHARGED: 3.11.34.

Three weeks in hospital. Progressing satisfactorily.
117.

CASE 40. (860).

ADMITTED: 25.10.34.

Primipara; 32-34 weeks maturity. Not well.

Yaws:

History negative.

SIGNS AND SYMPTOMS.

Anaemia. Temperature 99; pulse 96; respirations 28.

Urine.

Epithelial cells; leucocytes and granular casts present.

Faeces.

Liquid specimen; ascaris ova E. Coli, S. Haematobium ova present.

BLOOD EXAMINATION.

Red cells: 1,600,000.
Haemoglobin: 36%
Colour Index: 1.1
Malaria parasites: negative.

TREATMENT.

Liver and iron and arsenic by mouth. Santonin.

PROGRESS.

Ran no temperature. Urine cleared up in hospital.

30.10.34. Faeces: Liquid specimen, flagellates only seen.
3.11.34. Blood examination.

Red cells: 3,000,000.
Haemoglobin: 54%
Colour Index: .9

DISCHARGED: 3.11.34.

After ten days in hospital; improving.
CASE 41. (88).

ADMITTED: 23.1.35.

Para 1; 28 weeks maturity. Complaining of giddiness and weakness.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Anaemia. Temperature 100; pulse 108; respirations 28.

Urine.

No albumin; a few leucocytes and epithelial cells.

Faeces.

Liquid specimen; E.Coli and flagellates present.

BLOOD EXAMINATION.

Red cells: 1,000,000.

Haemoglobin: 22%.

Colour Index: 1.1

Nucleated red cells present.

Malaria parasites: scanty.

Wassermann: plus.

TREATMENT.

Raw liver and quinine by mouth; iron and arsenic injections.

PROGRESS.

Satisfactory.

29.1.35.

Haemoglobin: 35%.

4.2.35.

Haemoglobin: 44%.

DISCHARGED: 6.2.35.

After two weeks in hospital. Progressing satisfactorily.
CASE 42. (234).

ADMITTED: 25.2.35.

Primipara; 32 weeks maturity. Complaining of swelling of feet.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Oedema of feet. Anaemia. Temperature 99.6; pulse 112; respirations 28.

Urine.

No albumin; a few leucocytes present.

Faeces.

Ascaris ova present.

BLOOD EXAMINATION.

Red cells: 1,820,000.
Haemoglobin: 40%
Colour Index: 1.1
Some irregularity in size of red cells;
nucleated red cells present.
Malaria parasites: negative.
Wassermann: negative.

TREATMENT.

Raw liver by mouth. Oil of Chenopodium.
Quinine.

PROGRESS.

No temperature except on admission.

4.3.35.

Haemoglobin: 45%.

11.3.35.

Haemoglobin: 56%.

DISCHARGED: 12.3.35.

After two weeks in hospital. Improving.
CASE 43. (259).

ADMITTED: 4. 3. 35.

Primipara; 20 weeks maturity. Complaining of not feeling well.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Anaemia. Temperature 101.4; pulse 122; respirations 38.

Urine.

Clear.

Faeces.

Ascaris ova present.

BLOOD EXAMINATION.

Red cells: 2,000,000.

Haemoglobin: 40%.

Colour Index: 1

Irregularity in size of red cells; poly-chromatophilia; nucleated red cells present.

Malaria parasites: negative.

Wassermann: negative.

TREATMENT.

Liver extract, and marmite, by mouth; iron and arsenic injections. Oil of Chenopodium.

PROGRESS.

Temperature came down within five days.

9. 3. 35.

Haemoglobin 38%.

12. 3. 35.

Scanty megaloblasts in blood film.

15. 3. 35.

Haemoglobin 40%.

DISCHARGED: 18. 3. 35.

After/
CASE 43. (Continued).

After two weeks in hospital.

No alteration in blood picture. Blood films show irregularity in size of red cells. Polychromatophilia and scanty megaloblasts.
CASE 44. (529)

ADMITTED: 29.4.35.

Primipara; 32 weeks maturity. Admitted with anaemia.

Yaws.

History positive.

SIGNS AND SYMPTOMS.

Anaemia; Temperature 100; pulse 136; respirations 24.

Urine.

Clear.

Faeces.

No ova on examination; stools loose.

BLOOD EXAMINATION.

Red cells: 1,208,000.
Haemoglobin: 29%.
Colour Index: 1.2
Malaria parasites: negative.

TREATMENT.

Raw liver and quinine, iron and arsenic by mouth; also iron and arsenic injections.

PROGRESS.

7.5.35. Haemoglobin: 33%
14.5.35. Haemoglobin: 28%
19.5.35. Haemoglobin: 49%
22.5.35. Premature labour. Delivered of living child, weight 2 lb. 10 ozs. The infant died after three days.

Puerperium. Some temperature for two weeks. Gradual improvement.

27.5.35. Red cells: 3,680,000.
Haemoglobin: 54%
Colour Index: .75

DISCHARGED/
CASE 44 (Continued).

DISCHARGED: 29.5.35.

After one month in hospital.
Progressing satisfactorily.

READMITTED: 20.9.36.

Following pregnancy.
Admitted three days after premature delivery outside. No history of bleeding.

SIGNS AND SYMPTOMS.

Patient was profoundly anaemic, and died shortly after admission. She had not been seen at all during pregnancy. The premature infant also died.
CASE 45. (653).

ADMITTED: 23.5.35.
Para 1; 34 weeks maturity. Complaining of swelling of feet.

Yaws.
History negative.

SIGN AND SYMPTOMS.
Anaemia; oedema of feet.

Urine.
Scanty; red blood cells and epithelial cells present.

Faeces.
No ova seen.

BLOOD EXAMINATION.
On admission Haemoglobin: 32%

TREATMENT.
Raw liver and iron and arsenic by mouth.

PROGRESS.
Delivered one week after admission of a slightly premature living child.

30.5.35. Red cells: 2,280,000
Haemoglobin: 43%
Colour Index: .97

DISCHARGED.
Two weeks in hospital.
Progressing satisfactorily.

READMITTED: 12.9.38.
In next pregnancy. Very sick.

SIGNS/
CASE 45 (Continued).

SIGNS AND SYMPTOMS.

Anaemia; oedema of feet, legs, face, and lower abdominal wall for ten days. Sore mouth and tongue for six weeks. Temperature 99; pulse 96; respirations 24.

Urine.

Trace of albumin. Epithelial and pus cells present. No organisms.

Faeces.

Semi-solid; ascaris ova present.

BLOOD EXAMINATION.

Red cells: 1,150,000.
White cells: 5,800.
Haemoglobin: 25%.
Colour Index: 1.1
Nucleated red cells present.
Malaria parasites: negative.

TREATMENT.

Liver, ½ lb. daily, iron and arsenic and quinine by mouth. Campolon injections.

PROGRESS.

Labour three days after admission; normal living child, weight 6 lb. 3 ozs. Though apparently healthy at birth it went steadily downhill and died on the eighth day.


26.9.38. Haemoglobin: 35%.
3.10.38. Haemoglobin: 50%.
10.10.38. Haemoglobin: 60%.

DISCHARGED: 11.10.38.

After one month in hospital.
Progress satisfactory.
CASE 46. (927).

ADMITTED: 15.7.35.

Primipara; 34 weeks maturity. Complaining of fever.

Yaws.

History positive.

SIGNS AND SYMPTOMS.

Anaemia; temperature 99.2; pulse 108; respirations 24.

Urine.

Clear.

Faeces.

Nothing abnormal detected.

BLOOD EXAMINATION.

Red cells: 1,720,000.
Haemoglobin: 38%.
Colour Index: 1.1.
Some irregularity in size of red cells; polychromatophilia.
Malaria parasites: negative.
Wassermann: double plus.

TREATMENT.

Treated with liver, marmite and iron by mouth; N.A.B. injections.

PROGRESS.

Satisfactory. No temperature after admission.

6.8.35. Red cells: 2,000,000.
Haemoglobin: 40%.
Colour Index: 1.

DISCHARGED: 9.8.35.

Three weeks in hospital.

Improving.

AFTER/
CASE 46. (Continued).

AFTER HISTORY.

Delivered 12.9.35, on district. Living child, weighing 6 lbs. 3 ozs.

