A COMPARATIVE STUDY OF IRON DEFICIENCY IN THE INDIAN AND THE AFRICAN IN DURBAN

Thesis submitted for the
Degree of Doctor of Medicine

by

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November, 1963.

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Chapter 1

INTRODUCTION

Anaemia due to iron deficiency in young women has been widely observed for centuries though not recognised as such. Beutler (1963) suggests that Shakespeare probably had such a picture in mind when he wrote:

"She never told her love,
But let concealment, like a worm i' th' bud,
Feed upon her damask cheek: she pin'd in thought;
And, with a green and yellow melancholy,
She sat like Patience on a monument."

(Twelfth Night Act II Sc. IV.)

It is not known when iron was first used in the treatment of this disease. It is reported that in ancient Greece anaemia was recognised and the empirical treatment for it consisted of drinking water in which a sword had been allowed to rust. If one goes through the literature one observes that although the condition of iron deficiency had long been recognised, very little headway was made in this field until the present century when Whipple and Robscheit-Robbins (1925) acknowledged the therapeutic efficacy of inorganic iron in the anaemia of blood loss.

The historical background has been well reviewed by Heath and Patek (1937) and Beutler et al. (1963). Amongst the more important

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names in the early literature they place those of Sydenham, Menghini and Blaud. Sydenham in 1661 was the first to recognise the therapeutic value of iron in chlorosis although he had a misconception of the true nature of the disease itself. Iron continued to be used without rationale until Menghini in 1746 drew attention to the presence of iron in the blood. In 1832 Blaud introduced his pills for the cure of chlorosis, at the same time emphasising the importance of using high iron dosage. All these observations were confirmed and established with more accurate techniques by Heath, Strauss and Castle (1932), who also demonstrated that only a minute fraction of an oral dose of iron is absorbed and utilised in haemoglobin synthesis.

While this was going on theories as to the aetiology of chlorosis were still archaic. This syndrome was not given recognition as a separate entity despite the above observations on iron therapy, and iron continued to be used empirically. Chlorosis was attributed among other things to such factors as the abnormal functioning of the ovaries and was regarded by some as an illness in young girls which was difficult to distinguish from hysteria.

It was not until very recently when Witts (1930) described this condition and put it into its proper perspective, that iron deficiency became established and recognised as a specific entity which required iron for its cure. He was probably among the first to clarify the various eponyms under which this syndrome masqueraded. He described the typical

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clinical picture of what we today regard as iron deficiency. He analysed 6 cases of chlorosis, defining the condition as a secondary type of anaemia occurring spontaneously in adolescent females, and 50 cases of simple achlorhydric anaemia which is far more common in females and has its highest incidence between the ages of 40 and 50 years. The chief symptoms and signs in order of frequency were: anaemia, glossitis, splenomegaly, dyspepsia, diarrhoea, sore mouth or throat, menorrhagia, dysphagia, oedema, amenorrhoea, koilonychia (4 patients), acroparaesthesiae and angina. Anaemia and symptoms such as generalised weakness, malaise, dyspnoea and palpitations were complained of in 39 cases. Some patients, even with a haemoglobin level of 50%, had no symptoms. The diagnosis was made by finding a reduced colour index in the presence of anaemia and in most cases achlorhydria. The differential diagnosis included diseases such as alimentary neoplasms and haemorrhoids producing secondary anaemia. Menorrhagia was often associated with simple achlorhydric anaemia but in many cases this was thought to be secondary to the anaemia and not primary.

In his papers at the time he had regarded achlorhydria, especially in the anaemia occurring in later life, as an essential feature of the illness; but more recently he no longer holds this view and recognises that achlorhydria was given undue prominence (Witts, 1956).

When the literature on simple achlorhydric anaemia, chlorosis and idiopathic hypochromic anaemia was reviewed it was found that most of the patients on analysis had evidence of some source of blood loss which probably, together with multiple pregnancies and a poor dietary intake, gave rise to iron deficiency (Gray et al., 1936; Heath and Patek, 1937).

Just as there was no clarity in the past about iron and its use in anaemia, up to very recently there have been very vague ideas about the fundamental concepts of iron metabolism, a state of affairs due largely to inadequate methods of study.

An average adult male has a total of 4-5 grams of iron in his body and this is distributed in different forms, the amount of iron in each of these components varying widely in health and disease. Most of the iron present in the human body is found in haemoglobin. Similarly Hahn (1937) estimated that 57% of the total iron in the dog was contained in blood haemoglobin, 23% was in the myohaemoglobin, cell enzymes and so on, while 20% was made up of storage iron, which is said to be the compartment the iron content of which fluctuates most. The total iron stored in man, available for blood formation, is estimated to be about 1200 mg. - 1500 mg. (Haskins et al., 1952).

Iron is obtained from the diet, and for its absorption several factors have to be considered. First of all its availability is of

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importance since phytates and phosphates in the diet appear to retard its absorption (McCance et al., 1943; Kinney et al., 1950; Sharpe et al., 1950). Ascorbic acid enhances its absorption from food (Moore et al., 1952) and hydrochloric acid, contrary to earlier belief, has no such effect (Beutler, 1965). Ferrous iron is better absorbed than ferric iron (Moore et al., 1939; McCance et al., 1943), and ferrous chloride is more easily absorbed than food iron (Chodos et al., 1957). Studies with radioactive techniques on absorption of iron when given with food (Pirzio-Biroli et al., 1958) have demonstrated that iron deficient subjects absorb more iron than do normal subjects. Iron absorption can occur through the stomach and almost any portion of the gastro-intestinal tract, but it appears to take place chiefly and most efficiently through the upper portion of the small intestine (Stewart et al., 1950; Brown et al., 1958).

Regarding the mechanism of transfer of iron across the bowel mucosa, this is unsettled as yet. Granick (1946) had suggested that the protein apoferritin is continually being formed in the mucosal cells. In response to iron orally, this substance increases in concentration. Ferritin accumulates in these cells. The iron, it was postulated, forms a complex with apoferritin to form ferritin. The mucosal cells regulate iron absorption by maintaining a certain level of ferrous iron. The latter was said to be in equilibrium with the ferritin in the mucosal

/cells ...

cells and the plasma iron in the blood stream. No further iron could be absorbed as long as this balance was maintained. According to this hypothesis a lowering of the plasma iron would result in more rapid movement of iron out of the mucosal cells, depleting the stores of ferritin iron and finally lowering the concentration of ferrous iron in the mucosal cells. Increased absorption from the gastro-intestinal tract would then take place. This theory of a "mucosal block", preventing absorption when iron is not needed, was widely accepted. According to Dubach et al. (1948) this theory that mucosal cells accept iron for absorption or block its assimilation provides the best known explanation, but patients with adequate stores may assimilate considerable quantities of the metal and the block must be regarded as relative.

Recently, however, with radioactive and other advanced techniques it has been shown that there is no "absolute block" to iron absorption (Bothwell et al., 1958; Brown et al., 1958; Smith et al., 1958). Multiple factors seem to be involved which Beutler et al. (1963) list. These include hypoxia (Mendel, 1961), the rate of erythropoiesis and the level of iron stores (Bothwell et al., 1958; Pirzio-Biroli et al., 1960), the latter two probably being the most important.

The level of saturation of the iron-binding protein has also been suggested to play a role in iron absorption (Laurell, 1952) but other work has not confirmed this (Dubach et al., 1948; Yuille et al., 1950; Bothwell et al., 1958; Pirzio-Biroli et al., 1960; Wheby et al., 1963).

According to Pirzio-Biroli et al. (1958), in addition to excessive iron stores and decreased erythropoiesis, there are conditions which may act at the mucosal level to impair iron absorption, such as idiopathic steatorrhoea, myxoedema, infection and post-gastrectomy state in the iron-deficient patient.

How these different factors operate is still not clear. All one can say is that iron is obtained by absorption from the gut, and that the gastro-intestinal mucosa is regarded as the chief regulator of iron balance.

Once absorbed, the iron is bound to the specific transport protein, transferrin, and is transported in the plasma (Hahn et al., 1939) to the tissues where it is stored as ferritin and haemosiderin. It goes largely to the bone marrow and the liver but some finds its way to all the tissues to fulfil the requirements for enzyme iron.

After gaining entry into the body, iron is very strictly conserved and is used over and over again for haemoglobin synthesis. This is possible because the loss of iron from the body is very small (Dubach et al., 1955; Finch, 1959). The total amount leaving the body is approximately 1 mg. daily and is found in the urine, faeces, sweat and hair. Much of this iron, particularly that in the sweat, probably represents the enzyme iron of the cells which are desquamated from body surfaces (Beutler, 1962).

The comparative study of iron metabolism which this thesis presents materialised not because iron deficiency is a world-wide problem to-day but because it has been said in Natal that iron deficiency is common in the Indian but rare in the African, if it occurs at all. I have attempted to discover on the one hand, to what extent iron deficiency is a problem in the Durban Indian, trying to unravel factors contributing to its production, and on the other hand, its incidence in the local African.

The thesis is divided into three main sections:

(i) The relative incidence, clinical picture and probable aetiological factors involved in the production of iron deficiency anaemia (Chapter 2).

(ii) An ante-natal study to observe the effects on the iron stores of the increased demand for iron during pregnancy in the different racial groups (Chapter 3).

(iii) The state of the iron stores as reflected in the bone marrow and liver specimens obtained at necropsy (Chapter 4).

Chapter 2

A STUDY OF PATIENTS WITH IRON DEFICIENCY ANAEMIA ADMITTED TO HOSPITAL

This part of the study comprises a full investigation of patients who were admitted for investigation of anaemia to a medical ward in which the proportion of Africans to Indian admissions was approximately 4:1.

During the period of study there were 54 patients diagnosed as suffering from iron deficiency anaemia, of whom 43 were Indian (79.6%) and 11 African (20.4%). This number was made up of 38 females of whom 32 (84.2%) were Indian, and 16 males 11 of whom (68.8%) were Indian.

AGE DISTRIBUTION

The mean for the total group was 33.3 years with a range of 7 to 78 years. Figure 2.1. gives a better idea of the age scatter of the group as a whole while figure 2.2. illustrates the age distribution amongst Indian females who form the bulk of the study.

DIAGNOSIS

This was based on peripheral blood picture, the levels of serum iron and iron binding capacity of the serum and lastly the bone marrow picture.

Methods

Haemoglobin (Hb) was estimated as oxyhaemoglobin, the haematocrit determined and reticulocytes counted by the methods of Dacie (1956).

FIG. 2.1. NOMOGRAM OF AGE DISTRIBUTION OF THE TOTAL GROUP.

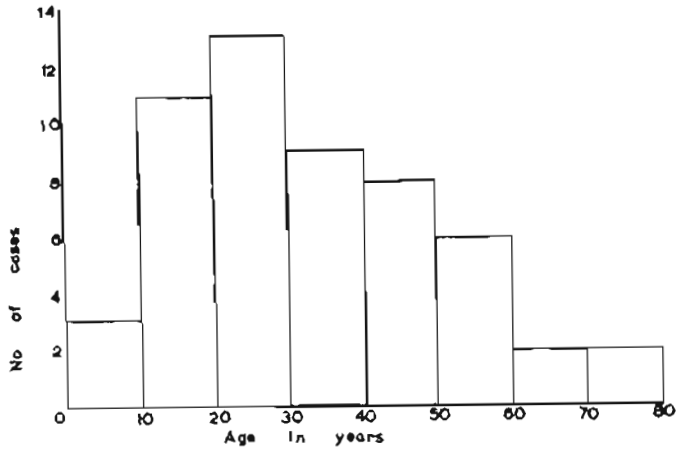
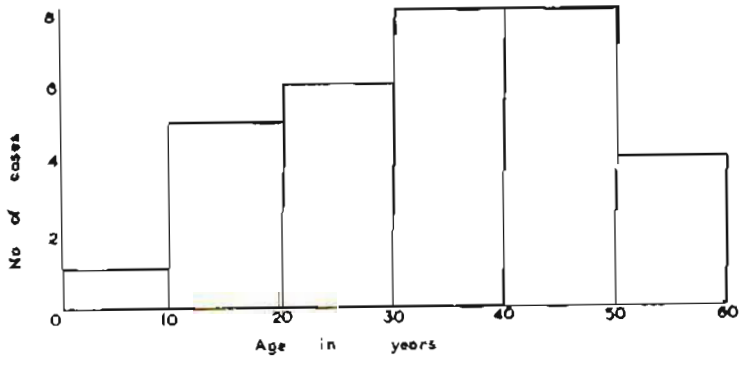


FIG. 2.2. AGE DISTRIBUTION AMONGST INDIAN FEMALES



Later the packed cell volume (P.C.V.) was estimated by the micro-haematocrit method (Wintrobe, 1963).

Blood and bone marrow smears were stained with May-Gruenewald stain and counter stained with Giemsa (Dacie, 1956).

All specimens of the bone marrow were obtained by sternal aspiration except in the case of children when iliac crest specimens were taken.

The marrow smears were also stained for iron stores using hydrochloric acid and 3% Potassium Ferrocyanide. These were graded in accordance with the amount of iron present: grade 0 representing the absence of iron with grade VI at the other extreme; grades I to III being regarded as normal amounts of iron (Rath and Finch, 1948). This investigation helps to exclude the cases of hypochromic anaemia associated with infection; the differential point being the deposition of normal or increased amounts of haemosiderin in the bone marrow in the other types of anaemia and that of infection as against its depletion in iron deficiency anaemia (Rath and Finch, 1948; Davidson et al., 1952; Stevens et al., 1953). All these were examined personally.

Serum iron was estimated by the method of Bothwell and Mallett (1955), while the radioactive Fe^{59} technique described by Bothwell et al. (1959) was used for the estimation of the unsaturated iron binding capacity (U.I.B.C.). The counting was done on 5 ml. specimens in a well-crystal scintillation counter. The total iron binding capacity (T.I.B.C.) was derived from the sum of the above two readings, and the

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percentage saturation calculated from the proportion of serum iron to total iron binding capacity. All glassware, including syringes, was made iron free before use and in almost all cases the blood was collected on the morning of the day after admission, bearing in mind the reported diurnal variation of serum iron levels - hypoferraemia occurring in the evenings (Powell, 1944; Patterson et al., 1952). These conditions and methods apply to all sections of this thesis. These estimations were initially done by the author and later by Miss A. Darling. All haematological investigations were done in the research laboratory of the Department of Medicine.

Results

Peripheral blood picture on admission: The Indian and African mean levels and ranges have been combined because these were comparable for each group.

The mean Hb level for the total group was 6.3g. per 100 ml., ranging from 2.1 - 9.6 g. per 100 ml., the lowest level amongst the Africans being 4g. per 100 ml.

The overall mean corpuscular haemoglobin concentration (M.C.H.C.) was 25.9% ranging from 22 - 31%.

The reticulocyte count ranged from 0.2 - 10.4% with a mean of 4.2%.

The peripheral smear contained hypochromic cells usually with microcytes.

Iron picture as assessed by the level of the serum iron and the iron binding capacity. The mean serum iron level in my patients was 17.4µg. per 100 ml., ranging from 0 - 39µg. per 100 ml. (µg.%). The T.I.B.C. was 409.5µg.%, with a range from 249-620µg.%. The overall mean percentage saturation was 4.4%, that for the Indians being 3.6% while the African figure was 5.1%.

Bone marrow examination: Most of the smears showed normoblastic erythropoiesis. As the histopathology of the bone marrow cannot be considered as diagnostic (Beutler et al., 1954) these smears were examined for iron stores. The smears were examined for iron in 50 patients and it was found that while 43 had no iron deposits in the marrow at all, 7 had traces (grade 0 - I). Hence the depleted stores in these patients confirmed the diagnosis of iron deficiency.

CLINICAL FEATURES

There may be a marked variation in the clinical manifestations of iron deficiency anaemia, and the incidence of the different symptoms and signs with which it commonly presents also varies in the descriptions given by different authors. The frequency with which the different symptoms and signs occurred in our patients is set out in table 2.1.

Symptomatology

The duration of illness ranged from a week to 8 years with an average of 8 months.

By far the commonest symptom was that of tiredness, and varying

/degrees ...

Table 2.1.

CLINICAL FEATURES
(in order of frequency)

Symptomatology	No. of cases
Dyspnoea on exertion and tiredness	40
Palpitations	20
Swelling	18
Malaise and weakness	17
Painful joints and backache	16
Dyspepsia, abdominal discomfort and pain	14
Headache	10
Frequency with or without dysuria	8
Loss of appetite	7
Oligo/hypomenorrhoea	7
Diarrhoea	7
Pain in the chest (?angina)	6
Sore throat and dysphagia	6
Dizziness	6
Cough	5
Feverishness	4
Amenorrhoea	4
Menorrhagia	3
Vomiting	3
Swelling in the neck (thyroid)	3
Constipation	3
Paraesthesiae	2
Pica	1
Skin desquamation	1
Melaena	1
Yellow eyes	1
Pruritus vulvae	1

Signs	No. of cases
Hepatomegaly	32
Koilonychia	29
Splenomegaly	26
Glossitis	17
Oedema	16
Skin and hair changes	6
Epigastric tenderness	5
Angular stomatitis and cheilosis	3
Thyroid enlargement	3
Mental changes	2
Sternal tenderness	2
Dental caries	1
Thyrotoxicosis	1

degrees of dyspnoea on exertion were present. Amongst the more frequent complaints were palpitations, malaise and weakness, swelling of the ankles, generalised pains and headache. About 26% (14 patients) had dyspeptic symptoms with or without vague abdominal pain, and only 2% of these patients had achlorhydria associated with these symptoms (cases 8 and 39).

Eleven per cent of the patients, all Indian females, complained of dysphagia. The average Hb for this group was 8.1 g. per 100 ml., their average age was 37.3 years while the duration of their illness varied from 2 months to about 8 years with a mean of 30 months. Four of these patients had glossitis, 2 (cases 5 and 8) had histamine-fast achlorhydria after maximal stimulation while 3 had splenomegaly. All except one (case 23), had koilonychia, thus demonstrating the correlation between koilonychia and other mucous membrane changes. The other gastro-intestinal symptoms complained of were loss of appetite, diarrhoea more frequently than constipation, and vomiting.

Six patients (11%) had pain in the chest which was suggestive of angina. Hunter (1946) found an incidence of 25% (8 out of 34 cases) and also that it was commoner in females. Five of my 6 patients were females, a little higher than expected taking the sex incidence of the whole series into account (38 females : 16 males) but the numbers are too small to draw firm conclusions.

Signs

Liver enlargement was detected in 59% of the patients while splenomegaly was present in 48%. There appeared to be no correlation between the Hb level and leucopaenia in patients with splenomegaly confirming the finding of Witts (1930).

Koilonychia figures prominently in all the earlier descriptions of iron deficiency anaemia (Witts, 1930; Wintrobe et al., 1933), but recent work suggests that it now occurs less frequently (Beutler, 1963). One possible explanation of this is that severe degrees of iron-deficiency are becoming less common, koilonychia being associated with severity of anaemia. Among my patients, however, the average Hb level of those with koilonychia was 5.7 g. per 100 ml. and 6.5 g. per 100 ml. among those without koilonychia, indicating that the correlation is at most only slight. The duration of the apparent illness, said to be an important factor, in my cases varied from a week to 8 years, with an average of about 9 months. It has been suggested that there may be an association between koilonychia and mucous membrane changes. Amongst my cases 5 of the Indian females with koilonychia had associated dysphagia. It has been postulated that unknown genetic factors may be responsible for its production, or is it perhaps a racial characteristic? Various degrees of this physical sign were present in 53.7% of my patients these being 63.6% of the Indian females, 54.5% of Indian males and 50% of African females, while none of the African males had koilonychia; the incidence among all the Indian patients being fairly high (60.5%) when compared with all the African patients (27.3%). Is

/this ...

this marked difference between the two groups perhaps due to the near depleted iron stores in the Indian as observed in the storage study, and hence an earlier demand on the iron present in the cell enzyme systems? The latter is probably the last port of call for iron after the stores have been depleted in the iron deficiency states. The reverse also applies here because, as the African has been found to have increased iron stores in the tissues (Gillman et al., 1945; Wainwright, 1957; Bothwell et al., 1960 and also from the iron storage studies reported in Chapter 4), it follows that the demand on the enzyme iron is not as great, and besides the koilonychia amongst the African patients has only been demonstrated in the females whose stores on the whole normally are not as heavily laden with iron as the males.

As far as the cardiovascular system is concerned, not all patients had tachycardia, pulse rates on admission ranging from 68 - 132 per minute. The average blood pressure was 125/70. Out of the 54 patients, clinical cardiomegaly was present in 12 and a gallop rhythm was heard in 7. Excluding one patient with chronic rheumatic heart disease and congestive cardiac failure (case 48), 43 patients had heart murmurs. A short early systolic murmur was present in 37 patients, 4 had a pansystolic murmur, while 2 had an early blowing diastolic murmur at the base. Five of these patients were in congestive cardiac failure. The Hb level in the 7 patients with pansystolic murmurs, early diastolic murmur or with congestive cardiac failure was

/3.7 g. ...

3.7 g. per 100 ml. while that of the 2 patients who had the early diastolic murmur was 2.3 g. per 100 ml. This demonstrates the correlation between the Hb level and the degree of the cardiac involvement (Wintrobe, 1946), which is not in agreement with the conclusion of Hunter (1946) that the duration of the illness is more important than the severity of the anaemia.

Thus the clinical picture observed was that of iron deficiency anaemia as described by other authors. Despite the ease with which the early diagnosis and treatment of iron deficiency anaemia can be established, it is surprising that one still sees all these gross manifestations of the disease, the picture very closely resembling the description given by Witts in 1930.

OBSTETRICAL AND GYNAECOLOGICAL HISTORY

Obstetrical history (Parity)

The average parity of the Africans was 3.8, ranging from 0 - 6, while that of the Indians was 4.3 ranging from 0 - 10 previous pregnancies. This figure excludes 11 of the Indian females (34.4%) who were single, 2 of them were premenarche (cases 6 and 19). Case 44, an African female, had 4 caesarean section deliveries. None of the patients was pregnant. Various forms of menstrual disorders were present, as can be seen from the table. In most instances it was decided that these were probably secondary to the anaemia. Whenever indicated, a diagnostic curettage was done in order to exclude local pathology.

Table 2.2.

FREQUENCY OF GYNAECOLOGICAL SYMPTOMS

Symptoms	No. of Cases	
	Indian	African
Oligo/Hypomenorrhoea	7	-
Post-menopausal	3	2
Amenorrhoea	3	1
Menorrhagia	3	-
Pre-menstrual	2	-

Case No. 9 was an Indian who was 51 years old, one year post-menopausal and nulliparous. She, however, gave a vague history of probable menorrhagia and on curettage she was found to have had endometrial polypi.

Of the 3 patients with menorrhagia one, who had reached her menarche about a year previously, had a good response to iron therapy and it was decided that she should be observed rather than investigated further. At follow up examination she said that she no longer had any menstrual disturbance (case 11). Another, a 34-year-old Indian female who had had 4 pregnancies, was found to have a proliferative endometrium (case 10), while the third showed a normal endometrium after curettage, and her menses were said thereafter to have become normal.

Of the 2 patients with irregular menses and a suggestion of increased blood loss during menstruation, one was shown to have a normal endometrium (case 16), and the other inflammatory changes (case 8).

DIETARY HISTORIES

The histories obtained are inadequate and probably unreliable in many instances. All one can attempt to do here is to give a broad classification of the type of diet consumed by dividing the patients up into groups. The diet was regarded as:-

(i) good when meat and vegetables were consumed daily,

(ii) adequate if these were taken more than once a week,

and (iii) poor if either one or both were consumed once a week or less.

According to these criteria 4.3% had a good meat intake while 43.5% and 52.2% had adequate and poor diets respectively. As for the consumption of vegetables, 34.3% had a good intake while 21.7% had adequate and 24% consumed vegetables seldom or not at all. Of these patients none of the Africans consumed meat daily, while only one had adequate amounts. Fifty per cent of the Africans seldom or never ate vegetables. One can conclude that the protein intake of both these groups is poor on the whole. Although a fair amount of vegetables was eaten by the Indians most of it was eaten cooked. In most of the patients studied, the bulk of the diet, it would appear, is made up of rice amongst the Indians and maize products amongst the Africans.

SPECIAL INVESTIGATIONS (BIOCHEMICAL AND RADIOLOGICAL)

As far as possible a series of investigations was done to exclude any cause of blood loss or to detect any other evidence which

/might ...

might account for iron deficiency anaemia.

Urine

Specimens of urine on 3 consecutive days were examined in all cases, and these were found to be abnormal in 13. Three patients had ova of Schistosoma haematobium (cases 35, 46, 51). A mild urinary infection was present in 7 patients while in 3 others scanty red blood cells were observed (cases 14, 18, 44).

Stool specimens

Three stools were examined in every case for the presence of parasites and their load, and for occult blood. The Gregersen test was used for detecting blood in the stools (Fordhaw et al., 1954) and the load was expressed as the number of ova per coverslip. The frequency of infestation is set out in table 2.3.

Table 2.3.

PARASITIC INFESTATION ON STOOL EXAMINATION

Ova	No. of cases
Trichocephalus	24
Ascaris	12
Hookworm	11
Entamoeba coli	6
Entamoeba hartmanni	2
Ova of Schistosoma, Giardia lamblia, Strongyloides and Enterobium	1 of each
Trophozoites of E.histolytica	

Eleven of the patients had normal stools while a variable number of ova

/of ...

of ascaris or trichocephalus were found in 28 cases. Fifteen patients had varying amounts of occult blood in their stools; 3 were associated with cirrhosis of the liver (cases 5, 22, 30); 6 associated with hookworm infestation (cases 6, 8, 29, 47, 49, 52); 2 with peptic ulceration (cases 36, 43); 2 with malabsorption (cases 45, 54); one with chronic gastrointestinal blood loss where the cause had not yet been determined (case 53); while one patient had a weakly positive occult blood test for which no cause was established (case 17).

Two patients (cases 27 and 51) had pus, blood and mucus in their stools while the latter also had trophozoites of Entamoeba histolytica for which treatment was given.

Hookworm ova were found in 11 patients of whom 7 were Indian females, one an Indian male, 2 African females and one African male. Wherever practicable, if hookworm ova were detected in any of the 3 stool specimens, the patients were given bephenium sulphate (a single dose of 5.0 g.) and a worm count was done on 2 consecutive specimens of 24-hour stools immediately after therapy. A week thereafter the ova count was repeated and this was done again a month later if the patient attended the follow up clinic, in order to assess the efficacy of treatment. In one patient (case 49) because of the persistence of the infection and the occult blood in the stool the therapy was repeated.

Table 2.4.

STOOL ANALYSIS ON PATIENTS WITH HOOKWORM INFESTATION

Case No.	Ova Count	Occult blood	Worm Count	Immediately after treatment: Ova Count	Immediately after treatment: Occult blood	One month after treatment: Ova Count	One month after treatment: Occult blood
49	70	++	265 adults	30	++		
	61	++	(135 M., 135 F.)	29	++		
	62	++					
29	95	++	26 adults	6	+	11	-
	68	-	(13 M., 13 F.)	27		9	-
	10	-		31			
52	18	+	10 adults	7	+		
	14	-	(5 M., 5 F.)	12	-		
	22	-	2 ascaris				
47	4	++	14 adults	18	++		
	1	+	(5 M., 9 F.)	c.	+		
	4	+	Taenia saginata				
20	c.	+					
	c.	-					
	-	-					
6	c.	+					
8	c.	-	no worms	nil	-	-	-
	c.	-	recovered				
	c.	-					
31	2	-	no worms	-	-		
	-	-	recovered				
	-	-					
32	2	-	2 adults (F.)	2	-	-	-
	c.	-	1 ascaris	c.	-		
	c.	-	19 Enterobius(F.)	6	-		
38	1	-	1 adult (F.)	-	+		
	1	-		c.	-		
	c.	-		c.	+		
14	-	-	no worms				
	-	-	recovered				
	-	-					

++ = strongly positive
+ = weakly positive
- = negative
c. = ova obtained on concentration of stool.

M = adult male
F = adult female

It will be noted that only 6 of the 11 patients with hookworm infestation had occult blood detected at the time of examination. Three of these patients (cases 29, 49 and 47) had a strongly positive test and from them 26, 265 and 14 adult worms of the Necator americanus species respectively were recovered. If one takes 5 - 20 ova as an indication of a moderate infection it will be seen that one patient who had a weakly positive occult blood and from whom 10 adult worms were recovered, falls into this category (case 52), while 2 others, an Indian and an African female, fall into the heavy infestation group, both having had strongly positive occult blood in the stools (cases 29, 49).

Liver function tests

Standard tests for serum bilirubin, alkaline phosphatase, zinc and thymol turbidities and serum proteins, together with the albumin : globulin ratio were done on all cases but one (case 45), while the serum proteins only were estimated on one African male (case 52) (see Joubert et al., 1959). The individual values are given in table 2 (Appendix, page A.57).

The percentage of patients with abnormal liver function tests (excluding those in whom associated liver disease was found on clinical grounds) is shown in table 2.5.

Table 2.5.

PERCENTAGE WITH ABNORMAL LIVER FUNCTION TESTS

Tests	Indian females	Indian males	Indian average	African females	African males	African average
Serum bilirubin	13.8	10	12.8	0	0	0
Alkaline phosphatase	0	10	2.6	0	50	16.7
Zinc turbidity	69	70	69.2	100	100	100
Thymol turbidity	41.7	20	35.9	50	50	50

Thus it can be concluded that the liver function tests, as judged from the above results, are grossly abnormal even in the absence of obvious liver disease. The zinc turbidity was abnormal in all Africans and although my series comprises only 6 cases these figures compare well with the 84% as found by Powell (1958) in a group of normal Africans from a similar socio-economic group. The turbidities are also abnormal in a fair proportion of Indians but this figure, however, differs from that obtained by Joubert et al. (1959) who consistently found the tests in the latter group to be within the European range. It is doubtful, however, if the subjects studied by the latter authors are representative of admissions to King Edward VIII Hospital. The serum proteins done by a chemical fractionation method (see Joubert et al., 1959) were also abnormal in both the racial groups, even more so in the African, as compared with the European.

Protein electrophoresis

Protein electrophoresis was done (see Joubert et al., 1959) on 19 Indian females, 8 Indian males, 4 African females and 4 African males. See table 3 (Appendix, page A.59). Although the total proteins are

/abnormal ...

abnormal in only 45.8% and 40% of the 24 Indians and 5 Africans respectively, in those without any obvious liver disease, the serum albumin is abnormally low in all the patients of both racial groups, with a reversal of the albumin : globulin ratio. As with chemical fractionation, the serum albumin is lower in the African compared with the Indian patients, while the serum globulin is elevated much more than in the latter group, giving an albumin : globulin ratio of 0.5/1 in the Indians and 0.3/1 in the African. Taking the individual globulin fractions, there is a gross generalised disturbance in both racial groups. The quantity of alpha$_1$ globulin is the same in the two groups while the Africans have a higher level of alpha$_2$ and beta globulins. The main difference between these two racial groups lies in the level of the gamma globulins which are much more elevated in the African.

Granted the numbers are small to draw any conclusions, particularly in the case of the Africans, the general trend of low serum albumins and elevated globulins in the latter is consistent with that found by other authors (Powell, 1958; Joubert et al., 1959). Reduction in plasma proteins and albumin fraction are reported to be common in anaemia (Heath et al., 1936) and the findings here are consistent with this. However, when one is dealing with a group whose serum proteins are usually reported to be abnormal (Powell, 1958; Joubert et al., 1959), one wonders if this plays any major role in the formation of the haemoglobin molecule or in the binding capacity of iron in the serum.

Vitamin C level

This was estimated by the dinitrophenylhydrazine method of King and Wootton (1956). Blood samples were taken prior to the consumption of any hospital food.

This estimation was done on 24 Indian females out of the 32, their mean being 0.49 mg. per 100 ml., ranging from 0 - 1.9 mg. per 100 ml. Four patients had no vitamin C in their serum (cases 4, 13, 14, 24). No other cause could be found for the iron deficiency in 3, whilst one was malnourished, had a light hookworm infestation and barium meal examination was suggestive of duodenal ulceration (case 14).

The mean for the 11 Indian males was 0.98 mg. per 100 ml., ranging from 0 - 2.4 mg. per 100 ml. One patient with no vitamin C in the serum had no other detectable factors causing his anaemia.

The estimations on the 5 African females ranged from 0.9 - 2.5 mg. per 100 ml., the mean being 1.6 mg. per 100 ml.

Of the African males the average on 3 cases was 0.45 mg. per 100 ml., ranging from 0 - 1.06 mg. per 100 ml. One patient who had no vitamin C in his blood, suffered from cirrhosis of the liver with congestive splenomegaly (case 50).

The vitamin C level in normal healthy individuals (in England) has been quoted variously as 0.4 to 2.0 mg. per 100 ml. and 0.3 to 1.3 mg. per 100 ml. with an average of 0.65 mg. per 100 ml.; but what is more

/significant ...

significant is the absence of vitamin C from the blood. If we regard 0.3 mg. per 100 ml. to be the lower limit of normality we find that amongst the total of 35 Indians on whom this estimation was done, 12 (34.3%) had readings below this level, 5 of these (41.6%) having no vitamin C in the serum.

Amongst the Africans on the other hand only one out of the 8 (12.5%) had no vitamin C in the blood, the rest having serum levels above 0.3 mg. per 100 ml. None of the patients in the series had clinical signs of scurvy.

Is it perhaps this apparent vitamin C deficiency in the Indians in my series, which is partly responsible for the high incidence of iron deficiency? The response to iron therapy by mouth in those patients with no vitamin C in the serum was good in all patients except one (case 24) in whom it was slow.

Radiological examination of the chest

Ball (1931) was amongst the first to demonstrate an increase in the heart size in anaemia radiologically and this has subsequently been confirmed by other workers. In a series of 34 cases (Hunter, 1946), 12 patients (35.3%) showed definite cardiomegaly as compared with my total of 14 out of 54 cases (25.9%).

Cardiac enlargement tends to occur more frequently in patients

/with ...

with low haemoglobin levels (Wintrobe, 1946; Blumgart et al., 1948); the average haemoglobin of the Indian female and male cases was 5.0 g. per 100 ml. and 5.9 g. per 100 ml. respectively, thus confirming the above statement (see table 2.6).

Table 2.6.

CORRELATION BETWEEN RADIOLOGICAL CARDIOMEGALY AND HAEMOGLOBIN LEVELS

Group	Total No.	Radiological cardiomegaly No. of cases	% age	Average Hb (g%) mean
Indian females	32	9	28	5
Indian males	11	3	27	5.9
African females	6	1	17	7.4
African males	5	1	20	6.0

An African female patient had cardiomegaly with 7.4 g. per 100 ml. Hb, but she had associated chronic rheumatic heart disease (case 48).

One patient had bilateral calcified primary complexes associated with a positive Mantoux reaction (case 2). However, there were no signs of activity both clinically and radiologically, while the haematological response to iron therapy was suggestive of simple iron deficiency anaemia rather than anaemia of infection. The same applied to another patient (case 10) who had a calcified primary complex with a positive Mantoux reaction, while a third had an ill-defined opacity at one apex again with a positive Mantoux reaction (case 18).

Other abnormal radiological findings were a small right basal effusion, related to cirrhosis of the liver (case 31), a probable

/tuberculous ...

tuberculous lesion in the left upper lobe which did not prevent a good response to iron therapy alone (case 45), and consolidation of the left lower lobe following amoebic abscess of the left lobe of the liver (case 54). In the last patient the haematological picture was consistent with iron deficiency anaemia on the findings in the bone marrow.

Electrocardiographic changes

In many cases of chronic anaemia electrocardiographic changes have been found; these are as a rule minor in degree and are not specific for anaemia. Thirty one Indians and 8 Africans were studied electrocardiographically. Amongst these 2 Indians (cases 11 and 38) and 2 Africans (cases 53 and 44), with an average Hb level of 6 g. per 100 ml., had normal electrocardiograms.

The T wave was commonly inverted in Lead III, being found in 11 Indians and 3 Africans; there was flattening of the T wave in Lead III together with some of the other leads in 9 cases. There were 8 Indians whose electrocardiograms showed depressed ST-segments, mainly in the chest leads, while elevation of the ST-segment was found in Lead I together with some of the other leads in 8 Indian and 1 African case. Left axis deviation was found in 4 Indians, one of whom had associated ischaemic heart disease with left bundle branch block. Five of the Indian patients, with an average Hb level of 7.4 g. per 100 ml. had low voltage, a finding consistent with that of Turner (1932). An Indian female of 9 years,

/with ...

with a Hb level of 2.1 g. per 100 ml., and an early diastolic and a short systolic murmur together with cardiomegaly, had electrocardiographic changes consistent with those of anterior myocardial infarction (case 6).

These findings are consistent with those of Wintrobe (1946) who, like Elliott (1934), stated that the most common change is a depression of the R-T (ST) junction and flat or inverted T waves, but without any corresponding change in the QRS complex. Hunter (1946) studied 25 cases of anaemia electrocardiographically and found flat T waves in one or more leads; and he quotes 2 females in the fifth decade with Hb levels of 40% and 50% each with grossly abnormal electrocardiograms. These changes were still present after one year despite therapy for the anaemia, and it was therefore thought that there was probably associated ischaemic heart disease.

The electrocardiographic changes have thus been found to be non-specific and resemble those known to occur with cardiac anoxia (Blumgart et al., 1948) and often those due to digitalis (Wintrobe, 1946).

Achlorhydria

Histamine fast achlorhydria is said to be very common in chronic iron deficiency anaemia. Lundholm (1939) found an incidence of 50% in a large series of cases of anaemias with a low colour index. Nine of the 39 Indian patients (23%) on whom this test was done, had

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no free acid after maximal stimulation with histamine (0.04 mg. per Kg. body weight); 7 of these were females and 2 were males. Of the latter, one (case 36) had a peptic ulcer, while the other (case 39) had a post gastrectomy syndrome with probable malabsorption. Amongst the females, one had cirrhosis of the liver, one had a duodenal ulcer with a mild hookworm infestation; in 2 of the cases there was a mild hookworm infestation with no evidence of blood loss, while 3 were labelled as "idiopathic" iron deficiency anaemia as no cause could be found.

This indicates that only in 44.4% of the cases of achlorhydria could a definite cause be found for the anaemia, which leaves 55.6% of cases of achlorhydria with anaemia in whom no definite diagnosis could be made. In 40% of the latter cases a mild hookworm infestation was present, but this was thought not to be of sufficient import as a specific cause for the anaemia.

