BANTU SIDEROSIS IN RHODESIA

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VOLUME I

Department of Pathology,
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February, 1960.

The Dean of the Faculty of Medicine,
The University,
Glasgow, W.2.

Dear Sir,

Re my thesis "BANTU SIDEROSIS IN RHODESIA" submitted for the Degree of Doctor of Medicine.

I hereby declare that the thesis was composed by me and that the work was done by me, except for the following technical procedures:-

1) the cutting and staining of histological sections
2) the haemoglobin values and staining of blood slides (Section VI)
3) twenty five out of the fifty red cell fragility tests (Section VII, Part I)
4) feeding the guinea pigs (Section VII, Part III).

These procedures were carried out by technical staff of the Department of Pathology, Harare Hospital and of the Public Health Laboratory, Salisbury.

Yours faithfully,

Wm. M. Buchanan

BANTU SIDEROSIS IN RHODESIA

SUMMARY

The thesis is divided into eight sections.

SECTION I

Introduction. The term Bantu siderosis is defined and there is a short discussion on what constitutes iron overload.

Previous Investigations. A review of previous work on Bantu siderosis, since it was first described by Strachan in 1929, is presented. Apart from a rather superficial investigation by Gelfand in 1955 in Rhodesia, all of this work has been done in South Africa.

Objects of the Study. a) to investigate in detail the incidence and degree of siderosis in Rhodesian Africans; b) to consider whether or not the high concentrations of iron in the tissues are harmful; c) to confirm that the various types of iron distribution in the body, noted in South Africa, also occur in Rhodesia; d) to investigate the factors which determine the sites of iron deposition in Bantu siderosis; e) to investigate the iron conte of the African diet in Rhodesia; f) to measure serum iron and total iron binding capacity values of a group of Rhodesian Africans.

SECTION II

Incidence & Degree of Siderosis in Rhodesian Africans

In a preliminary investigation, the iron content of liver, pancreas, heart and skin from autopsies on 200 Africans was assessed histologically.

This was followed by a combined histo-pathological and chemical study of iron concentrations in livers and spleens of 661 Africans and 101 Europeans seen at autopsy.

The results show that concentrations of iron in livers and spleens of Rhodesian Africans, and incidence of siderosis among them, is very similar to that found in the Africans of South Africa. The incidence and degree of siderosis in Rhodesian Europeans is similar to that found in non-African subje elsewhere in the world.

SECTION III

Pathological Effects of Iron on the Tissues

The material used in Section II was examined to see if there was any evidence in Rhodesian Africans to support the view that the iron in the tissue of siderotics is harmful, as suggested by some South African workers.

The findings are presented and discussed in conjunction with the results of experimental work on animals by other investigators. It is concluded that if excess iron in the liver is fibrogenic at all, it is so only to a very slig degree.

The incidence of tuberculosis is greater in severe siderotics than in those with mild siderosis or normal iron stores. Unexplained peritonitis is seen at autopsy in some cases with severe siderosis. It is suggested that these phenomena may result from lowered body resistance to infection in subjects with the more extreme degrees of siderosis.

SECTION IV

Distribution of Iron in the Body of Subjects with Bantu Siderosis

Details of iron distribution in the body, and in individual tissues, were obtained by histological examination of a large number of tissues derived from autopsies on 42 Africans with varying degrees of siderosis.

The possible causes of the widespread epithelial deposits of iron, found in some cases of Bantu siderosis with fine cirrhosis, but uncommon in absence of cirrhosis, are discussed. The most probable cause is considered to be the high percentage saturation of transferrin commonly found in these cases. The heavy reticulo-endothelial deposits of iron in Bantu siderosis probably result from the high incidence of infection in Africans.

SECTION V

Iron Content of the African Diet

An analysis was made of the iron content of a number of samples of cooked African food and home-brewed African beer. It is concluded that there is enough iron ingested in food and beer to account for the degree of siderosis found in Rhodesian Africans.

SECTION VI

Serum Iron Studies

The serum iron and total iron binding capacity values were estimated in 341 African out patients. This was done in an attempt to see if the raised S.I. and T.I.B.C. values, reported in some groups of Africans in South Africa, also occurred in Rhodesia. Such high values were not encountered in the group examined.

Post mortem serum iron values in a number of siderotic subjects who died of shock were found to be extremely high. It is suggested that the shock resulted from acute iron poisoning.

SECTION VII

Experimental Work

A number of investigations were carried out in an attempt to confirm or refute some of the theories relative to iron distribution in the bodies of subjects with Bantu siderosis.