Puerperium:

Non morbid.
CASE 47. (1097).

ADMITTED: 28.8.35.

Primipara; 30 weeks maturity. Complaining of frequent stools, cough, headache and loss of appetite. First attendance.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Anaemia; Temperature 100.4; pulse 146; respirations 32.

Urine.

Trace of albumin.

Faeces.

Semi-solid; no ova seen.

Sputum

No tubercle bacilli.

BLOOD EXAMINATION.

Red cells: 1,000,000
Haemoglobin: 20%.
Colour Index: 1
Malaria parasites: negative.

TREATMENT.

Quinine, iron, liver and marmite by mouth. Campolon injections.

PROGRESS.

Satisfactory. Temperature for one week.

9.9.35. Red cells: 2,100,000.
Haemoglobin: 38%.

20.9.35. Red cells: 2,350,000
Haemoglobin: 48%.

DISCHARGED: 23.9.35.

After one month in hospital.

Note: This was the first case for which Campolon was used.
CASE 48. (1111).

ADMITTED: 28.3.35.

Para 2; 26 weeks maturity. Complaining of palpitation, giddiness, breathlessness, weakness, and high coloured urine.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Anaemia; temperature 99.2; pulse 136; respirations 28.

Urine.

Clear, high coloured.

Faeces.

No ova nor protozoa seen.

BLOOD EXAMINATION.

Red cells: 1,500,000.
Haemoglobin: 28%. 
Colour Index: .9.
Malaria Parasites: negative.

TREATMENT.

Raw liver, quinine and iron by mouth, and injections of Campolon.

PROGRESS.

Satisfactory.

30.8.35. Red cells: 2,350,000.
Haemoglobin: 44%.
Colour Index: .9

9.9.35. Red cells: 3,000,000.
Haemoglobin: 58%.
Colour Index: .9

DISCHARGED/
CASE 48. (Continued).

DISCHARGED: 13.9.35.
Much improved.
After two weeks in hospital.

READMITTED: 25.10.35.
With fever and anaemia.
Red cells: 3,250,000.
Haemoglobin: 63%.

DISCHARGED: 2.11.35.
After one week in hospital.

AFTER HISTORY: 12.11.35.
Delivered of a living child, on district, weight 6 lbs. 14 ozs.
CASE 49. (1375).

ADMITTED: 24.10.35.

Primipara; 23 weeks maturity. Complaining of constipation and cough. Seen three weeks before. At that time anaemia not noticed.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Anaemia; Temperature 100; pulse 128; respirations 28.

Urine.

Clear.

Faeces.

No Ova nor protozoa seen.

BLOOD EXAMINATION

Red cells: 1,500,000.
Haemoglobin: 30%.
Colour Index: 1
Malaria parasites: present.

TREATMENT:

Liver, marmite, quinine and iron, by mouth.

PROGRESS:

No temperature after first day. Progress satisfactory.

5.11.35. Red cells: 2,250,000.
Haemoglobin: 40%.
Colour Index: .9

DISCHARGED: 6.11.35.

After two weeks in hospital.
Satisfactory.

AFTER/
CASE 49. (Continued).

AFTER HISTORY:

6.2.36. Seen again after normal delivery at home.

27.9.37. Another child delivered normally, weight 7 lb. 14 oz.

No anaemia in this pregnancy.
CASE 50. (1574).

ADMITTED: 6.12.35.

Primipara; 26 weeks maturity. Not well. Anaemia noted two weeks before at Antenatal Clinic.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Anaemia; temperature 99.8; pulse 100; respirations 24.

Urine.

Clear.

Faeces.

A few hookworm ova.

BLOOD EXAMINATION.

Red cells: 1,470,000.
Haemoglobin: 30%.
Colour Index: 1.
Malaria parasites: a few.

TREATMENT.

Liver, marmite, quinine, Blaud's pills, Ascaridol, by mouth.

PROGRESS.

Satisfactory.

16.12.35. Red cells: 2,000,000.
Haemoglobin: 38%.
Colour Index: .9

23.12.35. Haemoglobin: 55%.

DISCHARGED: 24.12.35.

Over two weeks in hospital.
CASE 51. (1585).

ADMITTED: 9.12.35.

Primipara; 24 weeks maturity. Complaining of headache and back pains. First attendance.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Anaemia; loud precordial murmurs; spleen enlarged; blood pressure 108/62; temperature 99.6; pulse 120; respirations 24.

Urine.

Clear.

Faeces.

No ova seen.

BLOOD EXAMINATION.

Red cells: 1,480,000.
Haemoglobin: 33%.
Colour Index: 1.1
Malaria parasites: A few.

TREATMENT.

Iron, quinine, liver and marmite by mouth.

PROGRESS.

Temperature for the first four days, thereafter normal.

16.12.35. Red cells: 1,830,000.
Haemoglobin: 48%.
Colour Index: 1.3

DISCHARGED: 24.12.35.

Two weeks in hospital.

Improving.
CASE 52. (206.)

ADMITTED: 20.2.36.

Para 1. 30 weeks maturity. Admitted complaining of sore mouth and cough.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Anaemia; mouth sore; temperature 100; pulse 128; respirations 32.

Urine.

Clear.

Faeces.

Ascaris ova present.

BLOOD EXAMINATION.

Red cells: 1,600,000.
Haemoglobin: 23%.
Colour Index: .7
Malaria parasites: negative.
Wassermann: negative.

TREATMENT.

Liver, marmite, santonin, and iron by mouth.

PROGRESS.

Some temperature for the first five days.

27.2.36. Red cells: 1,896,000.
Haemoglobin: 42%.
Colour Index: 1.1

DISCHARGED: 2.3.36.

Two weeks in hospital.

Improving.
CASE 53. (336).

ADMITTED: 19.3.36.

Primipara; 32 weeks maturity. Complaining of general pains and headache.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Anaemia; temperature 99.4; pulse 88; respirations 28.

Urine.

Trace of albumin. Epithelial cells present. No ova seen.

Faeces.

Ascaris and hookworm ova present.

BLOOD EXAMINATION.

Haemoglobin: 55%, (Talquist)

TREATMENT.

At first treated with quinine and light diet.

25.3.36. Put on liver, marmite, and iron by mouth; injections of Campolon.

PROGRESS.

Patient was treated as a case of malaria for one week until 25.3.36, when severe anaemia was noted.

Blood/
CASE 53. (Continued).

**Blood Examination.**

Red cells: 1,360,000.
Haemoglobin: 38%.
Colour Index: 1.4.

28.3.36. White cells: 13,400.
Marked polychromatophilia.
Nucleated red cells present.

3.4.36. Red cells: 1,936,000.
Haemoglobin: 35%.
Colour Index: .9.

14.4.36. Red cells: 2,144,000.
Haemoglobin: 50%.
Colour Index: 1.1.

Patient ran irregular temperature for two weeks,
as high as 100.4°.

**DISCHARGED:** 18.4.36.

One month in hospital.
Improving.

**READMITTED:** 6.5.36.

Normal delivery; weight of infant 5 lbs.
12 ozs.

**Puerperium.**

Non morbid.
CASE 54. (405).

ADMITTED: 6.4.36.

Para 3; 24 weeks maturity. Complaining of cough and debility. Patient refused offer of admission one week before.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Anaemia; temperature 98.2; pulse 108; respirations 24.

Urine.

Trace of albumin.

Faeces.

Ascaris ova present.

Sputum.

No tubercle bacilli.

BLOOD EXAMINATION.

Red cells: 1,600,000.
Haemoglobin: 38%.
Colour Index: 1.1
Malaria parasites: negative.
Wassermann: negative.

TREATMENT.

Liver, marmite, iron and Ascaridol by mouth; Campolon injections.

PROGRESS.

Improved.

21.4.36. Red cells: 3,206,000.
Haemoglobin: 52%.
Colour Index: .8

DISCHARGED: 26.4.36.

Three weeks in hospital.
CASE 55. (517).

ADMITTED: 4.5.36. -

Primipara; 26 weeks maturity. Complaining of pain in breast, and fever. Seen two weeks before. Anaemia not then noted.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Mastitis; anaemia; oedema of feet; temperature 99.4; pulse 104; respirations 28; spleen enlarged.