The average age of patients with achlorhydria without any specific cause for the anaemia was 35.6 years. As far as the associated symptomatology is concerned only two of them complained of dyspepsia (cases 8 and 39), two (22%) had dysphagia (cases 8 and 5) while koilonychia was present in all but 3 of the 9 patients. Bethell et al. (1934) observed dysphagia in 5 out of 33 patients of iron deficiency anaemia with achlorhydria. In 2 of my patients (cases 4 and 14) vitamin C was absent from the blood and therefore provided a probable

/additional ...

additional factor which might interfere with the absorption of dietary iron.

I did not find the large female preponderance amongst my cases of achlorhydria with iron deficiency anaemia reported by Bethell et al. (1934) in their series of 33 cases of achlorhydria.

Here again it should be noted that none of the African cases had histamine-fast achlorhydria, irrespective of the cause of the anaemia.

Barium studies

Barium studies in the form of a meal and a follow-through examination were done in order to exclude conditions such as a hiatus hernia, peptic ulceration, malabsorption syndrome and any other gastro-intestinal lesion that may give rise to blood loss and hence iron deficiency anaemia.

Amongst the Indian females, there were 3 cases with probable duodenal ulcers (cases 14, 28 and 30) while case 19 had changes which were possibly due to duodenal ulceration. Two patients (cases 16 and 17) demonstrated pylorospasm, cholecystitis having been excluded by the finding of a normal cholecystogram, while in one barium enema examination was normal (case 17). A flocculation pattern suggestive of malabsorption was observed in one patient (case 11).

Of the Indian males, duodenal ulcers were demonstrated in 3 patients (cases 35, 36 and 43); the first two of whom were 11 and 14 years old respectively. One patient who had had a gastrectomy done

/some ...

some time previously showed some flocculation after 24 hours (case 39), while another showed some delay in the emptying of the stomach but no other associated pathology was demonstrated (case 38).

One African female (case 47) demonstrated rapid emptying of the stomach, evidence of gastrointestinal hurry, which is said to be consistent with avitaminosis.

African males: In a 17-year-old patient with amoebic dysentery and bilharziasis, the barium meal examination showed a slight deformity of the duodenal cap which was suggestive of an ulcer near the left fornix. There was, however, no occult blood detected in his stool (case 51). A second patient (case 50) showed nothing definite although oesophageal varices were not excluded. Another (case 53) had chronic intestinal blood loss and his barium meal showed a filling defect in the central area of the stomach. Unfortunately, he could not be fully investigated as he was uncooperative.

The incidence of peptic ulceration as demonstrated on barium studies was 16% (7 probable cases) amongst the Indians while only one case (9%) was found among the Africans and even this was doubtful. In this hospital peptic ulceration is much less common among Africans.

Vitamin A absorption test

The vitamin A absorption test of Patterson and Wiggins (1954) was done as a screening test to detect malabsorption on all cases except

/two ...

two Indian females (cases 1 and 20) and one African male (case 53). Absorption was regarded as being poor if at the end of four hours of fasting the vitamin A content of the blood failed to increase by at least 500 units as compared with the control specimen. The absorption was poor in 17 patients in 2 of whom this was confirmed by a fat balance study (cases 45 and 47) while the latter test was suggestive of malabsorption in 3 further patients (cases 14, 19 and 54), one of whom, however, had a normal urinary radioactive vitamin B12 excretion test.

Fat balance studies

Fat balance studies were done, wherever possible, when malabsorption was suspected. Eleven patients were studied in this way including 9 of the 17 on whom the vitamin A absorption test was poor. The method used was that described by King and Wootton (1956), 5 specimens of 24-hour stools having been collected on 5 consecutive days. The findings were suggestive of malabsorption in 3 of these cases (Nos. 14, 19 and 54) in all of whom the vitamin A absorption test was poor. In 2 further cases (cases 45 and 47), the fat analysis confirmed the malabsorption which was suggested by the vitamin A absorption test (see table 2.7).

From this analysis it can be said that case 45, an African female, probably had subclinical steatorrhoea giving rise to iron deficiency anaemia. She also had an associated chest infection but her iron picture and response to therapeutic iron were not those of the anaemia of infection. Case 47 also showed malabsorption, but

/here ...

Table 2.7.

CASES WITH POOR ABSORPTION ON FAT BALANCE STUDIES

	Case No.	Average fat excretion/day over 5 days in Gms.	Associated Disease/ probable cause of iron deficiency
MALABSORPTION	45	11.3	probable pulmonary tuberculosis
	47	13.3	pellagra, hookworm infestation (intestinal hurry on barium meal examination)
? MILD MALABSORPTION	14	6.0	? duodenal ulcer, hookworm infestation, malnutrition
	19	5.0	? cause of anaemia
	54	6.6	amoebic liver abscess

here it could well have been secondary to pellagra from which she was also suffering; barium meal examination demonstrated intestinal hurry, said to be consistent with avitaminosis.

Urinary radioactive vitamin B12 excretion test

This further test for detecting malabsorption was done on only 5 patients (cases 18, 19, 21, 39 and 50). The test was done by the method established by Schilling (1953), using 5 ml. of urine in a well-crystal type scintillation counter. Excretion below 5% was regarded as definite evidence of malabsorption up to 10% as doubtful and above 10% as normal excretion, allowing for a maximum error of 2%. The quantities excreted in the urine by these patients were 15.9%, 14.1%, 22.6%, 9.1% and 14.5% respectively. Thus normal absorption was found in a patient (case 19) who had a poor vitamin A absorption test and whose fat analysis was suggestive of mild malabsorption. An Indian male who had a post gastrectomy syndrome excreted 9.1%, and although this is within experimental error it could well be compatible with a mild impairment of absorption.

Serum vitamin B12 levels

Serum vitamin B12 levels were estimated on some of the cases by the method of Hutner et al. (1956) (see table 2.8 for the results).

Table 2.8.

VITAMIN B12 LEVELS

	No. of Cases	Mean	Range
Indian females	14	313.2	127 - 1440
Indian males	5	491.2	256 - 787
African females	4	360.5	228 - 445

Experience in the same laboratories shows that healthy adults generally have serum levels higher than 200 µµg. per ml., values between 100 µµg. and 200 µµg. per ml. being of doubtful significance (Adams et al., 1962). In no case in this series was the level below 100 µµg. per ml. and only 2 were below 200 µµg. per ml. (cases 2 and 9). In rats it has been found that iron deficiency results in decreased vitamin B12 absorption and plasma levels (Yeh et al., 1961). This, however, does not apply to my patients amongst whom only 2 Indian females had somewhat low levels. It can therefore be assumed that the epithelial changes and other clinical features present in my patients are not due to associated vitamin B12 deficiency.

Other relevant investigations such as barium enema examination and blood urea were done wherever indicated; results of these may be found in the individual case histories. The Mantoux skin test was done on 32 of the Indian and 9 of the African patients, and was found to be

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positive in 34.4% and 77.7% respectively, indicating a fairly high exposure rate especially amongst the Africans.

DISCUSSION

Besides the finding of hypochromic anaemia, other criteria were used in the diagnosis of iron deficiency in the present series - reduced serum iron level in the blood and elevated iron binding protein of the serum, giving a low percentage saturation. As hypochromic and iron deficiency anaemia are not necessarily synonymous terms, a further tool was used in order to confirm the diagnosis. This involved the examination of the bone marrow for iron stores, a useful index in differentiating true iron deficiency from the picture found, for example, in the anaemia of infection. The distinguishing feature between these two conditions, which may both present with the picture of hypochromic anaemia (Stevens et al., 1953), is that the iron stores are normal or increased in the anaemia of infection while they are diminished or absent in the other. The final criterion was the response of the anaemia to medicinal iron.

RACE DISTRIBUTION

There is an obvious preponderance of Indian cases in the present series, namely 79.6% of 54 cases. This is especially so when one realises that the overall admission rate of Indians is one quarter

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of that of Africans. This, once again, confirms one's impression of the relatively high incidence of iron deficiency among Indians.

SEX DISTRIBUTION

As expected the incidence is higher in females, who formed 70.4% of the total group. Amongst the Indians, where a fair proportion remained undiagnosed ("idiopathic" iron deficiency anaemia), females constituted 74.4%, while there were only 54.5% females in the small African group, that is in a group where no cases of so-called "idiopathic" iron deficiency anaemia were found, there was almost an equal sex distribution.

AETIOLOGICAL FACTORS

The diagnosis of iron deficiency anaemia is not an end in itself but requires a search for the underlying cause since this anaemia only occurs when the demand for iron is greater than its supply to the body. This disturbance in demand and supply may be a result of chronic blood loss, some disorder of the gastrointestinal tract, faulty utilisation of iron or possibly defective diet. Any one or several of these factors may be involved in the production of the iron deficiency.

Chronic blood loss

Iron deficiency anaemia commonly occurs when there has been slow bleeding over a long period of time. The most common causes in women of the child bearing age are said to be multiple pregnancies and bleeding from the genital tract, in the form of menorrhagia or

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metrorrhagia, and this can frequently be underestimated as a cause.

Parity may have been a contributing factor in the production of anaemia in my cases. The role of parity in the local hospital population will be discussed in further detail later.

A full gynaecological investigation, including diagnostic curettage, was done in all cases where there was an indication. This procedure proved fruitful in one post-menopausal patient in whom endometrial polypi were found (case 9). One of the patients with menorrhagia had a proliferative endometrium and this 34-year-old female who had had 4 pregnancies, would otherwise have probably been referred to as a case of "idiopathic" iron deficiency anaemia (case 10). The 14-year-old girl had no diagnostic curettage done in view of her age, and so some local pathology was not excluded; nevertheless, judging from her response one is tempted to conclude that the menorrhagia was secondary to the anaemia since it later appeared to have corrected itself (case 11). The incidence of gynaecological abnormalities is not as high as that of Gray et al. (1956) in their gynaecological study of 40 cases of "idiopathic" iron deficiency anaemia.

On the other hand, in males and also probably in post-menopausal women, iron deficiency is most frequently due to bleeding from the gastro-intestinal tract. Amongst the lesions found to be probably responsible were peptic ulceration, cirrhosis of the liver with chronic bleeding from oesophageal varices and ulcerative colitis. Hookworm ova were found in some cases.

Peptic ulceration was demonstrated in 7 patients and was doubtful in one. The latter (case 19) was a 14-year-old Indian girl who sought medical advice purely because she was observed to be pale at school. Barium meal examination showed some distortion of the duodenal cap but no tenderness was elicited; at no stage was occult blood detected in her stools nor had she reached the menarche. If one includes this case, the incidence of peptic ulceration was 16.7%, while 87.5% of these were among Indians. There was only one African male (case 51). He was 17-years old and barium meal examination suggested duodenal ulceration; he also had amoebic dysentery and a urinary tract infection with bilharziasis. Two of the Indian males with peptic ulceration were 11 and 14 years old respectively and neither of them had abdominal pain related to meals. Peptic ulceration should be included in the search for a cause of iron deficiency anaemia in childhood, particularly in view of the high incidence reported by Girdany (1953).

There were 5 cases of cirrhosis of the liver based on clinical and biochemical findings. Three of these patients were Indian females, one an African female and the last an African male. One Indian female had associated signs of malnutrition and mild hookworm infestation (case 31), while the African female had an associated bilharziasis (case 46).

An Indian female (case 27) was admitted with dysentery which was thought to be due to ulcerative colitis, a recognised cause of

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iron deficiency resulting presumably from the loss of blood from the ulcerated areas.

There were 2 cases of severe gastrointestinal haemorrhage, one an Indian and the other an African male, in neither of whom was the diagnosis established. The former had a fatal melaena while in hospital and the latter, besides having had an abnormal liver function test, appeared to have had a filling defect in the stomach which could not be confirmed as the patient refused to have any further investigations.

Disorders of the gastrointestinal tract i.e. disorders in which absorption and nutrition may be impaired.

Malabsorption: There were 2 patients (cases 45 and 47), both African females, whose fat balance studies showed an average fat excretion of 11.3 and 13.3 g. per day respectively. The former probably had pulmonary tuberculosis while the latter had a mild hookworm infestation associated with signs of pellagra. Both of them were post-menopausal and had 4 and 5 children respectively. It is known that steatorrhoea can give rise to iron deficiency anaemia due to a failure in absorption but this may not be a sole factor (Badenoch et al., 1954). On this basis did these cases (especially the former) have sub-clinical steatorrhoea giving rise to iron deficiency, or was the malabsorption secondary to the associated disease (particularly in Case 47)?

Similarly, the small degrees of malabsorption as suggested by barium meal examination and fat balance tests in some of the other

/cases ...

cases was probably due to an associated disease, such as peptic ulceration or some infection.

Gastrectomy: A 28-year-old Indian male (case 39) who had a gastrectomy for a benign peptic ulcer 4 years previously, gave a five month history of vague abdominal pain and diarrhoea. Besides the finding of iron deficiency anaemia, he had histamine-fast achlorhydria, some flocculation on a 24-hour film after administration of barium and the urinary radioactive vitamin B12 excretion test suggested some impairment of absorption. At no stage was there any occult blood in his stool. It was therefore concluded that he most probably had iron deficiency anaemia with malabsorption as an important factor. Whether this was contributed to by the achlorhydria or by the gastrectomy cannot be resolved as both these conditions have been shown to be related to iron deficiency anaemia (Heulengracht, 1934). There is no doubt that iron deficiency anaemia can occur after partial gastrectomy (Witts, 1956; Hobbs, 1961); however, its incidence and the theories regarding the mechanism of production vary. Hobbs (1961) found half his males and all his females to be anaemic after partial gastrectomy and all his patients responded to iron therapy. He concluded that two thirds of his cases of anaemia were due to poor absorption while one third were due to blood loss, poor intake and such factors. Pirzio-Biroli et al. (1958), using radioactive techniques, also showed that after gastrectomy patients tend to absorb food iron poorly.

The case described above is consistent with the postulate

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of malabsorption of iron as a mechanism for the production of the anaemia. This is probably chiefly due to the rapid emptying of the gastric contents and may be contributed to by the histamine-fast achlorhydria.

Achlorhydria: The association between gastric atrophy and iron deficiency anaemia has long been recognised but which came first is still being debated. Supporting the one school of thought, Lees and Rosenthal (1958) conclude that gastric lesions precede the anaemia and are in some way concerned in the etiology of the iron deficiency. Their work, however, throws no light on the mechanism by which the gastric mucosa or secretions influence the absorption of iron. On the other hand, the view that the gastric mucosal changes are the result of iron deficiency anaemia is held by Davidson et al. (1955). As far as achlorhydria is concerned both these groups of workers found a similar incidence (55% and 48% respectively). Witts (1956) states that whereas it was formerly believed that achlorhydria gives rise to iron deficiency, it is now believed that iron deficiency causes atrophic gastritis and achlorhydria; and hence where it was previously considered to be an essential feature of this type of anaemia (Witts, 1930) it is now more or less agreed that while achlorhydria is commonly associated with it, it is by no means a fundamental factor. The role of achlorhydria in absorption is also a controversial point as there have been conflicting reports in the literature. Although the hydrochloric acid of the gastric secretion favours the solution of iron in the food and

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its conversion into the ferrous state, it is not indispensable for this purpose, and there is no evidence that anacidity causes anaemia (Witts, 1956).

Although the acidity of the gastric juice may play some part in iron absorption (Barer et al., 1937; Granick, 1954). Moore (1955) was unable to increase the absorption of food iron in patients with hypochlorhydria or achlorhydria by adding hydrochloric acid. However, as Wintrobe (1961) states, "when the requirement for iron becomes relatively high the presence of free hydrochloric acid may be important in making additional dietary iron available for absorption", but to what extent this is a factor in my cases is not known.

Associated diseases

There are some disease processes with which iron deficiency anaemia may be associated. Amongst these is the presence of an infection in which the utilisation of iron is impaired, and the iron deficiency may partly be due to this factor. Two Africans (cases 45 and 54) had infection of the chest and amoebic liver abscess respectively, but in both the haematological picture and the response to iron therapy were consistent with iron deficiency anaemia rather than the anaemia of infection. In addition fat balance studies demonstrated malabsorption in the former. An African female (case 48) had chronic rheumatic heart disease which is an accepted associated condition.

Besides the patients who showed various signs of malnutrition, there were 2 females, an 18-year-old Indian and a 62-year-old post-menopausal African who demonstrated signs of pellagra (cases 1 and 47). The former who is alleged to have been on an adequate diet had amenorrhoea

/and ...

and no cause for the iron deficiency was established, for she neither had evidence of having lost blood nor was there anything to suggest malabsorption. Pellagra is said to be associated with iron deficiency anaemia mainly due to the gastrointestinal hurry with resulting malabsorption. The African female had hookworm infestation besides the radiological evidence of gastrointestinal hurry and a poor fat absorption test. Her diet consisted mainly of maize and its products while meat was rarely consumed.

A mild urinary infection was present in 2 of the Indian females (cases 20 and 32), but in neither was this thought to be responsible for the iron deficiency. There was also mild hookworm infestation in both.

There were 2 patients with urinary bilharziasis (cases 46 and 51). One also had cirrhosis of the liver while the other had amoebic dysentery and probable duodenal ulceration both of which may cause iron deficiency.

The incidence of iron deficiency anaemia in rheumatoid arthritis has been reviewed by McCrae (1958). Based on bone marrow examination and response to therapy, he found iron deficiency anaemia in 33.3% of his cases. In my series there was one patient who had mild hookworm infestation but associated with this was ankylosing spondylitis (case 38). There was no other cause for the iron deficiency, which is assumed to be related to his spinal disease, the mild hookworm infestation being unlikely alone to have caused anaemia.

An Indian female who presented with mental symptoms was found to have iron deficiency anaemia for which no cause could be found except myxoedema (case 25). Iron deficiency anaemia has been described as occurring with myxoedema, Larsson (1957) finding 2 cases of true iron deficiency anaemia out of a total of 48 cases. As in my case he found no achlorhydria in his patients. The mechanism of production of the anaemia in this case, however, was not determined. It has been suggested (Piraio-Biroli et al., 1958) that it may be due to impaired absorption at the mucosal level, but this has not yet been adequately proven. On the other hand, it is unlikely to be related to interference with the synthesis of transferrin (Larsson, 1957) as my patient had a very high transferrin level.

HOOKWORM INFESTATION AND IRON DEFICIENCY

This association merits special discussion because of the tendency of incorrectly labelling a large number of cases as "hookworm anaemia". The aetiology of hookworm anaemia is well discussed by Rhoads et al. (1934) who state that this anaemia is due largely to iron deficiency, and this has been confirmed by other workers in the field. It seems as though the worms contribute to the formation of the anaemia exclusively by the mechanism of blood sucking and not through their toxins as was previously believed. This anaemia responds well to oral iron therapy only, without the prior removal of the worms (Cruz et al., 1948; Foy and Kondi, 1958). With isotopic methods Roche et al. (1957,1957(a),1959) found that a considerable amount of blood may be lost into the gastro-intestinal tract by patients infected with

/hookworm ...

hookworm. Although much of this is reabsorbed the net loss is, however, "capable of putting the body out of iron balance in tropical countries" (Foy and Kondi, 1958). Roche and his colleagues found a rough correlation between the number of ova in the stools and the amount of blood lost - on the average patients lost 2.74 ml. per 1,000 ova per gram of stool per day. Judging from the literature, there appears to be no doubt that in cases of hookworm infestation blood loss caused by the worm is only one of the factors involved in the production of the iron deficiency anaemia in a large number of cases. Several workers (Napier et al., 1941; Cruz et al., 1948) have shown that poor nutrition is a potentiating factor, while other contributory factors such as repeated pregnancies, and poor absorption and possibly achlorhydria, may also be present.

The finding of hookworm ova in the stools of an anaemic patient does not necessarily mean that the anaemia is caused by the hookworm infestation; its occurrence should be related to the infestation rate in the community. Nonetheless, it has been shown that the hookworm is capable of causing anaemia and the degree of anaemia correlates with the grade of infestation. Out of the 54 cases in this series, 11 (20.4%) were found to have hookworm ova in their stools; this figure being made up by 18.6% of the Indians and 23.7% of the Africans. Regarding the finding of 5 - 20 ova in the stool specimens as a moderate infestation, it was found that 2 of the patients (cases 29 and 49) had a heavy infestation while a third had a moderate infection (case 52). Those with heavy loads had stools which were strongly positive for occult

/blood ...

blood; and treatment produced 26 and 265 adult hookworms from them respectively. The case with a moderate load had a weakly positive test for occult blood and 10 adult hookworms were recovered after treatment. The rest of the patients had only mild loads, 3 with a weakly positive test for occult blood while 5 passed no occult blood. All the hookworms recovered belonged to the Necator americanus species, and usually occurred with an equal sex distribution.

There have been conflicting views about the symptomatology of hookworm anaemia as distinct from iron deficiency anaemia from other causes. Some authors (Rhoads et al., 1934) have drawn attention to the high incidence of epithelial changes in their cases of hookworm anaemia, but as Witts (1952) has stated these are due to the iron deficiency which is the common factor in this type of anaemia and which gives rise to various epithelial changes, irrespective of the cause of the anaemia.

Two of the 11 patients complained of abdominal pain, 3 had glossitis while one had dysphagia; all of these have been stated to be common in hookworm anaemia.

Achlorhydria was reported in 24% of a series of 54 cases of hookworm anaemia from Puerto Rico (Rhoads et al., 1934) while I found an incidence of 33.3% in my small series of 11 cases. Their average age was 28 years, and all had mild hookworm infestation.

The average Hb level in the two cases with heavy infestation was 3.5 g. per 100 ml. while that for the whole group was 4.8 g. per 100 ml., suggesting that the degree of infestation is related to the

/severity ...

severity of the anaemia. The eosinophil count varied from 48 - 2520 cells (the latter figure was associated with a heavy infestation in the African female). Their average reticulocyte count on admission was 2.9% and it is interesting to note that amongst the Africans there was a fairly marked reticulocytosis which, according to Stransky et al. (1947), is indicative of active erythropoiesis and, together with eosinophilia, is a feature of iron deficiency anaemia due to hookworm infestation.

Four of my patients had various signs of malnutrition, while a fifth had pellagra, giving an incidence of 45.5%. This is not surprising since it has been said that the classical picture of "hookworm anaemia" may not be produced unless there is also malnutrition (Witts, 1956). The latter probably accounts for some of the clinical features which are alleged to be found in hookworm anaemia.

Amongst the Indian females with hookworm infestation, there was only one with a heavy load. The test for occult blood in her stools was strongly positive. No other cause for the deficiency was determined. Her anaemia appears to have been due to the hookworms because she was 21 years old, had oligomenorrhoea, no pregnancies and was alleged to have had an adequate diet. Her serum vitamin C level was 0.1 mg. per 100 ml. and she had no achlorhydria. The rest had mild loads only. One had cirrhosis of the liver with signs of malnutrition, another was malnourished and probable duodenal ulceration was demonstrated on

/barium ...

barium studies, while in 4 of these no other lesion was found.

The Indian male with ankylosing spondylitis and mild hookworm infestation has already been mentioned (case 38).

Hookworm ova were found in the stools of 2 African females. One, who had a heavy infestation with occult blood in the stools, was post-menopausal, nulliparous, and had a diet consisting almost entirely of maize products, was probably an example of hookworm anaemia (case 49). The other, who had a mild worm load, was a pellagrin with mal-absorption (case 47).

A moderate load of hookworm ova, with a weakly positive occult blood test, was found in a 9-year-old anaemic African male with mal-nutrition and oedema, once again illustrating this combination (case 52).

My findings thus confirm those of other workers. When present, hookworm infestation is not necessarily the sole cause of iron deficiency anaemia, more often this is due to a multiplicity of factors. Hookworm ova may even be just an incidental finding. If the anaemia is due to hookworm it appears to produce the regular iron deficiency anaemia with its characteristic clinical features differing in no way except perhaps for eosinophilia.

DIET

After excluding blood loss and faulty absorption or utilisation of iron as the cause for iron deficiency anaemia, there remains the possibility of a defective diet as a contributory if not an aetiological

/factor ...

factor. All my patients belonged to the lower socio-economic group. The dietary histories obtained appeared to be unreliable in certain cases, especially when an attempt is made to correlate them with family income. However, this will have to suffice until such time as an organised field survey can be done.

The most striking point emerging from the brief dietary histories available is the lack of meat; about half of the patients either seldom or never ate any meat. About a quarter of the patients seldom or never consumed vegetables, the proportion in Africans being higher (50%).

Since both iron and protein are important factors in the production of the Hb molecule their presence or absence from the diet is presumably of some significance. It has been suggested (Powell, 1958) that deficiency of protein in the diet may be a factor in the production of the abnormal protein patterns and hypoalbuminaemia observed in normal Africans of the lower socio-economic group. The patients in my series also had abnormal protein patterns, but the complicating feature here is that anaemia in itself commonly causes a reduction in the plasma proteins and albumin fraction (Heath et al., 1936), the total proteins being reported to return to more normal values after the treatment of the anaemia (Adams, 1958). Unfortunately the serum protein estimations were not repeated after treatment in my cases. The question to pose here is whether or not this disturbance

/in ...

in the protein pattern, to some degree at least, occurs normally in the Indian of the lower socio-economic group, in the same way as it does in the African. Does this protein deficiency, as is apparent from my dietary histories, contribute to the production of the anaemia? Cartwright (1947) had suggested that a diet deficient in protein or in a specific amino acid might result in anaemia. Hahn and Whipple (1939) have shown that in dogs, made anaemic by repeated bleedings, a sufficient degree of limitation of protein intake (despite an excess of iron intake), can limit the production of Hb. Bethell et al. (1939) have suggested that a combined type of anaemia in pregnancy is presumably dependant on the need of protein and iron. Hence the probable importance of an adequate protein intake, particularly in individuals with low iron reserves.

That iron is a constituent of the Hb molecule and that a deficiency of iron in the body gives rise to anaemia are both well established facts. It is said to be rare to find cases with diets lacking in iron-containing foods as the sole cause of the iron deficiency (Beutler et al., 1963) but Gray et al. (1936) found that 71.4% of their 35 patients took a diet deficient in iron-containing foods. As the major source of iron is vegetables and seeing that 76% of my cases had a good or adequate vegetable intake, it is surprising, especially amongst the Indians, to have such a high incidence of iron deficiency, when one looks at the problem superficially. There are, however, other factors which may interfere with the iron content or the absorption of iron. There may, for instance, be present certain food idiosyncrasies

/interfering ...

interfering with the intake of iron; or the soil may be iron deficient and therefore vegetables may be low in iron content (Wintrobe, 1961). On the other hand, although iron may be present in the food it may not be available for absorption. This might be due to the presence of other constituents in the diet which may interfere with the absorption of iron. It has been suggested that phytates, which are present in various cereals, may do so by reacting with the iron as it passes through the intestinal tract, to form an insoluble iron phytate (Widdowson et al., 1942; McCance et al., 1943). A high phosphorus content has also been shown by Hegsted et al. (1949) to interfere with absorption. They concluded that the absolute amount of iron or phosphorus in the diet as well as the iron-phosphorus ratio influences the amount of iron absorbed. These insoluble compounds are particularly prone to form in a medium which is not strongly acid (Wintrobe, 1961), hence the theory of achlorhydria being a possible aetiological factor in iron deficiency. There is some evidence that excess calcium inhibits iron assimilation (Anderson et al., 1940; Sharpe et al., 1950), but this is still a controversial point as there has been counter evidence to the effect that calcium in moderate amounts can have a favourable effect on iron absorption by combining with the phosphate ions (Cubler, 1956). Unfortunately, I am in no position to comment, not only on the phytates, phosphorus and calcium, but also on the iron content of the diets consumed by my patients. I am, therefore, ignorant of the dietary iron intake of my patients and its availability for absorption.

An interesting fact which emerges from these investigations

/is ...

is the high proportion of Indian patients with low vitamin C levels. It is not postulated that iron deficiency anaemia was due to scurvy, as it is generally recognised that a low vitamin C level in the plasma does not necessarily indicate scurvy. It is questioned, however, whether the reduced vitamin C levels in these cases interferes with the absorption of iron, since vitamin C has been stated to enhance the absorption of iron from food (Moore et al., 1952; Moore, 1955; Moore et al., 1956). This it presumably does by reducing the ferric iron to the ferrous state (Witts, 1956). However, it has not been settled as to whether the amounts present normally in food have a significant effect.

"IDIOPATHIC" IRON DEFICIENCY ANAEMIA

After having been through the whole battery of investigations, it was found that there was left a certain proportion of patients in whom no single satisfactory cause for the iron deficiency was found. These consisted of 21 Indian females (65.6%) and 5 Indian males (45.5%), giving a total of 60.3% of the Indian cases. On the other hand there were none amongst the Africans. One female who was 34 years old was a possible exception. There was no evidence of gross blood loss, but 4 Caesarean sections in fairly rapid succession could have been responsible for the iron deficiency. She had normal gastric acidity and her diet consisted of daily vegetables and meat once a week only.

There were 21 Indian females (ranging in age from 9 - 56 years with an average age of 31.7 years) in whom no adequate pathological blood loss could be established. Their average Hb level was 6.1 g. per 100 ml.

The incidence of koilonychia and dysphagia was 61.9% and 23.8% respectively; dysphagia being twice as frequent as in the whole group of Indian females while the incidence of koilonychia was more or less the same.

Looking for the possible aetiological factors in these cases one observes firstly that the dietary intake was poor in 11 (52.4%) while 5 cases (23.8%) had achlorhydria. Table 2.9 shows the relevant clinical data on these cases.

The average serum vitamin C level was 0.6 mg. per 100 ml. Three patients had no vitamin C in the serum, only one of them having achlorhydria as well. Subclinical scurvy as a cause of iron deficiency anaemia in some cases cannot therefore be excluded.

Decreased intake and poor absorption of iron may have been responsible for the deficiency in a fair proportion of these cases. Five patients had doubtful evidence of blood loss. Of two of these patients, neither of whom had reached the menarche, one had a mild hookworm infestation while the other had a very doubtful radiological picture of duodenal ulceration after barium meal examination. In the latter case there were no symptoms related to ulceration nor was there any evidence of blood loss. A 19-year-old female with a normal menstrual history and no pregnancies had a poor diet, achlorhydria and mild hookworm infestation with no occult blood in the stools. The remaining 2 cases with mild hookworm infestations were in the older age group.

One had a poor diet and achlorhydria, while both had had more than 4 pregnancies. These 5 cases illustrate the multiplicity of factors which may be operative and so, with minimal blood loss, give rise to iron deficiency. The low dietary iron intake and the associated achlorhydria, probably by virtue of its influence on absorption, may render the quantity of iron absorbed insufficient to make up the deficiency produced by the losses.

This leaves 16 out of 32 Indian females, still a substantial number, in whom the diagnosis of "idiopathic" iron deficiency anaemia has been made. Their average age was 34.6 years while the average number of pregnancies was 3.7, 6 of them having had more than 4 pregnancies.

With regard to the gynaecological histories of these 16 patients, the majority had amenorrhoea or hypomenorrhoea. One case was post-menopausal. Two others had menorrhagia, but the latter symptom disappeared after iron therapy; in both of these patients, aged 14 and 45 years, menorrhagia was assumed to be secondary to anaemia (cases 11 and 17). The latter patient had had 5 pregnancies and suffered from mild diabetes.

Among the other associated findings was an 18-year-old patient with signs of pellagra but no achlorhydria or malabsorption. One of the patients presented with thyrotoxicosis and amenorrhoea. She was 42-years old and had had 3 pregnancies. One is not aware of any direct relationship between iron deficiency anaemia and thyrotoxicosis but Heath and Patek (1937) quote a similar case for which no definite

/explanation ...

explanation was advanced.

Although anaemia for which no cause can be found is said to be very uncommon in men, 5 Indian males (46%) fell into this category. Table 2.10 gives the relevant details on these patients. Their average age was 15.2 years (range 7 - 27 years), none being in the older age group. No patient had achlorhydria and the diet was poor in only one case. The average vitamin C level was 0.8 mg. per 100 ml., one patient having no vitamin C in the serum. Three patients (60%) had koilonychia and none dysphagia. One of the patients had infective hepatitis while another had bronchial asthma but these two conditions were thought not to be related.

My findings are consistent with those of Witts (1930) in his discussion of chlorosis in males. He analysed 5 cases of anaemia in males, aged from 18 - 28 years. None had achlorhydria and no cause for anaemia was discovered.

These features, both in males and females, seem to fit into the syndromes of chlorosis and idiopathic hypochromic anaemia as previously described. Today there is a tendency to diagnose these conditions less frequently. There have been several instances (Gray et al., 1936; Heath and Patek, 1937) where cases previously diagnosed as idiopathic hypochromic anaemia have been reviewed and in the majority some gastrointestinal, gynaecological or other evidence of blood loss, including multiple pregnancies, have been discovered. In only 7 of the

/60 ...

Table 2.10.

CASES OF "IDIOPATHIC" IRON DEFICIENCY ANAEMIA AMONGST MALES

(No evidence of blood loss)

Case No.	Age	Hb (g/100ml)	Diet	Gastric acidity	Vitamin C (mgm/100ml)	Associated Disease
41	7	4.0	Good	+	0.5	Infective hepatitis
40	12	5.2	Poor	+	0.2	Bronchial asthma
42	12	3.4	Adequate	+	2.4	-
34	18	4.9	Adequate	+	1.0	-
37	27	4.8	Adequate	+	Nil	-
Mean	15.2	4.5			.82	

+ = Present

60 cases of idiopathic hypochromic anaemia analysed by Heath and Patek (1937) did they fail to discover evidence of pathological blood loss.

Do my cases fall into this category which is now rapidly going out of favour? If so, I am at a loss to explain its high incidence. As for its occurrence in males, this is most unusual but it has been suggested that this anaemia is due to the strain of puberty (Witts, 1930).

The factor that appears to be common to both male and female patients is the diet, and one wonders if this does not play a major role in the production of the anaemia; while the achlorhydria and possibly the low vitamin C levels may be potentiating factors. In females multiple pregnancies probably add a further strain.

In the event of even minimal bleeding there should already be present in the Indian the stimulus for increased absorption of iron in view of their low iron stores as observed in the iron storage study (see chapter 4); but despite this they become iron deficient. On the other hand, in the African whose iron consumption is said to be high and in whom iron deficiency anaemia is reported to be rare (Walker, 1953; Gerritsen et al., 1954; Walker, 1955; Bronte-Stewart, 1957), I found no cases without an aetiological factor. I can therefore postulate that in the African, unlike the Indian, there is almost always a discernable cause for iron deficiency anaemia.

Chapter 3

ANAEMIA IN PREGNANCY

Iron deficiency is generally regarded as the most common cause of anaemia in pregnancy (Strauss et al., 1933; Scott et al., 1949; Ventura et al., 1951; Gatenby et al., 1955; Allaire et al., 1961). Since the level of the serum iron and the iron-binding protein have been found to be a useful index of iron metabolism (Rath et al., 1950), and likewise the estimation of the iron stores as judged by bone marrow examination (Scott et al., 1952), it was hoped that their measurement in anaemic pregnant women would reflect the state of iron balance in the different racial groups.

The results of contributions made in this field from different places vary considerably (see table 3.1). Whereas the incidence of the anaemia of pregnancy has decreased in some areas in recent years (Davidson et al., 1935, 1942, 1944), it is still high in other parts of the world. It varies from a very low incidence in Australia (Morgan, 1961) to about 80% in India if 11 g. per 100 ml. is taken as the critical level (Krishna Menon, 1962). The incidence of anaemia in Hyderabad (Shankar, 1962) is an underestimation as this study excluded all cases who were obviously clinically anaemic. Krishna Menon et al. (1960) treated 112 patients with anaemia of pregnancy over a 12-month

/period ...

Table 3.1.

INCIDENCE OF ANAEMIA IN PREGNANCY

(Hb less than 10.0 g. per 100 ml.)

	% with anaemia
BRITAIN	
Glasgow Scott & Govan (1949)	20
Bradford Benstead et al. (1952)	6.5
Dublin Gatenby and Lillie (1955)	23.8
North Staffordshire Giles and Burton (1960)	14.6
UNITED STATES	
Allaire et al. (1961)	15.3
AUSTRALIA	
Morgan (1961)	0
INDIA	
Shankar (1962)	46 and over
Krishna Menon et al. (1962)	±80 (Hb 11 g. per 100ml. & less)

peried and found that about 38% had iron deficiency while 60% had a dimorphic anaemia with macrocytic hypochromic anaemia and a normoblastic or a megaloblastic bone marrow. Although the total number of cases seen is not given in the latter study, the analysis, however, is interesting, as the overall incidence of iron deficiency in this series is probably much higher than 38%. This once again confirms the high incidence of iron deficiency in India.

A pregnancy survey was carried out in Durban in order to find out how important anaemia is as a complication of pregnancy in the different racial groups and what proportion is due to iron deficiency.

MATERIAL

The clinical material consisted of women attending King Edward VIII Hospital ante-natal clinic for the first time during pregnancy, who had not had any prior iron therapy. Due to technical reasons blood specimens from the Indian women were only available in the afternoons. Despite the fact that hypoferraemia may normally occur in the latter part of the day, low serum iron levels were always accompanied by low levels of Hb and by the appearance of blood smears which indicated iron deficiency anaemia. The cases were divided into 3 trimester groups, a random sample of cases having been taken for both the racial groups. There were 75 patients in each of the second and

/third ...

third trimesters and 25 in the first, a total of 175 Indians and the same number of Africans. Because of the difficulty in obtaining patients in the first trimester I have presumed that no real change takes place haematologically this early in pregnancy, as was shown by Ventura and Klopper (1951). As first trimester cases I therefore included non-pregnant patients within the reproductive age groups who were attending the casualty department for conditions unlikely to affect them haematologically. If the Hb level was found to be below 10 g. per 100 ml., a sternal puncture was done wherever possible. Examination of the bone marrow for haemosiderin appears to be an accurate means of assessing the status of the iron stores (Hutchison et al., 1953; Beutler, 1957) and this proved very useful in diagnosis. No follow up examinations were done on any of the women because of their poor attendance rate and the routine prescription of iron and vitamin tablets at the first visit to the ante-natal clinic.

RESULTS

Tables 4 and 5 (see Appendix, pages A.60 and A.65) show the values obtained in individual cases with relevant details.

Age and parity

The average age of Indians in this series was 24 years in the first trimester and 26 years in both the second and third trimesters, ranging from 15 - 39, 18 - 40 and 18 - 41 years in each trimester

/respectively...

respectively. The average number of pregnancies was 1.9 in the first, 3 in the second and 4 in the third trimester giving an overall average parity of 3.1.