Part I: The red cell life span and red cell fragility in healthy male African adults was measured. These values are within normal limits. It is concluded therefore, that the heavy iron deposits in the reticulo-endothelial system in Bantu siderosis are not due to abnormal red cell destruction so probably are due to infection as suggested in Section IV.

Part II: The iron concentration in heads and tails of pancreas in autops material from 15 Africans with cirrhosis, and 15 without cirrhosis, was estimated. No significant difference in concentration is found between the two sites in either group. It is felt that mechanical shunting of blood, caused by the cirrhotic liver, cannot be responsible for the widespread epithelial deposits of iron found in Bantu siderotics with cirrhosis.

Part III: Twenty guinea pigs were fed with African home-brewed beer and an adequate diet for three months. Results at autopsy show that this produce a moderate siderosis with an iron distribution similar to that found in Bantu siderosis.

Part IV: Human serum was treated with ^{59}Fe in such a way that, in one aliquot, the transferrin was approximately 50% saturated, and in a second aliquot, approximately 90% saturated. Various human tissues were incubated in the sera. The results show that the iron uptake by all tissues is greater

in the serum with the higher percentage saturation of transferrin.

Part V: The effect of an oral dose of iron on serum iron and percentage saturation values in Africans was investigated. The results are discussed with reference to the small extra-hepatic epithelial deposits of iron sometime found in Africans with normal percentage saturation of transferrin.

SECTION VIII

General Discussion & Conclusions

It is concluded that:

1) Bantu siderosis in Rhodesia is the same in all major respects as that found in South Africa.

2) As in South Africans, Bantu siderosis in Rhodesian Africans results from the ingestion of large amounts of iron in cooked food and home-brewed bee

3) Bantu siderosis and idiopathic haemochromatosis can be distinguished from one another by the iron distribution in the tissues, even when fine cirrhosis is present in the former.

4) Probably high percentage saturation of transferrin is the most important single factor in producing widespread epithelial deposits of iron in certain cases of Bantu siderosis. An alternative theory is discussed.

5) Probably iron in the tissues is almost inert and therefore does not produce hepatic fibrosis and cirrhosis but, in very severe cases, does lower the resistance of the body to infection.

C O N T E N T S

C O N T E N T S (CONTD.)

SECTION I

INTRODUCTION

Bantu siderosis is a condition found in African subjects in which excessive amounts of storage iron are found in certain body tissues. It appears to be most common in Southern Africa though cases have been reported also from Ghana, (Edington, 1959) and Tanzania, (Haddock, 1965). It is now generally accepted that the source of the iron is the abnormal large amount ingested in the diet.

It is difficult to define dogmatically a level above which iron concentration can be said to be excessive in any tissue. The liver is probably the organ whose iron content has been most extensively studied. It is the most important single organ in the body for storing iron containing as it does, between one quarter and one third of the total storage iron (Bothwell & Finch, 1962) and because of this its iron content is frequently used as an indication of the body's storage iron level. Many investigators consider that the upper limit of normal liver storage iron concentration is 0.1 g. per 100 g. dry weight* (Sheldon 1935, Gross, Sandberg & Holly, 1942; Gillman & Gillman, 1948; Bothwell & Bradlow, 1960; Bothwell & Isaacson, 1962) and it is at about this concentration that iron becomes visible histologically in suitably stained sections (Gillman, Mandelstam & Gillman, 1945; Higginson, Gerritsen & Walker, 1953; Bothwell & Bradlow, 1960).

In view of this therefore, in a large number of studies the authors have used the incidence of stainable iron in livers, or liver storage

* 0.1 g. per 100 g. dry weight approximately equals 0.25 mg./g. wet wei

iron concentration in excess of 0.10 g./100 g. dry weight, as a reflection of the incidence of siderosis in the population under consideration. In order to facilitate comparison between the present investigation and previous work on this subject it was decided to continue this practice. It is felt however that small amounts of stainable iron in the liver are completely physiological because, as will be shown, examination of material obtained at autopsy from European adults in Salisbury showed that 40% had stainable iron in their livers. None of these people were suffering from haemochromatosis, were chronic alcoholics, nor was there any history of blood transfusions or iron medication either oral or parenteral. Furthermore, as can be seen from Table IXstainable iron is a common finding in non-African subjects in various parts of the world. Indeed, in some places the incidence of stainable iron in the liver is greater than that found in a number of South African Bantu studies. In such non-African subjects however the amount of stainable iron is in most cases small, whereas in Bantu siderosis the amount is usually considerable and in many cases enormous