Urine.

Clear.

Faeces.

Nothing abnormal detected.

BLOOD EXAMINATION.

Red cells: 1,912,000.
Haemoglobin: 37%.
Colour Index: .9
Malaria parasites: negative.

TREATMENT.

Injections of Campolon; iron and liver by mouth.

PROGRESS.

Ten days after admission patient developed an attack of Flexner dysentery from which she recovered.

3.6.36. Red cells: 2,896,000.
Haemoglobin: 52%.
Colour Index: .9

DISCHARGED: 8.6.36.

Five weeks in hospital.

AFTER HISTORY: 31.7.36.

Delivered normally on district.
CASE 56. (464)

ADMITTED: 22.4.36.

Primipara; 24 weeks maturity. Not well.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Anaemia. Temperature 99; pulse 96; respirations 24.

Urine.

High colour. No albumin.

Faeces.

No ova, no protozoa.

BLOOD EXAMINATION.

Red cells: 2,100,000.
Haemoglobin: 56%.
Colour Index: 1.3
Malaria parasites: negative.
Wassermann: negative.

TREATMENT.

Liver and iron by mouth.

PROGRESS

Satisfactory.

DISCHARGED: 28.4.36.

One week in hospital.

READMITTED: 10.7.36.

Normal delivery of premature living child, weight 4 lbs. 9 ozs.

Puerperium: non-morbid.

Haemoglobin: 80%, (Talquist).
ADMITTED: 4.5.36.

Primipara; 22-24 weeks maturity. Complaining of fever.

Yaws:

History negative.

SIGNS AND SYMPTOMS.

Anaemia; slight jaundice; temperature 100; pulse 130; respiration 28.

Urine.

No albumin. Culture sterile.

Faeces.

Ascaris and hookworm ova present.

BLOOD EXAMINATION.

Red cells: 1,480,000.
White cells: 9,800.
Haemoglobin: 30%.
Colour Index: 1.
Some polychromatophilia. Nucleated red cells.
Malaria parasites: negative.

TREATMENT.

Liver, marmite, Ascaridol, iron and quinine by mouth.

PROGRESS.

Satisfactory.

21.5.36. Red cells: 2,328,000.
Haemoglobin: 53%.
Colour Index: 1.1

DISCHARGED: 30.5.36.

Over three weeks in hospital.

AFTER HISTORY. 6.7.36.

Progressing satisfactorily.
CASE 58. (575).

ADMITTED: 17.5.36.

Para 1; 30 weeks maturity. Complaining of headache, palpitation and giddiness. Was an in-patient 3 weeks before with bronchitis; anaemia not then noted.

Yaws:

History negative.

SIGNS AND SYMPTOMS.

Anaemia; oedema of legs; Temperature 99.6; pulse 116; respirations 32.

Urine.

Trace of albumin.

Faeces.

Liquid. No ova seen.

BLOOD EXAMINATION.

Red cells: 1,536,000.
Haemoglobin: 25%.
Colour Index: .8
Nucleated red cells present.
Malaria parasites: negative.
Wassermann: negative.

TREATMENT.

Liver, marmite, and iron by mouth. Iron and arsenic injections.

PROGRESS.

Satisfactory.

8.6.36. Red cells: 3,608,000.
Haemoglobin: 58%.
15.6.36. Red cells: 3,880,000.
Haemoglobin: 75%.
Colour Index: 1.

DISCHARGED: 17.6.36.
One month in hospital.

READMITTED: 1.7.36. For labour. Premature living child, weight 4 lbs.

Malaria parasites: plus.
Haemoglobin: 70%.
CASE 59. (732).

ADMITTED: 20.6.36.

Para 1; 30 weeks maturity. Complaining of palpitation and swelling of feet. Not seen before.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Anaemia; oedema of feet; temperature 99; pulse 124; respirations 28. Spleen enlarged.

Blood Pressure. 134/68.

Urine.


Faeces.

Semi solid. Scanty trichuris ova. No mucus. No amoebae.

BLOOD EXAMINATION.


TREATMENT.

Light diet at first, because of urinary condition.

PROGRESS.

24.6.36. Premature labour. Delivered of a macerated infant, weight 2 lbs. 4 ozs.

Puerperium.

Non morbid. Campolon injections begun on 24.6.36.

29/
CASE 59. (Continued).

29.6.36. Red cells: 2,376,000.
Haemoglobin: 37%

6.7.36. Red cells: 2,704,000.
Haemoglobin: 40%
Sickling noticed.

13.7.36. Red cells: 3,474,000.
Haemoglobin: 60%
Colour Index: .8

DISCHARGED: 17.7.36
One month in hospital.
ADMITTED: 6.7.36.

Primipara; 30 weeks maturity. Complaining of headache, palpitation and swelling of feet.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Oedema of feet, legs and abdominal wall. Face puffy. Temperature 100; pulse 124; respirations 28.

Blood Pressure. 122/56. Liver and spleen enlarged.

Urine.

Albumin plus. An occasional leucocyte and granular casts present. No ova.

On culture B. pyocyaneus and a few staphylococci.

Faeces.

No ova nor protozoa seen.

BLOOD EXAMINATION.

Red cells: 1,312,000.
Haemoglobin: 30%
Colour Index: 1.1
Malaria parasites: negative.
Wassermann: double plus.

TREATMENT.

At first treated with quinine and light diet. Injections of Campolon started 8.7.36. Later liver, marmite, and full diet added.

PROGRESS.

Progress at first not satisfactory.
CASE 60. (Continued).

14.7.36. Red cells: 1,032,000.  
White cells: 8,200.  
Haemoglobin: 26%.

18.7.36  Surgical induction of labour carried out.  
Delivery 19.7.36. Infant living, weight 3 lb. 4 ozs.

Puerperium.  
Morbid for one week, thereafter temperature normal.

25.7.36. Haemoglobin: 38%.

6.8.36. Haemoglobin: 54%. No sickling.

DISCHARGED: 11.8.36.

Five weeks in hospital.

Mother and child satisfactory, except for boils on baby's head.
CASE 61. (801).

ADMITTED: 9.7.36.

Primipara; 28 weeks maturity. Complaining of frequent stools. First attendance.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

No enteritis on admission. Temperature 101.4; pulse 128; respirations 28.

Urine.

Clear.

Faeces.

Solid. No ova nor protozoa seen.

BLOOD EXAMINATION.

On admission: Haemoglobin 60% (Talquist) Malaria parasites; plus. Wassermann: negative.

TREATMENT.

Quinine and fluids at first; anaemia noticed 17.7.36. Full diet; liver and marmite by mouth; iron and arsenic injections.

PROGRESS.

13.7.36. Haemoglobin: 50%.

17.7.36. Haemoglobin: 40% (Talquist)

Blood Examination:

Red cells: 2,160,000.
Haemoglobin: 37%.
Colour Index: 0.86.

24.7.36. Red cells: 2,488,000.
Haemoglobin: 60%.
Colour Index: 1.2. No sickling.

DISCHARGED: 28.7.36.

Three weeks in hospital.

AFTER HISTORY: 1.11.36.

Delivered normally, weight of infant 6 lbs. 4 ozs.
ADMITTED: 20.7.36.

Primipara; 18 weeks maturity. Complaining of general pains and dizziness. First attendance.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Anaemia. Temperature 100; pulse 112; respirations 28. Spleen enlarged.

Urine.

Trace of albumin.

Faeces.

Semi-solid; no ova nor protozoa.

BLOOD EXAMINATION.

Red cells: 1,240,000.
Haemoglobin: 30%
Colour Index: 1.2
Malaria parasites: negative.
Wassermann: double plus.

TREATMENT.

Liver by mouth; iron and arsenic and N.A.B. injections.

PROGRESS.

Improved.

1.8.36. Haemoglobin: 40%
6.8.36. Haemoglobin: 45%

DISCHARGED: 7.8.36.

Over two weeks in hospital.

To attend for N.A.B.
CASE 65. (1070).

ADMITTED: 14.9.36.

Para 2; 26 weeks maturity. Complaining of upper abdominal and side pains; seen one week before with fever, but patient refused offer of admission.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Temperature 100; pulse 124; respirations 28. Both previous infants delivered in hospital. No anaemia noticed then.