The average age of the African group was 28 years in the first, 25 years in the second and 27 years in the third trimester, their ages varying from 17 - 58 in the first, 16 - 41 years in the second and 16 - 43 years in the third trimester; while the average number of pregnancies was 2.3, 2 and 3 in each of the three trimesters respectively and an average parity of 2.7 for the whole group.

The haematological data on these are given in table 3.2.

Regarding a Hb level of 10 g. per 100 ml. or less for pregnant women as indicative of anaemia, it was found that 18.6% of the Indians in the second, and 34.6% in the third trimester fell into this category, while only 4% (3 cases) of the Africans in the third trimester were so affected. In anaemic patients, peripheral smears were examined and wherever possible, bone marrow was aspirated and the smears examined for the type of anaemia and the presence of haemosiderin.

Peripheral blood smears were examined on 38 cases of the 40 anaemic Indians in the second and the third trimesters. Thirty-one had various degrees of hypochromia. The average M.C.H.C. was 28.8% as compared with the overall M.C.H.C. of 31.4%.

Bone marrow aspirations, done on 36 of these patients, confirmed the presence of iron deficiency anaemia in the majority. In one patient (an African) erythropoiesis was megaloblastic.

Table 3.2.

HAEMATOLOGICAL VALUES IN PREGNANCY

	INDIANS		AFRICANS	
	mean	range	mean	range
First trimester				
Hb (g%)	12.5	10.3 - 15.6	13.0	10.8 - 15.9
M.C.H.C. (%)	32.0	28 - 36	32.1	30.0 - 35.5
Serum iron	72.0	12 - 173	85.8	20.0 - 191
T.I.B.C.	357.9	168 - 483	332.2	240 - 463
% saturation	20.5	3.3 - 50.6	25.5	8.4 - 59.3
Second trimester				
Hb (g%)	10.9	6 - 14.6	12.2	10.1 - 14.2
M.C.H.C. (%)	31.6	24 - 36	32.5	30 - 35
Serum iron	54.6	2 - 170	87.3	41 - 171
T.I.B.C.	511.2	318 - 733	430.7	256 - 679
% saturation	10.9	2.9 - 30.2	21.0	6.61 - 58.56
Third trimester				
Hb (g%)	10.8	7.5 - 13.4	12.2	8.2 - 15.2
M.C.H.C.	31.5	27 - 36	32.7	30 - 36
Serum iron	46.7	9 - 121	92.4	31 - 179
T.I.B.C.	513.9	318 - 651	464.0	284 - 670
% saturation	9.2	0.7 - 30.6	20.0	6.4 - 32.6

Amongst the Indians, out of the 32 marrow specimens stained for iron, 18 had no haemosiderin, 10 contained a decreased amount (grade 0 - I) while 4 had normal amounts of iron present (grade I - II). The average M.C.H.C. of the 28 women with depleted stores was 29%; 2 of them had an M.C.H.C. of 32%. The average M.C.H.C. was 31.5% in the 4 with normal iron stores. Therefore one can say that on the whole the M.C.H.C. has been a fairly reliable index of depletion of iron stores.

Of the 28 pregnant Indian women with decreased or absent stores the average serum iron, T.I.B.C. and percentage saturation levels were: 27.3 µg. per 100 ml., 500 µg. per 100 ml. and 5.5% respectively. The average serum iron and T.I.B.C. levels for the 4 individuals with normal stores in the presence of anaemia were, 36 µg. per 100 ml. and 543 µg. per 100 ml. respectively, giving a percentage saturation of 6.6, making them appear to be iron deficient. It can be concluded that there is only a fair correlation between the iron stores and the serum iron and T.I.B.C. levels, but it is not absolute.

Of the 3 African patients with anaemia only 2 had hypochromia. Of the 2 patients on whom bone marrow aspirations were done, one with normoblastic erythropoiesis had no haemosiderin granules while the other with megaloblastic erythropoiesis had iron on staining of the marrow (grade II - III).

Study of European cases

Simultaneously with this study, 139 pregnant European women were also investigated, under the same conditions except that no bone marrow aspirations were done on the anaemic patients. There were 39 women in the first trimester, and 50 in each of the other two trimesters. It was unnecessary here to include non-pregnant women in the first trimester group. All these were in the lower socio-economic strata of the Durban European population. (The results are set out in table 6 in the Appendix, page A.70). The average age was 24.3, 24.7, and 25.2 years in each of the 3 trimesters respectively while the average parity was 2.5, 2.1 and 2.4 respectively with an overall average of 2.3.

The average Hb levels in the 3 trimesters were 13.3 g., 12.3 g. and 11.9 g. per 100 ml. while the M.C.H.C. varied from 32.7% in the first to 32.3% in the second and 32.5% in the third trimester. The serum iron levels were 111.6 µg. in the first, 100.3 µg. in the second and 82.6 µg. per 100 ml. in the third trimester, while the T.I.B.C. values were 365.3 µg., 404.8 µg. and 463.1 µg. per 100 ml., giving a saturation of 31.6%, 26.5% and 18.5% respectively. This indicates the trend towards iron deficiency in the last trimester.

There were 4 cases of anaemia, one in the second and three in the third trimesters; none was severe. The haematological results on the 4 cases of anaemia were as shown in table 3.3.

Table 3.3.

HAEMATOLOGICAL VALUES IN PREGNANT EUROPEAN WOMEN WITH ANAEMIA

Case No.	No. of previous pregnancies	Hb (g./100ml.)	M.C.H.C. %	Plasma iron (µg./100 ml.)	T.I.B.C.	% saturation	Duration of pregnancy (trimester)
412	0	9.9	29	19	485	3.9	2nd
450	7	9.8	29	33	543	6.1	3rd
473	3	9.2	29.6	39	544	7.2	3rd
474	1	9.2	32	54	609	8.9	3rd

The M.C.H.C., (except in one), the serum iron, T.I.B.C. and percentage saturation in these cases indicate iron deficiency.

The average parity of 2.8 in this group is more or less equal to the group as a whole; once more demonstrating that there is no correlation between parity and the occurrence of anaemia in the present series.

DISCUSSION

These data obtained during pregnancy will be analysed after having established the normal values of Hb, serum iron and T.I.B.C. in the non-pregnant woman of the different racial groups.

First trimester cases

Except for the European cases the others are healthy non-pregnant women within the reproductive age group. The incidence of the Hb level is given in figure 3.1.

FIG. 3.1.

INCIDENCE OF HAEMOGLOBIN LEVELS IN FIRST TRIMESTER CASES

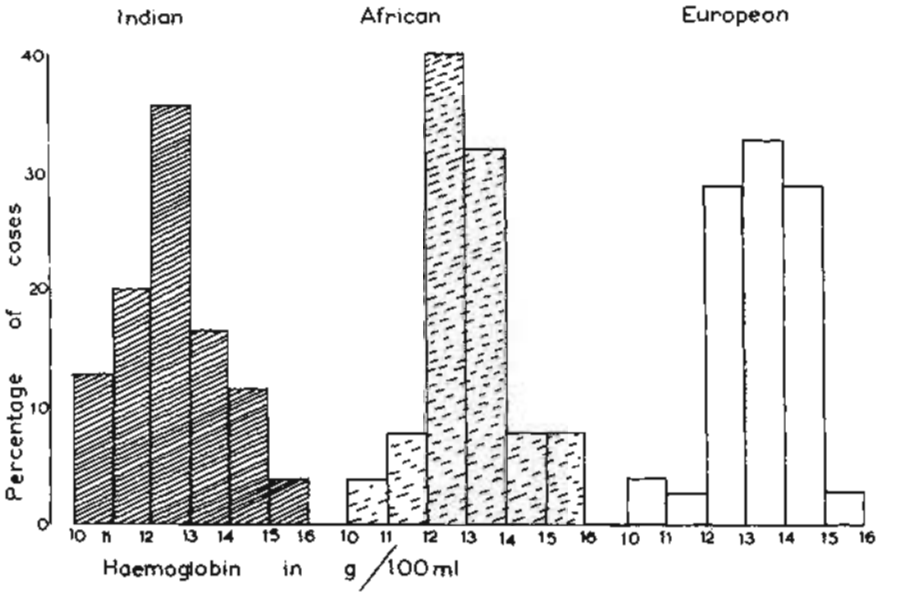


Table 3.4.

DISTRIBUTION OF HAEMOGLOBIN LEVELS

Haemoglobin level (g./100ml.)	Indian %	African %	European %
12g% or more	68	88	92.3
Below 12g%	32	12	7.7
Below 11.5g%	20	12	7.7
Below 11g%	12	4	5.1
Below 10.5g%	4	-	-

Although none of the patients was severely anaemic, it is worth noting that amongst the Indians especially there is already present a degree of anaemia, and so it is not surprising that the incidence of anaemia increases as pregnancy advances. Taking the normal Hb level of a non-pregnant adult female as 12 - 16 g.% (Wintrobe, 1961), it is seen that 32% fall below this level amongst the Indians while in the Africans and the Europeans it is 12% and 7.7% respectively (see table 3.4). This is of importance since Young et al. (1946) concluded it is possible to forecast the Hb estimation in later pregnancy as judged from the levels in early pregnancy. The average M.C.H.C.s were 32.0%, 32.1% and 32.9% for the Indian, African and European cases respectively.

Corresponding with these results, although the average serum iron and T.I.B.C. levels are within the normal limits for all the racial groups, 24% of the Indians, 4% of the Africans while none of the Europeans in the first trimester, had a percentage saturation of 10 and less -

/thus ...

thus emphasising the fairly high incidence of iron deficiency among the Indians already present before the drain on their iron stores due to pregnancy occurs. The iron storage study on post-mortem material (presented in chapter 4) correlates well with the relatively high incidence of iron deficiency among them as shown by the above findings.

This again demonstrates the apparent lack of correlation between parity and iron deficiency in the present study; since there is evidence of iron deficiency in a group of non-pregnant Indians whose parity is the lowest out of the 3 racial groups (1.9 compared with 2.3 and 2.5).

Comparison of haematological results in the 3 racial groups

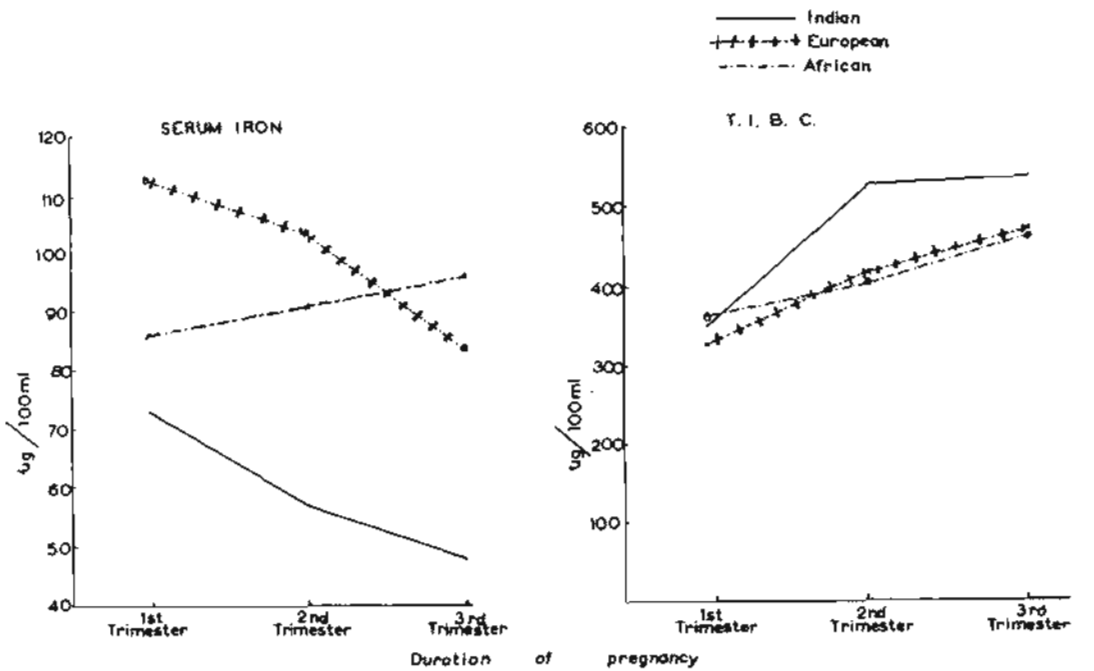
The serum iron and T.I.B.C. levels in the African and European patients are within normal limits (Ventura and Klopper 1951 - non-pregnancy levels) but in Indians the serum iron level appears to be a little low while the T.I.B.C. is within normal limits. There is a progressive fall in the serum iron level in the Indian and European patients unlike the case of the Africans. Regarding the T.I.B.C. there is a parallel progressive rise in all the racial groups as pregnancy advances, a finding which conforms with that of other authors (see figure 3.2).

There is at the moment no unanimity regarding the changes in the serum iron and the T.I.B.C. levels during pregnancy. While there is more or less general agreement that the T.I.B.C. rises progressively in pregnancy (Laurell, 1947; Fay et al., 1949; Rath et al., 1950;

/Ventura ...

FIG. 3.2.

SERUM IRON & TOTAL IRON BINDING CAPACITY LEVELS IN PREGNANCY IN THE THREE RACIAL GROUPS



Ventura et al., 1951) opinion differs as to the change in serum iron levels. Results from Britain show a fall at mid-term (Ventura et al., 1951) but Rath et al. (1949, 1950) and Fay et al. (1949) showed no such changes. My results support both groups of workers. On the one hand amongst the Indians, in whom iron deficiency appears to be widespread, there is a progressive fall in serum iron during pregnancy while on the other hand the level in Africans remains more or less constant. This illustrates the relative absence of iron deficiency in the latter group.

The Hb levels in the 3 racial groups decreased in mid term, as expected (Wintrobe, 1961), but here again this was most marked in the Indian. The M.C.H.C. remained fairly constant throughout, the Indians having had a lower basal reading.

The incidence of anaemia in pregnancy in the 3 racial groups, during the second and third trimesters

Considering patients in the second and third trimesters only, it was found that 26.7% of all the Indians, 2% of the Africans and 4% of the Europeans were anaemic at their first visit to the ante-natal clinic (see table 3.5). As expected the incidence increased as pregnancy advanced in all three groups, being highest among the Indians (35% in the third trimester), presumably due to their poor iron stores when pregnancy commenced.

In the majority of patients anaemia was only of moderate degree. Regarding a Hb level of 8 g. per 100 ml. or less as indicative of severe anaemia, it was found that neither the African nor the

/European ...

Table 3.5.

INCIDENCE OF ANAEMIA

(Number of cases with Hb of 10 g. per 100 ml. or less)

Pregnancy	Indian %	African %	European %
Second Trimester	18.6	-	2
Third Trimester	34.6	4	6
Overall incidence	26.7	2	4

European women fell into this category while 25% of the Indians (10 cases), did so, most of them being in the second trimester (7 cases). The incidence of anaemia in the African and European groups is extremely low and compares well with studies in Australia (Morgan, 1961). The high incidence in the Indian group, however, though probably not as high as that reported from India, is more akin to that found in populations elsewhere in the world (Gatenby et al., 1955).

Other South African studies

The incidence of anaemia has been studied by various workers in different parts of South Africa. Most of the work done has been on Africans and unfortunately there is only one set of figures available for Indians in pregnancy with which to compare my results.

In the present study of 150 pregnant African women in the second and third trimesters 3 were found to be anaemic. This incidence is a little above that of 1% observed by Theron et al. (1961) on pregnant African women in Pretoria. According to Gerritsen and Walker (1954), who compared the haematological findings in the non-pregnant African female with those of the pregnant female, there was no significant difference in the haemoglobin, haematocrit and serum iron levels in the different groups. In fact their Hb concentration, haematocrit and serum iron level remained constant through-out while the T.I.B.C. level

/rose ...

rose significantly during pregnancy. They attribute this lack of change to their high iron intake. My results for African females are consistent with the latter finding.

Bothwell (1962) estimated the Hb, the haematocrit, the serum iron and T.I.B.C. levels of 103 African women at term and found that the average Hb was 13 g. per 100 ml., serum iron 97 µg. per 100 ml., T.I.B.C. 492 µg. per 100 ml., while the percentage saturation was 21. Only 2% of these women were anaemic. Here again although the incidence of anaemia is lower, their average serum iron and T.I.B.C. results at this stage of pregnancy are almost the same as in the present series (third trimester).

In Durban in randomised samples Adams (1958) found that 4% of 100 African and 40.6% of 32 Indian women at term were anaemic. These results tally well with the present series in which the values for Africans and Indians in the third trimester were 4% and about 35% respectively.

As against the above observations in South African studies, in Cape Town Lanskowski (1960), regarding an M.C.H.C. of 30% as a critical lower limit of normal, came to the conclusion that 76.2% of Europeans, 81.4% of Coloureds and 93.9% of Africans at term were iron deficient, and advocated the need for prophylactic iron therapy. A criticism of this work is that a low M.C.H.C., without an estimation of the iron state, is not sufficient for the diagnosis of iron deficiency (Cartwright et al., 1946; Stevens et al., 1953).

Thus we see that my observations conform with the findings of most of the other authors regarding the incidence of the anaemia of pregnancy in the South African population groups studied.

Physiological changes during pregnancy

During pregnancy there is frequently much difficulty in establishing whether or not anaemia is present, due to certain physiological changes which normally occur in pregnancy and as a result of which the blood Hb level usually falls. This is an apparent change secondary to changes in blood volume from about the 26th to the 35th week of pregnancy. Both the plasma volume and the red cell mass have been shown to increase significantly in pregnancy, and as the plasma volume increases at a more rapid rate than the red cell mass, there is an apparent fall in the Hb level (Dieckmann et al., 1934; McLennan et al., 1948; Caton et al., 1951). Although there has been a variation in the percentage increase obtained by different workers, there is general agreement on the principle of "hydraemia" in pregnancy, which causes the so-called "physiological anaemia of pregnancy" in which the total red cell mass and the circulating Hb are increased so that the M.C.H.C. remains normal (Whitby and Britton, 1957). These are now considered normal phenomena and "we should retain the concept of a physiological anaemia of pregnancy and with it the possibility of a teleological explanation of the dilution of the Hb, such as the promotion of the absorption of iron or the diminution of the tendency to thrombosis" (Witts, 1962).

Among the other changes in the blood during pregnancy, which

/further ...

further complicate the diagnosis, are those of the protein content. The plasma proteins are reported to fall progressively during pregnancy (Paaby, 1960).

This hydraemia of pregnancy with its apparent anaemia should not be confused with the true anaemias of pregnancy, the commonest of which is hypochromic. It is more or less generally accepted that a Hb level below 10 g. per 100 ml. indicates true anaemia.

There is also a marked disturbance in iron metabolism since the demands of the foetus must be met from the maternal iron stores. During pregnancy about 273 mg. iron are accounted for by the foetus (Widdowson et al., 1951), 75 mg. by the cord and placenta (McCoy et al., 1961) and approximately 170 mg. by maternal bleeding at the time of delivery (Newton et al., 1961), amounting to some 500 mg. lost during pregnancy. On the other hand about 200 mg. are conserved because of the amenorrhoea which occurs during pregnancy and lactation, so there is a net deficit of about 300 mg. In addition there is a further loss estimated during 6 months of lactation to be about 180 mg. (Davidson et al., 1938). According to the latter authors there is a daily loss of just under 2 mg. during pregnancy and lactation for six months. There thus appears to be a fair drain on maternal stores during pregnancy and the daily need for iron for these purposes increases throughout pregnancy. Rath et al. (1950) calculated that one mg. per day is required in the first trimester, 4 mg. per day in the second and 10-12 mg. per day during the latter part of the third trimester. If these needs are successfully met in pregnancy no iron deficiency occurs especially if the dietary intake is adequate.

Aetiological factors

The primary cause of anaemia during pregnancy is a pre-existing iron deficiency (Rath et al., 1950), and this has been borne out by my Indian and African cases.

The diet has been much stressed by different authors as an important factor in the production of the anaemia of pregnancy. A diet deficient in iron has understandably been incriminated (Gray et al., 1936; Young et al., 1946); but the protein intake in addition to dietary iron has been shown to be responsible by some workers (Strauss and Castle, 1932; Bethell et al., 1939; Gatenby and Lillie, 1955).

One cannot help but suspect that the diet plays an important role as the dietary histories obtained from the anaemic cases were found to be poor on the whole. This may account for the high incidence of anaemia in the Indian when compared with the African who is known to have a high dietary iron intake (Gerritsen and Walker, 1954).

As regards the correlation between parity and anaemia, this appeared to have made no contribution in the present series. There was no relationship between parity and the development or the severity of the anaemia. Among the Indians, 40 patients with an average parity of 2.5, had Hb levels of 10 g. per 100 ml. or less while those with Hb

/levels ...

levels of 8 g. per 100 ml. and less had an average parity of 2.7 as compared to a parity of 3.3 for the whole group in the second and third trimesters. Of the 3 African cases with anaemia the average parity was 1.5 but the numbers are too small to be conclusive. This lack of correlation of parity with the degree of anaemia is consistent with the findings of Bland et al. (1930).

As far as the overall parity is concerned, it was highest in the Indian (3.1), lowest in the European (2.3) while the African was in between (2.7).

This demonstrates that the precise aetiology of the anaemia of pregnancy is still not clear except that there are several factors such as a pre-existing anaemia, the drain on the maternal iron stores, poor intake, and possibly multiple pregnancies especially if in rapid succession; and blood loss, whether or not related to the pregnancy, may also contribute. Here one has to bear in mind the possibility of pelvic congestion giving rise to occult blood in the stools as suggested by Dickens et al. (1959). They found an incidence of 32.6% in 58 pregnant women.

Despite all the controversies one probably ought to accept the fact that the incidence of iron deficiency varies greatly in different communities and the frequency with which the iron deficiency anaemia of pregnancy occurs, is an indication of a pre-existent iron deficiency at the onset of pregnancy (Rath et al., 1950). The latter authors state

/that ...

that this deficiency becomes exaggerated as the patient's total blood volume increases and the foetus strips the mother of iron. Unfortunately I was not able to follow up any of my cases throughout the present pregnancy, partly because of irregular and infrequent attendance and partly due to the routine administration of vitamins and haematinics at the ante-natal clinic.

My results appear to agree with the conclusion drawn by Scott and Govan (1949), in that the presence of a relative deficiency of iron or other factors before pregnancy, accounts for the development of anaemia in the latter part of pregnancy when the foetal storage of iron is greatest, and increasing demands are made on the maternal iron stores. Looking at the results for the first trimester one observes that these patients are already on the brink of iron deficiency. It is therefore not surprising to find that in Durban, particularly among Indians, anaemia is one of the commonest complications of pregnancy.

Chapter 4

IRON STORAGE STUDY

The body usually maintains an iron reserve which is present in the liver, bone marrow, spleen and other tissues. All these divisions for practical purposes are regarded as a single physiological unit. The iron is stored intracellularly in a protein-complex as ferritin and haemosiderin. These are functionally indistinguishable from each other (Shoden et al., 1953) and both are available for haemoglobin synthesis when the need arises (Finch et al., 1950).

The iron reserves in man as assessed quantitatively after repeated bleedings, (in one subject) have been found to be about 600 mg. by Hynes (1949) and about 1200 - 1500 mg. by Haskins et al. (1952). This level has been shown to vary from individual to individual.

The iron stores are called upon in the event of any blood loss in order to prevent iron deficiency anaemia from developing (Finch et al., 1950). It has been demonstrated that storage iron is depleted in iron deficiency (Rath et al., 1948; Davidson et al., 1952; Hutchinson, 1953; Stevens et al., 1953; Beutler et al., 1954). At the other end of the spectrum is the condition of iron excess which can also be assessed from the examination of the stores. This occurs in such diseases as haemochromatosis and haemosiderosis, and in "siderosis" in the African

/population ...

population.

Iron stores may be determined by many different methods and any of the storage tissues may be studied. In the present investigation I have selected the chemical analysis of the liver and bone marrow specimens, including a histological examination of the latter.

MATERIAL

This was obtained at necropsy, predominantly from medico-legal cases. Due to the shortage of material, especially Indian, hospital necropsies had to be included. The respective diagnosis and relevant clinical and pathological details on the latter cases are all available.

Two hundred Africans and 58 Indians were studied. Included amongst these were 40 African females and 21 Indian females. The Indian subjects ranged in age between 18 and 76 years, with an average age of about 44 years; while the Africans had a mean age level of about 36 years ranging from 16 - 80 years.

The average body weight of the Indian males was 119 pounds as compared to 126 pounds for the African males. The Indian females weighed 107 pounds while their counterpart amongst the Africans had an average weight of about 131 pounds.

There were also 16 European males studied, ranging in age from 17 - 65 years with a mean of about 45 years.

METHODS

Bone marrow specimens were obtained from the sternum and examined for iron content histologically, using the technique previously mentioned. The specimens were graded according to the amount of iron observed ranging from grade 0 - VI, on the following basis:-
0 ... no iron visible under oil immersion; I ... iron just visible under oil immersion; II ... small sparsely distributed particles in reticulus cells usually visible under low power; III ... numerous small particles throughout the marrow particles; IV ... larger particles with tendency to clump throughout the marrow particles; V ... dense large particles throughout the marrow particles; VI ... very large deposits of iron both intra- and extra-cellular, which tend to obscure the cellular detail of the marrow particles (the slide is a dense blue to the naked eye). Due to the difficulty of accurate definition these were expressed as grade 0 - I for absent to decreased amounts, I - II and II - III for normal amounts while the grades from III upwards were regarded as representing increased amounts.

For the chemical analysis of the bone marrow the method described by Gale et al. (1963) was used.

Iron concentrations in the liver specimens were estimated as described by Gale et al. (1963). Both the total and the haem iron were determined and the tissue iron was derived from them. These results are related to the dry weights of the tissue specimens and are expressed as g. per 100 g. dry weight of liver.

RESULTS

The detailed individual results with the relevant data are given in table 7, pages A.74 - A.80 in the appendix.

Liver specimens

A chemical analysis was done on liver specimens from 200 Africans of whom 40 were females. Table 4.1 shows the mean values of iron concentration in liver specimens in the groups studied. Table 4.2 indicates the proportion of cases in the different ranges.

Taking the normal range of tissue iron to be 0.01% - 0.09%, and regarding 1% as the very siderotic level, the following iron concentrations were found in the liver.

African males: Of the 160 subjects, 26.8% had iron concentrations within the normal range, 24.4% had moderately increased iron stores while severe siderosis was found in 16.3%. The average age for the subjects with severe siderosis was 45.5 years as compared to a mean of 36 years for the whole group of African males.

Amongst the African females 42.9% had concentrations within the normal limits while 5% were moderately siderotic and 7.5% severely so.

On the other hand 55.3% of the Indian males and 66.7% of Indian females had levels within the normal range. Moderate siderosis was found in 5.3% of Indian males while severe siderosis was found in one of the Indian females (4.8%). The latter patient, aged 30 years, had died of acute renal failure, which is known to give rise to increased

/iron ...

Table 4.1.

MEAN VALUES OF IRON CONCENTRATION IN THE LIVER SPECIMENS

Group	No. of cases	Average age	Average wet weight of liver (g.)	Mean Tissue Iron (g./100 g.) dry wt.
African males	160	36	1633	0.507
African females	40	34.4	1578	0.284
Indian males	37	45.6	1434	0.100
Indian females	21	41.4	1346	0.169
European males	16	44.6	1862	0.101

Table 4.2.

PERCENTAGE OF SUBJECTS WITH DIFFERENT IRON CONCENTRATIONS IN THE LIVER

Iron concentration %	Assessment	GROUPS (%) African males	African females	Indian males	Indian females	European males
Below 0.01%	-	0.6%	15%	13.1%	23.8%	12.5%
0.01 - 0.09%	Normal range	26.8%	42.5%	55.3%	66.7%	43.8%
0.1 - 0.39%	Mild siderosis	31.9%	30%	26.3%	4.8%	37.5%
0.4 - 0.99%	Moderate siderosis	24.4%	5%	5.3%	-	6.3%
Above 1%	Severe siderosis	16.3%	7.5%	-	*4.8%	-

* 1 case of chronic renal disease.

iron stores, although as a rule not as high a level as this.

Hepatic iron concentration at different age levels: Amongst the males, both African and Indian, there was a tendency for the iron concentrations to rise with age; maximum concentrations having been reached between the ages of 30 - 50 years in the Africans and 30 - 45 years in the Indians. There was a tendency for a second rise in the later age groups but the series was too small to draw any firm conclusions. This trend, however, appears to be followed by the female subjects to some extent in the 25 - 40 year age group. Table 8, page A.81 in the appendix shows the mean hepatic iron concentrations at different ages.

Bone marrow

The chemical analysis for iron content was done on 152 African males and 57 Indian males, in whom the mean levels were 542.6 µg. per g. (Standard deviation 234.3) and 86.6 µg. per g. (Standard deviation 41.3) respectively. The average iron concentration of the bone marrow for the European males was 129.9 µg. per g. The mean levels for the 38 African and 21 Indian females were 146.2 and 87.4 µg. per g. respectively.

Table 4.3 shows the percentage of subjects for each group at the different levels of iron concentration.

Association between iron concentration in the bone marrow

/and ...

Table 4.3.

PERCENTAGE OF SUBJECTS WITH DIFFERENT IRON CONCENTRATIONS IN THE BONE MARROW

Iron concentration (µg./g.)	Grade	African males	African females	Indian males	Indian females	European males
Less than 125		28.5	63.2	81.6	76.2	43.8
125 - 225	Normal range	18.5	21.1	15.8	23.8	43.8
225 - 999		42.5	15.8	5.3	-	12.5
Above 1000	Severe siderosis	10.6	-	-	-	-

and the age (see table 9, page A.82 in the appendix): As with the hepatic iron concentration, there is also an association between these two, particularly in the case of the Africans.

Histological analysis of the bone marrow iron: Table 4.4 shows the percentage with different gradings of marrow iron. Sixteen percent of the African males, 60% of Indian males, 43.8% of the European males, 50% of African females and 33.3% of Indian females had either traces or no iron deposition (Grade 0 - I). Moderately increased stores were found in 32.6% of the African males, 10% of Indian males, 6.3% of European males, 14.7% of African females and 19% of Indian females. Severe siderosis equivalent to the levels described in haemochromatosis (grade V - VI) was found in 11.1% of the African males and 2.9% of the African females, while neither of the other two racial groups had any histological evidence of this.

Comparison between histological estimations and chemical iron concentrations in the bone marrow: These results are shown in table 4.5. The figures were derived from the analysis of 246 subjects, including all racial groups. The mean iron concentration levels were correlated with the histological grading for iron. Figure 4.1 shows the fairly good overall correlation between these two methods of examination. However, when individual results are considered it is observed that marked disagreement may occur between these two methods (see table 7, page A.74 : cases no. 29, 23, 32, 234, 228, 224).

Table 4.4.

HISTOLOGICAL ANALYSIS OF BONE MARROW

Grade	African males	African females	Indian males	Indian females	European males
0 - I	16%	50%	60%	33.3%	43.8%
I - II & II - III (normal range)	40.3%	32%	30%	47.6%	50%
Increased stores (III - V)	32.6%	14.7%	10%	19%	6.3%
Severe siderosis (Haemochromatosis) (V - VI)	11.1%	2.9%	-	-	-

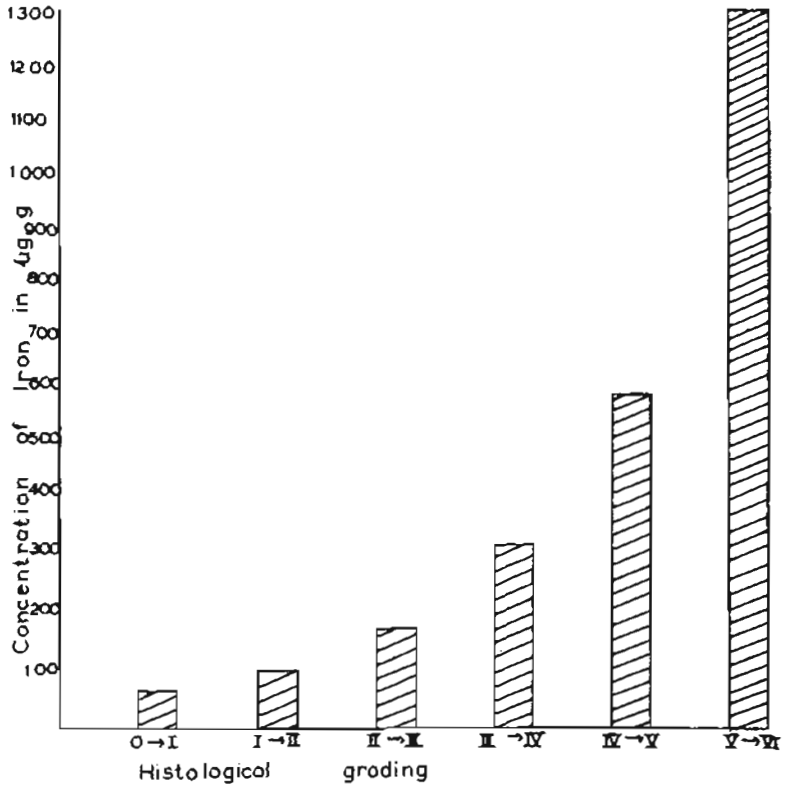
Table 4.5.

RELATIONSHIP BETWEEN MEAN IRON CONCENTRATION LEVEL AND THE HISTOLOGICAL GRADING IN THE BONE MARROW

	0 - I	I-II	II-III	III-IV	IV-V	V-VI
No. of cases	75	46	50	38	19	18
Mean iron concentration µg./g.	63	111	206	321	577	1302

FIG 4 1.

RELATIONSHIP BETWEEN THE IRON CONCENTRATION IN THE BONE MARROW ON CHEMICAL AND HISTOLOGICAL EXAMINATION



Comparison between the concentration of iron in the liver and bone marrow

The African males are considered here, numbers in the other groups being too small to draw any conclusions. Only subjects on whom both organs were analysed have been selected. The 40 subjects with hepatic iron concentrations between 0.01 g. and 0.09 g. per 100 grams (i.e. those within the normal range) had a mean bone marrow iron concentration of 145 µg. per g.; those between 0.1 and 0.3 g. per 100 g. had 294 µg. per g. iron, those with the moderately siderotic liver iron concentration had 426 µg. per g. iron in the bone marrow while the severely siderotic group had iron concentration of 890 µg. per g. in the marrow specimens. This illustrates the fairly good correlation of iron concentration as obtained in the liver and the bone marrow.

Comparing the percentage of subjects regarded as severely siderotic according to the concentration of iron in the liver and bone marrow, one finds that 16.3% of African males, 7.3% of African females and 4.8% of Indian females were severely siderotic according to hepatic iron concentration; while only 10.6% of African males were in this category on chemical analysis of the bone marrow (an arbitrary level of a 1000 µg. per g. was taken). On histological examination of the bone marrow 11.1% of African males and 2.9% of African females were observed to be in the severely siderotic group (see table 4.4). This once again shows fairly close correlation between histological and chemical analysis of the marrow.

DISCUSSION

This study demonstrates the racial differences in iron concentration.

Hepatic iron concentration

The probability of the observed difference between the means of iron concentrations in the liver for Indian and African males is much less than 0.01. The observed figures may be accepted, because the observed difference would be obtained by chance in much fewer than 1 instance in 100. The critical ratio of 18.4 at 195 degrees of freedom is significant at the 5% level, because it is much greater than 1.96 - also at the 1% level, because it is much greater than 2.58. Thus the difference between the means of the liver iron concentration for Indian and African males can be considered significant or highly reliable.

The probability of the observed difference between the means of the hepatic iron concentration for Indian and African females is 0.50 - 0.10. The observed figures may be rejected, because we would obtain the observed difference in 50 to 10 instances in 100, by accidents of sampling (by pure chance alone). The critical ratio of 1 at 59 degrees of freedom is not significant at the 5% level because it is less than 1.96; nor is it significant at the 1% level because it is less than 2.58. Thus the difference between the means for Indian and African females cannot be considered significant or reliable.

Age and hepatic iron concentration

Males: There is much less than 1 instance in a 100 that the contingency between age and iron concentration is due to chance. Therefore I conclude that a significant association exists between age and hepatic iron concentration for both Indian and African males.

Females: There is much less than 1 chance in 100 that the contingency between age and hepatic iron was accidental. Therefore I conclude that a significant association exists between age and hepatic iron concentration in both Indian and African females.

Bone marrow iron

The probability of the observed difference between the means of bone marrow iron for Indian and African males is much less than 0.01. The observed figures may be accepted because the observed difference would be obtained by chance in much fewer than 1 instance in 100. The critical ratio of 22.5 at 187 degrees of freedom is significant at the 5% level, because it is much greater than 1.96 - also at the 1% level, because it is much greater than 2.58. Thus the difference between the means of the bone marrow iron for Indian and African males can be considered to be significant or highly reliable.

The probability of the observed difference between the means of bone marrow iron for Indian and African females is much less than 0.01. The observed figures may be accepted, because the observed difference would be obtained by chance in much fewer than 1 instance in 100. The

/critical ...

critical ratio of 3.18 at 57 degrees of freedom is significant at the 5% level, because it is greater than 1.96 - also at the 1% level, because it is greater than 2.58. Thus the difference between the means of the bone marrow iron for Indian and African females can be considered significant or reliable.

Age and bone marrow iron

In both Indian and African males there is much less than 1 instance in 100 that the contingency between age and bone marrow iron is due to chance. Therefore I conclude that a significant association exists between age and marrow iron. This same significant association exists in the Indian and African females.

The Africans have a high degree of siderosis. All grades of iron overload have been found, varying from mild siderosis to amounts described in haemochromatosis. Moderate to severe siderosis was found in 41% of African males and 12% of African females on hepatic iron concentration while 44% of the former and 17% of the latter were in this category on histological examination of the bone marrow. As for the Indians they demonstrated normal to decreased stores in the majority of the subjects. There was only 1 Indian female who had markedly increased hepatic iron concentration. She was a hospital patient who had died in acute renal failure. Bath et al. (1948) have shown that in uraemia the iron reserves may be increased (grade 0 to ++++). While this marked difference exists between these two racial groups it is noted from

/the ...

the above statistical analysis that the difference between the female groups is not significant for hepatic iron concentration while the significant difference in the bone marrow iron is less than that in the males of the two racial groups.