PREVIOUS INVESTIGATIONS INTO BANTU SIDEROSIS

Attention was first drawn to the high incidence of siderosis in South African natives by Strachan in 1929. In 1,100 autopsies performed on Africans in Johannesburg between 1924 and 1928 he found 33 cases of iron pigmentation of a degree comparable with that of idiopathic haemochromatosis. 21 of these cases were males and 12 females. In 745 of the autopsies iron stains were applied systematically and it was found that, in addition to the 33 cases of "haemochromatosis", 49.4% had haemosiderin in the liver, spleen and lymph glands. In subjects over 30 years of age obvious haemosiderin occurred in 81%. Males and females appeared to be equally liable to the condition. In the 745 autopsies there was cirrhosis of the liver in 10.3%. A cellular infiltrate and some degree of portal fibrosis was found in most cases. Primary carcinoma of liver was present in 3%.

In an attempt to find an explanation for the high incidence of siderosis in the African he examined their diet. He noted the essentially carbohydrate nature of this and also their heavy consumption of kaffir beer. He pointed out that due to absence of control of time, temperature and materials in its preparation this beer may contain considerable quantities of acetic acid. Also, as it is largely prepared in paraffin tins, he felt that it would be contaminated with metals dissolved from the vessel, particularly stressing the importance of zinc and tin. He found the concentration of these metals to be high in many African foods and believed that they had a toxic

effect on the erythrocytes. The metals, he felt, combined with haemoglobin to produce haemofuscin which was deposited in the tissues. He shared the belief of von Recklinghausen and Mallory that haemosiderin resulted from the unmasking of iron in the haemofuscin. The iron content of the African diet was not examined, presumably because he believed that he had already accounted for the source of the large amounts of haemosiderin.

He found that there was a slight but definite increase in the fragility of red cells in Africans as compared with Europeans especially in subjects over 30 years of age. This, he thought, might play a small contributory part in the production of the haemosiderosis. Rabbits were fed with relatively small doses of salts of zinc, tin and copper in an attempt to produce haemochromatosis. Resultant changes in the liver included fibrosis and cellular infiltration of the portal areas. Accumulations of haemofuscin were found in livers and spleens but no haemosiderin was discovered in any of the animals. It was suggested that prolonged administration of the metalic salts might lead to cirrhosis.

He concluded that haemochromatosis was not uncommon in the South African native and that all stages occurred from pigmentation alone to a complete picture of bronzed diabetes. He believed that the chief factor in its production was the excess of carbohydrate in the diet contaminated with salts of zinc and tin. He also considered that heavy deposits of haemosiderin produced cirrhosis of the liver.

Gillman, Mandlestam and Gillman (1945) examined the livers of 500 Africans dying as a result of accidents in Johannesburg. They stained specimens of each liver for iron and estimated chemically the concentrations of iron and copper in the livers.

There was a close correlation between the amount of iron seen histologically and that estimated by chemical procedures. When the iron concentration in the liver exceeded 0.1 g/100 g dry weight it was always visible in suitably stained sections. On the other hand, at times iron was seen histologically when the liver iron concentration was less than 0.1 g/100 g dry weight. The iron concentrations found in their adult livers ranged from 0.01 to 5.44 g/100 g dry weight. In five infants with normal livers the concentration varied from 0.02 to 0.05 g/100 g dry weight. Comparing these results with the findings of Ramage, Sheldon and Sheldon (1933) in European infants they concluded that "in the first six months of life, the livers of African babies can contain up to 20 times less iron than babies in Europe dying from similar types of disease".

These authors considered that the large amounts of iron pigment seen in the liver and other organs were not derived from haemoglobin as postulated by Strachan, but resulted from altered intracellular metabolism caused by chronic malnutrition. Because of this they suggested that the iron pigment instead of being called haemosiderin should be called cytosiderin and the condition cytosiderosis. They stated that "the diet of the African is not particularly rich in iron" but quoted no reference in support of this.

It was found that though in many cases with cirrhosis there was a very high liver iron concentration there were also livers without any evidence of cirrhosis which had iron levels corresponding to those described in haemochromatosis. The authors felt therefore that iron pigment did not play a direct part in the production of cirrhosis.

They envisaged "cytosiderosis" as evolving in three stages: 1) the appearance of pigment in liver cells without any increase in the amount of iron; 2) a progressive increase in the pigment in the liver cells with a corresponding quantitative increase in the amount of iron; 3) further accumulation of pigment in the liver cells associated with its appearance in Kupffer cells and portal tract histiocytes, at which stage the iron content of the tissue estimated chemically was very high.