Urine.

Clear.

Faeces

Liquid. No ova nor protozoa.

BLOOD EXAMINATION. 29.9.36.

Red cells: 1,030,000. No sickling.
Haemoglobin: 30%.
Colour Index: 1.5.
Malaria parasites: plus.

TREATMENT.

Quinine at first. Liver and iron by mouth from 29.9.36.

PROGRESS.

Ran temperature for ten days.

7.10.36. Red Cells: 2,340,000.
Haemoglobin: 37%.

14.10.36. Haemoglobin: 37%.

DISCHARGED: 15.10.36.

Insisted on going out; improved. One month in hospital.

AFTER/
CASE 63. (Continued).

AFTER HISTORY: 16.1.37.

Delivered normally, weight of child 7 lbs. 8 ozs.

Note: Anaemia in this case was not noticed until the patient had been in hospital for two weeks for an attack of malaria.
CASE 64. (1136).

ADMITTED: 19.10.36.
Primipara; 28-30 weeks maturity. Not well. 
Seen one week before. Anaemia not then noted.

Yaws.
History negative.

SIGNS AND SYMPTOMS.

Anaemia; temperature 100; pulse 118; respirations 28.

Urine.
Clear.

Faeces.
Ascaris ova present; some hookworm ova also present.

BLOOD EXAMINATION.

Red cells: 1,370,000.
Haemoglobin: 27%.
Colour Index: 1.
Malaria parasites: negative.
Wassermann: negative.

TREATMENT.
Liver and iron by mouth.

PROGRESS.

Ran temperature until aborted.

12.11.36 Red cells: 3,080,000.
Haemoglobin: 56%.
Colour Index: .9.

7.12.36 Haemoglobin: 70%. Sickling of red cells present.

7.12.36 Aborted macerated foetus 2 lbs. 8 ozs.

Puerperium: morbid, for seven days. Cause of temperature not clear.

DISCHARGED: 18.12.36.

Two months in hospital.
Satisfactory.
CASE 65. (1198)

ADMITTED: 9.11.36. -

Para 1; 20 weeks maturity. Complaining of stomach-ache, headache, general pains and dizziness. Seen one week before and was then noted to be anaemic.

Yaws.

History positive.

SIGNS AND SYMPTOMS.

Anaemia. Temperature 99; pulse 136; respirations 24.

Urine.

Clear.

Faeces.

Solid specimen; hookworm ova present.

BLOOD EXAMINATION.

Red cells: 1,536,000.
Haemoglobin: 30%.
Colour Index: 1.
Malaria parasites: negative.

TREATMENT.

Quinine, iron, oil of Chenopodium by mouth. Raw liver by mouth.

PROGRESS.

Satisfactory. Ran temperature for 2 - 3 days after admission.

18.11.36. Red cells: 3,088,000.
Haemoglobin: 40%.
Colour Index 6.

DISCHARGED: 22.11.36.

Two weeks in hospital.

Much improved.
CASE 66. (1249).

ADMITTED: 26.11.36.

Primipara; 28-30 weeks maturity. Complaining of headache and general pains. Seen one week before as out-patient, when some anaemia noted.

Yaws:

History negative.

SIGNS AND SYMPTOMS.

Anaemia. Temperature 99; pulse 116; respirations 28.

Urine.

Trace of albumin.

Faeces.

Liquid. Ascaris ova, flagellates present.

BLOOD EXAMINATION.

Haemoglobin: 26%.
Malaria parasites: negative.
Wassermann: negative.

TREATMENT.

Liver, marmite, oil of Chenopodium, iron and arsenic by mouth.

PROGRESS.

Ran slight temperature while in hospital, not over 100°.

10.12.36. Haemoglobin: 40%.

DISCHARGED: 11.12.36.

Two weeks in hospital.

Much improved.
CASE 67. (1354).


Primipara; 26 weeks maturity. Complaining of swelling of feet. First attendance.

Yaws.

History negative.

SIGNs AND SYMPTOMs.

Anaemia. Swelling of feet, legs and anterior abdominal wall. Temperature 98.4; pulse 120; respirations 28.

Urine.

Clear.

Faeces.

Solid specimen; white blood cells present. No parasites seen.

BLOOD EXAMINATION.

Red cells: 1,808,000. 
Haemoglobin: 27%.
Colour Index: .75.
Malaria parasites: negative.
Wassermann: negative.

TREATMENT.

Liver and iron by mouth.

PROGRESS.

Satisfactory. Some temperature for first few days.

28.12.36.

Red cells: 2,616,000.
Haemoglobin: 55%.
Colour Index: 1.

DISChARGED: 2.1.37.

Two/
CASE 67. (Continued).

Two weeks in hospital.

AFTER HISTORY.

CASE 68. (111).

ADMITTED: 2.1.37.

Para 3; 34 weeks maturity. Complaining of swelling of feet. Last seen one month before.

Yaws.

History negative.

SIGNS AND SYMPTOMS.


Blood Pressure. 150/68.

Urine.

Albumin present; leucocytes and epithelial cells. No ova.

Faeces.

Not examined.

BLOOD EXAMINATION.

Haemoglobin: 47%.
Malaria parasites: negative.

TREATMENT.

At first on albuminuria diet. After delivery Campolon injections; liver and iron by mouth.


PROGRESS.

11.1.37. Delivered premature living infant, weight 5 lbs.


14.1.37. Red cells: 1,368,000.
Haemoglobin: 40%.
Colour Index: 1.5.

21./
CASE 68. (Continued).

Haemoglobin: 28%.
Colour Index: 1.5
  No sickling. Blood culture sterile.


29.1.37. Red cells: 2,256,000.
Haemoglobin: 37%.
Temperature down after transfusion.

DISCHARGED: 3.2.37.
  One month in hospital.
  Improving.
ADMITTED: 5.2.37.

Primipara; 30 weeks maturity. Complaining of cough and high coloured urine. Seen four days before, when anaemia not noted.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Anaemia; temperature 99; pulse 128; respirations 28.

Urine.

Clear; high coloured. Granular casts and epithelial cells.

Faeces.

Solid specimen. No ova nor protozoa.

Sputum.

No tubercle bacilli. On culture short chain non-haemolytic streptococci.

BLOOD EXAMINATION.

Red cells: 1,480,000.
Haemoglobin: 45%.
Colour Index: 1.6
Malaria parasites: plus.
Wassermann: negative.

TREATMENT.

Liver and iron by mouth. Campolon injections; iron and arsenic injections.

PROGRESS.

Satisfactory.

15.2.37. Red cells: 2,472,000.
Haemoglobin: 49%.
Colour Index: 1.

DISCHARGED/
DISCHARGED: 22.3.37.
Over two weeks in hospital.
Improving.

AFTER HISTORY.
10.5.37. Delivered of living child, weight 5 lbs.
Puerperium. Non morbid.
CASE 70. (172).

ADMITTED: 18.2.37.

Para.1; 30 weeks maturity. Complaining of giddiness, palpitation and feeling tired. Seen one week before, and found to be anaemic.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Anaemia. Temperature 99; pulse 100; respirations 28.

Urine.

Trace of albumin; epithelial and pus cells present. On culture B. Coli plus.

Faeces.

Hookworm ova and strongyloides larvae present.

BLOOD EXAMINATION.

Red cells: 1,000,000. No sickling.
Haemoglobin: 30%.
Colour Index: 1.5.
Malaria parasites: negative.

TREATMENT.

Liver by mouth and Campolon injections. Iron and arsenic by mouth.

PROGRESS.

Satisfactory.

19.3.37. Red cells: 2,050,000.
Haemoglobin: 49%.
Colour Index: 1.2.

DISCHARGED: 22.3.37.

One month in hospital. Satisfactory.

AFTER/
CASE 70. (Continued).

AFTER HISTORY:

Readmitted. 8.5.37. Premature delivery of living child, weight 4 lb. 10 ozs.
CASE 71. (189).

ADMITTED: 25.2.37.

Primipara; 26 weeks maturity. Complaining of headache, backache, giddiness, palpitation, and vomiting. Found to be anaemic one month before.

Yaws.

History negative.

SIGNs AND SYMPTOMS.

Anaemia. Temperature 99; pulse 120; respirations 42.

Urine.