The European males, although not analysed statistically, appeared to follow a middle line between these two racial groups, particularly as observed on bone marrow examination.

This high incidence of siderosis in the African (especially in the male) is further illustrated when calculating the approximate average total storage iron present. This was obtained by relating the average dry weight of the liver (approximately 25% wet weight) to the average hepatic iron concentration and the average weight of the liver to the iron concentration in the bone marrow (the average weight of the liver was assumed to be equal to the weight of the bone marrow in man (Wintrobe, 1963)). It was observed that the total tissue iron in the liver was 1.87 g. in the African, 0.32 g. in the Indian and 0.47 g. in the European subjects studied. Similarly in the bone marrow the total iron concentrations were 0.75 g., 0.12 g. and 0.24 g. respectively. This gives a total storage iron in the liver and bone marrow (which form the major proportion of iron stores in man) of 2.62 g. in the African, 0.44 g. in the Indian and 0.71 g. in the European.

Comparing the levels for the males in the three racial groups, it was found that the total body reserve as estimated above was 2.96 g., 0.48 g. and 0.71 g. for the African, Indian and European subject respectively.

There was a marked sex difference as expected (Stevens et al., 1953; Finch, 1959) amongst the Africans, viz. a total iron reserve of 1.35 g. in the African female. On the other hand the Indian females on the whole appeared to have a higher reserve than the Indian males, their estimated total iron reserve having been 0.69 g.

Thus we have, amongst our subjects Indians at one end with low iron stores and Africans at the other end of the scale with increased stores while the European levels appear to be in between and follow more or less the normal expected pattern.

These findings of a high incidence of siderosis in Africans in South Africa are in agreement with those of Gillman et al. (1945, 1951); Walker et al. (1950, 1953); Wainwright (1957); Bothwell et al. (1960, 1962). Siderosis has also been described in African subjects in other parts of Africa (Higginson et al., 1953; Edington, 1954, 1959). Bothwell et al. (1960) and Gillman et al. (1945) had initially used cases of sudden traumatic deaths as their subjects. Bothwell (1962) subsequently showed that the incidence of siderosis in the hospital group of patients is very similar to that of the medico-legal subjects.

Although the incidence observed by different authors is not exactly the same, the results indicate the high incidence of siderosis in the African (particularly the male).

The use of the bone marrow as a sensitive index of iron balance (Hutchison, 1953) is thus confirmed. This finding is of clinical

/significance ...

significance since the bone marrow is readily accessible for examination. The latter together with the serum iron and T.I.B.C. levels can be used routinely for the assessment of iron balance clinically, and to determine whether any imbalance, if present, is due to deficiency or faulty utilization of iron.

Chapter 5

DISCUSSION

Iron metabolism is of current interest particularly because of the problems it poses both in Southern Natal and elsewhere in the world. According to the World Health Organization iron deficiency anaemia is one of the main health problems of the world today. The incidence of iron deficiency varies in different areas. It depends on the one hand on factors such as the dietary intake of iron and its availability and on the other on physiological and pathological blood loss.

In South Africa hypochromic anaemia has been often stated to be rare in the African. This statement was supported by the abnormally high serum iron values observed in some African population groups; and also by the abnormal deposition of iron frequently seen in the tissues of the adult African at necropsy.

The plasma iron is reported to vary considerably and while some workers observed a significant difference between the sexes (Pirrie, 1952) others did not (Cartwright et al., 1949). Values obtained by different workers on subjects in South Africa, Britain, America and the Scandinavian countries are set out in table 5.1. The iron after absorption is transported by transferrin, which is said to be a β1 globulin (Surgenor et al., 1949). Transferrin

/normally ...

Table 5.1.

MEAN VALUES OF SERUM IRON AND T.I.B.C. IN NORMAL ADULTS

	Serum Iron (µg. per 100ml.)	T.I.B.C. (µg. per 100ml.)
Powell (1944)	143	-
Holmberg et al. (1945)	-	300
Cartwright et al. (1949)	125	359
Rath et al. (1949)	100	300
Ventura (1952)	-	300
Squires (1952)		
Bechuanaland	133	-
Gerritsen & Walker		
Europeans in Johannesburg	114	335
Africans from Pondoland, Northern Transvaal, Tanganyika.	105	375
Africans from Mozambique, Angola, Nyasaland	285	520

normally is only one third saturated with iron (Heath and Finch, 1949). Mean values reported elsewhere for the total iron binding capacity are also given in the table, together with results in Africans in South Africa. Although serum iron levels in African groups are high there is also an elevation of the T.I.B.C., giving a relatively normal per cent saturation, a situation which distinguishes "siderosis" from haemochromatosis where saturation tends to rise to almost 100%.

Since all the iron lost from the body has to be replaced by absorption from the diet, this is a very important aspect of iron metabolism. An average Western diet contains 10 - 15 mg. of iron per day and about 10% of this is absorbed, that is 1 - 1.5 mg. per day. The dietary iron requirements of adults have been estimated normally between 0.5 mg. and 1.5 mg. per day for males and an extra 0.5 mg. to 1.0 mg. per day for menstruating females. The maximum that can be absorbed in the presence of deficient stores is about 30%, and this (provided that the diet is adequate) is sufficient. However, the requirements vary with age and sex and there are stages in life when the need for iron increases. This is well discussed by Heath and Patek (1937), who state the peaks to be in infancy (when dietary intake is limited) and during adolescence (because of rapid growth), while menstruation and pregnancy add a further strain.

As has been previously mentioned, it is not only the amount of the metal present in the diet but also the form in which it is presented, the presence or absence of other constituents, and the local conditions in the bowel, which determine the amount of iron available for absorption.

The dietary data in the present study are quite inadequate. Dietary analyses from India indicate that the main source of iron appears to be from cereals and pulses (Balasubramanian et al., 1962; Srivastava et al., 1962). Some of the cereals, despite their high iron content, contained a low proportion of ionisable iron. The phytate content of the type of cereal consumed was found to be fairly high. These factors make for poor availability of iron. As for the vitamin C content in the Indian diet in a series of 40 normal subjects with a good standard of living, Srivastava and his colleagues found that there were mild symptoms of vitamin C deficiency in some although their vitamin C intake was within the normal range. They postulated that the losses of this vitamin can be accounted for by the process of cooking.

To a certain extent all these factors are applicable here as the foodstuffs consumed by the Indian population in Durban are more or less the same as those eaten in India. A point of interest is the low level of vitamin C in my Indian patients, in 5 of whom this vitamin was absent from the serum. Amongst the Indians in

/Durban ...

Durban one would expect an increased rate of absorption because of their low iron stores. Despite the presence of this stimulus they tend to become readily iron deficient. It therefore appears that although the stimulus for increased absorption is present in these cases, the raw material is not available in adequate amounts to overcome this deficiency. Furthermore, in the presence of low iron reserves it is possible that poor protein intake aggravates the anaemia.

Twenty three per cent of the 39 Indians on whom the test was done, had histamine-fast achlorhydria after maximal stimulation. In 55.6% of these patients (all females) with achlorhydria and iron deficiency anaemia, no definite cause was found, and they were labelled as cases of idiopathic iron deficiency anaemia. The role of hydrochloric acid in the absorption of iron has already been discussed. To what extent achlorhydria is a factor in my Indian cases is not known; there is certainly a larger incidence in the idiopathic group. None of the Africans on whom this test was done had achlorhydria, and with one possible exception, no African had idiopathic iron deficiency anaemia.

The African diet, on the other hand, has been well studied in South Africa. Although the diet is deficient in many respects according to accepted standards it is characterised by an "habitually" high iron content. It contains 100 mg. per day and may even go up to

/200 mg. ...

200 mg. per day. The iron is derived not only from the diet but also from the utensils in which the food is prepared (Walker et al., 1950). This is more marked in the male section who consume greater quantities of the iron rich indigenous beer. When such large quantities of iron are present in the diet there may be an absorption abnormality which results in increased deposition of iron in the tissues. This can either be due to the fact that the "mucosal block" theory does not apply in these cases or there may be some other factors stimulating increased absorption. Kinney et al. (1950) showed that ligation of the pancreatic ducts in dogs resulted in increased absorption of iron from the gastro-intestinal tract with marked increase in liver iron values. This was confirmed in man by Davis et al. (1962) who found that the absorption of iron was increased in chronic pancreatitis. It is possible that in the African in whom the incidence of malnutrition is high, chronic pancreatitis may contribute to the increased absorption of iron, but definite information on this point is lacking. Gillman et al. (1958) on the other hand, believed that basically the cellular changes due to malnutrition probably in association with the type of diet eaten, are responsible for the increased deposition of iron in the tissues.

The total amount of iron lost in the urine, stool and from body surfaces is estimated to be between 0.5 and 1.5 mg. It has been demonstrated that the small amount of iron which is normally excreted

/is ...

is not altered with alteration in the dietary intake (McCance et al., 1937) and the urinary excretion of iron in African subjects studied by Walker et al. (1953), was observed to be normal. Thus "siderosis" in the African probably develops as a result of normal excretion in the presence of high iron intake and absorption. A small amount of iron is reported to be excreted in the sweat and most of this on chemical analysis has been found to be in the cellular debris (Hussain et al., 1960). However, with isotopic techniques, Dubach et al. (1955) have shown that although only small amounts of iron are lost by this route, the fact remains that some loss does occur. What part this plays in the racial groups in Durban under climatic conditions where sweating is common, is not yet known.

Thus the major local problems of iron metabolism are those of iron deficiency on the one hand and iron overload on the other. This is practically a racial problem since the Indian represents the deficiency group while overload is almost confined to the African. This difference in iron stores has been shown to be statistically significant. On approximate estimation it was found that the overall mean iron reserve in the 200 Africans studied was 2.62 g. while that in the 58 Indians was 0.44 g. Whereas the normal total iron content of the body is reported to be 5 g., it is seen that in the African in the present series the iron content in the stores alone reaches 2.62 g. The liver is regarded as the most important storage organ containing iron both in the parenchymal cells and in the reticulo-endothelial system. Amongst the Indians 17% had decreased iron concentration in the liver, showing that a high proportion of Indians have depleted stores. Thus it is not surprising that the incidence of iron deficiency among them is so high.

In order to maintain the stores at an optimal level (Pirzio-Biroli et al., 1960) the rate of absorption of iron varies under normal circumstances according to the needs of the body. The maximum that can be absorbed from an average Western diet is about 2 - 4 mg. per day (Finch et al., 1950). If normal individuals were to lose blood beyond this rate, a negative iron balance would occur. Males do not readily go into this negative balance because of the limited excretion rate of iron. Females, on the other hand, lose 1 - 2 mg. per day and therefore it is not surprising that their iron stores are lower and iron deficiency anaemia much more common than in males. If a negative iron balance persists the stores become depleted and clinical evidence of iron deficiency occurs.

The importance of the above observations is the fact that they can be applied clinically to assess the iron stores. Sternal bone marrow is easily accessible and, seeing that it is a reliable index, its examination for stainable iron is very useful and important. In iron deficiency no stainable iron is present in the tissues. The findings in this thesis indicate that a fairly large proportion of Indians are in a state of latent iron deficiency. Iron deficiency can be recognized on examination of the bone marrow for storage iron - decreased amounts indicating iron depletion and when associated with anaemia, it is almost always due to iron deficiency.

A sequence of events occurs as iron deficiency develops.

Initially the body iron stores progressively become reduced. This is referred to as pre-clinical iron deficiency and is recognised by the examination of the bone marrow for haemosiderin. The serum iron and T.I.B.C. levels remain normal for as long as the bone marrow requirements are satisfied by the storage iron. Most of the Indian subjects appear to be in this phase of iron balance. The rate of iron absorption should increase provided that there is an adequate amount of available iron in the diet. Subsequently the stores are so depleted that bone marrow activity becomes limited. At this stage the serum iron level falls and the T.I.B.C. level rises and when the percentage saturation becomes markedly reduced, haemoglobin synthesis is impaired and the M.C.H.C. becomes reduced. Only at this stage does anaemia become manifest.

If this state persists then the tissues become depleted of iron as the last resort. This causes alterations in certain cellular functions and produces changes in epithelial structures. This it is suggested, is due to changes in certain enzyme reactions, since cytochrome C particularly and perhaps some other enzymes as well have been shown to be altered in iron deficiency (Beutler, 1957, 1959; Gubler et al., 1957).

The specific tissue changes described in iron deficiency anaemia include koilonychia, Plummer-Vinson syndrome and oral changes. Analysing the 54 patients with anaemia (Chapter 2), it was found that

/11% ...

11% had dysphagia, with other mucous membrane changes in some of them. All these were Indian females. Koilonychia of varying degree was present in 53.7% of my patients being made up of 60.5% of Indians and 27.3% of Africans. The latter were all females.

The finding of these epithelial changes, more commonly in the Indian, tempts one to believe that this may be a racial characteristic. However, it is more reasonable to conclude that this difference is probably due to the low iron stores in the Indians. Due to the limited stores available, it would appear that an earlier demand is made on the cellular enzyme systems in the tissues. Among the Africans koilonychia was only demonstrated in the females whose iron stores have been shown to be lower than those of the males. As these epithelial changes are usually a late manifestation of iron deficiency, it indicates that there has been a long-standing deficiency in the affected individuals.

All the patients in this series (Chapter 2) presented with anaemia and not with iron deficiency as such. The criterion used for the diagnosis were the clinical picture and certain laboratory aids. The average haemoglobin level was 6.5 g. per 100 ml. with an average M.C.H.C. of 25.9%. The serum iron level was 17.4 µg. per 100 ml., while the average level of the T.I.B.C. was 409.5 µg. per 100 ml., giving an overall mean saturation of 4.4%. These figures are well within iron deficiency levels. However, they can be misleading at times as the anaemia of infection may also give low serum iron and

/percentage ...

percentage saturation values. It is therefore necessary to assess the iron stores so as to confirm the presence of iron deficiency. Bone marrow examination for haemosiderin was done on 50 patients of whom 43 had no iron deposits while 7 had reduced amounts. The demonstration of haemosiderin in the latter cases in the presence of iron deficiency is rather paradoxical but it has been suggested that this is presumably due to the fact that this iron for some unknown reason is not readily mobilised for use.

Normally there is a balance maintained between iron intake and its physiological demand. Should there be any abnormality in absorption from the diet or blood loss due to any cause, a state of imbalance may arise. It therefore follows that if an individual, consuming a normal Western diet (10 - 30 mg. iron), were to lose more than 3 - 4 mg. per day, he would inevitably become iron deficient. This is so because the maximum amount that can be absorbed from the diet is 3 - 4 mg. Chronic blood loss is thus commonly associated with iron deficiency anaemia. In males and probably in post-menopausal women bleeding from the gastro-intestinal tract is the most likely cause while gynaecological and obstetrical causes are frequently found in women of the child bearing age.

In my series there were 54 patients with iron deficiency anaemia of whom 43 were Indians and 11 were Africans. There were 38

/females ...

females and 16 males giving a ratio of 2.3 : 1. The ages varied from 7 years to 78 years with an average of 33.3 years. This demonstrates the female sex preponderance and the high incidence of iron deficiency anaemia amongst Indians.

Of the gynaecological causes of chronic blood loss there was one patient with endometrial polypi and another with a proliferative endometrium. Amongst the lesions thought to be probably responsible for bleeding from the gastrointestinal tract were peptic ulceration (7 patients), cirrhosis of the liver in 5 patients, ulcerative colitis in one, while hookworm ova were found in 11 patients. Two patients with severe gastro-intestinal haemorrhage remained undiagnosed.

Of the 5 African males in the series, 2 had evidence of gastro-intestinal bleeding, in one of whom cirrhosis of the liver with splenomegaly was found while in the other no cause for the bleeding was established. A grossly malnourished boy of 9 years had a moderate hookworm infestation which was thought to be responsible for the anaemia. There was evidence of malabsorption in one patient who also had an amoebic liver abscess. The fifth patient had amoebic dysentery, a urinary infection with bilharziasis and probable duodenal ulceration, all of which factors could have contributed to the iron deficiency anaemia.

A heavy load of hookworm ova with occult blood in the stools was found in one African female while another had cirrhosis of the liver and bilharziasis. Two of the African females had malabsorption, the one having sub-clinical steatorrhoea while the other had associated pellagra as well as hookworm infestation. One of the females had associated chronic rheumatic heart disease while in the remaining one there was no evidence of blood loss except for a history of 4 Caesarean sections in rapid succession.

Out of the 11 Indian males evidence of gastro-intestinal haemorrhage was found in 4 patients, 3 of whom had peptic ulceration while the other had a fatal melaena. There was one patient in whom a gastrectomy had been done previously and malabsorption rather than bleeding was found to have caused the anaemia. One patient with a poor nutritional state and a mild hookworm infestation also had ankylosing spondylitis.

There were 32 Indian females in the series out of whom 8 had evidence of bleeding from the gastro-intestinal tract, there having been 3 patients with cirrhosis of the liver, 3 with peptic ulceration, one with ulcerative colitis and one with a heavy hookworm infestation. One of the patients had myxoedema while one of the remainder had endometrial polypi and another a proliferative endometrium.

Since iron deficiency anaemia is as a rule secondary to either

/blood ...

blood loss or malabsorption, these factors were established in all the Africans whilst in 60.5% of Indians they were not detected. Whereas minimal evidence of blood loss was found in 5 of these Indian females, in 16 of them together with 5 Indian males I failed to discover any evidence of pathological blood loss. The question arises as to whether my patients in the younger age group, both male and female, belong to the so-called chlorotic anaemia. It can be postulated that the normal physiological stress of growth and menstruation during adolescence is too great for the limited iron stores available. Since they have to depend on the diet to make up for these added requirements, they tend to become readily iron deficient as the quantities of iron available in the diet are probably inadequate for the demands made. Both in the latter group and in the rest of the females with idiopathic iron deficiency anaemia there was no evidence of malabsorption and all responded well to iron therapy. An inadequate diet giving rise to iron deficiency occurs most commonly during the "stress periods" of life. Therefore it is common during rapid growth, with menstruation and in pregnancy. I feel that the diet plays a fairly important role in the production of anaemia in these Indian patients. This problem can only be unravelled by the use of modern radio-isotope studies.

Realising that bleeding from the uterus is one of the

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frequently missed causes of iron deficiency anaemia, every patient with any gynaecological complaint was investigated accordingly. Only 3 patients had complained of menorrhagia. One was found to have a proliferative endometrium while the rest had no abnormality. The menorrhagia in the latter cases was thought to be secondary to the anaemia in view of the response to iron therapy. A post-menopausal patient who had given a vague history of menorrhagia, was found to have endocervical polypi.

Hookworm anaemia is regarded as one of the major causes of blood loss in many parts of the world. Amongst the 54 patients with iron deficiency anaemia, 11 (20.3%) had hookworm ova in their stools while only 6 of these had occult blood present at the time of examination. Severe infestations were found in 2 patients while in one it was only moderate. The remainder had mild degrees of infestation which were thought not to be sufficient to produce iron deficiency anaemia, except perhaps in the 4 Indian females who otherwise would have been labelled as having idiopathic iron deficiency anaemia.

To study the incidence of anaemia in the presence of hookworm infestation it would be worth while quoting some observations made in this department on this subject (unpublished data). At about the same period as this study was undertaken, 149 patients who were admitted to the same ward for conditions other than iron deficiency anaemia or

/gastro-intestinal ...

gastro-intestinal blood loss, had 3 consecutive stools examined for the presence of hookworm ova and occult blood. It was found that 34 of these patients (22.8%) had hookworm ova in their stools while 4 had occult blood at the time of examination. One patient had a moderate infestation while the rest were mild. If these cases are used as a control group, this demonstrates that the incidence of hookworm infestation in patients with iron deficiency anaemia is very similar to that in a sample of non-anaemic hospital patients. This similar incidence together with a light degree of infestation indicates that hookworm anaemia is probably not a major cause of iron deficiency in the hospital population group.

A pregnant woman with normal stores is well equipped to meet the extra demands of pregnancy. However, in the Indian women who have been shown to have depleted stores with which to start pregnancy, iron balance is difficult to maintain unless they can absorb at least 3 mg. (30%) daily from the diet. This quantity of iron is probably not available in adequate amounts in their diet hence the high incidence of iron deficiency anaemia during pregnancy. Amongst the first trimester Indian patients it has been shown that there is already present a degree of anaemia, while in 2% of these cases the percentage saturation was 10 and less. Therefore it is not surprising that the incidence of anaemia in the Indians during pregnancy is 26.7% as compared to 2% in in the African and 4% in the European. Multiple pregnancies, especially

/if ...

if at frequent intervals and commencing at an early age, are said to predispose to iron deficiency anaemia. Amongst my patients parity does not appear to have contributed. However, early marriage and pregnancies in rapid succession could possibly have been responsible for the high incidence in the Indian as compared with the African. Once again one feels that the dietary intake in the Indians is probably responsible in part, because these subjects become iron deficient despite the fact that the absorption of iron is enhanced in pregnancy (Hahn et al., 1951).

From the above considerations, it is apparent that a multiplicity of factors may be involved in the production of iron deficiency. Whichever factor is involved in a given case, the ultimate result is the same, viz. a negative iron balance with ultimate depletion of the iron stores of the body. This occurs irrespective of race or sex and is confirmed in the different racial groups studied. I hope I have achieved the purpose of correcting many misconceptions such as iron deficiency anaemia in the Indian is commonly due to hookworm infestation and also that the condition does not occur in the African. These have been impressions in the past. It has been found that iron deficiency, although uncommon, does occur in the African, and when it occurs, the picture does not differ from that of other racial groups.

SUMMARY

The thesis is comprised of a comparative study of iron metabolism with particular reference to iron deficiency in the Indian and the African; Europeans were included when material was available.

Fifty four patients with iron deficiency anaemia were studied. There were 43 Indians as compared with 11 Africans although the overall proportion of Indians to Africans admitted to the same ward was 1 : 4.

Amongst the Indians the commonest cause of anaemia from blood loss was peptic ulceration (6 patients); while 3 had cirrhosis of the liver, one had hookworm anaemia and one was a case of ulcerative colitis. Gynaecological lesions were found in 2 patients, one had a proliferative endometrium and the other had endometrial polypi. Idiopathic iron deficiency anaemia was found in 60.5% of the Indian patients (both male and female).

Amongst the Africans on the other hand, there were 2 cases of hookworm anaemia and 2 of cirrhosis of the liver while peptic ulceration was suspected in one patient who also had amoebic dysentery and urinary bilharziasis. None of the Africans had idiopathic iron deficiency anaemia with the possible exception of one who had 4 Caesarean sections in rapid succession.

There were 175 Indians, 175 Africans and 139 Europeans who were studied haematologically during pregnancy. The incidence of

/iron ...

iron deficiency anaemia among them was 26.7%, 2% and 4% respectively.

The third aspect of the thesis is confined to an analysis of necropsy material for iron stores. Two hundred Africans and 58 Indians were studied. It was found that the incidence of "siderosis" in the African was high. There was a significant difference in the iron concentrations in the stores of the 2 racial groups.

It was concluded that iron deficiency anaemia is common in the Indian. Diet appears to play an important role in its production.

APPENDIX I

CASE HISTORIES

Only positive findings are usually recorded in the summaries which follow.

Case 1

B.S. Indian female aged 18 years (I.6422/60)
Admitted: 15.9.60 Single Occupation: Housewife

History: Amenorrhoea for 7 months (at menarche). Desquamation hands and arms for 2 months. Painful calves and feet with paraesthesiae for 1 month. Dyspnoea and palpitations on marked exertion. Menses C15 3/28; dysmenorrhoea.

Social and dietary history: Lives with siblings. Both parents died while she was young. Diet: meat twice a week; vegetables - cabbage, potatoes, peas, beans, carrots - almost daily; fish once a week; fruit occasionally; eggs occasionally; milk with tea only.

Examination: Thin. Pale. Koilonychia. Angular stomatitis and cheilosis. Desquamation of skin of arms. C.V.S.: P. 80. B.P. 120/80. Slight cardiomegaly. Soft ejection systolic murmur in all areas. Chest: clear. Abdomen: splenomegaly. C.N.S.: Hypoaesthesia on feet.

Investigations: Hb 6.2 g. per 100 ml., M.C.H.C. 25%, reticulocyte count 1.9%.
Serum iron: 23 µg. per 100 ml., T.I.B.C. 490 µg. per 100 ml.
Stool: ascaris and trichocephalus ova.
Urine: normal.
Liver function test: serum bilirubin 0.5 mgm. per 100 ml., alkaline phosphatase 8 K.A. units, zinc turbidity 15 units, thymol turbidity 8 units, total proteins 3.9 g. per 100 ml., albumin 2.5 g. per 100 ml., globulin 1.4 g. per 100 ml., A/G ratio: 1.5/1.
Free acid present on gastric analysis.
Barium meal: normal.
Vitamin B12: 500 µµg. per ml.
Mantoux reaction: negative.

Diagnosis: Idiopathic iron-deficiency anaemia.
Pellagra.

Case 2

L.B. Indian female aged 25 years (I.8341/60)
Admitted: 6.12.60 Married

History: Sore throat and dysphagia for 5-6 years. Angular stomatitis for 2-3 years. Thyroid swelling for 2-3 years. Off-colour and loss of weight. Amenorrhoea for 2 months.

Social and dietary history: Husband, a labourer, earns R2.00 - R4.00 per month and separate food rations. 2 children who are 4 and 5 years old respectively. Diet: meat twice a week; eggs 2-3 times a week; no milk; no cheese. All vegetables regularly.

Examination: Pale mucosae. Slightly built. Nails brittle. Smooth diffuse thyroid enlargement, no toxicity. C.V.S.: P. 82. B.P. 100/60. Grade 2 ejection systolic murmur in pulmonary area. R.S.: sternal tenderness present. Abdomen: splenomegaly.

Investigations: Hb 9.6 g. per 100 ml., M.C.H.C. 28%, reticulocyte count 1.5%.
Bone marrow contained no iron granules.
Serum iron: 29 µg. per 100 ml., T.I.B.C. 596 µg. per 100 ml.
Stool: normal.
Urine: normal.
Liver function tests: serum bilirubin 0.8 mgm. per 100 ml., alkaline phosphatase 5 K.A. units, zinc turbidity 10 units, thymol turbidity 2 units, total proteins 9 g. per 100 ml., albumin 3.3 g. per 100 ml., globulin 5.7 g. per 100 ml., A/G ratio: 0.6/1.
Vitamin A absorption: first specimen 252 I.U. per 100 ml. rising to 7,257 I.U. per 100 ml. after 4 hours.
Free acid present on gastric analysis.
Barium meal: normal.
Vitamin B12: 170 µµg. per ml.
Mantoux reaction: positive.

Diagnosis: Idiopathic iron-deficiency anaemia.

Case 3

D.S. Indian female aged 24 years (I.8609/60)
Admitted: 15.12.60

History: Right sided frontal headache, feverishness, occasional "cramp" like pain in the right iliac fossa aggravated by menses, for 1 month. Menses 8/30; has missed 1 period. Slight dyspnoea on exertion for 1 month with ankle oedema.

Social and dietary history: No children. Married in February 1960. Husband earns R4.00 per week. Diet: meat 3 times a week, liver once a month; green vegetables daily; fruit daily; milk and cheese occasionally.

Examination: Pale mucosae. Nails not brittle. R.S.: clear. C.V.S.: P. 96. B.P. 135/65. No cardiomegaly. Ejection systolic murmur at pulmonary area. Left ventricular impulse. Peripheral pulses palpable.

Investigations: Hb 5.7 g. per 100 ml., M.C.H.C. 28%, reticulocyte count 1.0%.
Serum iron: 39 µg. per 100 ml., T.I.B.C. 544 µg. per 100 ml.
Stool: ascaris ova.
Urine: normal.
Liver function test: serum bilirubin 0.6 mgm. per 100 ml., alkaline phosphatase 4 K.A. units, zinc turbidity 8 units, thymol turbidity 2 units, total proteins 8 g. per 100 ml., albumin 4 g. per 100 ml., globulin 4 g. per 100 ml. A/G ratio: 1/1.
Vitamin C: 0.81 mgm. per 100 ml.
Vitamin A absorption test: first specimen 310 I.U. per 100 ml. rising to 1050 I.U. per 100 ml. after 4 hours.
Free acid present on gastric analysis.
Barium meal: normal.
Vitamin B12: 400 µµg. per ml.
Mantoux reaction: negative.

Initially a dimorphic picture but later an uncomplicated iron deficiency on peripheral smears.

Diagnosis: Idiopathic iron-deficiency anaemia.

Case 4

L.P. Indian female aged 35 years (I.924/61)
Admitted: 2.2.61 Occupation: Housewife

History: 4 days malaise and low backache aggravated by movement. Dyspnoea on exertion and palpitations. Oligomenorrhoea. Occasional headache.

Social and dietary history: Does not drink. Husband is a driver earning R12.00 per week. Lives with the family of 3 children. Diet: no meat; vegetables daily; fish once a week; eggs 3 times a week; milk with tea; fruit twice a week.

Examination: Marked pallor. Early koilonychia. C.V.S.: not dyspnoeic. P. 76. B.P. 110/40. Heart: soft ejection systolic murmur. Abdomen: splenomegaly.

Investigations: Hb 5.2 g. per 100 ml., M.C.H.C. 26%, reticulocyte count 2.8%.
Bone marrow contained no free iron.
Serum iron: 8 µg. per 100 ml., T.I.B.C. 421 µg. per 100 ml.
Stool: normal.
Urine: normal.
Liver function tests: serum bilirubin 0.6 mgm. per 100 ml., alkaline phosphatase 5 K.A. units, zinc turbidity 3 units, thymol turbidity 1 unit, total proteins 6.7 g. per 100 ml., albumin 4.4 g. per 100 ml., globulin 2.3 g. per 100 ml., A/G ratio: 1.9/1.
Vitamin C: Nil.
Vitamin A absorption test: first specimen 294 I.U. per 100 ml. rising to 1024 I.U. per 100 ml. after 4 hours.
Histamine fast achlorhydria after maximal stimulation.
Barium meal: normal.
Vitamin B12: 260 µµg. per ml.

Diagnosis: Idiopathic iron-deficiency anaemia.

Table 2.9.

CASES OF "IDIOPATHIC" IRON-DEFICIENCY ANAEMIA AMONGST FEMALES

Case No.	Age	Hb (g./100ml.)	Diet	Gastric acidity	Vitamin C (mgm./100ml.)	No. of Pregnancies	Menstrual Disturbance	Associated Diseases
6	9	2.1	Poor	+	1.3	-	Pre-menarche	Mild hookworm infestation with positive occult blood
32	19	3.9	Poor	Nil	-	-	-	Mild hookworm infestation with no occult blood
20	30	8.1	Good	-	-	6	Post-menopausal	Mild hookworm infestation with positive occult blood
8	40	7.4	Poor	Nil	0.1	7	Post-partum haemorrhage 6 years ago	Mild hookworm infestation with no occult blood Endometrium - inflammatory changes
19	14	5.5	Poor	Nil	0.7	-	Pre-menarche	??? Duodenal ulcer
11	14	5.8	Poor	+	1.9	-	Menorrhagia	-
1	18	6.2	Adequate	+	-	-	Amenorrhoea	Pellagra
16	22	6.1	Poor	+	1.3	-	Hypomenorrhoea	Normal endometrium
3	24	5.7	Adequate	+	0.8	-	Amenorrhoea	-
2	25	9.6	Good	+	-	2	Amenorrhoea	-
23	32	9.1	Adequate	-	-	9	-	All pregnancies ended as stillbirths
13	32	7.8	Poor	+	Nil	4	Hypomenorrhoea	-
21	34	8.1	Poor	+	0.1	5	-	Transfused for an abortion 1 year ago
4	35	5.2	Poor	Nil	Nil	3	Oligomenorrhoea	-
7	40	3.6	Good	-	0.7	5	Oligomenorrhoea	-
12	40	2.4	Poor	Nil	0.3	4	-	-
26	42	8.1	Adequate	+	0.8	3	Amenorrhoea	Thyrotoxicosis
15	45	5.0	Adequate	+	0.5	8	-	-
17	45	7.2	Adequate	+	0.6	5	Menorrhagia	Mild diabetes
18	49	6.0	Poor	+	0.7	1	Post-menopausal	-
24	56	5.8	Adequate	+	Nil	10	-	-
Mean	31.66	6.1			0.6	3.7		

+ = Present

Case 5

A.R. Indian female aged 51 years (I.1634/61)
Admitted: 27.2.61

History: Painful joints for 6 months. Tiredness and difficulty with swallowing solids (discomfort at level of angle of Louis) for 3 months. Also has retrosternal pain with exertion.

Social and dietary history: meat 3 times a week; vegetables daily; mealie rice; milk occasionally.

Examination: Pale. Thin nails. Tongue pale, smooth and fissured. C.V.S.: P. 76. B.P. 120/60. Heart: apex beat in the 5th left intercostal space just outside the mid-clavicular line; loud ejection systolic murmur down left sternal border. Chest: breath sounds decreased at both bases. Abdomen: liver 1 fingerbreadth enlarged and spleen 4 fingerbreadths enlarged.

Investigations: Hb 6.2 g. per 100 ml., M.C.H.C. 27%, reticulocyte count 4.2%, W.C.C. 3,700 per c.mm., platelets 380,000 per c.mm.
Bone marrow: rather acellular marrow with small erythroblasts; myelopoiesis normal. Trace of iron present.
Serum iron: 14 µg. per 100 ml., T.I.B.C. 377 µg. per 100 ml.
Stool: trace of occult blood; ascaris and trichocephalus ova.
Urine: pus cells+.
Liver function tests: serum bilirubin 0.4 mgm. per 100 ml., alkaline phosphatase 10.4 K.A. units, zinc turbidity 12 units, thymol turbidity 3 units, total proteins 7.3 g. per 100 ml., albumin 3 g. per 100 ml., globulin 4.3 g. per 100 ml., A/G ratio: 0.7/1.
Vitamin C: 0.4 mgm. per 100 ml.
Vitamin A absorption test: first specimen 319 I.U. per 100 ml. rising to 386 I.U. per 100 ml. after 4 hours.
Histamine fast achlorhydria after maximal stimulation.
Barium meal: normal.
Vitamin B12: 270 µµg. per ml.

Diagnosis: Iron-deficiency anaemia probably due to cirrhosis of the liver with congestive splenomegaly.

Case 6

S.H. Indian female aged 9 years (I.2729/61)
Admitted: 11.4.61 Scholar: Class II

History: Vague abdominal pain, backache and painful legs for 2 weeks. Swelling of face and body for 2 weeks. Becomes easily tired and dyspnoeic on exertion. For a few months has had pica for sand. Poor appetite. Often feels faint and giddy. No bleeding.

Dietary history: Meat once a week; fish occasionally; milk 1 cup 3 times a week at school; curry and rice daily; vegetables 3-4 times a week.

Examination: Very marked pallor and koilonychia. Extremities were warm. C.V.S.: P. 120, good volume. Jugular venous pressure elevated to 4 cm. Feet and legs oedematous. B.P. 120/65. Heart: 5th left intercostal space outside mid-clavicular line; very active heart. Gallop rhythm. Ejection systolic murmur and short early diastolic murmur. Chest: clear. Abdomen: 3 fingerbreadths non tender hepatomegaly. C.N.S.: normal.

Investigations: Hb 2.1 g. per 100 ml., M.C.H.C. 25%, reticulocyte count 1.8%, W.C.C. 6,500 per c.mm., eosinophils 9%.
Bone marrow: no free iron, normoblastic, most of the cells small.
Serum iron: 0 µg. per 100 ml., T.I.B.C. 620 µg. per 100 ml.
Stool: ova of trichuris trichiura and hookworm; occult blood positive.
Urine: normal.
Liver function tests: serum bilirubin 0.75 mgm. per 100 ml., alkaline phosphatase 6 K.A. units, zinc turbidity 23 units, thymol turbidity 5 units, total proteins 7.4 g. per 100 ml., albumin 2.9 g. per 100 ml., globulin 4.5 g. per 100 ml., A/G ratio: 0.65/1.
Vitamin C: 1.3 mgm. per 100 ml.
Vitamin A absorption test: first specimen 403 I.U. per 100 ml., rising to 1,142 I.U. per 100 ml. after 4 hours.
Free acid present on gastric analysis.
Barium meal: normal.
Vitamin B12: 204 µµg. per ml.

Diagnosis: Iron-deficiency anaemia associated with a mild hookworm infestation.

Case 7

G.M. Indian female aged 40 years (I.4020/61)
Admitted: 5.6.61 Occupation: Housewife

History: Progressive weakness and dyspnoea on exertion after walking a few yards, for 3 months. Unproductive cough for 1 week. Slight swelling of the feet. Oligomenorrhoea for a "long time".

Social and dietary history: Married with 1 live child, 4 had died. Diet: Breakfast - curry, bread and tea with milk. Lunch - curry and rice; vegetables and meat daily. Supper - meat, rice and curry. Does not drink fresh milk. Cheese occasionally.

Examination: Obese. Pale. Koilonychia. Minimal ankle oedema. C.V.S.: Jugular venous pressure elevated. P. 88. B.P. 120/80. Apex beat in the 5th left intercostal space just outside the mid-clavicular line, with a presystolic gallop rhythm at the apex and systolic murmur in all areas. R.S.: clear. Abdomen: 3 fingerbreadths firm splenomegaly; ± 3 fingerbreadths firm hepatomegaly.

Investigations: Hb 3.6 g. per 100 ml., M.C.H.C. 27.5%, reticulocyte count 0.5%, W.C.C. 7,500 per c.mm., with 2% eosinophils, platelets 170,000 per c.mm.
Bone marrow: not very cellular. Myeloid series normal, also erythroid although most of the normoblasts are small. Trace of iron present.
Serum iron: 17 µg. per 100 ml., T.I.B.C. 300 µg. per 100 ml.
Stool: trichocephalus ova.
Urine: normal.
Liver function tests: serum bilirubin 0.4 mgm. per 100 ml., alkaline phosphatase 5.4 K.A. units, zinc turbidity 11 units, thymol turbidity 4 units, total proteins 6.5 g. per 100 ml., albumin 3.1 g. per 100 ml., globulin 3.4 g. per 100 ml., A/G ratio: 0.9/1.
Vitamin C: 0.7 mgm. per 100 ml.
Vitamin A absorption test: first specimen 202 I.U. per 100 ml. rising to 2,587 I.U. per 100 ml. after 4 hours.
Vitamin B12: 260 µµg. per ml.

Diagnosis: Idiopathic iron-deficiency anaemia with mild congestive cardiac failure.