Gillman and Gillman (1945) assessed 400 liver biopsies from 120 patients suffering from pellagra. Pigmentary cirrhosis was found in 15% of these adult pellagrins chiefly in patients under 40 years of age. They were indistinguishable clinically and pathologically from haemochromatosis. Iron pigment and cirrhosis were encountered as frequently in females as males. "Pigment" first appeared in the liver cells nearest the central vein. In more severe cases it spread to involve the whole lobule with increasing intensity until the most heavily pigmented cells were seen in the periportal region. If by pigment they meant haemosiderin this finding is at variance with all later work which shows initial haemosiderin deposits occurring in the periportal liver cells. It is just possible however that they meant

haemofuscin which is frequently seen in the centrilobular liver cells even when haemosiderin is absent.

As a result of this investigation they concluded that: 1) "haemochromatosis can be regarded as one of the commonest sequelae of pellagra", 2) the iron pigment in the liver originated within the liver cells resulting from an upset of intracellular metabolism caused by dietary imbalance. Both haemosiderin and haemofuscin have a common origin from mitochondria, 3) haemosiderin is not the main factor responsible for the production of cirrhosis nor does it cause primary carcinoma of liver.

In another study Gillman and Gillman (1948), examined the livers of 261 Africans and 90 Europeans over 10 years of age who died as a result of trauma in Johannesburg. 40.0% Europeans and 81.6% Africans had livers which contained stainable iron. Chemical estimations were carried out in almost 100 livers. Normal livers were found to have iron concentrations of less than 0.1 g/100 g dry weight.

They concluded from this investigation that though haemosiderin and cirrhosis were more common and of a greater degree in the livers of pellagrins than in the general population, haemosiderin could occur in absence of overt manifestations of pellagra. One adverse effect that the large quantities of iron had on the body, they believed, was the blocking of the reticulo-endothelial system by large inert iron-containing molecules which might explain the rapid progress and acute character of tuberculosis in the African.

Strachan had previously noted the prevalence of tuberculosis in siderotic subjects and did not think that it played an important part in the production of siderosis. He did not however discuss the effect of the iron on the tuberculous process.

Kinney, Hegsted and Finch (1949), experimenting on rats, found that when excess iron was fed to those animals considerably more was absorbed when their diet was deficient than when it was adequate and only in the former were deposits of iron found in the liver.

Following this, in 1950, Walker and Arvidsson investigated the diet of Africans living in Johannesburg. They found that, so far from the African diet being "not particularly rich in iron" as stated by Gillman et al., (1945), the reverse was true. They discovered that "a typical diet cooked in the traditional manner may contain 100-150 mg of iron per diem". The iron content of a 2000 calorie diet before cooking contained only between 15 and 30 mg of iron so it became apparent that the excess iron found in the cooked food was derived from the iron cooking pots so frequently used by the local Africans. Analysis of stools of Africans from various institutions showed an excretion of between 65 and 145 mg of iron a day confirming the high intake of this metal.

They suggested that the habitual ingestion of large amounts of iron associated with the type of malnutrition affecting the African might be an aetiological factor in the "haemochromatosis" so commonly found among these people. It was explained later (Walker & Arvidsson, 1953) that they had used the term haemochromatosis in

error and had meant haemosiderosis.

Walker and Arvidsson (1953) confirmed the high iron content of the African diet in a further investigation. In this they noted especially the low pH of kaffir beer (3.0-3.5) which enhanced dissolving of the iron from the containers in which it was prepared. They reviewed published experimental evidence showing that at a high level of iron intake the "mucosal block" (Granick, 1946) regulating the absorption of this metal is overcome allowing the absorption of excessive iron which is deposited in various tissues of the body.

They concluded that the excess of tissue iron found in Bantu siderosis could be accounted for by the habitually high oral iron intake alone and that there was no need to postulate a lesion of the digestive tract, malnutrition or other causes to account for it. They could find no evidence that "iron overload per se was detrimental to well-being".

The serum iron (S.I.) and total iron binding capacities (T.I.B.C.) of various African groups were estimated by Gerritsen and Walker (1953 a,b). They found the mean S.I. values for male Africans from the Johannesburg area, Mozambique, Angola and Nyasaland were appreciably higher than found in European controls. They found also that in these subjects the T.I.B.C. average values were raised so that the percentage saturation was not particularly high in most cases. The average values of S.I. and T.I.B.C. were not significantly raised in female Africans from the Johannesburg area or male Africans from North Transvaal, Pondoland, Swaziland,

Tanganyika or Basutoland.