Clear.

Faeces.

Solid specimen. No ova nor protozoa.

BLOOD EXAMINATION.

Red cells: 1,512,000. No sickling.
Haemoglobin: 35%.
Colour Index: 1.1.
Malaria parasites: plus.

TREATMENT:

Liver and marmite by mouth; iron and arsenic injections.

PROGRESS.

Slight temperature for first three days only. Progress satisfactory.

8.3.37. Red cells: 2,670,000.
Haemoglobin: 50%.
Colour Index: .9.

DISCHARGED: 13.3.37.

Two weeks in hospital.
Improved.

AFTER/
CASE 71. (Continued).

AFTER HISTORY:

Readmitted. 27.5.37. Surgical induction of labour; labour long, with primary inertia. Infant weighed 6 lbs. 12 ozs; still-born.

Puerperium. Morbid.
CASE 72. (263).

ADMITTED: 18.3.37.

Para 1; 22 weeks maturity. Complaining of breathlessness, cough, sore mouth and indigestion. Attended two weeks before. Anaemia not then noted.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Sore mouth, anaemia. Temperature 100.2; pulse 110; respirations 28.

Urine.

Clear.

Faeces.

Semi-solid, flagellates present.

BLOOD EXAMINATION.

Red cells: 1,820,000. No sickling.
Haemoglobin: 38%.
Colour Index: 1.
Malaria parasites: negative.
Wassermann: negative.

TREATMENT.

Liver and marmite by mouth; Campolon and iron and arsenic injections.

PROGRESS.

Satisfactory.

31.3.37. Red cells: 3,170,000.
Haemoglobin: 55%.
Colour Index: .88.

DISCHARGED: 5.4.37.

Nearly three weeks in hospital. Improving.

AFTER HISTORY:

CASE 73. (656).

ADMITTEN: 17.6.37

Para 3; 28 weeks maturity. Complaining of general pains, headache and giddiness for one week. Anaemia noted 2 weeks before as out-patient.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Very anaemic. Temperature 99.8; pulse 152; respirations 40.

Urine.

Light cloud albumin; granular casts and scanty leucocytes present. Culture sterile.

Faeces.

Liquid; no ova nor protozoa seen. Stools frequent.

BLOOD EXAMINATION.

Red cells: 700,000. No sickling.
Haemoglobin: 15%.
Colour Index: 1.
Malaria parasites: negative.
Wassermann: negative.

TREATMENT.

Liver and iron by mouth; Campolon injections.

PROGRESS.

23.6.37. Red cells: 1,040,000.
Haemoglobin: 18%.
25.6.37. Delivered of macerated foetus, weight 1 lb. 2 ozs.

Puerperium. Morbid. Temperature for three weeks.

5.7.37. Red cells: 1,280,000.
Haemoglobin: 30%.
12.7.37. Red cells: 1,340,000.
Haemoglobin 35%.
6./
CASE 73. (Continued).

6.8.37. Red cells: 2,910,000
Haemoglobin 75%.

Had abscess of left buttock and some boils.

DISCHARGED: 9.8.37.

Nearly two months in hospital.

Well.

AFTER HISTORY: 1938.

Has had no child since.
CASE 74. (737).

ADMITTED: 8.7.37.

Para 1; 28 weeks maturity. Complaining of cough, palpitation, general weakness and swelling of legs. First attendance.

Yaws.

History positive.

SIGNS AND SYMPTOMS.

Anaemia. Oedema of feet and legs and puffiness of face. Temperature 99.4; pulse 128; respirations 40.

Blood Pressure. 125/55.

Urine.

Trace of albumin.

Faeces.

Liquid specimen; no ova; some flagellates present.

BLOOD EXAMINATION.

Red cells: 700,000.
White cells: 15,600.
Haemoglobin: 20%.
Colour Index: 1.3.
Marked irregularity in size of red cells; polychromatophilia; macrocytes present; nucleated red cells present.

Malaria parasites: negative.

TREATMENT.

Liver and iron and marmite by mouth; Campolon injections.

PROGRESS.

Satisfactory.

15./
CASE 74. (Continued).

15.7.37. Red cells: 1,580,000.
Haemoglobin: 32%.
22.7.37. Haemoglobin: 47%.
29.7.37. Red cells: 3,000,000.
Haemoglobin: 66%.
Colour Index: 1.1.

DISCHARGED: 29.7.37.

Much improved.
Three weeks in hospital.
CASE 75. (747).

ADMITTED: 12.7.37.

Para 1; 32-34 weeks maturity. Complaining of general pains, cough for one week and fever at night. First attendance.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Anaemia. Temperature 100.2; pulse 100; respirations 24.

Urine.

Clear.

Faeces.

Ascaris and hookworm ova and E. Histolytica present.

BLOOD EXAMINATION.

Red cells: 1,540,000.
White cells: 3,000.
Haemoglobin: 42%.
Colour Index: 1.4
Irregularity in size of red cells; nucleated red cells present.
Malaria parasites: negative.

TREATMENT.

Liver, marmite, and iron by mouth; Emetin started 26.7.37.

PROGRESS.

19.7.37. Haemoglobin: 43%.
28.7.37. Haemoglobin: 49%.
4.8.37. Red cells: 3,080,000.
Haemoglobin: 70%.
Colour Index: 1.

DISCHARGED: 9.8.37.

Satisfactory.
One month in hospital.

Note: Improvement much more rapid after Emetin treatment was started.
CASE 76. (817)

ADMITTED: 1.8.37.

Primipara; 36-38 weeks maturity. Supposed to be in labour. Attended five weeks before.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Anaemia. Oedema of feet; temperature 99.4; pulse 108; respirations 28.

Blood pressure: 118/72.

Urine.

Light cloud albumin. Culture sterile.

Faeces.

Semi-solid. Flagellates present and some mucus. Stools frequent. On culture, no non-lactose fermenters present.

BLOOD EXAMINATION.

Red cells: 1,020,000.
White cells: 8,200.
Haemoglobin: 25%.
Colour Index: 1.2.
Malaria parasites: plus.

TREATMENT.

Injections of Campolon; raw liver and iron by mouth.

PROGRESS.

Went into labour one week after admission. Forceps applied for maternal distress; delivered of still-born infant; weight 5 lbs. 2 ozs.

Patient died in third stage as a result of anaemia and heart failure.
CASE 77. (827).

ADMITTED: 5.8.37.

Para 1; 32 weeks maturity. First attendance. Complaining of giddiness, palpitation, cough for four days, swelling of feet and loss of appetite.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Anaemia. Oedema of feet. Temperature 98.2; pulse 88; respirations 24.

Blood Pressure. 132/72.

Urine.

Trace of albumin.

Faeces.

No ova seen.

BLOOD EXAMINATION.

Red Cells: 1,180,000. Sickling of red cells.

Haemoglobin: 27.2.

Colour Index. 1.2

Marked irregularity in size of red cells; nucleated red cells present.

Malaria parasites: negative.

TREATMENT.

Campolon injections; liver, marmite and iron by mouth.

PROGRESS.

Ran some temperature not over 99.8 for a week.
Case 77. (Continued).

Haemoglobin: 47%.
Colour Index: 1.1.

22.8.37. 16 days after admission delivered of living child, weight 4 lbs. 10 ozs.

Puerperium: Non morbid.

31.8.37. Haemoglobin: 53%.

DISCHARGED: 1.9.37.

Over three weeks in hospital.
Satisfactory. Infant not yet reached birth weight.
CASE 78. (864).

Primipara; 38 weeks maturity. Complaining of palpitation and vomiting. Attended one week before and found to be anaemic then.

SIGNS AND SYMPTOMS.

Oedema of feet. Temperature 98.4; pulse 82; respirations 28.

Blood Pressure: 130/68.

Urine.

Cloud of albumin; leucocytes and epithelial cells present. No ova seen.

Faeces.

Liquid. No ova nor protozoa seen.

BLOOD EXAMINATION.

Red cells: 1,310,000.
Haemoglobin: 31%.
Colour Index: 1.1.
Malaria parasites: negative.

TREATMENT.

Campolon injections; liver and marmite by mouth.

PROGRESS.

Ran some temperature, up to 99.4, before delivery.