Case 8

R.S. Indian female aged 40 years (I.4775/61)
Admitted: 5.7.61

History: Dizziness, backache, dysphagia and pruritis vulvae for about 8 years. Dyspnoea on exertion with palpitations for about 1 year. Dysuria with frequency. Epigastric pain aggravated by meals; relieved by alkalis for about 1 year. No menorrhagia; irregular menses.

Social and dietary history: Has 7 children with ages ranging from 8 years to 21 years, the last one was a post partum haemorrhage. Diet: cabbage, beans, occasional fruit. No meat.

Examination: Not ill. Ankle oedema. Koilonychia - hands and feet. Mucous membranes pale. Angular stomatitis. Tongue smooth and pale. P. 84. B.P. 120/90. Heart: soft ejection systolic murmur in all areas. Gynaecologically inflammatory changes were found in the endometrium.

Investigations: Hb 7.4 g. per 100 ml., M.C.H.C. 26%, reticulocytes 0.8%, W.C.C. 12,800 per c.mm., platelets 350,000 per c.mm.
Bone marrow: active cellular, erythropoiesis normoblastic, myeloid series and megakaryocytes normal. No haemosiderin.
Serum iron: 7 µg. per 100 ml., T.I.B.C. 300 µg. per 100 ml.
Stool: hookworm ova.
Urine: 10 leucocytes per high power field.
Liver function tests: serum bilirubin 0.3 mgm. per 100 ml., alkaline phosphatase 4.5 K.A. units, zinc turbidity 10 units, thymol turbidity 3 units, total proteins 5.5 g. per 100 ml., albumin 2.3 g. per 100 ml., globulin 3.2 g. per 100 ml., A/G ratio: 0.8/1.
Protein electrophoresis (in g. per 100 ml.): albumin 1.9; globulins: α_1 0.7, α_2 1.4, β 1.3, gamma 2.4, total protein 7.7.
Vitamin C: 0.1 mgm. per 100 ml.
Vitamin A absorption test: first specimen 910 I.U. per 100 ml., rising to 2,330 I.U. per 100 ml. after 4 hours.
Histamine fast achlorhydria on gastric analysis.
Barium meal: normal.
Vitamin B12: 220 µµg. per ml.
Blood sugar: 135 mgm. per 100 ml.

Diagnosis: Iron-deficiency anaemia associated with a mild hookworm infestation.

Case 9

N.P. Indian female aged 51 years (I.4797/61)
Admitted: 6.7.61

History: Generalised pains for about 10 years. Tiredness with "weakness" for 1 year. Dyspnoea on exertion and slight dependent oedema for about 1 month. Menopausal for 1 year, menorrhagia previously.

Social and dietary history: Is looked after by a brother. Has no children. Single. "Sickly". Stays with brother with a big family and a low income. Diet: meat 2-3 times a week; eggs occasionally; fish occasionally; milk occasionally. Bread and rice daily. Vegetables daily.

Examination: Psychological overlay. Mucous membranes pale. Koilonychia. C.V.S.: P. 80. B.P. 165/100. Heart: heaving. Apex beat in the 6th interspace, 1½" outside the mid-clavicular line. Ejection systolic murmur in all areas. Abdomen: 3 fingerbreadths hepatomegaly. On curettage was shown to have endometrial polypi.

Investigations: Hb 6.8 g. per 100 ml., M.C.H.C. 26%, reticulocyte count 1.5%, W.C.C. 5,300 per c.mm.
Bone marrow: erythropoiesis normoblastic, most cells small. Myeloid series normal. No iron granules.
Serum iron: 0 µg. per 100 ml., T.I.B.C. 460 µg. per 100 ml.
Stool: normal.
Urine: normal.
Liver function tests: serum bilirubin 0.7 mgm. per 100 ml. alkaline phosphatase 8.5 K.A. units, zinc turbidity 4 units, thymol turbidity 2 units, total proteins 6.8 g. per 100 ml., albumin 3.3 g. per 100 ml., globulin 3.5 g. per 100 ml., A/G ratio: 0.9/1.
Vitamin C: 0.2 mgm. per 100 ml.
Vitamin A absorption test: first specimen 680 I.U. per 100 ml., rising to 1,640 I.U. per 100 ml. after 4 hours.
Free acid present on gastric analysis.
Barium meal: normal.
Vitamin B12: 127 µµg. per ml.

Diagnosis: Iron-deficiency anaemia due to endometrial polypi.

Case 10

D.G. Indian female aged 34 years (I.5284/61)
Admitted: 25.7.61

History: Generalised aches and pains for 12 years. Cough for 2 weeks with slightly blood-stained sputum on 2 occasions. Dyspnoea on exertion for 1 month and weakness for 2 weeks. Menses regular but doubtful about amount of blood loss.

Social and dietary history: Had 4 children, 2 of whom are alive. Diet: meat twice a week; milk seldom; no fish; eggs seldom; green vegetables occasionally.

Examination: Mucous membranes pale. Tongue smooth with a few patches of pigmentation. Koilonychia. Small areas of depigmentation on skin over trunk and legs. C.V.S.: P. 88. B.P. 125/75. Apex beat in the 5th interspace within the mid-clavicular line. Short soft ejection systolic murmur and a third heart sound. Abdomen: 1 fingerbreadth hepatomegaly, soft, and non tender. A proliferative endometrium found on curettage.

Investigations: Hb 9.0 g. per 100 ml., M.C.H.C. 29%, reticulocyte count 6.0%, W.C.C. 9,500 per c.mm., platelets 310,000.
Bone marrow: trace of iron present. Cellular marrow with normoblastic erythropoiesis, many of the normoblasts are small. White cell series and megakaryocytes normal.
Serum iron: 43 µg. per 100 ml., T.I.B.C. 530 µg. per 100 ml.
Stool: normal.
Urine: normal.
Liver function tests: serum bilirubin 0.8 mgm. per 100 ml., alkaline phosphatase 8 K.A. units, zinc turbidity 10 units, thymol turbidity 2 units, total proteins 7.0 g. per 100 ml., albumin 4.8 g. per 100 ml., globulin 2.2 g. per 100 ml., A/G ratio: 2.1/1.
Vitamin C: 0.3 mgm. per 100 ml.
Vitamin A absorption test: first specimen 134 I.U. per 100 ml. rising to 3,066 I.U. per 100 ml. after 4 hours.
Free acid present on gastric analysis.
Barium meal: normal.
Vitamin B12: 446 µµg. per ml.

Diagnosis: Iron-deficiency anaemia - had a proliferative endometrium.

Case 11

G.S. Indian female aged 14 years (I.6044/61)
Admitted: 23.8.61

History: Burning retrosternal pain for 3 days. Malaise and temporal headaches for 3 days. Dyspnoea on exertion for 6 months. Dysuria for 1 week. Menses 6/30 for 1½ years (3-4 pads per day).

Social and dietary history: Has 3 brothers and 3 sisters, all of whom are well. Parents are also well. Diet: meat once a week; fish occasionally; cabbage, green beans, peas and cauliflower often; fresh fruit daily; milk 1 glass per day.

Examination: Mucous membranes pale. Brittle thin nails. Tongue normal. P. 90. B.P. 120/65. Ejection systolic murmur in all areas.

Investigations: Hb 5.8 g. per 100 ml., M.C.H.C. 25%, reticulocyte count 2.5%, W.C.C. 5,530 per c.mm. with 2% eosinophils, platelets 760,000 per c.mm.
Bone marrow: no free iron. Most of the red cell precursors are small normoblasts and isolated larger cells present. Some of the metamyelocytes rather large.
Serum iron: 0 µg. per 100 ml., T.I.B.C. 355 µg. per 100 ml.
Stool: ova of ascaris, trichocephalus and E.hartmanni.
Urine: normal.
Liver function tests: serum bilirubin 1.1 mgm. per 100 ml., alkaline phosphatase 7 K.A. units, zinc turbidity 1 unit, thymol turbidity 1 unit, total proteins 5.8 g. per 100 ml., albumin 2.6 g. per 100 ml., globulin 3.2 g. per 100 ml., A/G ratio: 0.8/1.
Vitamin C: 1.9 mgm. per 100 ml.
Vitamin A absorption test: first specimen 403 I.U. per 100 ml. rising to 6804 I.U. per 100 ml. after 4 hours.
Free acid present on gastric analysis.
Barium meal: showed a flocculation pattern suggestive of malabsorption syndrome.
Vitamin B12: 344 µµg. per ml.

Diagnosis: Idiopathic iron-deficiency anaemia ? due to menorrhagia.

Case 12

S.R. Indian female aged 40 years (I.6539/61)
Admitted: 11.9.61 Occupation: Housewife

History: Generalised pains, progressive dyspnoea on exertion and headache for 3 months. Palpitations for 3 months together with swelling of ankles. Has a poor appetite. Menses 4/30, regular. No menorrhagia (2 pads per day).

Social and dietary history: Has 4 children of whom the youngest is 2½ years old. Diet: meat once a week; green vegetables daily; fish occasionally.

Examination: Mucous membranes pale. Koilonychia. Slight ankle oedema. P. 114. B.P. 130/80. Apex beat in the 6th intercostal space in the anterior axillary line. A systolic thrill with a pan-systolic murmur at the apex and an early diastolic murmur in the 4th left intercostal space. Abdomen: 1 fingerbreadth non tender hepatomegaly. Spleen easily palpable.

Investigations: Hb 2.4 g. per 100 ml., M.C.H.C. 26%, reticulocyte count 6.0%, W.C.C. 4,600 per c.mm., platelets 190,000 per c.mm.
Bone marrow: no free iron. Rather an acellular marrow in which both erythroid and myeloid cells are small in size. Erythropoiesis is normoblastic.
Serum iron: 18 µg. per 100 ml., T.I.B.C. 362 µg. per 100 ml.
Stool: trichocephalus ova.
Urine: normal.
Liver function tests: serum bilirubin 1.1 mgm. per 100 ml., alkaline phosphatase 12 K.A. units, zinc turbidity 9 units, thymol turbidity 3 units, total proteins 5.4 g. per 100 ml., albumin 1.8 g. per 100 ml. globulin 3.6 g. per 100 ml., A/G ratio: 0.5/1.
Protein electrophoresis (in g. per 100 ml.): albumin 1.4, globulins: α_1 0.5, α_2 0.7, β 1.0, gamma 2.2, total protein 7.7.
Vitamin C: 0.3 mgm. per 100 ml.
Vitamin A absorption test: 134 I.U. per 100 ml., rising to 369 I.U. per 100 ml. after 4 hours.
Barium meal: suggestive of intrinsic gastric neoplasm. (At laparotomy stomach found to be normal but displaced by a marked splenomegaly).
Histamine fast achlorhydria after maximal stimulation.
Vitamin B12: 399 µµg. per ml.

Diagnosis: Idiopathic iron-deficiency anaemia.

Case 13

P.R. Indian female aged 32 years (I.6559/61)
Admitted: 12.9.61

History: Fever and generalised pains associated with headache for 2 weeks. Micturition $\frac{D}{N}$ $\frac{4}{2}$. Menses: hypomenorrhoea.

Social and dietary history: Husband well. 2 siblings well. 3 children lost. One child who is well is 9 years old. Husband works in a knitting mill. Diet: meat once a week; fish once a month; no milk; vegetables 3 times a week; fruit occasionally.

Examination: Mucosae pale. Koilonychia. Dry skin and hair. C.V.S.: P. 88. B.P. 130/80. Apex beat in the 5th intercostal space within the mid-clavicular line. A soft ejection systolic murmur was present. Abdomen: 1 fingerbreadth hepatomegaly and 2 fingerbreadths firm splenomegaly.

Investigations: Hb 7.8 g. per 100 ml., M.C.H.C. 31%, reticulocyte count 2.5%.
Bone marrow: no free iron. Not a very cellular marrow. Erythropoiesis is normoblastic.
Serum iron: 16 µg. per 100 ml., T.I.B.C. 560 µg. per 100 ml.
Stool: normal.
Urine: normal.
Liver function tests: serum bilirubin 0.7 mgm. per 100 ml., alkaline phosphatase 7 K.A. units, zinc turbidity 6 units, thymol turbidity 3 units, total proteins 6.3 g. per 100 ml., albumin 1.9 g. per 100 ml., globulin 4.4 g. per 100 ml., A/G ratio: 0.4/1.4.
Vitamin C: nil.
Vitamin A absorption test: first specimen 277 I.U. per 100 ml. rising to 1,882 I.U. per 100 ml. after 4 hours.
Free acid present on gastric analysis.
Barium meal: normal.
Blood urea: 36 mgm. per 100 ml.

Diagnosis: Idiopathic iron-deficiency anaemia.

Case 14

S.R. Indian female aged 18 years (I.7511/61)
Admitted: 16.10.61 Occupation: Works in a clothing factory

History: Joint pains for 4 months. Tiredness on the slightest exertion for 1 week. Loss of appetite for 3 days and loss of weight for 4 months. Menses ? 7/1-3 months.

Social and dietary history: 8 siblings, all of whom are well. Lives with her parents. Diet: Fairly poor. Does not eat meat. No eggs. Drinks a glass of milk 3 times a week. Fish bi-weekly. Green vegetables frequently. Rice for bulk.

Examination: Hair ginger and lustreless. Crazy pavementing of skin of legs. Very pale. Koilonychia. Tongue smooth. C.V.S.: P. 120. B.P. 115/70. Slight cardiomegaly. The 1st heart sound at the apex was loud and a pansystolic murmur was present. The pulmonary sound was accentuated. Abdomen: 2 fingerbreadths non tender firm liver enlargement and a 3 fingerbreadths splenomegaly.

Investigations: Hb 3.2 g. per 100 ml., M.C.H.C. 27%, reticulocyte count 6%, W.C.C. 1,800 per c.mm., 2% eosinophils, platelets 190,000 per c.mm.
Bone marrow: micronormoblastic; no iron present.
Serum iron: 13 µg. per 100 ml., T.I.B.C. 370 µg. per 100 ml.
Stool: ova of ascaris, trichocephalus, hookworm and E.coli.
Urine: occasional red blood cells.
Liver function tests: serum bilirubin 0.5 mgm. per 100 ml., alkaline phosphatase 3 K.A. units, zinc turbidity 4 units, thymol turbidity 2 units, total proteins 5.8 g. per 100 ml., albumin 2.6 g. per 100 ml., globulin 3.2 g. per 100 ml., A/G ratio: 0.8/1.
Protein electrophoresis (in g. per 100 ml.): albumin 3.3; globulins: α_1 0.6, α_2 0.5, β 1.0, gamma 2.0, total protein 7.4.
Vitamin C: Nil.
Vitamin A absorption test: first specimen 168 I.U. per 100 ml., rising to 554 I.U. per 100 ml. after 4 hours.
Fat balance: total faecal fat content: 12.4 g., 4.2 g., 5.6 g., 3.4 g. per 24 hours stool each.
Histamine fast achlorhydria after maximal stimulation.
Barium meal: suggestive of duodenal ulcer crater.

Diagnosis: Iron-deficiency anaemia due to duodenal ulceration.
Malnutrition.
Mild hookworm infestation.

Case 15

C.N. Indian female aged 45 years (I.7862/61)
Admitted: 27.10.61

History: Swelling of the feet for 2 years. Palpitations and dyspnoea on exertion for 3 months. Menses 6/28; regular. Has 8 children, the youngest being 12 years old.

Dietary history: Meat twice a week; fish twice a week; milk with tea only; no eggs, vegetables daily (all cooked); fruit occasionally.

Examination: Mucosae pale. Feet oedematous. C.V.S.: P. 90. B.P. 135/85. Ejection systolic murmur in all areas. Abdomen: Liver 1 fingerbreadth enlarged.

Investigations: Hb 5 g. per 100 ml., M.C.H.C. 24%, reticulocyte count 8%, W.C.C. 7,900 per c.mm., platelets 260,000 per c.mm.
Bone marrow: active cellular. Normoblastic erythropoiesis with many micro-normoblasts. No haemosiderin.
Serum iron: 20 µg. per 100 ml., T.I.B.C. 385 µg. per 100 ml.
Stool: trichocephalus ova.
Urine: normal.
Liver function tests: serum bilirubin 0.4 mgm. per 100 ml., alkaline phosphatase 5 K.A. units, zinc turbidity 7 units, thymol turbidity 3 units, total proteins 6.4 g. per 100 ml., albumin 3.4 g. per 100 ml., globulin 3.0 g. per 100 ml., A/G ratio: 1.1/1.
Protein electrophoresis (in g. per 100 ml.): albumin 1.6; globulins: α_1 0.7, α_2 1.0, β 1.1, gamma 2.4, total protein 6.8.
Vitamin C: 0.5 mgm. per 100 ml.
Vitamin A absorption test: first specimen 344 I.U. per 100 ml. rising to 10,439 I.U. per 100 ml. after 4 hours.
Free acid present on gastric analysis.

Diagnosis: Idiopathic iron-deficiency anaemia.

Case 16

L.C. Indian female aged 22 years (I.8198/61)
Admitted: 11.11.61 Single Occupation: Housewife

History: Weakness, palpitations and dyspnoea on exertion for 2 to 3 months. Swelling of the legs for 4 days. Menses irregular (hypomenorrhoea). No menorrhagia.

Dietary history: Vegetarian. Poor diet, predominantly rice.

Examination: Marked pallor and oedema. C.V.S.: P. 108, irregular. Jugular venous pressure elevated. Heart: slight cardiomegaly. Systolic thrill and an ejection systolic murmur at the apex. A gallop rhythm was heard. Abdomen: 2 fingerbreadths hepatomegaly and splenomegaly. Gynaecologically normal.

Investigations: Hb 6.1 g. per 100 ml., M.C.H.C. 26.5%, W.C.C. 9,000 per c.mm., with 2% eosinophils, platelets 540,000 per c.mm.
Bone marrow: active cellular. Normoblastic erythropoiesis, the cells being micronormoblasts. White cell series and megakaryocytes normal. No free iron observed.
Serum iron: 10 µg. per 100 ml., T.I.B.C. 415 µg. per 100 ml.
Stools: ascaris and trichocephalus ova.
Urine: albumin trace; approximately 10 leucocytes per high power field.
Liver function tests: serum bilirubin 0.5 mgm. per 100 ml., alkaline phosphatase 7 K.A. units, zinc turbidity 6 units, thymol turbidity 3 units, total proteins 6.7 g. per 100 ml., albumin 3.2 g. per 100 ml., globulin 3.5 g. per 100 ml., A/G ratio: 0.9/1.
Protein electrophoresis (in g. per 100 ml.): albumin 2.5; globulins: α_1 0.5, α_2 0.7, β 0.9, gamma 2.3, total protein 6.9.
Vitamin C: 1.3 mgm. per 100 ml.
Vitamin A absorption test: first specimen 201 I.U. per 100 ml., rising to 1,344 I.U. per 100 ml. after 4 hours.
Free acid present on gastric analysis.
Barium meal: some pylorospasm demonstrated.

Diagnosis: Idiopathic iron-deficiency anaemia in congestive cardiac failure.

Case 17

B.S. Indian female aged 45 years (I.8587/61)
Admitted: 22.11.61 Occupation: Housewife

History: "Burning" epigastric pain aggravated by meals and not relieved by milk or antacids, for 2 months. Occurs for 2-3 days with remissions of 2-3 days. Diabetic. Gripping precordial pain for a few days. Menses regular 8/28. Bleeds excessively for first 3 days for 1 year (12 pads per day). Para 4, gravida 5.

Dietary history: Is "on an ulcer diet". Boiled foods with decreased amounts of starch.

Examination: Mucous membranes pale. Tongue smooth. C.V.S.: P. 112. B.P. 190/90. Heart: a soft ejection systolic murmur was heard. Abdomen: epigastric and right hypochondrial tenderness. Spleen just palpable. On curettage no abnormality was noted in the uterus.

Investigations: Hb 7.2 g. per 100 ml., M.C.H.C. 26%, reticulocyte count 3.2%, W.C.C. 8,500 per c.mm. with 3% eosinophils, platelets 290,000 per c.mm.
Bone marrow: active cellular. Erythropoiesis normoblastic with micronormoblasts. Very scanty iron deposits seen.
Serum iron: 11 µg. per 100 ml., T.I.B.C. 470 µg. per 100 ml.
Stool: occult blood present.
Urine: normal.
Liver function tests: serum bilirubin 0.3 mgm. per 100 ml., alkaline phosphatase 7 K.A. units, zinc turbidity 6 units, thymol turbidity 2 units, total proteins 7.9 g. per 100 ml., albumin 1.5 g. per 100 ml., globulin 6.4 g. per 100 ml., A/G ratio: 0.2/1.
Protein electrophoresis (in g. per 100 ml.): albumin 1.2; globulins: α_1 0.7, α_2 1.4, β 1.5, gamma 1.6, total protein 6.6.
Vitamin C: 0.6 mgm. per 100 ml.
Vitamin A absorption test: first specimen 420 I.U. per 100 ml., rising to 604 I.U. per 100 ml. after 4 hours.
Free acid present on gastric analysis.
Barium meal demonstrated pylorospasm - ? secondary to other pathology (?cholecystitis)
Cholecystogram: normal.
Barium enema: normal.
Glucose tolerance test: fasting blood sugar of 164 mgm. per 100 ml., and curve was suggestive of mild diabetes.

Diagnosis: Idiopathic iron-deficiency anaemia.
Mild diabetes mellitus.

Case 18

B.S. Indian female aged 49 years (I.3579/62)
Admitted: 26.4.62

History: Epigastric pain associated with low back ache for 2 weeks. Dyspnoea on exertion for 2 weeks. Occasional cough. Occasional palpitations. Menses: 1 year irregular periods occurring every 2-3 weeks, bleeds little for 2-3 days; no menorrhagia.

Social and dietary history: Husband is unemployed for last 3 years. She earns 60c. a week for washing. Occasionally helped by neighbours. Has one child who is living away. Child was at a tuberculosis sanatorium. Post-menopausal. Diet: mainly rice, bread and porridge. Meat once a week; occasional eggs; fish once a week; vegetables - beans, cabbage and dhall; fruit occasionally.

Examination: Scars right neck (discharged pus years ago). Pallor of mucosae. Early koilonychia. Tongue smooth and pigmented. C.V.S.: P. 80. B.P. 120/80. Abdomen: spleen palpable. 1 finger-breadth hepatomegaly.

Investigations: Hb 6.0 g. per 100 ml., M.C.H.C. 22%, reticulocyte count 2.8%, W.C.C. 5,800 per c.mm., platelets 170,000 per c.mm.
Bone marrow: no haemosiderin. Normoblastic erythropoiesis, cells mostly small normoblasts.
Serum iron: 8 µg. per 100 ml., T.I.B.C. 394 µg. per 100 ml.
Urine: no abnormality.
Stool: ova of ascaris.
Liver function tests: serum bilirubin 1.01 mgm. per 100 ml., alkaline phosphatase 8 K.A. units, zinc turbidity 7 units, thymol turbidity 3 units, total proteins 6.8 g. per 100 ml., albumin 3 g. per 100 ml., globulin 3.8 g. per 100 ml., A/G ratio: 0.8/1.
Protein electrophoresis (in g. per 100 ml.): albumin 2.2; α_1 0.6, α_2 1.0, β 1.9, gamma 2.2, total protein 7.9.
Mantoux positive.
Vitamin C: 0.7 mgm. per 100 ml.
Vitamin A absorption test: first specimen 117.6 I.U. per 100 ml., rising to 1159.6 I.U. per 100 ml. after 4 hours.
Free acid present on gastric analysis.
Barium meal: normal.
Radio-active vitamin B12 absorption test: normal.

Diagnosis: Idiopathic iron-deficiency anaemia.

Case 19

K.S. Indian female aged 14 years (I.1941/62)
Admitted: 1.3.62 Occupation: Scholar

History: Feels quite well but attended hospital because she was observed by the teacher to have "weak" blood. Admitted on direct questioning to a year of shortness of breath when going up steps. Pre-menarche.

Social and dietary history: Has 8 siblings. Diet: meat once a week; eggs once a week; fish nil; milk 1 cup per day; cheese and butter frequently; vegetables - peas, beans, cabbage; peanuts (some type daily); fruit 2-3 times a week.

Examination: Not ill. Marked pallor of mucosae. No koilonychia. Tongue normal. Abdomen: liver just palpable. 2 fingerbreadths splenomegaly. C.V.S.: P. 100. Triple rhythm heard. Short early systolic murmur in all areas.

Investigations: Hb 5.5 g. per 100 ml., M.C.H.C. 22%, reticulocyte count 2.0%, W.C.C. 8,000 per c.mm., platelets 139,000 per c.mm.
Bone marrow: no iron observed. Moderate cellularity showing normoblastic erythropoiesis, normal myeloid series.
Serum iron: 1 µg. per 100 ml., T.I.B.C. 497 µg. per 100 ml.
Urine: no abnormality.
Stool: ascaris and trichocephalus ova.
Liver function tests: serum bilirubin 0.6 mgm. per 100 ml., alkaline phosphatase 10 K.A. units, zinc turbidity 5 units, thymol turbidity 3 units, total proteins 6.3 g. per 100 ml., albumin 3.2 g. per 100 ml., globulin 3.1 g. per 100 ml., A/G ratio: 1/1.
Protein electrophoresis (in g. per 100 ml.): albumin 2.2; globulins: α_1 0.2, α_2 0.5, β 0.5, gamma 0.9, total protein 4.3.
Mantoux: negative.
Vitamin C: 0.7 mgm. per 100 ml.
Vitamin A absorption test: first specimen 336 I.U. per 100 ml., rising to 779.4 I.U. per 100 ml. after 4 hours.
Fat balance: total faecal fat content: 6 g., 5.8 g., 5.2 g., 3.1 g., 2.9 g. per 24 hours stool each.
Histamine fast achlorhydria after maximal stimulation.
Barium meal: duodenal cap distorted but not tender - a duodenal ulcer, though unlikely, has not been excluded.
Radio-active vitamin B12 absorption test: within normal limits.
Hb electrophoresis: Hb A. Alkaline denaturation = foetal Hb less than
Hb A 2% of total Hb.

Diagnosis: Idiopathic iron-deficiency anaemia but a duodenal ulcer suggested on barium meal examination.

Case 20

K.M. Indian female aged 30 years (I.932/62)
Admitted: 29.1.62 Occupation: Housewife

History: Painless swelling in the upper abdomen - gradually progressing in size. Last menstrual period 1½ years ago. Does not smoke or drink.

Social and dietary history: Married. 6 children ranging from 13 - 1½ years of age, all well. Husband a salesman. Diet: Breakfast - bread, tea with milk and sugar. Lunch - rice, bread, vegetables, meat or fish. Supper - as for lunch.

Examination: Not ill. Mucosae pale. C.V.S.: P. 84. B.P. 110/80. Abdomen: distended. Large mass in upper abdomen extending down to umbilicus, with a sharp edge and moving on respiration, felt to be hepatomegaly. Spleen just tipped. No ascites.

Investigations: Hb 8.1 g. per 100 ml., M.C.H.C. 29%, reticulocyte count 0.8%, W.C.C. 4,300 per c.mm., platelets 290,000 per c.mm.
Bone marrow: no iron deposits. Cellular, normoblastic erythropoiesis, normal myeloid series.
Serum iron: 28 µg. per 100 ml., T.I.B.C. 610 µg. per 100 ml.
Urine: albumin+, pus cells++, occasional red blood cells. Heavy growth of B.coli and A.aerogenes.
Stool: no occult blood; hookworm ova. Culture: negative.
Chest X-ray: Normal except for elevation of right dome.
X-ray abdomen: large soft tissue mass in upper abdomen on right. more anteriorly than posteriorly situated.
Liver function tests: direct van den Bergh slight trace, serum bilirubin 0.7 mgm. per 100 ml., alkaline phosphatase 8 K.A. units, zinc turbidity 4 units, thymol turbidity 2 units, total protein 6 g. per 100 ml., albumin 2.7 g. per 100 ml., globulin 3.3 g. per 100 ml., A/G ratio: 0.8/1.
Mantoux: positive.
Barium meal: normal.
Blood urea: 30 mgm. per 100 ml.
Fasting blood sugar: 71 mgm. per 100 ml.
Vitamin B12: 1440 µµg. per ml.
Cholecystogram: normal.
Intravenous pyelogram: mass appears to arise outside the kidney and pressing on it, obstructing upper calyces on the right.

Diagnosis: Idiopathic iron-deficiency anaemia associated with a mild hookworm infestation and urinary infection.

Case 21

C.P. Indian female aged 34 years (I.610/62)
Admitted: 18.1.62 Occupation: Housewife

History: Dysphagia for solids; hoarseness; malaise; retrosternal pain - ? anginal, and gets easily tired, for 3 months. Last menstrual period 4 days ago. No menorrhagia. Menses 16, 5-6/28.
Numbness right face - 6 years ago. Appendicectomy - 19 years ago.
Abortion in February 1961, when she was transfused.

Social and dietary history: Married. Husband and 4 children - youngest 8 years old. Husband a weaver in a textile factory. Diet: meat 5 lbs. a week; fish occasionally; milk 1 pint a day; cheese 1 lb. a week, butter 1½ lb. a week; fruit daily.

Examination: Very thin. Pallor of mucous membranes. Nails flattened.
C.V.S.: P. 84. B.P. 120/90. Abdomen: splenomegaly - just palpable.
E.N.T. opinion: normal.

Investigations: Hb 8.1 g. per 100 ml., M.C.H.C. 27%, reticulocyte count 0.2%, W.C.C. 3,400 per c.mm., platelets 255,000 per c.mm.
Bone marrow: contained no iron. Erythropoiesis normoblastic; most normoblasts rather small. Megakaryocytes and myeloid series normal.
Serum iron: 35 µg. per 100 ml., T.I.B.C. 364 µg. per 100 ml.
Urine: normal.
Stool: normal.
Liver function tests: direct van den Bergh slight trace, serum bilirubin 0.9 mgm. per 100 ml., alkaline phosphatase 5 K.A. units, zinc turbidity 4 units, thymol turbidity 1 unit, total proteins 7.2 g. per 100 ml., albumin 3.3 g. per 100 ml., globulin 3.9 g. per 100 ml., A/G ratio: 0.8/1.
Protein electrophoresis (in g. per 100 ml.): albumin 2.4, globulins: α_1 0.6, α_2 0.7, β 0.5, gamma 1.6, total protein 5.8.
Mantoux: negative.
Vitamin C: 0.1 mgm. per 100 ml.
Vitamin A absorption test: first specimen 252 I.U. per 100 ml., rising to 688 I.U. per 100 ml. after 4 hours.
Barium meal: normal, including a thin barium swallow.
Free acid present on gastric analysis.
Radio-active vitamin B12 absorption test: normal.

Diagnosis: Idiopathic iron-deficiency anaemia.

Case 22

H.B.M. Indian female aged 56 years (I.3578/62)
Admitted: 26.4.62 Occupation: Housewife

History: An asthmatic for "many years". Swelling of abdomen intermittently, relieved by injections, for 8 years. Exacerbation for 1 month. About 8 years post-menopausal. Dyspnoea on exertion for 2 years. Swelling of feet for 2 weeks.

Dietary history: Meat 2-3 times a week; 2 eggs a week; fish 3 times a week; no milk; vegetables - beans, cabbage, tomatoes, potatoes.

Examination: Pale muoosae. Oedema of ankles. Pellagrinous dermatoses on hands. C.V.S.: P. 76. B.P. 120/70. Chest: crepitations at the right base. Abdomen: marked ascites. After paracentesis 3 fingerbreadths splenomegaly and 3 fingerbreadths hepatomegaly, firm and irregular palpated.

Investigations: Hb 5.3 g. per 100 ml., M.C.H.C. 25%, reticulocyte count 4.2%, W.C.C. 7,100 per c.mm., platelets 220,000 per c.mm.
Bone marrow: no free iron. Normoblastic erythropoiesis.
Serum iron: 9 µg. per 100 ml., T.I.B.C. 249 µg. per 100 ml.
Urine: normal.
Stool: weakly positive for occult blood.
Liver function tests: serum bilirubin 0.8 mgm. per 100 ml., alkaline phosphatase 9 K.A. units, zinc turbidity 9 units, thymol turbidity 3 units, total proteins 5.4 g. per 100 ml., albumin 1.6 g. per 100 ml., globulin 3.8 g. per 100 ml., A/G ratio: 0.4/1.
Protein electrophoresis (in g. per 100 ml.): albumin 1.6; globulins: α_1 0.8, α_2 0.1, β 1.0, gamma 2.2, total protein 6.2.
Mantoux: negative.
Vitamin C: 0.2 mgm. per 100 ml.
Vitamin A absorption test: first specimen 403.2 I.U. per 100 ml., rising to 2,192.4 I.U. per 100 ml. after 4 hours.
Barium meal: normal.
Free acid present after maximal stimulation on gastric analysis.
Ascitic fluid: protein 0.9 g. per 100 ml., sterile. Few lymphocytes, occasional polymorphs.
Serum electrolytes: Na 133, K 4.7, Cl 133 mEq/l.
Blood Wassermann reaction: negative.

Diagnosis: Iron-deficiency anaemia with cirrhosis of the liver.

Case 23

A.B. Indian female aged 32 years (I.4442/62)
Admitted: 28.5.62

History: Sensation of food sticking in the throat and difficulty with breathing for 2 months. Deafness since childhood. Dyspnoea even at rest. Dysphagia. Polyuria $\frac{D}{N}$ $\frac{6-8}{0}$. Menses normal 5/28.
9 full-term stillbirths; last one 10 years ago; no live births.

Social and dietary history: Husband working in a laundry. Diet: meat twice a week; fish occasionally; rice, bread, mealie rice and vegetables usually.

Examination: Pallor of mucous membranes. Mild thyroid swelling. Smooth tongue. C.V.S.: P. 96. B.P. 150/100. Soft short early systolic murmur at base. No signs of thyrotoxicosis.

Investigations: Hb 9.1 g. per 100 ml., M.C.H.C. 27%, reticulocyte count 1.6%, W.C.C. 6,900 per c.mm.
Bone marrow: no haemosiderin. Small normoblasts.
Serum iron: 35 µg. per 100 ml., T.I.B.C. 444 µg. per 100 ml.
Urine: normal.
Stool: normal.
Liver function tests: serum bilirubin 0.8 mgm. per 100 ml., alkaline phosphatase 6 K.A. units, zinc turbidity 6 units, thymol turbidity 2 units, total proteins 8.0 g. per 100 ml., albumin 3.3 g. per 100 ml., globulin 4.7 g. per 100 ml., A/G ratio: 0.7/1.
Protein electrophoresis (in g. per 100 ml.): albumin 2.3; globulins: α_1 0.7, α_2 1.0, β 1.3, gamma 1.8, total protein 7.1.
Mantoux: negative.
Vitamin A absorption test: first specimen 92.4 I.U. per 100 ml., rising to 2856.0 I.U. per 100 ml. after 4 hours.
Barium meal: no post cricoid web. Slight reflux from stomach in Trendellenburg position; no hiatus hernia demonstrated.
Blood Wassermann reaction: negative.
Serum cholesterol: 200 mgm. per 100 ml.
Protein-bound iodine: 4.8 µg. per 100 ml.

Diagnosis: Idiopathic iron-deficiency anaemia.

Case 24

G.N. Indian female aged 56 years (I.8154/62)
Admitted: 10.10.62 Occupation: Housewife

History: Malaise, fatigability and dyspnoea on exertion, all progressively getting worse, for 6 months. Has been anorectic throughout. Vague generalised abdominal discomfort for 6 days. 6 months constipation. 1 week dysuria. Menses 2-3/28; no inter-menstrual bleeding. 2 days frontal headaches.

Social and dietary history: Married. Has 10 children ranging in age from 22 years to 8 years. Normal deliveries. Husband earns R10.00 per week, one son earns R7.00 per week. Pay monthly instalments on a house R7.00 per month. Diet: meat and fish 2-3 times a week; no eggs, milk, cheese; vegetables 2-3 times a week (beans, carrots, cabbage). (Does not eat supper herself).

Examination: T. 103. Not distressed. Warm periphery. Pallor of mucosae. Flattening of nails. Smooth tongue. C.V.S.: Hyperdynamic circulatory state. P. 112. B.P. 130/80. Abdomen: 1 fingerbreadth non tender hepatomegaly. No haemorrhoids nor ulcers on rectal examination. On vaginal examination - chronic cervicitis.

Investigations: Hb 5.8 g. per 100 ml., M.C.H.C. 23%, reticulocyte count 5.8%, W.C.C. 7,000 per c.mm., platelets 193,000 per c.mm.
Bone marrow: no haemosiderin. Rather acellular with micronormoblasts.
Serum iron: 33 µg. per 100 ml., T.I.B.C. 326 µg. per 100 ml.
Urine: non catheter specimen showed: pus++ (probably due to contamination)
Stools: ova of trichocephalus.
Chest X-ray: normal.
Liver function tests: direct van den Bergh positive, serum bilirubin 1.6 mgm. per 100 ml., alkaline phosphatase 5 K.A. units, zinc turbidity 7 units, thymol turbidity 3 units, total proteins 7.5 g. per 100 ml., albumin 2.9 g. per 100 ml., globulin 4.6 g. per 100 ml., A/G ratio: 0.6/1.
Protein electrophoresis (in g. per 100 ml.): albumin 2; globulins: α_1 0.4, α_2 0.8, β 1.1, gamma 1.7, total protein 6.0.
Mantoux: positive.
Vitamin C: nil.
Vitamin A absorption test: first specimen 386 I.U. per 100 ml., rising to 789 I.U. per 100 ml. after 4 hours.
Barium meal: normal.
Free acid present on gastric analysis.
Blood urea: 27 mgm. per 100 ml.

Diagnosis: Idiopathic iron-deficiency anaemia. Chronic cervicitis also present.

Case 25

E.A. Indian female aged 47 years (I.6994/62)
Admitted: 30.8.62 Occupation: Housewife

History: Inability to concentrate and sundry aches. Further history unreliable because highly suggestible. Dyspnoea on exertion of long duration. Menses 4-5/30, not excessive. Was at a mental institution for about 3 months for similar complaints.

Social and dietary history: 1 of 7 siblings. Unmarried. ? Mixed adequate diet.

Examination: T. 96. Mucosae pale. Plaintive. Dry skin and hair. "Puffy" facies. C.V.S.: P. 68. B.P. 125/75. C.N.S.: mental change suggestive of hypochondriasis and depression with slowness of activity. Ankle jerks delayed relaxation and return phase.