19.8.37. Red cells: 2,430,000.
Haemoglobin: 49%.
Colour Index: 1.


Puerperium/
CASE 78. (Continued).

Puerperium. Temperature for 4 days after delivery.


DISCHARGED: 30.8.37.

Two weeks in hospital.

Condition satisfactory.
CASE 79. (1137).

ADMITTED: 24.10.37. -

Para 4; 38 weeks maturity. Complaining of giddiness and vomiting for three days. First attendance.

Yaws:

History negative.

SIGNS AND SYMPTOMS.

Anaemia. Temperature 100.8; pulse 124; respirations 22.

Urine.

Light cloud albumin. A few granular casts present. Culture sterile.

Faeces.

Semi-solid. No ova nor protozoa.

BLOOD EXAMINATION.

Red cells: 850,000.
Haemoglobin: 22%.
Colour Index: 1.3.
Malaria parasites: present.

TREATMENT.

Campolon injections; liver, iron, and arsenic by mouth. Had blood transfusion 27.10.37.

PROGRESS.

Satisfactory.

2.11.37. Red Cells: 1,490,000.
Haemoglobin: 35%.

18./
CASE 79. (Continued).

18.11.37. Red cells: 2,260,000.
Haemoglobin: 51%.
Colour Index: 1.1.

DISCHARGED: 2.12.37.
Five weeks in hospital.

AFTER HISTORY:
13 ozs.

Blood Examination.
Red cells: 4,030,000.
Haemoglobin: 75%.
Colour Index: .9.
CASE 80. (1178).

ADMITTED:

Primipara; 28-30 weeks maturity. Complaining of cough for ten days and sore mouth. Three weeks before was noted to be anaemic.

Yaws:

History negative.

SIGNS AND SYMPTOMS.

Anaemia; swelling of feet; sore tongue and lips. Temperature 99; pulse 104; respirations 28. Spleen enlarged.


Urine.

Clear.

Faeces.

Liquid. A few ascaris and hookworm ova present.

BLOOD EXAMINATION.

Red cells: 1,090,000. No sickling.
Haemoglobin: 31%.
Colour Index: 1.5.
Malaria parasites: negative.

TREATMENT.

Injections of Campolon. Liver, marmite and iron by mouth.

PROGRESS.

Satisfactory.
CASE 80. (Continued).

18.11.37. Red cells; 2,570,000.
Haemoglobin: 56%.
Colour Index: 1.1.

DISCHARGED: 24.11.37.

Three weeks in hospital.

AFTER HISTORY:

Reported as having delivered normally by herself.
CASE 81. (1198).

ADMITTED: 8.11.37. -

Para 2; 16 weeks maturity. Complaining of fever at night. First attendance.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Anaemia; oedema of feet; temperature 99.6; pulse 100; respirations 32.

Urine.

Clear.

Faeces.

Ascaris ova present.

BLOOD EXAMINATION.

Red cells: 1,650,000.
Colour Index: .9.
Malaria parasites: negative.

TREATMENT.

Injections of Campolon; liver, marmite and iron by mouth.

PROGRESS.

Satisfactory.

13.11.37. Haemoglobin: 47%.
20.11.37. Red cells: 2,600,000.
Haemoglobin: 53%.
Colour Index: 1.

DISCHARGED: 23.11.37.

Two weeks in hospital.

AFTER HISTORY: 31.8.38.

Delivered normally, living child, weight 6 lbs. 5 ozs.
CASE 82. (1304).


Para 2; 32–34 weeks maturity. Complaining of palpitation and cough for four days. Attended one week before when oedema of feet was present.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Anaemia; oedema of feet; temperature 99.2; pulse 116; respirations 24.

Urine.

Clear.

Faeces.

E. Coli present.

BLOOD EXAMINATION.

Red cells: 1,890,000.
Haemoglobin: 34%.
Colour Index: .9.
Malaria parasites: negative.
Wassermann: negative.

TREATMENT.

Liver, marmite and iron by mouth.

PROGRESS.

Satisfactory.

17.12.37. Red cells: 2,890,000.
Haemoglobin: 46%.
Colour Index: .8.


Two weeks in hospital.
Primipara; 36-38 weeks maturity. Complaining of giddiness and palpitation. Seen one month before; very anaemic but refused offer of admission.

Yaws. History negative.

SIGNS AND SYMPTOMS.

Anaemia. Temperature 100; pulse 100; respirations 28.

Urine.


Faeces.

Ascaris and scanty hookworm ova present, also E. Coli.

BLOOD EXAMINATION.

Red cells: 1,630,000.
Haemoglobin: 35%.
Colour Index: 1.
Malaria parasites: negative.
Wassermann: negative.

TREATMENT.

Campolon injections. Liver, marmite and iron by mouth.

PROGRESS.

Delivered four days after admission, living child, weight 6 lbs. 1 oz.


Haemoglobin: 58%.
Colour Index: .8.


Six weeks in hospital. Progressing satisfactorily.
CASE 84. (1297).


Primpara; 24-26 weeks maturity. Complaining of headache, giddiness and palpitation for four days. First attendance.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Anaemia. Temperature 103.4; pulse 104; respirations 32; spleen enlarged.

Urine.

Clear.

Faeces.

Ascaris ova and flagellates present.

BLOOD EXAMINATION.

Red cells: 1,120,000.
Haemoglobin: 28%.
Colour Index: 1.2.
Macrocytes present.
Nucleated red cells present.
Malaria parasites: plus.

TREATMENT.

Campolon injections; liver, marmite, quinine and iron by mouth.

PROGRESS.

Satisfactory.

20.12.37. Haemoglobin: 45%.
            White cells: 15,000.
            Haemoglobin: 55%.
            Colour Index: 1.


Three weeks in hospital. Improving.

AFTER HISTORY:

Delivered normally; infant weighed 5 lbs. 12 ozs.
Haemoglobin 65%. (Talquist).
CASE 85.  (1293).


Para 1; 28 weeks maturity. Complaining of fever, palpitation and cough for five days. Attended two months before, when anaemia was noticed.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Anaemia. Oedema of feet, face also looks puffy. Temperature 99.6; pulse 124; respirations 26.

Urine.

Trace albumin. Pus and epithelial cells present. No ova seen. Culture sterile.

Faeces.


Sputum. No tubercle bacilli.

BLOOD EXAMINATION.

Red cells: 1,090,000.
White cells: 23,400.
Haemoglobin: 20%.
Colour Index: 1.
Polychromatophilia: nucleated red cells present.
Malaria parasites: negative.

TREATMENT.

Campolon injections; liver, by mouth.

PROGRESS.

Ran temperature to over 100° for two weeks. Stools rather frequent; Widal negative.

20.12.37.

Red cells: 1,630,000.
Haemoglobin: 30%.
White cells: 4,800.
CASE 86. (1316).

**ADMITTED:** 13.12.37.

Para 1; 28 weeks maturity. Complaining of cough and palpitation for one week. Seen two weeks before when anaemia not noted.

**Yaws.**

History positive.

**SIGNS AND SYMPTOMS.**

Anaemia. Oedema of feet. Temperature 99.4; pulse 130; respirations 28.

**Urine.**

Albumin present. Pus and granular casts. On culture B. Coli present.

**Faeces.**

Solid. No ova nor protozoa seen.

**BLOOD EXAMINATION.**

Red cells: 910,000.
White cells: 9,800.
Haemoglobin: 18%.
Colour Index: 1.
Irregularity in shape of red cells; polychromatophilia; nucleated red cells present.
Malaria parasites: negative.

**TREATMENT.**

Campolon injections. Liver, marmite and iron by mouth.

**PROGRESS.**

Ran temperature for five days. Steady improvement.

13.12.37 Red cells: 2,490,000.
Haemoglobin: 54%.
Colour Index: 1.1

**DISCHARGED:** 3.1.38. Much improved. Three weeks in hospital.

**AFTER HISTORY:** 28.2.38.

Haemoglobin: 70%. (Talquist).
CASE 87. (88).

ADMITTED: 13.1.38.

Para 1. 32 weeks maturity. Complaining of lower abdominal pains and weakness.

Yaws. History negative.

SIGNS AND SYMPTOMS.

Anaemia. Temperature 100.2; pulse 120; respirations 28.

Urine.

Albumin present. Pus and granular casts. Culture sterile.

Faeces.

Semi-solid. No ova nor protozoa seen.

BLOOD EXAMINATION.

Red cells: 960,000.
White cells: 8,200.
Haemoglobin: 26%.
Colour Index: 1.4.
Nucleated red cells present.
Malaria parasites: negative.

TREATMENT.

Campolon injections. Liver and iron by mouth.

PROGRESS.

Ran temperature until 19.1.38.

19.1.38. Delivered of macerated foetus, weight 3 lbs. 2 ozs.

Puerperium. Non morbid.

27.1.38. Red cells: 2,380,000
             Haemoglobin: 48%
             Colour Index: 1.
1.2.38. Haemoglobin: 68%

DISCHARGED: 1.2.38.

Two weeks in hospital.

AFTER HISTORY: 2.3.38.

Satisfactory. Haemoglobin 70% (Talquist).
CASE 88. (216).

ADMITTED: 21.2.38.

Primipara; 32 weeks maturity. Attended two weeks before, anaemia not then noted.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Anaemia. Temperature 100; pulse 96; respirations 24.

Blood Pressure: 110/60.

Urine.

Trace of albumin. Scanty epithelial cells and leucocytes present.

Faeces.

Liquid; no ova nor protozoa seen.

BLOOD EXAMINATION.

Red cells: 1,290,000. No sickling.
Haemoglobin: 24%.
Colour Index: 1.
Malaria parasites: scanty.
Wassermann: negative.

TREATMENT.

Campolon injections. Liver, marmite and iron by mouth.

PROGRESS.

Ran temperature for two weeks.

4.3.38. Haemoglobin: 38%.
14.3.38. Red cells: 2,270,000.
Haemoglobin: 51%.
Colour Index: 1.1
21.3.38. Haemoglobin: 59%.

DISCHARGED: 23.3.38.

One month in hospital.
Improving.

AFTER HISTORY.

15.4.38. Normal delivery; weight of child 6 lbs.
CASE 89. (212).

ADMITTED: 21.2.38.

Primipara; 36 weeks maturity. Complaining of slight lower abdominal pains and swelling of feet. Last seen one week before, anaemia not noted.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Anaemia. Oedema of feet. Temperature 99.2; pulse 104; respirations 24.

Blood Pressure. 120/74.

Urine.

Trace of albumin. Culture sterile.

Faeces.

Flagellates present.

BLOOD EXAMINATION.

Red cells: 1,560,000.
Haemoglobin: 28%.
Colour Index: .9
Malaria parasites: negative.

TREATMENT.

Campolon injections; iron by mouth.

PROGRESS.

Satisfactory. Delivered three days after admission; weight of child 5 lbs. 10 ozs.

Puerperium. Non morbid.

11.3.38. Haemoglobin: 33%. No sickling.
25.3.38. Red cells: 2,750,000.
           Haemoglobin: 57%.
           Colour Index: 1.

DISCHARGED: 1.4.38.

Progressing satisfactorily.
Five weeks in hospital.
CASE 90. (237).

ADMITTED: 1.3.38.

Primipara; 23 weeks maturity. Swelling of feet for two weeks. First attendance.

Yaws.

History negative.

SIGNS AND SYMPTOMS.


Urine.

Trace of albumin. A few leucocytes and epithelial cells present. Culture sterile.

Faeces.

No ova. No protozoa.

BLOOD EXAMINATION.

Red cells: 1,960,000.
Haemoglobin: 38%.
Colour Index: 1.
Malaria parasites: negative.

TREATMENT.

Campolon injections; liver, marmite and iron by mouth.

PROGRESS.

Satisfactory.

14.3.38. Red cells: 2,370,000.
Haemoglobin: 51%.
Colour Index: 1.1.

21.3.38. Haemoglobin: 60%.

DISCHARGED: 21.3.38

Three weeks in hospital.

AFTER/
CASE 90. (Continued).

AFTER HISTORY. 21.5.38.

Admitted for labour.

Urine.

Trace of albumin in the urine.
Malaria parasites: plus.

Blood Pressure. 144/90.

Normal delivery in hospital, weight of child
6 lbs. 10 ozs.

CASE 91. (271).

ADMITTED: 14.3.38.

Para 9; 34 weeks maturity. Swelling of feet for one week. Attended one week before, anaemia not noted.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Anaemia. Oedema of feet. Temperature 99.4; pulse 120; respirations 24.

Blood Pressure. 118/70.

Urine.

Trace of albumin, granular casts present. Culture sterile.

Faeces.

Liquid. No ova nor protozoa.

BLOOD EXAMINATION.

Red cells: 1,220,000.
Haemoglobin: 25%.
Colour Index: 1.
Malaria parasites: negative.
Wassermann: negative.

TREATMENT.

Campolon injections. Liver, marmite and iron by mouth.

PROGRESS.

Satisfactory.

22./
CASE 91. (Continued).

22.3.38. Haemoglobin: 32%.

30.3.38. Haemoglobin: 32%.

6.4.38. Red cells: 2,150,000.
Haemoglobin: 58%.
Colour Index: 1.3.
Urine. Trace of albumin. No casts.
Leucocytes present.


DISCHARGED: 28.4.38.

Improved.

Six weeks in hospital.
CASE 92. (290).

ADMITTED: 24.3.38.

Primipara; 26-28 weeks maturity. Complaining of constipation. First attendance.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Anaemia. Temperature 99; pulse 120; respirations 24.

Blood Pressure. 100/60.

Urine.

Clear.

Faeces.

Liquid specimen; scanty hookworm ova present.

BLOOD EXAMINATION.

Red cells: 2,150,000.
Haemoglobin: 48%.
Colour Index: 1.1.
Malaria parasites: negative.

TREATMENT.

Campolon injections; liver and marmite by mouth and Ascaridol.

PROGRESS.

Satisfactory.

29.3.38. Red cells, 3,000,000.
Haemoglobin: 70%.
Colour Index: 1.1.

DISCHARGED: 5.4.38.

Nearly two weeks in hospital.

AFTER HISTORY.

Normal premature delivery on district.
Weight of child, 5 lbs. 6 ozs.
CASE 93. (296).

ADMITTED: 22.3.38.

Primipara; 28 weeks maturity. Fever and pain in the chest for three days. Seen one week before; anaemia not then noted.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Anaemia. Temperature 100.4; pulse 140; respirations 36.

Urine.

Clear.

Faeces.

Scanty hookworm ova present.

BLOOD EXAMINATION.

Red cells: 790,000.
Haemoglobin: 22%.
Colour Index: 1.5.
Malaria parasites: plus. Plasmodium malariae.
Wassermann: negative.

TREATMENT.

Campolon injections; liver, marmite and iron by mouth.

PROGRESS.

Ran temperature before and after delivery for 10 days.

26.3.38. Premature still-birth, 2 lbs. 4 ozs.
30.3.38. Haemoglobin: 38%.
6.4.38. Red cells: 2,200,000.
Haemoglobin: 44%.
Colour Index: 1.
13.4.38. Haemoglobin: 50%.
20.4.38. Haemoglobin: 55%.

DISCHARGED: 26.4.38.

Satisfactory.
Five weeks in hospital.
CASE 94. (352).

ADMITTED: 8.4.38.

Primipara; 30-32 weeks maturity. Fever; headache and cough.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Anaemia. Temperature 101.2; pulse 116; respirations 24.

Blood Pressure. 110/60.

Urine.

Granular casts present.

Faeces.

Flagellates present.

Sputum.

No tubercle bacilli. A few streptococci present.

BLOOD EXAMINATION.

Red cells: 1,600,000.
Haemoglobin: 40%.
Colour Index: 1.2.
Malaria parasites: negative.
Wassermann: negative.

TREATMENT.

Campolon injections. Liver and marmite by mouth; iron and arsenic injections.

PROGRESS.

Not satisfactory at first.

19.4.38. Haemoglobin: 50%.
26.4.38. Haemoglobin: 45%.
3.5.38. Haemoglobin: 48%.
10.5.38. Haemoglobin: 65%.

Blood Pressure. 124/96.

DISCHARGED: 11.5.38.

Did not attend again.
One month in hospital.
CASE 95. (445).