Investigations: Hb 7.8 g. per 100 ml., M.C.H.C. 31%.
Bone marrow: normoblastic with no stainable iron.
Serum iron: 22 µg. per 100 ml., T.I.B.C. 507 µg. per 100 ml.
Urine: normal.
Stool: normal.
Chest X-ray: minimal cardiomegaly.
Liver function tests: serum bilirubin 0.65 mgm. per 100 ml., alkaline phosphatase 7.0 K.A. units, zinc turbidity 4 units, thymol turbidity 4 units, total proteins 6.2 g. per 100 ml., albumin 2.9 g. per 100 ml., globulin 3.3 g. per 100 ml., A/G ratio: 0.9/1.
Mantoux: positive.
Vitamin A absorption test: first specimen 638 I.U. per 100 ml., rising to 3634 I.U. per 100 ml., after 4 hours.
Barium meal: normal.
Free acid present on gastric analysis.
Electrocardiogram: low voltage.
Serum cholesterol: 350 mgm. per 100 ml.
Protein-bound iodine: 2.3 µgm. per 100 ml. I_{131} uptake was 4% up to 24 hours. Clearly in hypothyroid range.

Diagnosis: Iron-deficiency anaemia associated with myxoedema.

Case 26

S.M. Indian female aged 42 years (I.8183/62)
Admitted: 11.10.62 Occupation: Housewife

History: Generalised aches and pains for 1 year, progressive over the last 6 months. Palpitations for 6 months. Sore throat and mouth with dysphagia, mainly for solids, for 3 months. Dyspnoea on exertion and swelling of the feet for 3 months. Frequency of micturition with burning for 6 months. Amenorrhoea for 6 months. Swelling in neck for 6 months. No voice changes.

Social and dietary history: Married. Husband employed in a factory earning R6.00 per week. Had 3 children but all died soon after birth. Diet: meat 2-3 times a week; rice frequently; eggs 1-2 per week. Vegetables - cabbage, beans, potatoes, peas - 2-3 times a week. Fruit occasionally. No fish, milk or cheese.

Examination: Not ill looking. Marked koilonychia. Smooth red tongue with angular stomatitis. Warm periphery. Legs oedematous. C.V.S.: P. 105. B.P. 130/60. Soft ejection systolic murmur - precordium. Multinodular thyromegaly with a bruit overlying the left half. Bilateral exophthalmos with lid lag and infrequent blinking. Some sluggishness of movements suggestive of myopathy.

Investigations: Hb 8.1 g. per 100 ml., M.C.H.C. 26%, reticulocyte count 2.8%.
Bone marrow: no haemosiderin. Normally cellular marrow, erythropoiesis normoblastic. Myeloid series and megakaryocytes normal.
Serum iron: 28 µg. per 100 ml., T.I.B.C. 361 µg. per 100 ml.
Urine: normal.
Stool: occult blood negative; ascaris and trichocephalus.
Chest X-ray: normal.
Liver function tests: serum bilirubin 0.5 mgm. per 100 ml., alkaline phosphatase 11 units, zinc turbidity 8 units, thymol turbidity 2 units, total proteins 7.4 g. per 100 ml., albumin 2.1 g. per 100 ml., globulin 5.3 g. per 100 ml., A/G ratio: 0.4/1.
Protein electrophoresis (in g. per 100 ml.): albumin 2.5; globulins: α_1 0.7, α_2 0.9, β 1.3, gamma 2.0, total protein 7.4.
Mantoux: positive.
Vitamin C: 0.8 mgm. per 100 ml.
Vitamin A absorption test: first specimen 184 I.U. per 100 ml., rising to 3074 I.U. per 100 ml. after 4 hours.
Barium meal: normal.
Free acid present on gastric analysis.
Protein-bound iodine: 9.3 µg. per 100 ml., I_{131} uptake 74% at 2 hours (upper normal 35%) and 59% at 24 hours.
Serum cholesterol: 190 mgm. per 100 ml.

Diagnosis: Idiopathic iron-deficiency anaemia.
Thyrotoxicosis.

Case 27

K.D. Indian female aged 21 years. (I.4345/62)
Admitted: 24.5.62 Single Occupation: Housewife

History: Loose mucoid, bloody stools 3-4 times daily intermittently for 2 months. Vague epigastric discomfort post-prandially for 1 week. Has had episodes of arthralgia in all large joints intermittently over the past year. Occasional substernal pain for 2 weeks. Menses: last menstrual period 2 weeks ago. 5-6/30. No menorrhagia. Was investigated for dyspepsia in November 1959 after a report of melaena stools, but barium examination at the time was negative.

Social and dietary history: Parents not alive - mother had a "stroke"; father had blood in the stools terminally. 5 siblings all of whom are well. Diet: meat twice a week; eggs occasionally; no fish. Condensed milk daily with tea. All kinds of vegetable. Curry and rice predominantly.

Examination: Not ill looking. Pale. C.V.S.: P. 90. B.P. 135/70. Abdomen: tender in epigastrium and both iliac fossae. Descending and ascending colon palpable. On proctoscopy: no haemorrhoids seen and mucosa finely granular. Sigmoidoscope passed to 15": Haemorrhagic areas mainly in lower rectum with mucus on mucosa. These changes compatible with any chronic diarrhoea.

Investigations: Hb 8.9 g. per 100 ml., M.C.H.C. 27%, reticulocyte count 3.2%, W.C.C. 8,000 per c.mm., platelets 240,000 per c.mm.
Bone marrow: no haemosiderin. Erythropoiesis is normoblastic.
Serum iron: 16 µg. per 100 ml., T.I.B.C. 405 µg. per 100 ml.
Urine: normal.
Stool: culture normal. Pus++ Blood+++.
Chest X-ray: normal.
Liver function tests: serum bilirubin 0.3 mgm. per 100 ml., alkaline phosphatase 8 K.A. units, zinc turbidity 4.8 units, thymol turbidity 1.2 units, total proteins 6.2 g. per 100 ml., albumin 3.0 g. per 100 ml., globulin 3.2 g. per 100 ml., A/G ratio: 0.9/1.
Protein electrophoresis (in g. per 100 ml.): albumin 1.8; globulins: α_1 0.6, α_2 0.9, β 1.0, gamma 1.6, total protein 5.9.
Vitamin A absorption test: first specimen 319 I.U. per 100 ml., rising to 1982 I.U. per 100 ml. after 4 hours.
Barium meal: normal.
Free acid present on gastric analysis.
Barium enema: within normal limits.

Diagnosis: Iron-deficiency anaemia associated with dysentery, probably ulcerative colitis.

Case 28

P.G. Indian female aged 45 years (I.9623/62)
Admitted: 29.11.62

History: Abdominal pain, mainly lower abdominal, unrelated to anything and constant, for 3 months. Vomited frequently, particularly after eating or drinking anything, for 3 weeks. 3 days loose watery stools. Dyspnoea on exertion for 3 months. Menses 4-5/30. No menorrhagia. No melaena.

Social and dietary history: Married. Husband died 8 years ago of "heart trouble". Has a 28 year old daughter. Therefore a widow living with relatives (is seeking a pension or grant to keep her going financially). Diet: mealie rice and vegetable curry daily. Vegetables daily - cabbage, beans, cauliflower. Meat once a week. No fish. Milk with tea only. No butter or cheese.

Examination: Not distressed. Mucosae pale. Koilonychia. Tongue dry and smooth. C.V.S.: P. 92. B.P. 125/75. Bilateral loin tenderness. Proctoscopy: normal.

Investigations: Hb 9.0 g. per 100 ml., M.C.H.C. 30%, reticulocyte count 1.0%, W.C.C. 7,000 per c.mm.
Bone marrow: no haemosiderin. Normal appearance of marrow except that erythroblasts are rather small.
Serum iron: 6 µg. per 100 ml., T.I.B.C. 346 µg. per 100 ml.
Urine: 3 leucocytes per high power field. Heavy growth of B.coli.
Stool: ascaris and trichocephalus ova.
Liver function tests: serum bilirubin 0.3 mgm. per 100 ml., alkaline phosphatase 8 K.A. units, zinc turbidity 2 units, total protein 6.9 g. per 100 ml., albumin 2.8 g. per 100 ml., globulin 4.1 g. per 100 ml. A/G ratio: 0.7/1.
Protein electrophoresis (in g. per 100 ml.): albumin 2.2; globulins: α_1 0.8, α_2 1.1, β 1.2, gamma 2.8, total protein 8.1.
Mantoux: negative.
Vitamin A absorption test: first specimen 270 I.U. per 100 ml., rising to 500 I.U. per 100 ml. after 4 hours.
Barium meal: scarred duodenal cap.
Free acid present on gastric analysis.
Blood urea 37 mgm. per 100 ml.
Serum electrolytes: Na 139, K 3.9, Cl 86 mEq/l.

Diagnosis: Iron-deficiency anaemia due to peptic ulceration.

Case 29

M.G. Indian female aged 21 years (I.9312/62)
Admitted: 19.11.62 Occupation: Housework

History: Puffiness of the face, lassitude and dyspnoea for 7 days. Palpitations. Menses: 3-4/28, regular, scanty blood loss. Was treated elsewhere eleven months ago for the same complaints - after which she had improved but then stopped taking treatment.

Social and dietary history: Unmarried. Has 6 siblings. No other member of family with similar complaint. Diet: meat twice a week; eats spinach and vegetables. Mainly rice, bread and potatoes.

Examination: Marked pallor. Koilonychia. Looks ill. Smooth tongue. C.V.S.: P. 90. B.P. 120/80. Grade 2 ejection systolic murmur over precordium and carotids. Abdomen: 1 fingerbreadth splenomegaly. 1½ fingerbreadth hepatomegaly, firm and sharp-edged.

Investigations: Hb 2.8 g. per 100 ml., M.C.H.C. 22%, reticulocyte count 0.2%.
Bone marrow: no haemosiderin.
Serum iron: 23 µg. per 100 ml., T.I.B.C. 311 µg. per 100 ml.
Urine: albumin nil, ± 5 leucocytes per high power field, scanty red blood cells.
Stool: ova of hookworm (heavy); occult blood positive.
Liver function tests: serum bilirubin 0.5 mgm. per 100 ml., alkaline phosphatase 10 K.A. units, zinc turbidity 5 units, thymol turbidity 1 unit, total proteins 6.1 g. per 100 ml., albumin 2.5 g. per 100 ml., globulin 3.6 g. per 100 ml., A/G ratio: 0.7/1.
Protein electrophoresis (in g. per 100 ml.): albumin 2; globulins: α_1 0.3, α_2 0.7, β 0.9, gamma 1.1.
Vitamin C: 0.1 mgm. per 100 ml.
Vitamin A absorption test: first specimen 80 I.U. per 100 ml. rising to 630 I.U. per 100 ml. after 4 hours.
Fat Balance: total faecal fat content: 5.8 g. per 24 hour stool.
Barium meal: normal.
Free acid present on gastric analysis.
Vitamin B12: 344 µµg. per ml.

Diagnosis: Iron-deficiency anaemia probably due to hookworm infestation.

Case 30

D.S. Indian female aged 42 years (I.5382/62)
Admitted: 3.7.62 Occupation: Housewife

History: Epigastric pain, post prandial, relieved by milk and alkalis for 9 months. Pain in the legs for 3 weeks. Progressive dyspnoea on exertion for 3 weeks; palpitations. Anorexia. Menses: last menstrual period 2 days ago. 3-4/28; no menorrhagia. "High blood pressure" for 1 year.

Social and dietary history: Married. 8 children, 2 daughters married; all others supporting her. Diet: ? adequate.

Examination: Not distressed. Mucosae pale. Rather obese. Minimal ankle and sacral oedema. P. 80. B.P. 160/95. Grade 2-3 ejection systolic murmur. Abdomen: slight epigastric tenderness.

Investigations: Hb 7.1 g. per 100 ml., M.C.H.C. 27%, reticulocyte count 5.4%, W.C.C. 10,200 per c.mm., platelets 510,000 per c.mm.
Bone marrow: no iron. Active cellular marrow in which the normoblasts are small.
Serum iron: 5 µg. per 100 ml., T.I.B.C. 525 µg. per 100 ml.
Urine: albumin nil; ± 10 leucocytes per high power field; coarsely granular casts and epithelial cells.
Stool: occult blood positive.
Chest X-ray: heart enlarged; plethoric lung fields; rather prominent pulmonary conus. Consistent with congestive cardiac failure ? due to mitral stenosis.
Liver function tests: serum bilirubin 0.7 mgm. per 100 ml., alkaline phosphatase 5 K.A. units, zinc turbidity 3 units, thymol turbidity 2 units, total proteins 7.2 g. per 100 ml., albumin 3.2 g. per 100 ml., globulin 4.0 g. per 100 ml., A/G ratio: 0.8/1.
Protein electrophoresis (in g. per 100 ml.): albumin 2.9; globulins: α_1 0.6, α_2 0.8, β 1.1, gamma 1.9, total protein 7.3.
Mantoux: negative.
Vitamin C: 0.5 mgm. per 100 ml.
Vitamin A absorption test: first specimen 176.4 I.U. per 100 ml., rising to 1864.8 I.U. per 100 ml. after 4 hours.
Barium meal: irritable spastic duodenal cap; chronic duodenal ulcer not excluded.
Free acid present on gastric analysis.

Diagnosis: Iron-deficiency anaemia probably due to bleeding duodenal ulcer.

Case 31

R.S. Indian female aged 42 years (I.8827/62)
Admitted: 1.11.62 Occupation: Housewife

History: Swelling of abdomen and legs for 1 week. No clear history of haematemesis and melaena. Menses 3-4/30; not excessive.

Social and dietary history: Married and has 6 children, the last of whom is 7 years old. Diet: meat once a week; no eggs. Diet restricted to vegetables, bread and rice mainly.

Examination: Thin, with gross ascites and oedema of legs. Afebrile. Pale. Mild koilonychia (no glossitis). C.V.S.: hyperkinetic circulatory state. P. 120. B.P. 120/80. Vigorous pulsations in neck, probably venous. Apex beat in the 6th interspace at mid-clavicular line. Ejection systolic murmur at base. Gross ascites, spleen ballotable.

Investigations: Hb 3.5 g. per 100 ml., M.C.H.C. 25%, reticulocytes 0.5%, W.C.C. 2,400 per c.mm., platelets 200,000 per c.mm.
Bone marrow: contained no haemosiderin.
Serum iron: 0 µg. per 100 ml., T.I.B.C. 258 µg. per 100 ml.
Urine: normal.
Stool: normal. 2 hookworm ova in one specimen.
Chest X-ray: small right basal effusion.
Liver function tests: serum bilirubin 1.4 mgm. per 100 ml., alkaline phosphatase 5 K.A. units, zinc turbidity 24 units, thymol turbidity 2 units, total protein 6.1 g. per 100 ml., albumin 2.1 g. per 100 ml., globulin 4.0 g. per 100 ml., A/G ratio: 0.5/1.
Protein electrophoresis (in g. per 100 ml.): albumin 1.3; globulins: α_1 0.6, α_2 0.4, β 0.7, gamma 1.6, total protein 4.6.
Mantoux: negative.
Vitamin C: 0.2 mgm. per 100 ml.
Vitamin A absorption test: first specimen 16.8 I.U. per 100 ml., rising to 840 I.U. per 100 ml., after 4 hours.
Barium meal: normal.
Free acid present on gastric analysis.
Glucose tolerance test (in mgm. per 100 ml.): Fasting blood sugar 118, 128 ($\frac{1}{2}$ hr.) 174 (1 hr.) 148 ($1\frac{1}{2}$ hr.) 164 (2 hr.) 82 ($2\frac{1}{2}$ hr.)
Peritoneal fluid: acellular; protein 2.2 g. per 100 ml.

Diagnosis: Iron-deficiency anaemia with cirrhosis of the liver and a mild hookworm infestation.

Case 32

G.P. Indian female aged 19 years (I.10958/63)
Admitted: 14.1.63. Occupation: Clothing factory worker

History: Swelling of legs and dyspnoea on exertion for 1 week.
Menses: last menstrual period 7.1.63. C 16 4/28; no menorrhagia.
Had similar complaints a year ago.

Social and dietary history: Earns R2.50 per week in a clothing factory. 4 in the family unit. Father died about 15 months ago of a cerebro-vascular accident. Mother gets grants of R6.00 and R4.00 per month for self and 2 sons. Total income R20.00 per month, of which rent is R4.00 per month and food costs R8.00 per month. Diet: predominantly vegetable. Meat once a week. Milk with tea only. Fish occasionally.

Examination: T. 98°F. Very pale. P. 88. B.P. 110/70. Short ejection systolic murmur in all areas.

Investigations: Hb 3.9 g. per 100 ml., M.C.H.C. 21.5%, reticulocyte count 2.9%, W.C.C. 9,000 per c.mm.
Serum iron: 11 µg. per 100 ml., T.I.B.C. 499 µg. per 100 ml.
Bone marrow: no iron.
Urine: 20 leucocytes per high power field and scanty red blood cells; albumin present; urine sterile.
Stool: ova of hookworm, ascaris and trichocephalus.
Chest X-ray: normal.
Liver function tests: serum bilirubin 0.2 mgm. per 100 ml., alkaline phosphatase 3 K.A. units, zinc turbidity 4 units, thymol turbidity 2 units, total protein 5.6 g. per 100 ml., albumin 2.9 g. per 100 ml., globulin 2.7 g. per 100 ml., A/G ratio: 1.1/1.
Protein electrophoresis (in g. per 100 ml.): albumin 2.1; globulins: α_1 0.5, α_2 0.9, β 1.1, gamma 2.1, total protein 6.7.
Mantoux: positive.
Vitamin A absorption test: first specimen 218 I.U. per 100 ml., rising to 2452 I.U. per 100 ml. after 4 hours.
Histamine fast achlorhydria after maximal stimulation.

Diagnosis: Idiopathic iron-deficiency anaemia associated with a urinary infection and mild hookworm infestation.

Case 33

A.N. Indian male aged 78 years (I. 772/61)
Admitted: 28.1.61.

History: Exacerbation of dyspnoea on effort, swelling of the legs and fever for 3 weeks. History of pain in the chest (? angina) and dyspnoea for about 4 years. Central chest pain on effort.

Social and dietary history: Drinks small amounts of alcohol. Smokes 20 cigarettes a day. Diet: meat once a week; milk and bread for breakfast; fish about once a week; vegetables occasionally.

Examination: Dyspnoeic. Somewhat confused. Marked pallor. Slight oedema of legs and sacrum. C.V.S.: P. 96. B.P. 120/60. Jugular venous pressure elevated. Heart: apex beat in the 7th intercostal space outside the mid clavicular line. Pansystolic murmur at apex conducted to the axilla. Chest: bilateral basal crepitations. Abdomen: 2 fingerbreadths hepatomegaly.

Investigations: Hb 3.9 g. per 100 ml., M.C.H.C. 23%, W.C.C. 8,000 per c.mm.
Bone marrow: contained no haemosiderin. Cellular bone marrow: normoblasts small, while leucopoiesis is normal.
Serum iron: 16 µg. per 100 ml., T.I.B.C. 432 µg. per 100 ml.
Stool: normal.
Urine: normal.
Liver function tests: serum bilirubin 0.8 mgm. per 100 ml., alkaline phosphatase 10 K.A. units, zinc turbidity 8 units, total proteins 7.5 g. per 100 ml., albumin 3.3 g. per 100 ml., globulin 4.2 g. per 100 ml., A/G ratio: 0.8:1.
Vitamin C: 1.8 mgm. per 100 ml.
Vitamin A absorption test: first specimen 268 I.U. per 100 ml. rising to 294 I.U. per 100 ml. after 4 hours.
Fat balance: total faecal fat content: average excretion of 0.7 g. per 24 hour stool.
Barium meal: normal pattern.
Vitamin B12: 256 µµg. per ml.
Electrocardiogram showed a right bundle branch block.

Diagnosis: Iron-deficiency anaemia due to gastro-intestinal haemorrhage.
Ischaemic heart disease with mild congestive cardiac failure.

Case 34

B.J. Indian male aged 18 years (I. 3319/61)
Admitted: 5.5.61 Occupation: Handyman

History: Progressive dyspnoea on exertion associated with palpitations for 1 year. Pain in the left chest, not pleuritic, and right abdomen for 1 week.

Social and dietary history: 1 sibling had a pleural effusion.
Diet: meat 2-3 times a week; milk with tea; rice, vegetables and curries frequently.

Examination: Not ill. Very pale. Tongue pale with smooth edges.
C.V.S.: P. 80. B.P. 120/60. Heart: soft ejection systolic murmur in all areas. Chest: slight sternal tenderness. "Barrel-shaped".
Abdomen: 1 fingerbreadth firm splenomegaly; liver just palpable.

Investigations: Hb 4.9 g. per 100 ml., M.C.H.C. 24.5%, reticulocyte count 3%, W.C.C. 3,400 per c.mm. with 5% eosinophils, platelets 160,000 per c.mm.
Bone marrow: no free iron. Somewhat acellular with relatively infrequent primitive cells; normoblastic.
Serum iron: 27 µg. per 100 ml., T.I.B.C. 430 µg. per 100 ml.
Stool: normal.
Urine: normal.
Liver function test: serum bilirubin 0.4 mgm. per 100 ml., alkaline phosphatase 5 K.A. units, zinc turbidity 7 units, thymol turbidity 2 units, total proteins 7 g. per 100 ml., albumin 4.4 g. per 100 ml., globulin 2.6 g. per 100 ml., A/G ratio: 1.7/1.
Protein electrophoresis (in g. per 100 ml.): albumin 2.9; globulins: α_1 0.6, α_2 0.7, β 1.2, gamma 1.6.
Vitamin C: 1.0 mgm. per 100 ml.
Vitamin A absorption test: first specimen 227 I.U. per 100 ml., rising to 3,444 I.U. per 100 ml. after 4 hours.
Free acid present on gastric analysis.
Barium meal: no oesophageal varices; no ulceration; pattern normal.
Vitamin B12: 636 µµg. per ml.

Diagnosis: Idiopathic iron-deficiency anaemia.

Case 35

H.R. Indian male aged 11 years (I.5840/61)
Admitted: 16.8.61

History: Dyspnoea on moderate exertion and palpitations for 3 weeks. Vomits occasionally. No history of any blood loss.

Social and dietary history: Adopted since he was a day old and brought up as a son. Has 3 sisters and 4 brothers, all of whom are well. Grandfather died 11 days ago. Diet: Breakfast - bread and tea. Lunch - bread or rice and curry. Supper - curry and rice. Meat twice a week (eats little); eggs 1-2 a week; fish seldom. Does not like green vegetables.

Examination: Mucosae pale. Koilonychia. C.V.S.: P. 108. B.P. 90/60. Triple rhythm, a soft ejection systolic murmur and a loud pulmonary 2nd sound. Abdomen: 1 fingerbreadth firm non tender hepatomegaly. 1 fingerbreadth firm splenomegaly.

Investigations: Hb 9.0 g. per 100 ml., M.C.H.C. 28%, reticulocyte count 9%, W.C.C. 11,900 per c.mm., platelets 480,000 per c.mm.
Bone marrow: no iron observed. A cellular marrow in which erythropoiesis is normoblastic. Myeloid series and megakaryocytes normal.
Serum iron: 30 µg. per 100 ml., T.I.B.C. 715 µg. per 100 ml.
Stool: trichocephalus and non viable S.mansoni ova.
Urine: S.haematobium ova; pus cells and red blood cells.
Liver function tests: serum bilirubin 0.5 mgm. per 100 ml., alkaline phosphatase 11 K.A. units, zinc turbidity 16 units, thymol turbidity 4 units, total proteins 7.5 g. per 100 ml., albumin 2.9 g. per 100 ml., globulin 4.6 g. per 100 ml., A/G ratio: 0.6/1.
Vitamin C: 1.4 mgm. per 100 ml.
Vitamin A absorption test: first specimen 1150 I.U. per 100 ml. rising to 2730 I.U. per 100 ml. after 4 hours.
Free acid present on gastric analysis.
Barium meal: duodenal ulcers. Duodenal cap deformed, 2 ulcer craters one near base, other near apex and mucosae of first and beginning of the second part of the duodenum was irregular. ? Mild malabsorption.
Vitamin B12: 258 µµg. per ml.
Blood urea: 18 mgm. per ml.

Diagnosis: Iron-deficiency anaemia with duodenal ulceration and bilharziasis.

Case 36

D.N. Indian male aged 14 years (I. 7764/61)
Admitted: 24.10.61 Occupation: Scholar

History: Tiredness for 5 months, pain in the left iliac fossa, not related to meals and aggravated by running for 18 months. No blood loss, nor melaena.

Social and dietary history: Has 7 siblings, all of whom are well. He is the second last child. Diet: meat 4 times a week; fish once a month; eggs daily; milk daily; vegetables and fruit regularly.

Examination: Mucosae pale. T. 98.4°F. P. 80. B.P. 105/60. A short systolic murmur in all areas. Liver 1 fingerbreadth enlarged and spleen was just tipped. No epigastric tenderness.

Investigations: Hb 5.9 g. per 100 ml., M.C.H.C. 26%, reticulocyte count 1.9%, W.C.C. 9,000 per c.mm., platelets 160,000 per c.mm.
Serum iron: 12 µg. per 100 ml., T.I.B.C. 366 µg. per 100 ml.
Stool: occult blood weakly positive; trichocephalus ova.
Urine: normal.
Liver function tests: serum bilirubin 0.4 mgm. per 100 ml., alkaline phosphatase 11 K.A. units, zinc turbidity 5 units, thymol turbidity 2 units, total proteins 6.6 g. per 100 ml., albumin 3.2 g. per 100 ml., globulin 3.4 g. per 100 ml., A/G ratio: 0.9/1.
Protein electrophoresis (in g. per 100 ml.): albumin 2.1; globulins: α_1 0.7, α_2 1.2, β 1.4, gamma 1.7, total protein 7.1.
Vitamin C: 0.8 mgm. per 100 ml.
Vitamin A absorption test: first specimen 336 I.U. per 100 ml. rising to 4082 I.U. per 100 ml. after 4 hours.
Barium meal: gastric ulcers demonstrated.
Histamine fast achlorhydria after maximal stimulation.

Diagnosis: Iron-deficiency anaemia due to peptic ulceration.

Case 37

M.G. Indian male aged 27 years (I.8181/61)
Admitted: 9.11.61

History: Progressive dyspnoea on exertion, palpitations, weakness, headache, dizziness and dryness of the tongue, for 6 months. Admitted to another medical ward 2 years previously when "hookworm anaemia" was diagnosed as based on the stool report.

Dietary history: Meat twice a week; fish once a week; eggs seldom; milk seldom; vegetables occasionally; rice frequently.

Examination: Marked pallor. P. 72. B.P. 95/50. Heart: a soft ejection systolic murmur. Abdomen: 1 fingerbreadth non-tender hepatomegaly and the spleen was just palpable.

Investigations: Hb 4.8 g. per 100 ml., M.C.H.C. 25%, reticulocyte count 5.2%, platelets 110,000, W.B.C. 4,200 per c.mm. with 2% eosinophils.
Bone marrow: normoblastic erythropoiesis with many micronormoblasts. Iron deposits very scanty. Normal erythropoiesis with many micronormoblasts.
Serum iron: 12 µg. per 100 ml., T.I.B.C. 414 µg. per 100 ml.
Stool: normal.
Urine: normal.
Liver function tests: serum bilirubin 0.2 mgm. per 100 ml., alkaline phosphatase 7 K.A. units, zinc turbidity 6 units, thymol turbidity 1 unit, total proteins 6.9 g. per 100 ml., albumin 3.9 g. per 100 ml., globulin 3.0 g. per 100 ml., A/G ratio: 1.3/1.
Protein electrophoresis (in g. per 100 ml.): albumin 2.8; globulins: α_1 0.3, α_2 0.4, β 0.8, gamma 1.0, total protein 5.3.
Vitamin C: Nil.
Vitamin A absorption test: first specimen 436 I.U. per 100 ml., rising to 2,100 I.U. per 100 ml. after 4 hours.
Free acid present on gastric analysis.
Barium meal: negative.

Diagnosis: Idiopathic iron-deficiency anaemia.

Case 38

M.M. Indian male aged 25 years (I.8416/61)
Admitted: 16.11.61

History: Generalised weakness, joint pains and dyspnoea for 2 years. Exacerbation of swelling of joints for 1 week. In D Ward in April 1960 for ankylosing spondylitis (clinically). Frequency of micturition but no dysuria.

Dietary history: vegetarian - no eggs, meat, milk or fish.

Examination: Dull. Koilonychia. Marked pallor of mucosae. Jugular venous pressure elevated to 2 cm. P. 90. B.P. 130/80. Ejection systolic murmur. Abdomen: spleen just tipped. Knees and spine: full movement elicited.

Investigations: Hb 6.1 g. per 100 ml., M.C.H.C. 22, reticulocyte count 2.0%, W.C.C. 7,300 with 12% eosinophils, platelets 230,000 per c.mm.
Bone marrow: normoblastic erythropoiesis. Cells micronormoblasts. Occasional giant metamyelocytes. No iron seen.
Serum iron: 9 µg. per 100 ml., T.I.B.C. 460 µg. per 100 ml.
Stool: light hookworm infestation and no occult blood.
Urine: normal.
Liver function tests: serum bilirubin 0.4 mgm. per 100 ml., alkaline phosphatase 6 K.A. units, zinc turbidity 14 units, thymol turbidity 2 units, total proteins 6.2 g. per 100 ml., albumin 3.0 g. per 100 ml., globulin 3.2 g. per 100 ml., A/G ratio: 0.9/1.
Protein electrophoresis (in g. per 100 ml.): albumin 2.2; globulins: α_1 0.3, α_2 0.9, β 1.3, gamma 1.8, total protein 6.5.
Vitamin C: 0.6 mgm. per 100 ml.
Vitamin A absorption test: first specimen 201 I.U. per 100 ml. rising to 2,394 I.U. per 100 ml. after 4 hours.
Free acid present on gastric analysis.
Barium meal: some delay in stomach emptying, i.e. in a 6-hour film barium was still present but nil else abnormal.

Diagnosis: Iron-deficiency anaemia associated with a mild hookworm infestation and ankylosing spondylitis.

Case 39

C.R. Indian male aged 28 years (I.2173/62)
Admitted: 8.3.62

History: Headache and vague abdominal pain with diarrhoea passing 1-2 stools per day, for 4-5 months. Progressive tiredness, insomnia and swelling of feet and legs for 1 month. Palpitations when going uphill. Appetite good. Nocturnal frequency of micturition. Perforated peptic ulcer in 1955. Gastrectomy in 1956. Haemorrhoidectomy in 1961 after a 3-year history.

Social and dietary history: One of a family of 13. Unmarried. Lives with family. Earns R13.00 per week. Smokes 20 cigarettes a day.
Diet: Meat once a week; fish once a week; eggs once a week; butter daily; no milk; vegetables - cabbage, carrots, peas, beans, potatoes, lettuce, pumpkin, spinach - daily (some type); fruit - daily (oranges, bananas, apples).

Examination: Not ill looking. Mucosae very pale. Flattening of nails. C.V.S.: P. 120. B.P. 140/60. Short systolic murmur over the precordium. Abdomen: healed operation scars. 2 fingerbreadths hepatomegaly. Slight epigastric tenderness.

Investigations: Hb 4.0 g. per 100 ml., M.C.H.C. 23%, reticulocyte count 2%, W.C.C. 9,000 per c.mm., platelets 1,580,000 per c.mm.
Bone marrow: no free iron. Fairly active marrow as regards erythrocytes and megakaryocytes. Red blood cell precursors mostly small normoblasts.
Serum iron: 7 µg. per 100 ml., T.I.B.C. 545 µg. per 100 ml.
Urine: normal.
Stool: ova of trichocephalus.
Liver function tests: serum bilirubin 0.4 mgm. per 100 ml., alkaline phosphatase 5 K.A. units, zinc turbidity 2 units, thymol turbidity 1 unit, total proteins 5.6 g. per 100 ml., albumin 3.1 g. per 100 ml., globulin 2.5 g. per 100 ml., A/G ratio: 1.2/1.
Protein electrophoresis (in g. per 100 ml.): albumin 2.6; globulins: α_1 0.5, α_2 1.0, β 1.8, gamma 2.0, total protein 7.9.
Vitamin C: 0.5 mgm. per 100 ml.
Vitamin A absorption test: first specimen 84.0 I.U. per 100 ml., rising to 806.4 I.U. per 100 ml. after 4 hours.
Histamine fast achlorhydria after maximal stimulation.
Barium meal: gastrectomy. Some flocculation on 2 hour film.
Radio-active vitamin B12 absorption test: suggests moderate impairment of absorption.
Serum calcium: 9.15 mgm. per 100 ml.
Blood Wassermann reaction: negative.

Diagnosis: Iron-deficiency anaemia associated with a post-gastrectomy syndrome.

Case 40

R.S. Indian male aged 12 years (I.5359/62)
Admitted: 2.7.62 Occupation: Scholar

History: Shortness of breath of many years duration. Vague pains in the legs for 1 week. Anorexia for a long time. An asthmatic for years. No history of any blood loss.

Social and dietary history: Has 5 siblings. Father has hypertension. Diet: has a small appetite. Bread, rice, milk occasionally; eats no meat.

Examination: Not distressed. Marked pallor. Flat nails. C.V.S.: P. 80. B.P. 110/60. Grade 2-3 ejection systolic murmur. Abdomen: 1 fingerbreadth hepatomegaly.

Investigations: Hb 5.2 g. per 100 ml., M.C.H.C. 22%, reticulocyte count 1.2%, W.C.C. 6,200 per c.mm., platelets 227,000 per c.mm.
Bone marrow: no haemosiderin granules. Erythropoiesis is normoblastic.
Serum iron: 12 µg. per 100 ml., T.I.B.C. 499 µg. per 100 ml.
Stool: no occult blood; ova of trichocephalus.
Chest X-ray: normal.
Liver function tests: serum bilirubin 0.5 mgm. per 100 ml., alkaline phosphatase 22 K.A. units, zinc turbidity 7 units, thymol turbidity 3 units.
Protein electrophoresis (in g. per 100 ml.): albumin 1.9; globulins: α_1 0.7, α_2 0.8, β 1.3, gamma 2.2.
Mantoux: positive.
Vitamin C: 0.2 mgm. per 100 ml.
Vitamin A absorption test: first specimen 1066 I.U. per 100 ml. rising to 1444 I.U. per 100 ml. after 4 hours.
Barium meal: normal.
Free acid present without stimulation on gastric analysis.
Vitamin B12: 519 µµg. per ml.

Diagnosis: Idiopathic iron-deficiency anaemia.
Bronchial asthma.

Case 41

G.N. Indian male aged 7 years (I.6353/62)
Admitted: 7.8.62

History: Progressive breathlessness and tiredness on exertion for 2 months. Fever, generalised joint pains and yellowness of eyes for 1 week. Had an epistaxis 3 days ago. Stools well coloured; no melaena nor any blood loss from elsewhere.

Social and dietary history: 5th child, others well. Diet: meat daily, vegetables daily.

Examination: In some distress showing pallor of mucous membranes, flattened nails and mild jaundice. T. 100.5. C.V.S.: P. 108. B.P. 90/65. Hyperdynamic pulsations over precordium with a grade 3 early short systolic murmur. Abdomen: 2½ fingerbreadths hepatomegaly and 2 fingerbreadths splenomegaly.

Investigations: 4.0 g. per 100 ml., M.C.H.C. 21%, reticulocyte count 5.6%, W.C.C. 10,600 per c.mm., platelets 445,000 per c.mm.
Bone marrow: trace of haemosiderin. Fairly cellular marrow with normoblastic erythropoiesis; the normoblasts being rather small.
Serum iron: 8 µg. per 100 ml., T.I.B.C. 446 µg. per 100 ml.
Urine: normal.
Stool: normal.
Liver function tests: direct van den Bergh positive, serum bilirubin 1.6 mgm. per 100 ml., alkaline phosphatase 37 K.A. units, zinc turbidity 11 units, thymol turbidity 9 units, total proteins 6.9 mgm. per 100 ml., albumin 2.5 mgm. per 100 ml., A/G ratio: 0.6/1.
Protein electrophoresis (in g. per 100 ml.): albumin 2.3; globulins: α_1 0.8, α_2 1.0, β 1.3, gamma 1.7, total protein 7.1.
Mantoux: negative.
Vitamin C: 0.5 mgm. per 100 ml.
Vitamin A absorption test: first specimen 218.4 I.U. per 100 ml., rising to 2,352.0 I.U. per 100 ml. after 4 hours.
Barium meal: normal.
Free acid present on gastric analysis.
Blood Wassermann reaction: negative.
Hb electrophoresis Hb A/Hb A.
Serum vitamin B12: 787 µµg. per ml.
Coomb's test: antierythrocyte antibodies not detected.

Diagnosis: Idiopathic iron-deficiency anaemia.
Infective hepatitis.

Case 42

S.M. Indian male aged 12 years (I.2753/62)
Admitted: 28.3.62 Occupation: Scholar

History: Intermittent attacks of diarrhoea since 9 months old. Occur at the rate of an episode every 3-5 weeks. Mentally "backward" and dull but milestones normal. Palpitations on exertion - 3-4 months, associated with dyspnoea on exertion and tiredness.

Social and dietary history: Fourth of a family of 9; 1 died at the age of 2½ years of "running stomach". One sister had ? "anaemia" 3 years ago. Father a taxi owner - used to earn up to R50.00 per month; now less because car old. Diet: meat 3 times a week; vegetables daily; fish twice a week; eggs 4-5 times a week.

Examination: Looks ill. Marked pallor. Koilonychia. Smooth tongue. C.V.S.: P. 108. B.P. 120/60. Jugular venous pressure elevated to angle of jaw. Ejection systolic murmur Grade II-III and 1st heart sound loud. Abdomen: 2 fingerbreadths non tender hepatomegaly. Spleen tipped. Fundi: pale.