ADMITTED: 9.5.38.

Para 1; 32-34 weeks maturity. Complaining of cough and pains in the thighs for two weeks. Attended one month before.

Yaws.

History negative.

SIGNS AND SYMPTOMS.

Anaemia. Temperature 99; pulse 112; respirations 24.

Urine.

Trace of albumin.

Faeces.

No ova nor protozoa.

BLOOD EXAMINATION.

Red cells: 2,050,000.
Haemoglobin: 45%. Colour Index: 1.1.
Malaria parasites: negative.
Wassermann: weak plus.

TREATMENT.

Liver and iron by mouth. N.A.B. injections.

PROGRESS.

Satisfactory.

16.5.38. Reticulocyte count 22% of red cells.
18.5.38. Haemoglobin: 55%.

DISCHARGED: 24.5.38.

Two weeks in hospital.

AFTER HISTORY: 4.8.38.

Normal delivery, weight of child 5 lbs. 10 ozs.
CASE 96. (629).

ADMITTED: 4.7.38.

Primipara; 22-24 weeks maturity. First attendance. Painful breasts and cough for a week.

Yaws.

History positive.

SIGNS AND SYMPTOMS.

Anaemia. Breasts engorged. Temperature 101.8; pulse 120; respirations 40.

Urine.

A trace of albumin. A few leucocytes and epithelial cells present.

Faeces.

Semi-solid; hookworm ova present.

Sputum.

No tubercle bacilli seen.

BLOOD EXAMINATION.

Red cells: 1,060,000.
Haemoglobin: 40%.
Colour Index: 2.
Malaria parasites: scanty.
Wassermann: double plus.

TREATMENT.

Liver and quinine by mouth and oil of chenopodium.

PROGRESS.

Satisfactory.

12.7.38. Haemoglobin: 55%.
18.7.38. Haemoglobin: 59%. No sickling.

DISCHARGED: 19.7.38.

Two weeks in hospital.
CASE 97. (728).

ADMITTED: 29.7.38.

Primipara; 36–38 weeks maturity. Complaining of palpitation, sleeplessness and headache for four days. Last seen five weeks before.

Yaws. History negative.

SIGNS AND SYMPTOMS.

Anaemia. Oedema of feet, face and abdominal wall. Temperature 98.6; pulse 88; respirations 36.

Blood Pressure. 158/106.

Urine.

Albumin plus. Numerous epithelial cells and leucocytes present. No ova nor organisms seen.

Faeces. Not examined.

BLOOD EXAMINATION.

On admission Haemoglobin 35%. (Talquist).

TREATMENT.

Patient at first treated as toxic albuminuria.

30.7.38. Labour induced by artificial rupture of membranes.

30.7.38. Delivered normally, living child, weight 5 lbs. 12 ozs.


3.8.38. Liver treatment started, by mouth.

7.8.38. Campolon injections started.

9.8.38. Red cells: 1,304,000.

Haemoglobin: 26%. 

Colour Index: 1.


Blood pressure dropped after delivery.

DISCHARGED: 15.8.38.

Two weeks in hospital.

Very much improved.
CASE 98. (750).

ADMITTED: 5.8.38.

Primipara; post-natal. Delivered on district 1.8.38. Complaining of pains in both sides and in the chest; cough and fever since the day after delivery. Had attended out patients two weeks before.

YAWS.

History negative.

SIGNS AND SYMPTOMS.

Anaemia. Oedema of feet. Unhealed perineal tear. Temperature 100.4; pulse 110; respirations 28. Infant alive, weight 5 lbs. 4 ozs.

BLOOD EXAMINATION.

Red cells: 1,000,000.
Haemoglobin: 24%.
Colour Index: 1.2.
Malaria parasites: negative.

TREATMENT.

Prontosil, two tablets three times a day for three days until temperature came down. Campolon injections, liver and iron by mouth.

13.8.38. Haemoglobin: 33%.
White cells: 12,782.
20.8.38. Red cells: 2,970,000.
Haemoglobin: 42%.
Colour Index: .75.

DISCHARGED: 26.8.38.

Progress satisfactory.
Three weeks in hospital.
CASE 92. (907).

ADMITTED: 21.9.38.

Primipara; 28' weeks maturity. First attendance. Admitted for observation.

Yaws.

History positive.

SIGNS AND SYMPTOMS.

Anaemia. Faint jaundice. Temperature 99.4; pulse 124; respirations 24; spleen enlarged.

Blood Pressure. 126/66.

Urine.

Albumin plus. A few leucocytes and granular casts present. No organisms seen.

Faeces.

A few hookworm ova.

BLOOD EXAMINATION.

Red cells: 1,920,000.
Haemoglobin: 34%.
Colour Index: .89.
Malaria parasites: A few.
Wassermann: Weak plus.

TREATMENT.

Liver, iron and quinine by mouth.

PROGRESS.

29.9.38. Haemoglobin: 39%.
7.10.38. Red cells: 2,580,000.
Haemoglobin: 50%.
Colour Index: 1.

DISCHARGED: 12.10.38. Progressing satisfactorily.
Three weeks in hospital.
CASE 100. (910).

ADMITTED: 22.9.38.

Primipara; 38 weeks maturity. Admitted for labour. Last seen nine days before, when discharged, after coming in with false pains. On that occasion malaria parasites plus; Haemoglobin: 60%. (Talquist).

_Yaws:

History negative.

SIGNs AND SYMPTOMS.

Oedema of feet. Temperature 98.6; pulse 80; respirations 24.

Urine.

Clear.

Faeces.

Semi solid. No ova, no protozoa.

BLOOD EXAMINATION.

On admission: Haemoglobin 45%. (Talquist) Malaria parasites: negative.

TREATMENT.


24.9.38. Treatment for anaemia with liver, by mouth, started.

PROGRESS.

24.9.38. A normal delivery. Weight 5 lbs. 11 ozs.

Puerperium. Non morbid.

23.9.38. Red cells. 1,680,000.

Haemoglobin: 30%.

Colour Index: .9.


DISCHARGED: 1.10.38.

Anaemia improving.

Ten days in hospital.
<table>
<thead>
<tr>
<th>TABLE 1.</th>
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</table>

PARITY, MATURITY, RED CELL COUNTS, HAEMOGLOBIN PERCENTAGES and COLOUR INDICES of 100 cases of Macrocytic Anaemia on admission as In-Patients at the Maternity Hospital, Accra, from 1928-38.
TABLE 1.

Re-ad. = re-admission.

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<tr>
<th>Case No.</th>
<th>Parity</th>
<th>Maturity in weeks</th>
<th>Red Cells.</th>
<th>Haemoglobin Percentage</th>
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Subsequent pregnancy.
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Subsequent pregnancy.
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Postpartum.
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Undifferentiated white cells 11.4%.

Megalocytes present.
TABLE 3.

ROUTINE BLOOD EXAMINATIONS made by the writer in Kumasi to discover to what extent anaemia was present without definite symptoms among the pregnant women. Only patients who did not at first sight appear to be anaemic were tested. Haemoglobin was estimated by a Sahli Haemoglobinometer.

SUMMARY OF FINDINGS.

NUMBER OF COUNTS carried out 100.

MALARIA PARASITES present in 23.

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COLOUR INDEX OF UNITY OR OVER - present in 23.

RED CELL COUNTS:

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TABLE 4.

ROUTINE ESTIMATIONS OF HAEMOGLOBIN carried out by the writer to discover the degree of anaemia without symptoms among the pregnant women attending the Maternity Hospital, Accra, as out-patients.

SUMMARY OF FINDINGS.

NUMBER OF EXAMINATIONS CARRIED OUT:— 381.

The Sahli Haemoglobinometer was used.

MALARIA PARASITES present in 94 cases, or 24.6%.

HAEMOGLOBIN VALUES in 381 CASES:—

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Routine Laboratory Examination of In-patients at the
Maternity Hospital, Accra.

Blood.

Malaria.
Number of examinations made 6925
Malaria parasites present in 27%.

Wassermann Reaction
Number of examinations made 5335
Positive Wassermann rate 32.5%.

Faeces.

Number of examinations made 1372
Ascaris ova present in 17%
Ankylostome ova " 16%
Taenia ova " 1 case.
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GÄNSSLER/
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