Investigations: Hb 3.4 g. per 100 ml., M.C.H.C. 20%, reticulocyte count 3.2%, W.C.C. 4,900 per c.mm., platelets 300,000 per c.mm.
Bone marrow: no haemosiderin. Erythropoiesis is normoblastic, the red cell precursors being rather small.
Serum iron: 17 µg. per 100 ml., T.I.B.C. 347 µg. per 100 ml.
Urine: normal.
Stool: trichocephalus ova and E.coli.
Chest X-ray: slight cardiac enlargement.
Liver function tests: serum bilirubin 0.4 mgm. per 100 ml., alkaline phosphatase 8 K.A. units, zinc turbidity 3 units, thymol turbidity 2 units, total proteins 7.0 g. per 100 ml., albumin 2.9 g. per 100 ml., globulin 4.1 g. per 100 ml., A/G ratio: 0.7/1.
Protein electrophoresis (in g. per 100 ml.): albumin 2.2; globulins: α_1 0.4, α_2 1.0, β 1.4, gamma 2.0, total protein 7.0.
Mantoux: negative.
Vitamin C: 2.4 mgm. per 100 ml.
Vitamin A absorption test: first specimen 109.2 I.U. per 100 ml., rising to 898.8 I.U. per 100 ml. after 4 hours.
Fat balance: total faecal fat content: 2.8 g., 1.6 g., 3.9 g., 2.0 g., 1.1 g. per 24-hours stool each.
Barium meal: normal.
Free acid present on gastric analysis.
Hb electrophoresis: Hb A/Hb A.
Blood urea: 18 mgm. per 100 ml.

Diagnosis: Idiopathic iron-deficiency anaemia.

Case 43

P.M. Indian male aged 76 years (I.4582/62)
Admitted: 4.6.62

History: Intermittent attacks of epigastric pain, relieved by alkalis, for 10 years. Numbness of little and ring fingers of both hands.

Examination: Not distressed. C.V.S.: P. 86. B.P. 145/70. Abdomen demonstrated some fullness and a succussion splash elicited. Left kidney palpable. Moderate benign hypertrophy of the prostate.

Investigations: Hb 7.8 g. per 100 ml., M.C.H.C. 28%, reticulocyte count 2.4%, W.C.C. 7,800 per c.mm., platelets 435,000 per c.mm.
Bone marrow: no iron. Erythropoiesis is normoblastic.
Serum iron: 22 µg. per 100 ml., T.I.B.C. 436 µg. per 100 ml.
Stool: trichocephalus ova and occult blood positive.
Urine: pus+.
Liver function tests: serum bilirubin 1.2 mgm. per 100 ml., alkaline phosphatase 6 K.A. units, zinc turbidity 8 units, thymol turbidity 2 units, total proteins 8.1 g. per 100 ml., albumin 3.4 g. per 100 ml., A/G ratio: 0.7/1.
Vitamin C: 1.6 mgm. per 100 ml.
Vitamin A absorption test: first specimen 604 I.U. per 100 ml., rising to 823 I.U. per 100 ml. after 4 hours.
Barium meal: duodenal ulcer with pyloric stenosis.
Free acid present on gastric analysis.

Diagnosis: Iron-deficiency anaemia with pyloric stenosis due to a chronic peptic ulcer.

Case 44

B.I. African female aged 34 years (27441/60)
Admitted: 13.9.60 Occupation: Teacher

History: Headache for 1 year. Dysmenorrhoea. Painful right leg for 3 weeks. Dyspnoea on exertion for 3 weeks. Palpitations. Constipation. Menses: C16 4/28. Diarrhoea with blood 1 month ago; 4 Caesarean sections.

Social and dietary history: Family income R5.00 per month; 4 children and self; no alcohol. Diet: meat once a week; fish twice a week; milk daily with porridge only and tea; eggs 1 a day; vegetables daily - potatoes, spinach, cabbage; fruit - oranges and bananas frequently.

Examination: Marked pallor. C.V.S.: P. 82. B.P. 110/70.
Heart: ejection systolic murmur. Abdomen: splenomegaly.

Investigations: Hb 7 g. per 100 ml., M.C.H.C. 27%, reticulocytes 2%, platelet count 250,000 per c.mm.
Bone marrow: contained no haemosiderin.
Serum iron: 30 µg. per 100 ml., T.I.B.C. 407 µg. per 100 ml.
Stool: normal.
Urine: scanty red blood cells.
Liver function tests: serum bilirubin 0.4 mgm. per 100 ml., alkaline phosphatase 5 K.A. units, zinc turbidity 14 units, thymol turbidity 7 units, total proteins 7.5 g. per 100 ml., albumin 3.2 g. per 100 ml., globulin 4.3 g. per 100 ml. A/G ratio: 0.7/1.
Vitamin C: 2.2 mgm. per 100 ml.
Vitamin A absorption test: first specimen 294 I.U. per 100 ml. rising to 890 I.U. per 100 ml. after 4 hours.
Free acid present on gastric analysis.
Mantoux reaction: weakly positive.
Vitamin B12: 325 µµg. per ml.

Diagnosis: Iron-deficiency anaemia probably due to repeated Caesarean sections.

Case 45

G.M. African female aged 53 years (7176/61)
Admitted: 4.3.61 Occupation: Housewife

History: Bronchial asthma for 20 years; exacerbation for 1 day. Swelling of legs and dizziness for 1 week.

Social and dietary history: Financially assisted by sister as has no income of her own. Two live children, 4 dead. Post-menopausal. Diet: meat twice a week; eggs and fish occasionally; vegetables 3 times a week; fruit once a week. (Probably a poor diet).

Examination: Dyspnoeic. Mucous membranes pale. Oedema of legs and sacrum. C.V.S.: P. 80. B.P. 130/90. Gallop rhythm. Chest: marked bronchospasm. Abdomen: 3 fingerbreadths hepatomegaly. Sigmoidoscope passed up to 15 cm. and no lesion seen.

Investigations: Hb 9.4 g. per 100 ml., M.C.H.C. 28%, reticulocyte count 6%, W.C.C. 7,200 per c.mm. with 17% eosinophils.
Serum iron: 19 µg. per 100 ml., T.I.B.C. 445 µg. per 100 ml.
Stool: weakly positive for occult blood.
Urine: normal.
Vitamin A absorption test: first specimen 117.6 I.U. per 100 ml. rising to 352.8 I.U. per 100 ml. after 4 hours.
Fat balance: total faecal fat content: 21.2 g., 5.8 g., 12.8 g., 4.7 g. per 24 hours stool each.
Free acid present on gastric analysis.
Barium meal: normal.
Vitamin B12: 228 µµg. per ml.
Barium enema: normal.

Diagnosis: Iron-deficiency anaemia due to sub-clinical steatorrhoea. Bronchial asthma.

Case 46

R.N. African female aged 30 years (16522/62)
Admitted: 22.5.62

History: Terminal haematuria associated with dysuria, frequency and pain in the left iliac fossa, for 2 months. Swelling of the feet and face for 2 weeks. Dyspnoea on exertion and tiredness for 1 week. Menses: normal. Occasional palpitations. Dizziness for 6 months. No history of blood loss.
1956/8 In King Edward VIII Hospital - "heart trouble" - swelling of feet without dyspnoea.
1959 " " " " " - antepartum haemorrhage for which she had required a blood transfusion.
1961 " " " " " - treated for abdominal pain.

Social and dietary history: 1953 - miscarriage; 1956 - stillbirth; 1959 a live birth. Father died - ? cause; mother well. 4 siblings all of whom are well. Neither drinks nor smokes. Diet: meat 3 times a week (3 lbs. weekly for 2 adults). No milk. Fish once a week. No eggs. Fruit - bananas and apples daily. Vegetables - cabbage, pumpkin, onion, potatoes. No beans, tomatoes, peas. Main diet: rice, mealie meal porridge, potatoes, cabbage and meat.

Examination: Not ill. Slightly built. Pallor and slight oedema of feet. Early koilonychia. Smooth tongue. C.V.S.: P. 80. B.P. 125/85. Short systolic murmur. Abdomen: 2-3 fingerbreadths smooth non tender hepatomegaly; 1 fingerbreadth splenomegaly. Rectal examination: normal.

Investigations: Hb 8.5 g. per 100 ml., M.C.H.C. 27%, reticulocyte count 3.6%, W.C.C. 7,700 per c.mm., platelets 653,000 per c.mm.
Bone marrow: no haemosiderin. Fairly active, normoblastic erythropoiesis. The normoblasts are small.
Serum iron: 17 µg. per 100 ml., T.I.B.C. 506 µg. per 100 ml.
Urine: albumin, pus, red blood cells; ova of S.haematobium; B.coli cultured.
Stool: no abnormality.
Chest X-ray: normal.
Liver function tests: serum bilirubin 0.9 mgm. per 100 ml., alkaline phosphatase 6 K.A. units, zinc turbidity 7 units, thymol turbidity 2 units, total proteins 6.9 g. per 100 ml., albumin 2.5 g. per 100 ml., globulin 4.4 g. per 100 ml., A/G ratio: 0.6/1.
Protein electrophoresis (in g. per 100 ml.): albumin 1.6; globulins: α_1 0.9, α_2 2.0, β 1.8, gamma 2.4, total protein 8.7.
Mantoux: positive.
Vitamin C: 2.5 mgm. per 100 ml.
Vitamin A absorption test: first specimen 268 I.U. per 100 ml. rising to 1797 I.U. per 100 ml. after 4 hours.
Barium meal: normal.
Free acid present on gastric analysis.
Blood urea: 18 mgm. per 100 ml.

Diagnosis: Iron-deficiency anaemia associated with urinary bilharziasis and probable cirrhosis of the liver with bleeding oesophageal varices.

Case 47

M.N. African female aged 62 years (34224/62)
Admitted: 13.11.62 Occupation: Housewife

History: Generally unwell for 1 month, feeling weak, mildly breathless; and watery diarrhoea for 1 week. Post-menopausal for years. No symptoms suggestive of peptic ulceration, no blood loss in stools or melaena.

Social and dietary history: Has 5 children, the youngest child being 15 years old. Diet does not include meat, fish or eggs - consisting largely of samp, beans, mealie rice, tea and bread.

Examination: Wasted. T. 99 - 100°F. Pellagra dermatitis forearms. Mucosae pale. Smooth tongue. P. 100. B.P. 90/60. Ejection systolic murmur at all areas.

Investigations: Hb 4.0 g. per 100 ml., M.C.H.C. 25%, reticulocyte count 7.0%, W.C.C. 7,000 per c.mm.
Bone marrow: no haemosiderin.
Serum iron: 20 µg. per 100 ml., T.I.B.C. 297 µg. per 100 ml.
Urine: normal.
Stool: ova of hookworm; occult blood strongly positive; Shigella flexner 2 cultured.
Chest X-ray: normal.
Liver function tests: serum bilirubin 0.4 mgm. per 100 ml., alkaline phosphatase 8 units, zinc turbidity 8 units, thymol turbidity 2 units, total proteins 6.8 g. per 100 ml., albumin 2.5 g. per 100 ml., globulin 4.3 g. per 100 ml., A/G ratio: 0.6/1.
Protein electrophoresis (in g. per 100 ml.): albumin 1.1; globulins: α_1 0.7, α_2 0.8, β 1.1, gamma 1.4, total protein 5.1.
Mantoux: negative.
Vitamin C: 1.0 mgm. per 100 ml.
Vitamin A absorption test: first specimen 50 I.U. per 100 ml., rising to 302 I.U. per 100 ml. after 4 hours.
Fat balance: total faecal fat content: 17.6 g., 8.3 g., 10.1 g. per 24 hours stool each.
Barium meal: stomach emptied at rapid rate, i.e. evidence of gastro-intestinal hurry, in keeping with avitamosis.
Free acid present on gastric analysis.
Glucose tolerance test (in mgm. per 100 ml.): Fasting blood sugar 100, 152 ($\frac{1}{2}$ hr.) 152 (1 hr.) 182 ($1\frac{1}{2}$ hr.) 126 (2 hr.) 108 ($2\frac{1}{2}$ hr.).
Barium enema: normal.

Diagnosis: Iron-deficiency anaemia associated with pellagra and hookworm infestation together with malabsorption.

Case 48

M.G. African female aged 30 years (28245/62)
Admitted: 18.9.62 Occupation: Housewife

History: Progressive dyspnoea on exertion associated for last 2 months with burning retrosternal pain, for 1 year. Palpitations for 1 year. Swelling of feet for 2 months. Activities limited for last 6 months because of dyspnoea. Anorexia for 2 months. Menses: 2/12 amenorrhoea; recalls heavy menses for 2 months in mid year immediately prior to amenorrhoea. Was treated for dyspnoea on exertion 3 months ago.

Social and dietary history: Married. 5 children, the youngest being 7 years old. Diet: poor. Mainly maize products with meat once or twice a month; occasionally vegetables; no milk, eggs or fish.

Examination: Distressed. Dyspnoeic at rest. Afebrile. Marked pallor. Smooth tongue. Flat nails. Oedema of legs and sacrum. Warm periphery. Jugular venous pressure elevated. C.V.S.: P. 112, collapsing. B.P. 150/50. Apex beat in the 7th intercostal space in mid axillary line and both a right and a left ventricular impulse felt. Murmurs of mitral incompetence and aortic incompetence and possibly aortic stenosis associated with a thrill in the neck. Abdomen: 4 fingerbreadths tender hepatomegaly. Spleen just palpable.

Investigations: Hb 7.4 g. per 100 ml., M.C.H.C. 27%, reticulocyte count 2.5%.
Bone marrow: no haemosiderin. Micronormoblastic erythropoiesis.
Serum iron: 25 µg. per 100 ml., T.I.B.C. 365 µg. per 100 ml.
Urine: normal.
Stool: cysts of E.coli and negative for occult blood.
Chest X-ray: massive generalised cardiomegaly with pulmonary congestion.
Liver function tests: serum bilirubin 0.6 mgm. per 100 ml., alkaline phosphatase 7 K.A. units, zinc turbidity 15 units, thymol turbidity 4 units, total proteins 7.9 g. per 100 ml., albumin 2.5 g. per 100 ml., globulin 5.4 g. per 100 ml., A/G ratio: 0.5/1.
Protein electrophoresis (in g. per 100 ml.): albumin 2.1; globulins: α_1 0.4, α_2 0.9, β 1.7, gamma 2.4, total protein 7.5.
Mantoux: positive.
Vitamin C: 0.9 mgm. per 100 ml.
Vitamin A absorption test: first specimen 218.4 I.U. per 100 ml. rising to 1486.8 I.U. per 100 ml. after 4 hours.
Free acid present on gastric analysis.
Electrocardiogram: left axis deviation with left ventricular hypertrophy.
Blood culture: Staph.saprophyticus.
Anti-streptolysen titre: 125 units.
Vitamin B12: 444 µµg. per ml.

Diagnosis: Iron-deficiency anaemia associated with congestive cardiac failure due to chronic rheumatic heart disease.

Case 49

B.M. African female aged 58 years (19555/62)
Admitted: 23.6.62 Occupation: Housewife

History: Dizziness, malaise, headache, for about 1 year. Dyspnoea on exertion. Palpitations. Post-menopausal for many years. No history of blood loss.

Social and dietary history: Married. Husband died long ago of "chest trouble". No children. Lives with nieces (6 in house). Unemployed. Diet: meat rarely; no milk; fruit rarely. Mainly mealie meal porridge, samp, beans, mealie rice. Does not get sufficient food herself.

Examination: Not ill looking. Pallor of mucous membranes. Tongue: normal. 1 finger-nail flat. C.V.S.: P. 108. B.P. 190/90. Short systolic murmur. Abdomen: 3 fingerbreadths soft non-tender hepatomegaly. No splenomegaly.

Investigations: Hb 4.2 g. per 100 ml., M.C.H.C. 23%, reticulocyte count 6.8%, W.C.C. 12,000 per c.mm. with 21% eosinophils, platelets 660,000 per c.mm.
Bone marrow: no iron seen. Fairly active erythropoiesis, micro-normoblasts.
Serum iron: 9 µg. per 100 ml., T.I.B.C. 325 µg. per 100 ml.
Urine: normal.
Stool: ova of hookworm and trichocephalus.
Chest X-ray: normal.
Liver function tests: serum bilirubin 0.5 mgm. per 100 ml., alkaline phosphatase 6 K.A. units, zinc turbidity 8 units, thymol turbidity 2 units, total proteins 6.2 g. per 100 ml., albumin 1.7 g. per 100 ml., globulin 4.5 g. per 100 ml., A/G ratio: 0.4/1.
Protein electrophoresis (in g. per 100 ml.): albumin 1.6; globulins: α_1 0.5, α_2 0.6, β 1.0, gamma 2.0, total protein 5.7.
Mantoux: positive.
Vitamin A absorption test: first specimen 638.4 I.U. rising to 1260 I.U. per 100 ml. after 4 hours.
Barium meal: normal.
Free acid present on gastric analysis.
Blood urea: 37 mgm. per 100 ml.

Diagnosis: Iron-deficiency anaemia due to hookworm infestation.

Case 50

M.M. African male aged 25 years (18891/62)
Admitted: 16.6.62 Occupation: electrician's assistant.

History: Weakness and tiredness for 2 months. Left upper quadrant pain and backache for 2 weeks. Unproductive cough for 3 days. Dyspnoea on exertion and palpitations.

Social and dietary history: Married and has 2 children. Works as an electrician's assistant earning R7.90 a week. Diet: predominantly maize and its products.

Examination: Looks ill. Mucous membranes pale. C.V.S.: P. 80, ? collapsing. B.P. 150/80. Ejection systolic murmur. Abdomen: 5 fingerbreadths mildly tender splenomegaly. 2 fingerbreadths non-tender hepatomegaly.

Investigations: Hb 6.0 g. per 100 ml., M.C.H.C. 24%, reticulocyte count 6.7%, W.C.C. 3,400 per c.mm., platelets 52,000 per c.mm.
Bone marrow: no haemosiderin seen. A normally cellular marrow with normoblastic erythropoiesis. Myeloid series normal. A few megakaryocytes observed.
Serum iron: 25 µg. per 100 ml., T.I.B.C. 410 µg. per 100 ml.
Urine: normal.
Stool: occult blood positive.
Chest X-ray: heart enlarged.
Liver function tests: serum bilirubin 0.7 mgm. per 100 ml., alkaline phosphatase 10 K.A. units, zinc turbidity 17 units, thymol turbidity 5 units, total proteins 7.4 g. per 100 ml., albumin 3.3 g. per 100 ml., globulin 4.1 g. per 100 ml., A/G ratio: 0.8/1.
Protein electrophoresis (in g. per 100 ml.): albumin 2.0; globulins: α_1 0.5, α_2 0.7, β 1.1, gamma 2.4.
Mantoux: positive.
Vitamin C: nil.
Vitamin A absorption test: first specimen 117.6 I.U. per 100 ml., rising to 478.8 I.U. per 100 ml. after 4 hours.
Barium meal: nil definite; oesophageal varices not entirely excluded.
Free acid present without stimulation on gastric analysis.
Electrocardiogram: normal.
Radio-active vitamin B12 absorption test: normal.

Diagnosis: Iron-deficiency anaemia associated with cirrhosis of the liver and congestive splenomegaly.

Case 51

P.M. African male aged 17 years. (34128/61)
Admitted: 27.10.61 Occupation: nil.

History: Diarrhoea with blood, passing 5-6 stools per day, for 4 months. Haematuria for a long time.

Social and dietary history: Lives with mother. Diet: meat, milk and fish occasionally. Also has peas, potatoes, mealies. Predominantly maize products.

Examination: Not ill looking. Fairly poor nutrition. Mucous membranes pale. C.V.S.: P. 108. B.P. 110/80. Abdomen: 1 fingerbreadth soft non tender hepatomegaly. Sigmoidoscopy: pink mucosa with superficial open ulceration.

Investigations: Hb 9.2 g. per 100 ml., M.C.H.C. 30%, reticulocyte count 2.4%, W.C.C. 10,300 per c.mm., platelets 620,000 per c.mm.
Bone marrow: no iron. Active cellular marrow with normoblastic erythropoiesis.
Serum iron: 10 µg. per 100 ml., T.I.B.C. 370 µg. per 100 ml.
Urine: red blood cells, pus cells, viable forms of S.haematobium.
Urine culture: gram-negative bacillus, non-haemolytic Streptococci, Staph. saprophyticus.
Stool: E.histolytica (trophozoites). Viable S.haematobium, non viable S.mansoni. Blood+++, pus+, mucus+.
Chest X-ray: normal.
Liver function tests: serum bilirubin 0.4 mgm. per 100 ml., alkaline phosphatase 14 K.A. units, zinc turbidity 7 units, thymol turbidity 2 units, total proteins 6.7 g. per 100 ml., albumin 2.9 g. per 100 ml., globulin 3.8 g. per 100 ml., A/G ratio: 0.7/1.
Protein electrophoresis (in g. per 100 ml.): albumin 2.5; globulins: α_1 0.4, α_2 1.0, β 1.1, gamma 2.5, total protein 7.5.
Mantoux: positive.
Vitamin C: 1.06 mgm. per 100 ml.
Vitamin A absorption test: first specimen 739 I.U. per 100 ml., rising to 924 I.U. per 100 ml. after 4 hours.
Fat balance: total faecal fat content: less than 5 g. per 24 hour stool.
Barium meal: duodenal cap a little deformed, suggesting an ulcer near the left fornix.
Free acid present on gastric analysis.
Intravenous pyelogram - Control: calcification of bladder; spina bifida occulta involving L5; kidneys normal.
24-hour urine: negative for acid-fast bacilli.
Blood urea: 36 mgm. per 100 ml.

Diagnosis: Iron-deficiency anaemia associated with amoebic dysentery, urinary infection with bilharziasis and a probable duodenal ulcer.

Case 52

I.M. African male aged 9 years (28760/62)
Admitted: 23.9.62

History: Loose watery stools 4-5 times a day for 1 month. Swelling of the abdomen and feet for 1 week.

Social and dietary history: No family history of tuberculosis. Poor diet.

Examination: Emaciated. Pale. Oedema of legs and sacrum. C.V.S.: B.P. 115/70. Abdomen: gaseous distension of abdomen. Shifting dullness present. 1 fingerbreadth hepatomegaly.

Investigations: Hb 7.3 g. per 100 ml., M.C.H.C. 30%, reticulocyte count 3.5%.
Bone marrow: no iron granules. Erythropoiesis active and normoblastic.
Serum iron: 0 µg. per 100 ml., T.I.B.C. 319 µg. per 100 ml.
Urine: albumin trace, pus+, scanty red blood cells.
Stool: ova of hookworm, ascaris and trichocephalus. No occult blood. Non viable ova of S.haematobium. Culture: negative.
Chest X-ray: left hilum enlarged and perihilar changes present - ?Kochs.
Serum proteins: total 5.2 g. per 100 ml., albumin 1.6 g. per 100 ml., globulin 3.6 g. per 100 ml., A/G ratio: 0.4/1.
Mantoux: positive.
Vitamin A absorption test: first specimen 126 I.U. per 100 ml. rising to 1512 I.U. per 100 ml. after 4 hours.
Barium meal: normal.
Sputa: negative for acid-fast bacilli.
Barium enema: normal except for gaseous distension of large bowel.

Diagnosis: Iron-deficiency anaemia with hookworm infestation.
Malnutrition with nutritional oedema.

Case 53

A.M. African male aged 61 years (27143/62)
Admitted: 7.9.62 Occupation: Farmhand

History: Vomiting after meals, associated with upper abdominal discomfort, for 3 months. Dyspnoea on exertion. No haematemesis but recalls melaena about 2 month's ago.

Social and dietary history: Married. 9 daughters. Worked as a farm-hand in the dairy earning R5.00 per month without having to pay rent on the room occupied. Unemployed for 1 year. For last 3 months lives with married daughter in Durban ? son-in-law's earnings. Diet: breakfast - porridge. Lunch - curry, meat, vegetable. Supper - rice, curry, mealie rice. Meat: 2-3 times a week; fish occasionally only; eggs previously daily now occasionally; milk - now with tea only; vegetables - potatoes, carrot, cabbage - 2-3 times a week. Used to drink Kaffir Beer twice a day (5c.) - no drinking for 3 months. Smokes moderately.

Examination: Not distressed. Pallor marked. Tongue smooth. Apyrexial. C.V.S.: P. 72. B.P. 110/65. Abdomen: dilated hypogastric veins filling from above. Rectal examination: normal.

Investigations: Hb 5.2 g. per 100 ml., M.C.H.C. 26%, reticulocyte count 2.5%, W.C.C. 11,500 per c.mm., platelets 156,000 per c.mm.
Bone marrow: no haemosiderin.
Serum iron 21 µg. per 100 ml., T.I.B.C. 306 µg. per 100 ml.
Urine: normal.
Stool: occult blood strongly positive: cysts of E.coli and E.hartmanni.
Chest X-ray: normal.
Liver function tests: serum bilirubin 0.4 mgm. per 100 ml., alkaline phosphatase 6 K.A. units, zinc turbidity 12 units, thymol turbidity 3 units, total proteins 6.0 g. per 100 ml., albumin 1.8 g. per 100 ml., globulin 4.2 g. per 100 ml., A/G ratio: 0.4/1.
Protein electrophoresis (in g. per 100 ml.): albumin 1.6; globulins: α_1 0.5, α_2 0.7, β 1.2, gamma 1.5, total protein 5.5.
Mantoux: negative.
Vitamin C: 0.3 mgm. per 100 ml.
Barium meal & swallow: normal. Appears to be some rigidity of antral area and ? a filling defect. (Mucosal studies of this area not done).
Electrocardiogram: normal.
Blood sugar: 84 mgm. per 100 ml.

Diagnosis: Iron-deficiency anaemia due to chronic gastro-intestinal blood loss. No final diagnosis was established because of patient's uncooperation.

Case 54

L.N. African male aged 29 years (10589/62)
Admitted: 24.3.62

History: Pain in the upper abdomen for 2 months. Cough, productive of white sputum, for 1 month. Haemoptysis for 2 days. Loss of appetite. No dysentery nor any blood loss.

Dietary history: Diet poor. Alcoholic intake heavy.

Examination: Ill. Pyrexial. Mucous membranes pale. C.V.S.: P. 132. Slight oedema of legs. B.P. 140/60. Chest: ? small left pleural effusion with left lower lobe collapse. Friction rub left base. Right fundus: haemorrhage. On sigmoidoscopy: mucosae pale.

Investigations: Hb 8.7 g. per 100 ml., M.C.H.C. 25%, reticulocyte count 10.4%, W.C.C. 18,100 per c.mm., platelets 660,000 per c.mm.
Bone marrow: trace of iron present. Erythropoiesis normoblastic; increase in plasma cells. Marrow activity normal.
Serum iron: 35 µg. per 100 ml., T.I.B.C. 359 µg. per 100 ml.
Urine: normal.
Stool: ova of trichocephalus; cysts of E.coli; weakly positive for occult blood.
Chest X-ray: consolidation left lower lobe.
Chest screening: changes at left base with elevation of diaphragm on left.
Liver function tests: serum bilirubin 0.52 mgm. per 100 ml., alkaline phosphatase 17.0 K.A. units, zinc turbidity 16 units, thymol turbidity 2.9 units, total protein 6.4 g. per 100 ml., albumin 1.2 g. per 100 ml., globulin 5.2 g. per 100 ml., A/G ratio: 0.2/1.
Protein electrophoresis (in g. per 100 ml.): albumin 1.1; globulins: α_1 0.6, α_2 1.3, β 1.3, gamma 3.3, total protein 7.6.
Mantoux: positive.
Vitamin A absorption test: first specimen 117.6 I.U. per 100 ml., rising to 252.0 I.U. per 100 ml. after 4 hours.
Fat balance: total faecal fat content: 9.2 g., 11.2 g., 2.8 g., 4.8 g. per 24 hours stool each.
Barium meal: normal.
Free acid present on gastric analysis.
Blood culture: negative.
Liver pus: contained trophozoites of E.histolytica.
Blood urea: 22 mgm. per 100 ml.

Diagnosis: Ruptured left lobe amoebic liver abscess.
Iron-deficiency anaemia with mild malabsorption.

APPENDIX II

DETAILED RESULTS

TABLES 1 - 9

Table 1

HAEMATOLOGICAL RESULTS IN 54 PATIENTS WITH IRON-DEFICIENCY ANAEMIA

Case No.	Hb (g./100 ml.)	M.C.H.C. %	Retics. %	Serum iron (µg./100ml.)	T.I.B.C. (µg./100ml.)	Bone marrow iron grading
1	6.2	25	1.9	23	490	
2	9.6	28	1.5	29	596	0
3	5.7	28	1.0	39	544	
4	5.2	26	2.8	8	421	0
5	6.2	27	4.2	14	377	I
6	2.1	25	1.8	0	620	0
7	3.6	27.5	0.5	17	300	I
8	7.4	26	0.8	7	300	0
9	6.8	26	1.5	0	460	0
10	9.0	29	6.0	43	530	0-I
11	5.8	25	2.5	0	355	0
12	2.4	26	6.0	18	362	0
13	7.8	31	2.5	16	560	0
14	3.2	27	6.0	13	370	0
15	5.0	24	8.0	20	385	0
16	6.1	26.5	3.6	10	415	0
17	7.2	26	3.2	11	470	0-I
18	6.0	22	2.8	8	394	0
19	5.5	22	2.0	1	497	0
20	8.1	29	0.8	28	610	0
21	8.1	27	0.2	35	364	0
22	5.3	25	4.2	9	249	0
23	9.1	27	1.6	35	444	0
24	5.8	23	5.8	33	326	0
25	7.8	31		22	507	0
26	8.1	26	2.8	28	361	0
27	8.9	27	3.2	16	405	0
28	9.0	30	1.0	6	346	0
29	2.8	22	0.2	23	311	0
30	7.1	27	5.4	5	525	0
31	3.5	25	0.5	0	258	0
32	3.9	21.5	2.9	11	499	0
33	5.4	31	7.0	16	432	0
34	4.9	24.5	3.0	27	430	0
35	9.0	28	9.0	30	715	0
36	5.9	26	1.9	12	366	
37	4.8	25	5.2	12	414	I
38	6.1	22	2.0	9	460	0
39	4.0	23	2.0	7	545	0

Case No.	Hb (g./100 ml.)	M.C.H.C. %	Retics. %	Serum iron (µg./100ml.)	T.I.B.C. (µg./100ml.)	Bone marrow iron grading
40	5.2	22	1.2	12	499	0
41	4.0	21	5.6	8	446	0-I
42	3.4	20	3.2	17	347	0
43	7.8	28	2.4	22	436	0
44	7.0	27	2.0	30	407	0
45	9.4	28	6.0	19	445	
46	8.5	27	3.6	17	506	0
47	4.0	25	7.0	20	297	0
48	7.4	27	2.5	25	365	0
49	4.2	23	6.8	9	325	0
50	6.0	24	6.7	25	410	0
51	9.2	30	2.4	10	370	0
52	7.3	30	3.5	0	319	0
53	5.2	26	2.5	21	306	0
54	5.7	25	10.4	35	359	0-I

Table 2

LIVER FUNCTION TESTS ON PATIENTS WITH IRON-DEFICIENCY ANAEMIA

Case No.	Serum bilirubin (mgm./100ml.)	Alkaline phosphatase (K.A. units)	Zinc turbidity (units)	Thymol turbidity (units)
1	0.5	8	15	8
2	0.8	5	10	2
3	0.6	4	8	2
4	0.6	5	3	1
5	0.4	10.4	12	3
6	0.75	6	23	5
7	0.4	5.4	11	4
8	0.3	4.5	10	3
9	0.7	8.5	4	2
10	0.8	8	10	2
11	1.1	7	1	1
12	1.1	12	9	3
13	0.7	7	6	3
14	0.5	3	4	2
15	0.4	5	7	3
16	0.5	7	6	3
17	0.3	7	6	2
18	1.01	8	7	3
19	0.6	10	5	3
20	0.7	8	4	2
21	0.9	5	4	1
22	0.85	9	9	3
23	0.8	6	6	2
24	1.6	5	7	3
25	0.65	7	4	4
26	0.5	11	8	2
27	0.3	8	4.8	1.2
28	0.3	8	5	2
29	0.5	10	5	1
30	0.7	5	3	2
31	1.4	5	24	2
32	0.2	3	4	2
33	0.8	10	8	2
34	0.4	5	7	2
35	0.5	11	16	14
36	0.4	11	5	2
37	0.2	7	6	1
38	0.4	6	14	2
39	0.4	5	2	1

Case No.	Serum bilirubin (mgm./100ml.)	Alkaline phosphatase (K.A. units)	Zinc turbidity (units)	Thymol turbidity (units)
40	0.5	22	7	3
41	1.6	37	11	9
42	0.8	8	3	2
43	1.2	6	8	2
44	0.4	5	14	7
45				
46	0.9	6	7	2
47	0.4	8	8	2
48	0.6	7	15	4
49	0.5	6	8	2
50	0.7	10	17	5
51	0.4	14	7	2
52				
53	0.4	6	12	3
54	0.52	17	16	2.9

Table 3

PROTEIN ELECTROPHORESIS ON PATIENTS WITH IRON-DEFICIENCY ANAEMIA

Case No.	Albumin (g./100 ml.)	Globulins (g./100 ml.) α1	α2	β	gamma	Total Proteins (g./100 ml.)
8	1.9	0.7	1.4	1.3	2.4	7.7
12	1.4	0.5	0.7	1.0	2.2	5.8
14	3.3	0.6	0.5	1.0	2.0	7.4
15	1.6	0.7	1.0	1.1	2.4	6.8
16	2.5	0.5	0.7	0.9	2.3	6.9
17	1.2	0.7	1.4	1.5	1.8	6.6
18	2.2	0.6	1.0	1.9	2.2	7.9
19	2.2	0.2	0.5	0.5	0.9	4.3
21	2.4	0.6	0.7	0.5	1.6	5.8
22	1.6	0.8	0.6	1.0	2.2	6.2
23	2.3	0.7	1.0	1.3	1.8	7.1
24	2.0	0.4	0.8	1.1	1.7	6.0
26	2.5	0.7	0.9	1.3	2.0	7.4
27	1.8	0.6	0.9	1.0	1.6	5.9
28	2.2	0.8	1.1	1.2	2.8	8.1
29	2.0	0.3	0.7	0.9	1.1	5.0
30	2.9	0.6	0.8	1.1	1.9	7.3
31	1.3	0.6	0.4	0.7	1.6	4.6
32	2.1	0.5	0.9	1.1	2.1	6.7
34	2.9	0.6	0.7	1.2	1.6	7.0
36	2.1	0.7	1.2	1.4	1.7	7.1
37	2.8	0.3	0.4	0.8	1.0	5.3
38	2.2	0.3	0.9	1.3	1.8	6.5
39	2.6	0.5	1.0	1.8	2.0	7.9
40	1.9	0.7	0.8	1.3	2.2	6.9
41	2.3	0.8	1.0	1.3	1.7	7.1
42	2.2	0.4	1.0	1.4	2.0	7.0
46	1.6	0.9	2.0	1.8	2.4	8.7
47	1.1	0.7	0.8	1.1	1.4	5.1
48	2.1	0.4	0.9	1.7	2.4	7.5
49	1.6	0.5	0.6	1.0	2.0	5.7
50	2.0	0.5	0.7	1.1	2.4	6.7
51	2.5	0.4	1.0	1.1	2.5	7.5
53	1.6	0.5	0.7	1.2	1.5	5.5
54	1.1	0.6	1.3	1.3	3.3	7.6

Table 4

HAEMATOLOGICAL VALUES OF INDIAN WOMEN IN PREGNANCY

FIRST TRIMESTER

Case No.	Age	No. of Pregnancies	Hb (g./100ml.)	M.C.H.C. %	Plasma iron (µg./100 ml.)	T.I.B.C.	% saturation
1	21	-	12.2	33	136	371	36.7
2	28	6	11.7	35	52	338	15.4
3	25		12.9	32	12	366	3.3
4	20		10.6	31	22	368	5.0
5	16	-	10.8	30	21	168	14.2
6	31	2	10.3	28	40	476	8.4
7	29	6	11.5	29	84	349	24.1
8	±32	5	13.2	32	64	442	14.5
9	38	5	13.4	30.5	173	397	43.6
10	15	-	12.0	30	72	308	23.4
11	17	-	12.0	30	28	347	8.1
12	16	-	14.6	32.5	120	363	33.1
13	39	10	11.6	32	53	483	11.0
14	21	1	12.0	32.5	110	339	32.4
15	38	6	13.1	34.5	74	406	18.2
16	19	-	12.4	35	77	332	23.2
17	21	-	15.6	36	25	281	8.9
18	21	-	11.8	32	68	365	18.6
19	32	-	14.3	31	73	365	20.0
20	15	-	12.3	34	54	296	18.2
21	20	2	12.9	32	44	396	11.1
22	29	4	11.1	31	30	349	8.6
23	19	-	14.0	33	92	255	36.1
24	20	-	12.8	31.5	115	473	24.3
25	20	-	13.8	32	160	316	50.6
Total number of cases - 25							
Mean	24	1.9	12.5	32.0	72	358	20.5
Range	15 to 39	0 to 10	10.3 to 15.6	28 to 36	173 to 12	168 to 483	3.3 to 50.6

SECOND TRIMESTER

26	25	6	12.4	33	63	733	8.6
27	27	2	10.4	30	35	554	6.3
28	28	-	12.8	35	45	569	7.9
29	22	-	9.2	29.6	27	577	4.7
30	30	5	7.9	28	22	529	4.2

Case No.	Age	No. of Pregnancies	Hb (g./100ml.)	M.C.H.C. %	Plasma iron (µg./100 ml.)	T.I.B.C.	% saturation
31	38	5	11.0	32	16	506	3.2
32	24	3	9.0	30	50	530	9.4
33	24	3	13.3	35	91	621	14.7
34	20	2	7.3	26	13	443	2.9
35	33	3	11.7	35	83	401	20.7
36	24	4	11.6	31	22	472	4.7
37	18	1	13.1	34	91	434	21.0
38	32	4	7.1	28	26	455	5.7
39	23	3	10.5	31	54	508	10.6
40	22	1	11.8	32	44	500	8.8
41	25	1	11.2	31	59	475	12.4
42	25	2	11.1	30	46	536	8.6
43	28	5	11.0	32	58	498	11.6
44	25	3	11.4	34	42	402	10.4
45	26	3	12.3	34	60	541	11.1
46	26	2	13.0	33	51	496	10.3
47	30	5	11.0	32	52	562	9.3
48	22	2	8.5	29	46	561	8.2
49	22	2	11.0	31	46	473	9.8
50	21	1	10.1	32	42	408	10.3
51	19	-	9.4	30	38	448	8.5
52	30	2	12.5	31	53	388	13.6
53	26	4	13.0	36	83	470	17.7
54	32	7	6.0	24	31	565	5.5
55	26	4	10.1	31	37	557	6.7
56	27	3	11.2	32	96	471	20.3
57	20	1	11.8	32	82	552	14.9
58	18	-	10.2	32	84	484	17.4
59	35	6	11.6	32	55	475	11.6
60	20	-	13.1	32	75	484	15.5
61	27	4	10.1	33	29	488	6.0
62	30	4	11.0	31	39	480	8.1
63	25	1	12.2	33	78	503	15.5
64	19	-	11.2	34	43	357	12.0
65	27	5	10.9	33	51	442	11.5
66	34	2	10.5	33	40	500	8.0
67	29	4	11.0	34	95	423	22.4
68	22	1	10.2	31	42	552	7.6
69	29	3	10.7	33	92	482	19.1
70	26	2	11.3	31	47	549	8.6
71	24	1	7.8	28	34	442	7.7
72	27	3	10.6	30	42	560	7.5
73	26	5	10.5	32	99	549	18.0
74	21	2	10.5	30	77	546	14.1
75	21	-	12.3	31.5	75	521	14.4

Case No.	Age	No. of Pregnancies	Hb (g./100ml.)	M.C.H.C. %	Plasma iron (µg./100 ml.)	T.I.B.C.	% saturation
76	22	2	12.0	34	71	531	13.3
77	23	3	6.9	27	40	610	6.6
78	24	3	11.0	32	42	562	7.5
79	19	-	13.2	34	67	318	21.0
80	25	4	10.9	33	39	529	7.4
81	28	4	10.7	33	54	428	12.6
82	26	3	11.6	33	79	528	15.0
83	30	6	11.9	33	55	544	10.1
84	18	-	12.3	31	86	443	19.4
85	19	1	10.8	32	62	582	10.7
86	31	5	12.2	33	38	498	7.7
87	36	4	11.1	32	34	574	5.9
88	33	5	12.1	33	58	627	9.3
89	31	8	12.2	32	50	551	9.1
90	25	2	8.8	29	2	543	0.4
91	28	7	9.6	31	26	564	4.6
92	19	-	8.7	30	17	569	3.0
93	40	7	10.1	32	91	457	19.9
94	33	-	6.8	26	30	520	5.8
95	30	2	11.0	32	85	565	15.1
96	20	1	11.4	31	33	643	5.1
97	19	-	14.6	32	170	565	30.2
98	31	6	11.6	33	55	545	10.1
99	21	1	12.7	33	54	516	10.5
100	26	2	11.2	31	55	550	10.0
Total number of cases - 75							
Mean	25.8	2.8	10.9	31.6	54.6	511.2	10.9
Range	18 to 40	0 to 8	6.0 to 14.6	24 to 36	2 to 170	318 to 733	0.4 to 30.2
THIRD TRIMESTER							
101	18	-	12.5	32	68	508	13.4
102	22	3	12.5	36	82	532	15.4
103	33	5	8.2	27	49	534	9.2
104	30	7	12.3	33	67	579	11.6
105	25	3	7.7	28	10	505	2.0
106		3	8.1	28	20	565	3.5
107	22	2	12.1	31	54	531	10.2
108		6	10.0	29	15	565	2.7
109	20	2	11.8	31	56	586	9.6

Case No.	Age	No. of Pregnancies	Hb (g./100ml.)	M.C.H.C. %	Plasma iron (µg./100 ml.)	T.I.B.C.	% saturation
110	34	9	11.8	32	102	612	16.7
111	26	5	8.9	29.6	21	531	4.0
112	19	-	12.2	32	36	550	6.6
113	22	-	14.3	32	53	547	9.7
114	24	4	11.0	34	16	540	3.0
115	27	5	11.7	33	19	569	3.3
116	26	6	9.8	35	14	574	2.4
117	33	8	11.9	34	71	651	10.9
118	20	2	9.8	31	33	452	7.3
119	25	1	9.6	32	38	416	9.1
120	22	1	9.2	31	19	318	6.0
121	41	9	10.1	32	43	507	8.5
122	23	1	11.0	30	41	470	8.7
123	30	3	12.7	33	39	434	9.0
124	34	6	10.6	32	39	449	8.7
125	24	1	8.0	30	23	353	6.5
126	26	5	11.2	33	41	356	11.5
127	31	5	11.4	33	46	396	11.6
128	32	1	7.5	28	16	446	3.6
129	28	4	12.2	31	62	506	12.3
130	21	1	10.9	32	28	428	6.5
131	19	-	13.0	32	63	457	13.8
132	27	2	9.8	32	23	451	5.1
133	35	7	11.2	34	40	410	9.8
134	29	2	9.3	29	3	408	0.7
135	25	2	9.1	28	12	413	2.9
136	23	2	9.4	31	62	466	13.3
137	25	3	11.4	33	55	488	11.3
138	26	2	10.5	31	43	472	9.1
139	27	3	12.2	30	67	472	14.2
140	29	5	10.1	32	43	509	8.5
141	23	2	12.3	31	45	545	8.3
142	29	8	10.7	33	64	603	10.6
143	34	8	11.1	33	66	541	12.2
144	28	5	8.2	29	38	492	7.7
145	23	2	11.6	33	61	389	15.7
146	39	7	11.2	32	59	569	10.4
147	24	2	8.7	29.5	25	536	4.7
148	21	2	9.9	29	41	486	8.4
149	28	3	13.4	33	42	534	7.9
150	22	3	12.7	33	65	564	11 5
151	19	1	10.9	31.5	34	532	6.4
152	23	3	12.9	35	79	475	16.6
153	40	8	10.2	30	32	542	5.9
154	23	3	10.3	33	37	567	6.5

Case No.	Age	No. of Pregnancies	Hb (g./100ml.)	M.C.H.C. %	Plasma iron (µg./100 ml.)	T.I.B.C.	% saturation
155	21	-	13.0	32	70	513	13.6
156	30	7	9.9	29	68	562	12.1
157	24	4	9.0	27	9	559	1.6
158	30	5	9.1	29	54	633	8.5
159	33	2	10.8	30	60	630	9.5
160	29	5	9.2	31	13	565	2.3
161	40	8	14.2	33	78	520	15.0
162	35	6	10.4	32	90	650	13.9
163	27	2	11.3	32	59	596	9.9
164	21	-	11.2	34	121	396	30.6
165	30	7	12.8	33	97	440	22.0
166	21	1	9.8	31	37	568	6.5
167	34	4	8.8	29	45	597	7.5
168	30	7	11.3	31	77	553	13.9
169	25	-	8.9	30	46	586	7.9
170	26	5	9.8	32	46	516	8.9
171	21	1	11.6	34	42	537	7.8
172	18	-	12.4	32	40	502	8.0
173	19	-	12.2	32	77	625	12.3
174	37	9	10.1	33	23	543	4.3
175	37	7	11.2	31	32	542	5.9
Total number of cases - 75							
Mean	26.2	3.7	10.8	31.4	46.7	513.9	9.2
Range	18 to 41	0 to 9	7.5 to 14.2	27 to 36	9 to 121	318 to 651	0.7 to 30.6

Table 5

HAEMATOLOGICAL VALUES OF AFRICAN WOMEN IN PREGNANCY

FIRST TRIMESTER

Case No.	Age	No. of Pregnancies	Hb (g./100 ml.)	M.C.H.C. %	Plasma iron (µg./100 ml.)	T.I.B.C.	% saturation
176	27	3	12.2	32	95	415	22.9
177	23	2	12.5	32	168	384	43.8
178	33	9	13.2	33	93	340	27.4
179	40	4	12.6	31	50	255	19.6
180	18	-	13.6	33	104	405	25.7
181	19	-	10.8	31	61	321	19.0
182	28	6	12.2	31	116	419	27.7
183	26	2	12.4	33	89	463	19.2
184	24	-	12.2	31	91	381	23.9
185	20	-	15.5	34	81	324	25.0
186	17	-	13.2	33	81	372	21.8
187	34	1	13.4	31	40	290	13.8
188	25	3	11.2	31	71	311	22.8
189	20	1	11.2	31	20	240	8.4
190	30	-	12.2	31	78	295	26.4
191	30	4	13.9	30	60	371	16.2
192	20	-	12.4	32	56	318	17.6
193	32	3	13.4	33.5	92	318	28.9
194	19	2	14.2	32	106	346	30.6
195	58	9	14.6	34	71	244	29.1
196	38	2	13.9	30	191	322	59.3
197	22	1	12.6	32	54	326	15.6
198	40	1	15.9	35.5	119	278	42.8
199	23	1	13.4	33	123	320	38.4
200	41	3	12.4	32	35	248	14.1
Total number of cases - 25							
Mean	28	2.3	13.0	32.1	85.8	332.2	25.6
Range	17 to 58	0 to 9	10.8 to 15.9	30 to 35.5	20 to 191	240 to 463	8.4 to 59.3

SECOND TRIMESTER

201	24	-	12.0	34	96	501	19.2
202	18	-	11.7	32	99	679	14.6
203	19	-	11.6	33	78	498	15.7
204	16	-	12.5	34	84	479	17.5
205	21	1	13.6	31	73	368	19.8

Case No.	Age	No. of Pregnancies	Hb (g./100ml.)	M.C.H.C. %	Plasma iron (µg./100 ml.)	T.I.B.C.	% saturation
206	26	1	12.0	33	78	448	17.4
207	28	7	12.9	32	93	447	20.8
208	22	1	12.7	33	85	469	18.2
209	19	-	11.1	33	65	459	14.2
210	23	1	12.0	34	52	454	11.5
211	16	-	11.2	35	75	443	16.9
212	33	9	13.9	33	97	289	33.5
213	38	8	10.9	32	56	444	12.6
214	22	-	13.4	33	89	359	24.8
215	29	5	12.2	32	87	476	18.2
216	34	2	12.3	32	56	366	15.3
217	21	1	11.6	33	90	319	28.2
218	17	-	12.1	34	70	510	13.7
219	20	-	12.9	35	106	372	28.5
220	27	6	12.6	33	66	440	15.0
221	35	5	12.2	34	114	339	33.6
222	23	2	10.8	33	163	488	33.4
223	20	-	12.2	32	88	464	19.0
224	18	-	13.4	32	45	451	10.0
225	17	-	13.4	33	124	448	27.7
226	20	1	13.1	33	60	392	15.3
227	20	-	11.2	32	111	410	27.1
228	29	6	10.5	32	51	257	19.9
229	39	8	13.8	34	134	514	26.1
230	23	2	12.7	34	52	267	19.5
231	27	1	11.6	33	64	309	20.7
232	31	5	11.3	32	122	416	29.3
233	24	3	12.6	32	113	538	21.0
234	21	1	12.6	35	131	401	32.7
235	20	2	10.5	31	52	468	11.1
236	19	-	11.0	32	108	394	27.4
237	36	5	10.4	32	99	409	24.2
238	21	4	11.4	30	68	401	16.9
239	34	5	11.8	31	67	465	14.4
240	32	3	12.0	32	63	487	13.0
241	29	1	13.1	32	75	395	19.0
242	20	-	12.7	30	107	362	29.6
243	19	-	13.1	31	98	468	20.9
244	22	-	12.3	30	95	420	22.6
245	21	1	12.0	31	73	434	16.8
246	25	4	13.6	33	112	339	33.1
247	29	4	13.1	33	164	405	40.5
248	23	1	12.6	33	85	410	20.7
249	28	4	11.0	35	126	436	28.9
250	28	3	12.9	31	102	449	22.8

Case No.	Age	No. of Pregnancies	Hb (g./100 ml.)	M.C.H.C. %	Plasma iron (µg./100 ml.)	T.I.B.C.	% saturation
251	27	2	10.6	32	41	521	7.9
252	22	2	11.4	33	113	383	29.6
253	19	-	12.7	34	47	448	10.5
254	21	1	14.1	33	101	477	21.2
255	22	3	13.8	34	104	360	28.8
256	27	3	12.3	34	95	405	23.5
257	21	-	13.4	34	109	376	29.0
258	30	4	13.4	34	109	384	28.4
259	25	5	14.2	35	94	430	21.9
260	28	2	12.0	32	123	533	23.1
261	41	9	11.7	31	45	447	10.1
262	23	2	10.5	32	171	292	58.6
263	32	4	10.1	31	40	605	6.6
264	18	-	12.0	31	71	441	16.1
265	32	4	10.6	30	42	519	8.1
266	28	2	13.9	35	98	450	21.8
267	27	1	11.9	35	53	536	9.9
268	22	-	11.0	33	79	397	19.9
269	23	2	11.4	31	112	382	29.3
270	32	1	10.2	30	92	486	18.9
271	20	-	13.0	33	75	370	20.3
272	36	7	12.6	32	59	439	13.4
273	40	9	10.9	31	78	432	18.1
274	18	-	12.2	31	55	414	13.3
275	22	1	13.4	30.5	83	524	15.8
Total number of cases - 75							
Mean	25	2.4	12.2	32.5	87.3	430.7	21.0
Range	16	0	10.1	30	41	256	6.6
	to	to	to	to	to	to	to
	41	9	14.2	35	164	679	58.6
THIRD TRIMESTER							
276	27	1	12.4	34	179	558	32.1
277	33	5	11.5	33	119	557	21.4
278	29	5	10.2	33	127	517	24.6
279	23	3	10.1	32	63	433	14.5
280	34	8	10.8	33	60	510	11.8
281	26	2	9.9	31	68	578	11.8
282	40	6	11.5	32	167	542	30.8
283	16	-	12.0	31	71	519	13.7
284	18	-	10.4	31.5	78	550	14.2

Case No.	Age	No. of Pregnancies	Hb (g./100ml.)	M.C.H.C. %	Plasma iron (µg./100 ml.)	T.I.B.C.	% saturation
285	17	-	10.2	32	150	569	26.4
286	37	8	12.4	32	82	602	13.6
287	23	-	12.9	31	88	619	14.2
288	42	8	12.0	31	86	496	17.3
289	23	-	12.4	34	124	670	18.5
290	24	3	12.5	34	65	380	17.1
291	30	3	12.6	32	95	475	20.0
292	20	-	14.5	35	59	571	10.3
293	32	6	12.5	34	82	601	13.6
294	25	2	11.7	36	36	566	6.4
295	26	1	12.3	35	50	562	8.9
296	26	1	13.0	33	48	300	16.0
297	23	1	10.9	33	100	409	24.4
298	26	1	11.9	32	75	385	19.5
299	26	-	13.4	32	76	353	21.5
300	18	-	11.7	32	39	449	8.7
301	29	3	12.2	32	122	386	31.6
302	21	1	11.5	32	77	353	21.8
303	27	3	10.0	32	59	284	20.8
304	30	4	13.2	33	70	303	23.1
305	30	3	13.1	33	92	357	25.8
306	23	2	12.9	32	141	457	30.9
307	17	-	14.1	34	72	380	18.9
308	26	1	12.5	34	68	346	19.7
309	20	1	8.2	30	60	462	13.0
310	20	3	12.2	34	75	437	17.2
311	34	4	13.1	34	154	472	32.6
312	26	1	11.2	32	31	376	8.2
313	27	4	10.9	32.5	135	490	27.6
314	21	2	12.3	32	34	416	8.2
315	20	-	13.4	34	82	426	19.2
316	28	1	12.7	34	82	426	19.2
317	25	2	12.3	32	95	415	22.9
318	30	5	12.7	34	97	322	30.1
319	43	10	11.1	35	108	398	27.1
320	25	-	12.7	33	112	359	31.2
321	38	6	10.8	32	47	557	8.4
322	30	4	11.8	32	100	504	19.8
323	30	2	12.0	32	124	529	23.4
324	40	12	14.4	33	156	532	29.3
325	36	5	12.3	33	90	490	18.4
326	30	2	12.0	30	117	418	28.0
327	21	1	11.8	34	134	438	30.6
328	32	3	13.2	36	112	407	27.5
329	21	2	12.3	34	71	557	12.7

Case No.	Age	No. of Pregnancies	Hb (g./100ml.)	M.C.H.C. %	Plasma iron (µg./100 ml.)	T.I.B.C.	% saturation
330	19	-	14.5	35	132	513	25.7
331	34	4	14.7	35	130	479	27.1
332	36	3	15.2	35	100	470	21.3
333	26	4	13.0	33	99	458	21.6
334	28	3	13.8	32	142	545	26.1
335	30	6	12.7	31	160	604	26.5
336	30	4	12.0	32	135	584	23.1
337	16	-	11.4	33	120	426	28.2
338	23	2	14.4	33	104	443	23.5
339	33	3	12.0	32	32	442	7.2
340	24	2	12.2	31	51	532	9.6
341	19	1	12.2	31	101	555	18.2
342	36	7	15.0	32	89	422	21.1
343	28	5	10.3	32	122	391	31.2
344	35	8	12.3	33	80	470	17.0
345	29	7	12.3	31	89	459	19.4
346	32	5	11.0	31	60	558	10.8
347	28	6	13.9	34	71	499	14.2
348	32	5	12.4	33	83	343	24.2
349	34	4	12.0	32	87	472	18.4
350	27	3	11.1	33	35	505	6.9
Total number of cases - 75							
Mean	27.5	3.1	12.2	32.7	92.3	468.9	20.0
Range	16 to 43	0 to 12	8.2 to 15.2	30 to 36	31 to 171	284 to 670	6.4 to 32.6

Table 6

HAEMATOLOGICAL VALUES OF EUROPEAN WOMEN IN PREGNANCY

FIRST TRIMESTER

Case No.	Age	No. of Pregnancies	Hb (g./100ml.)	M.C.H.C. %	Plasma iron (µg./100 ml.)	T.I.B.C.	% saturation
351	19	1	12.0	34	79	398	19.8
352	25	4	11.3	35	99	319	31.0
353	18	-	14.2	36	77	422	18.2
354	35	3	14.0	34	74	304	24.3
355	19	1	12.8	33	29	206	14.1
356	33	3	14.6	32	80	236	33.9
357	27	4	12.4	33	76	347	21.9
358	23	1	14.2	33	90	268	33.6
359	19	-	12.6	33	97	287	33.8
360	25	2	13.6	33	153	359	42.6
361	35	5	14.3	33	164	303	54.1
362	30	4	14.7	35	99	433	22.9
363	21	3	15.4	33	77	393	19.6
364	22	3	13.7	33	96	419	22.9
365	19	2	12.6	32	126	546	23.1
366	17	-	14.0	32	80	289	27.7
367	24	3	12.2	33	146	458	31.9
368	24	1	12.0	33	193	409	47.2
369	24	1	13.5	32	139	497	28.0
370	29	2	14.2	33	79	451	17.5
371	24	2	14.2	33	162	408	39.7
372	31	7	14.4	32	115	320	35.9
373	21	1	12.6	32	99	246	40.2
374	23	1	13.1	31	148	295	50.2
375	29	7	10.9	31	124	308	40.3
376	22	-	12.2	31	125	302	41.4
377	24	2	10.9	29.5	81	467	17.3
378	19	1	13.2	34	110	384	28.6
379	23	-	13.7	33	182	452	40.3
380	29	4	13.3	33	56	554	10.1
381	23	5	13.6	35	127	497	25.5
382	21	-	13.3	35	167	362	46.1
383	30	11	13.6	33	93	319	29.2
384	24	2	12.9	33	106	305	34.8
385	26	2	13.3	34	127	321	39.6
386	22	-	13.9	30	77	335	23.0
387	27	2	13.1	31	117	422	27.7

Case No.	Age	No. of Pregnancies	Hb (g./100ml.)	M.C.H.C. %	Plasma iron (µg./100 ml.)	T.I.B.C.	% saturation
388	21	1	14.4	33.5	143	283	50.5
389	21	6	12.8	33	143	324	44.1
Total number of cases - 39							
Mean	24.3	2.5	13.3	32.9	111.7	365.3	31.6
Range	17 to 35	0 to 11	10.9 to 15.4	29.5 to 36	29 to 193	206 to 554	10.1 to 54.1
			SECOND TRIMESTER				
390	22	5	10.9	32	35	310	11.3
391	28	3	12.9	32	91	346	26.0
392	29	4	12.9	34	109	399	27.3
393	20	-	12.2	34	114	318	35.8
394	24	-	12.1	32	122	282	43.3
395	18	-	13.2	34	122	279	43.7
396	21	-	14.5	32	110	238	46.2
397	24	2	14.0	33	90	398	22.6
398	22	3	13.6	33	115	356	32.3
399	27	2	12.9	32	108	323	33.4
400	22	3	14.3	34	140	328	42.7
401	19	-	13.2	32	112	413	27.1
402	19	-	11.6	34	82	406	20.2
403	20	2	11.5	33	48	565	8.5
404	29	3	13.8	32.5	118	360	32.8
405	23	1	11.6	33	56	462	12.1
406	24	1	11.5	33	93	346	26.9
407	25	7	12.1	32	126	312	40.4
408	29	2	11.8	35	95	360	26.4
409	22	2	10.9	30	45	479	9.4
410	23	2	12.4	31	80	378	21.6
411	26	4	12.6	31.5	190	362	52.5
412	23	-	9.9	29	19	485	3.9
413	21	-	11.9	31	103	333	30.9
414	16	2	11.9	30.5	40	414	9.7
415	21	-	11.8	32	75	371	20.2
416	22	-	12.6	33.5	163	403	40.4
417	27	2	12.4	33.5	103	407	25.3
418	23	2	12.8	34	81	393	20.6
419	32	2	11.9	33	103	473	21.8
420	22	1	12.6	33	128	348	36.8
421	24	1	13.0	33	134	420	31.9

Case No.	Age	No. of Pregnancies	Hb (g./100ml.)	M.C.H.C. %	Plasma iron (µg./100 ml.)	T.I.B.C.	% saturation
422	30	3	11.6	32	144	394	36.5
423	22	-	13.1	31	88	316	27.8
424	35	2	11.9	32	116	404	28.7
425	18	1	11.9	33	104	420	24.8
426	35	9	10.2	30	95	514	18.5
427	25	1	13.2	30	108	408	26.5
428	37	5	10.5	29.6	57	576	9.9
429	24	1	10.9	30	60	550	10.9
430	24	3	11.6	33	178	399	44.6
431	34	4	12.8	32	126	461	27.3
432	30	5	14.3	35	136	350	38.9
433	18	-	10.2	30	29	542	5.4
434	20	1	12.0	32	127	487	26.1
435	37	7	12.2	33	47	537	8.8
436	26	2	13.5	33	117	381	30.7
437	27	3	12.6	34	132	456	28.9
438	21	1	11.9	32	46	561	8.2
439	25	3	11.1	33	154	419	36.6
Total number of cases - 50							
Mean	24.7	2.1	12.3	32.3	100.3	404.8	26.5
Range	16 to 37	0 to 9	9.9 to 14.5	29 to 35	19 to 190	238 to 576	3.9 to 52.5
			THIRD TRIMESTER				
440	19	-	11.8	33	44	414	10.6
441	23	1	11.4	32	34	475	7.2
442	19	-	11.6	33	77	391	19.7
443	28	4	13.1	32	62	461	13.4
444	24	2	13.1	31	110	320	34.4
445	25	2	12.2	33	64	499	12.8
446	29	2	11.8	34	129	476	27.1
447	31	4	12.9	32	104	367	28.3
448	30	1	11.6	32	79	527	15.0
449	28	3	11.4	33	96	370	25.9
450	35	7	9.8	29	33	543	6.1
451	25	7	11.6	31	98	432	22.7
452	19	-	10.6	31	45	417	10.8
453	20	1	11.2	32	42	469	9.0
454	20	2	10.2	32	63	423	14.9

Case No.	Age	No. of Pregnancies	Hb (g./100ml.)	M.C.H.C. %	Plasma iron (µg./100 ml.)	T.I.B.C.	% saturation
455	41	7	12.6	33	51	540	9.4
456	32	2	11.6	32	107	599	17.9
457	21	1	10.3	31	57	447	12.8
458	19	1	13.5	34	68	329	20.7
459	23	3	10.8	33	118	454	26.0
460	27	2	13.7	33	99	455	21.8
461	19	1	13.0	32	76	473	16.1
462	34	8	13.8	32.5	91	443	20.5
463	20	-	12.8	33	113	395	28.6
464	29	3	10.9	33	79	465	17.0
465	28	1	11.5	34	40	440	9.1
466	21	1	12.4	33	63	436	14.4
467	27	7	11.3	31	70	470	14.9
468	21	2	11.6	33	71	457	15.5
469	18	1	11.9	34	69	489	14.1
470	30	2	11.4	34	103	483	21.3
471	26	2	12.6	31	167	487	34.3
472	26	1	13.6	34	91	507	17.9
473	28	3	9.2	29.6	39	544	7.2
474	32	1	9.2	32	54	609	8.9
475	20	1	13.7	35	68	508	13.4
476	29	6	11.2	33	84	422	19.9
477	20	-	12.4	34	96	359	26.7
478	23	3	10.8	32	31	591	5.2
479	40	11	10.2	31	40	449	8.9
480	20	1	11.2	32	77	502	15.3
481	20	-	12.0	32.5	60	575	10.4
482	24	1	13.2	34	156	468	33.4
483	22	1	14.2	34	107	532	20.1
484	22	2	11.6	32	124	332	37.4
485	22	1	12.8	33	90	564	16.0
486	21	1	12.9	32	112	402	27.9
487	30	3	11.0	29	156	432	36.1
488	23	1	13.0	33	149	483	30.8
489	25	1	12.4	34	76	428	17.8
Total number of cases - 50							
Mean	25.2	2.4	11.9	32.5	82.6	436.1	18.6
Range	18 to 41	0 to 11	9.2 to 14.2	29 to 35	31 to 167	320 to 609	5.2 to 37.4

Case No.	Age	Height (ft.	inchs.)	Weight (lbs.)	We (
73	40	5	7	147	1
58	22	5	9	133	1
24	49	6	0	149	1
193	16	5	7	97	1
75	28	5	9	160	1
141	30	5	8	132	1
2	30	5	6	132	1
62	28	5	10	153	1
29	55	5	10	142	1
183	18	5	6	112	1
80	28	5	6	130	1
143	42	5	11	185	1
89	28	5	8	153	1
54	31	5	7	144	1
27	30	5	10	125	
130	28	5	5	145	
81	38	5	9	190	
110	18	5	6	113	
50	23	5	9	135	
13	36	6	0	97	
131	25	5	5	130	
206	27	5	7	124	
133	70	5	6	135	
57	40	5	8	126	
108	30	-		-	
139	50	5	9	185	
161	35	5	6	150	
43	16	5	8	123	
135	33	5	9	143	
194	42	5	5	107	
204	25	6	0	146	
69	38	6	0	165	
85	25	5	9	140	
10	23	5	8	121	
101	26	5	7	125	
84	25	5	9	140	
14	50	5	9	125	
40	45	5	6	110	

		eight (ft.	inchs.)	eight (lbs.)
	042	5	9	130
	27	5	9	116
	26	5	9	128
	30	5	10	150
,3	16	5	11	114
23	19	5	11	123
96	30	5	9	136
188	38	5	8	124
148	25	5	9	145
68	19	5	11	184
185	26	5	5	121
63	45	5	9	125
90	45	5	11	136
16	20	5	8	137
159	26	5	7	145
142	32	5	9	125
138	20	5	9	141
1	30	-		-
153	25	5	4	150
48	48	5	10	90
22	41	5	9	125
70	30	5	3	154
128	43	5	11	118
155	46	5	8	117
91	25	6	3	176
134	32	5	9	102
154	30	5	3	145
165	28	5	9	117
41	45	5	9	119
55	28	5	8	131
67	48	5	6	134
125	29	5	8	127
132	35	5	8	145
112	30	5	6	150
175	39	5	5	95
201	40	6	0	164
26	41	5	11	133
32	40	5	5	135
168	35	5	7	160
77	54	5	3	86
145	50	5	8	165
66	30	5	9	137
99	22	5	6	116
100	30	6	0	142
136	22	5	6	140
150	40	5	6	145

Case No.	Age	Height (ft. inchs.)	Weight (lbs.)
17	40	6 0	145
8	38	5 9	151
200	38	5 8	107
9	30	5 3	117
86	40	5 8	134
88	43	5 7	147
152	50	5 4	145
164	40	5 6	121
182	29	5 9	132
144	40	5 10	170
174	39	5 8	114
83	38	5 9	131
92	34	5 4	106
45	32	6 0	148
65	38	5 11	161
60	28	5 6	112
124	26	5 7	118
158	20	5 5	140
59	36	5 10	136
140	34	-	-
95	47	5 6	112
123	60	5 6	127
39	38	5 9	136
42	35	5 8	117
121	35	5 10	165
56	40	5 10	138
171	35	5 2	160
117	35	5 8	-
18	38	5 9	102
122	30	5 6	145
97	32	5 7	109
172	40	5 9	125
156	46	5 8	123
93	40	6 0	152
167	50	5 10	160
107	40	well built	
186	42	5 9	105
176	80	5 7	137
207	50	5 7	103
151	20	5 2	125
52	46	5 9	140
33	40	5 10	132
4	30	5 9	130
15	61	5 11	161

Case No.	Age	Height (ft.	inchs.)	Weight (lbs.)
137	43	5	8	142
149	30	5	4	114
35	40	5	9	120
195	68	5	8	124
5	30	5	7	129
113	30	-		-
72	50	5	9	99
127	45	5	6	110
61	68	5	9	131
173	35	5	4	100
170	30	5	4½	150
255	16	5	6	109
240	30	5	10	131
222	80	5	7	144
245	13	5	0	80
234	34	6	0	149
253	50	5	6	117
233	42	5	6	121
219	24	5	3	90
217	25	5	11	129
246	40	5	10	127
225	28	5	10	144
250	48	5	6	92
214	25	6	3	135
232	58	5	9	118
258	36	5	3	103
243	32	5	9	118
227	47	5	3	109
251	42	6	3	117
228	35	5	10	132
254	59	5	9	96
244	78	5	6	90
241	25	5	6	136
209	30	5	6	128
236	18	5	0	102
226	30	5	2	142
242	29	5	6	152
215	28	5	6	146
221	40	5	4	151
248	36	5	3	89
213	35	5	1	101
238	50	5	6	104

Case No.	Age	Height (ft.inchs.)	Weight (lbs.)	Weight (g.)	Haem (g./l
37	28	5 5	125	1570	0.0
47	24	5 6	124	1510	0.0
118	30	5 0	90	1277	0.0
169	24	5 1	120	1206	0.0
120	20	5 0	110	1319	0.0
11	40	5 6	203	2370	0.0
157	20	4 10	95	1099	0.01
12	22	5 5	120	1450	0.03
203	60	5 6	136	1550	0.03
187	34	5 8	236	1890	0.02
166	36	5 0	119	1640	0.03
51	29	5 6	152	1860	0.01
162	30	5 2	120	1454	0.07
184	26	5 6	132	1740	0.02
109	30	-	-	1504	0.03
119	56	5 6	121	1760	0.04
36	60	5 9	138	1780	0.01
64	26	5 5	133	1580	0.03
116	37	5 4	170	2327	0.08
19	25-30	5 3	119	-	0.03
28	45	5 7	148	-	0.021
105	40	5 4	190	-	0.044
38	68	5 4	116	1150	0.054
180	35	5 6	185	1780	0.025
87	29	5 0	110	1440	0.051
71	31	5 3	196	1900	0.026
190	23	5 3	110	1450	0.029
106	30	5 0	140	1589	0.034
49	78	5 6	121	1510	0.028
74	18	5 6	118	1670	0.032

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163	26	5 3	135	1149	0.051
202	70	5 0	94	1640	0.040
208	31	6 3	125	1360	0.034
6	61	6 0	120	1650	0.027
20	74	5 8	117	1400	0.028
102	47	5 8	107	1190	0.036
146	45	5 4	140	1362	0.093
94	18	5 11	99	1060	0.041
191	52	6 0	127	1550	0.033
78	68	5 11	129	1230	0.052
104	30	5 3	120	1050	0.045

Case No.	Age	Height (ft.inchs.)	Weight (lbs.)	Liver Weight (g.)	Liver Haem iron (g./100 g.)	Liver Topical (glea)
181	66	5 6	84	1030	0.046	I
103	60	5 3	120	-	0.044	II
46	69	5 8	134	1430	0.021	
25	50	5 8	109	1420	0.032	IV
160	58	-	129	1760	0.047	III
79	65	5 3	90	1075	0.035	
76	21	6 3	132	1670	0.040	
3	28	-	-	-	0.049	
98	50	5 9	103	1890	0.039	
197	50	5 9	117	1650	0.047	I
34	50	5 9	148	1740	0.032	III
44	65	6 0	125	1640	0.047	I
129	30	5 6	107	1240	0.033	I
115	43	5 3	140	1178	0.039	I
7	31	5 5	124	1430	0.061	
126	40	5 6	125	1554	0.044	I
82	45	5 8	129	1650	0.036	I
230	68	5 9	130	1600	0.090	
252	45	5 6	175	2010	0.014	
223	50	5 6	149	880	0.081	
237	18	5 5	85	1320	0.017	
229	40	5 7	129	1460	0.038	II
259	25	6 0	105	1340	0.015	III
210	27	5 6	100	1420	0.027	I
220	39	5 9	173	2340	0.019	
211	76	5 6	119	1050	0.020	
					INDIAN FEMAL	
257	52	5 6	85	955	0.030	
256	63	5 6	112	1030	0.048	
224	28	5 3	112	1640	0.041	
231	20	5 5	103	800	0.048	I
249	60	5 9	125	1960	0.034	
218	51	5 3	81	1060	0.039	
212	56	5 2	156	850	0.019	
235	16	5 0	96	1630	0.022	I
247	54	5 3	123	1400	0.014	II
216	60	5 0	85	1230	0.036	
239	30	5 6	106	1250	0.054	IV
114	40	4 10	125	2227	0.045	I
30	22	5 0	112	1550	0.058	II
192	46	5 2	111	1610	0.058	II

Case No.	Age	Height (ft.	inchs.)	Weight (lbs.)	Weigh (g.)
177	18	5	4	109	1020
189	18	5	3	91	1570
21	25	5	3	100	1300
179	36	5	0	91	1000
178	54	5	3	98	1510
31	68	4	11	100	1120
198	53	5	6	126	1560
2	42	6	0	160	2341
8	65	5	10	185	2199
9	29	5	11	175	2227
13	35	5	11	160	2137
10	55	5	8	145	1666
14	50	5	11	195	2000
18	50	5	8	176	2000
1	50	5	10	190	2961
6	17	5	0	115	1258
3	60	5	10	150	1844
12	65	5	7	150	1731
4	25	5	7	145	1390
7	40	6	0	170	1184
5	40	5	8	140	1242
11	40	5	10	140	1958
17	50	6	0	190	1660

Table 8

HEPATIC IRON CONCENTRATION IN DIFFERENT AGE GROUPS

MALES

Age Group	INDIANS No. of Cases	INDIANS Average Tissue Iron(g./100g.)	AFRICANS No. of Cases	AFRICANS Average Tissue Iron(g./100g.)
20.5 & under	2	0.044	13	0.210
20.5 - 25.5	2	0.076	15	0.160
25.5 - 30.5	5	0.079	38	0.479
30.5 - 35.5	2	0.166	19	0.589
35.5 - 40.5	3	0.231	30	0.533
40.5.- 45.5	4	0.253	17	0.565
45.5 - 50.5	6	0.068	16	0.573
50.5 - 55.5	1	0.029	2	0.180
55.5 - 60.5	2	0.045	3	0.748
60.5 & over	10	0.048	7	1.318
Total No. of Cases	37		160	
Mean		0.100		0.507
Standard Deviation		0.075		0.226

FEMALES

20.5 & under	4	0.024	4	0.620
20.5 - 25.5	2	0.016	5	0.085
25.5 - 30.5.	2	1.306	14	0.140
30.5.- 35.5	-	-	4	0.237
35.5 - 40.5	2	0.016	6	0.122
40.5.- 45.5	-	-	1	0.096
45.5 - 50.5	1	0.015	1	2.976
50.5 - 55.5	5	0.050	-	-
55.5 - 60.5	3	0.158	3	0.052
60.5 & over	2	0.021	2	0.786
Total No. of Cases	21		40	
Mean		0.169		0.284
Standard Deviation		0.372		0.475

Table 9

BONE MARROW IRON CONCENTRATION IN DIFFERENT AGE GROUPS

MALES

Age Group	INDIANS No. of Cases	INDIANS Average Amount of Iron (µg./g.)	AFRICANS No. of Cases	AFRICANS Average Amount of Iron (µg./g.)
20.5 & under	2	63.5	13	171.3
20.5 - 25.5	2	233.0	15	195.3
25.5 - 30.5	5	60.4	35	242.7
30.5 - 35.5	2	37.5	19	403.5
35.5 - 40.5	3	130.0	28	513.6
40.5 - 45.5	4	96.0	16	653.4
45.5 - 50.5	6	73.3	15	536.5
50.5 - 55.5	1	103.0	2	387.5
55.5 - 60.5	2	107.0	2	535.0
60.5 & over	10	70.2	7	912.4
Total No. of Cases	37		152	
Mean		86.6		542.6
Standard Deviation		41.27		234.3

FEMALES

20.5 & under	4	102.5	4	59.5
20.5 - 25.5	2	27.0	5	76.8
25.5 - 30.5	2	82.5	14	174.3
30.5 - 35.5	-	-	4	220.8
35.5 - 40.5	2	56.0	5	119.8
40.5 - 45.5	-	-	1	52.0
45.5 - 50.5	1	122.0	1	72.0
50.5 - 55.5	5	108.2	-	-
55.5 - 60.5	3	104.6	3	62.3
60.5 & over	2	59.0	1	700.0
Total No. of Cases	21		38	
Mean		87.4		146.19
Standard Deviation		27.67		106.12

ACKNOWLEDGMENTS

I wish to express my deep appreciation to Professor E.B. Adams, whose advice and criticisms I found most helpful and encouraging.

I am also indebted to Theresa Eleanore Vieyra Currin, Lecturer in the Department of Sociology, University of Natal, for assistance with the statistical analyses, Miss A. Dorling for her help with the iron estimations, Mrs. M. Walters for the haematological investigations, Miss P. Smith, of the Amoebiasis Research Unit, who undertook the stool examinations, Mrs. J.R. Rudder for the typescript and the staff of the Department of Pathology, King Edward VIII Hospital, Durban, for the routine biochemical investigations.

Thanks are also due to the Obstetrical units of King Edward VIII and Addington Hospitals, the Government Pathologist and his staff and the Medical Superintendent of King Edward VIII Hospital, Durban, for facilities made available.

My grateful thanks to Dr. T.H. Bothwell of the Department of Medicine, University of the Witwatersrand, who taught me the techniques and for the assistance obtained through him from the World Health Organization.

This thesis was undertaken while the author was a medical registrar in the Department of Medicine, University of Natal. As such she attended to and examined all the patients who are included here.

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