Higginson et al., (1955) investigated 44 necropsies on siderotic subjects of varying severity. A number of organs were examined histologically, especially for iron content, and in 21 cases iron concentrations in selected organs were determined chemically. Liver specimens from autopsies on a further 72 Africans living in other territories in Southern Africa were also examined. 252 additional necropsies were reviewed to establish the incidence of siderosis and its relationship to hepatic fibrosis. They also examined 110 liver biopsies, many of which were from undernourished subjects.

It was found that under 20 years of age siderosis was very uncommon and never severe. After this its incidence and severity increased with age to the fifth decade and from then onwards both remained relatively constant. Unlike previous investigators who had found the sexes equally affected they found females were less severely affected than males. No relationship was found between siderosis and malnutrition. Haemosiderin deposits were present in the livers of Africans from the Rhodesias, Nyasaland, Bechuanaland, Swaziland and Mozambique suggesting for the first time that siderosis was not confined to the Africans of South Africa.

The highest concentration of iron found in a liver in this series was 5.52 g/100 g dry weight. It was noted that though in many cases severe siderosis was accompanied by portal fibrosis and cirrhosis, this was not always so and some cases with heavy iron deposits in the liver showed little or no fibrosis. Because of

this, in common with Gillman et al., (1945), they believed that "fibrosis cannot be regarded as dependent upon haemosiderin deposition"

They considered that iron was first deposited in the reticulo-endothelial cells and shortly after in the parenchymal cells of the liver, believing that the pattern of iron distribution evolved in the same manner as in mice injected with saccharated iron oxide (Cappell, 1930).

Greater concentrations of iron were found in the spleen than the liver in almost all cases. Only 7 out of 82 pancreases examined microscopically had iron pigment in islet and acinar cells and in all of these except one the deposits were scanty. It is not stated whether those patients with pancreatic deposits were suffering from cirrhosis of liver. Few hearts contained stainable iron and, in those that did, the deposits were scanty. In the kidneys of both moderate and severe cases of siderosis scanty haemosiderin granules were commonly seen in the distal convoluted tubules and loops of Henle.

They contrasted their findings of iron distribution and concentrations with those given by Sheldon (1935) for European subjects with idiopathic haemochromatosis and found the following differences: 1) In Bantu siderosis the concentration of iron in the spleen was usually higher than in the liver and much higher than the concentrations quoted by Sheldon for classical haemochromatosis, with two exceptions. 2) In their cases haemosiderin was present in the pancreas in only severe cases and pancreatic fibrosis was rare, both

findings being in marked contrast to haemochromatosis. 3) There was an absence of significant haemosiderin deposits in the epithelial cells of the stomach, thyroid, salivary and suprarenal glands and in the heart muscle fibres, unlike the heavy deposits found in haemochromatosis. 4) Haemosiderin deposits in duodenal and jejunal villi are scanty in haemochromatosis but extremely heavy in most cases of Bantu siderosis. 5) In the bone marrow heavy deposits of iron are usual in Bantu siderosis while in haemochromatosis deposits are usually slight.

Because of the different patterns of iron deposition between idiopathic haemochromatosis and Bantu siderosis, they felt that these conditions did not have a common aetiology. Also in considering the aetiology of Bantu siderosis they decided that "while undernutrition and infection may accentuate pre-existing siderosis in the Bantu", these factors are not of major importance in its production. They rejected the possibility of metallic poisoning or parasites as aetiological factors but considered that the excessive amounts of iron found in the African diet by Walker and Arvidsson (1953) might be important and that this aspect deserved further study.

Bothwell, van Doorn Wittkampf, Du Preez and Alper (1953) measured the absorption of orally administered radioactive iron in a small number of Bantu siderotics. No increase in absorption was found.

The first investigation into siderosis in Rhodesia was carried out by Gelfand in 1955. Tissues were examined from 105 unselected autopsies on Africans dying in Salisbury native hospital.

Macroscopic methods only were used to demonstrate the presence of haemosiderin and no chemical estimations were made. Among the 75 adults, 57.3% had stainable iron in the liver, 52% in the spleen, 16% in lung. Stainable iron was found in pancreas, suprarenal, heart and small intestine in a small number of cases. In the 30 autopsies on children under 5 years stainable iron was found in the liver in 56.7%, and in the spleen in 30%. Positive results were found in 56.6% of the 23 infants under 18 months. It is not stated whether these last results refer to liver, spleen or to both.

Walker and Higginson (1956) cast doubt on the validity of Gelfand's results in infants, pointing out the great rarity of siderosis in very young African subjects on the Rand. Furthermore, using identical methods to Gelfand they were able to demonstrate positive results on livers of 2 African infants in which chemical and histopathological investigations showed iron concentrations which were well within the normal range.

Gillman, Muthorn and Lamont (1957) in an analysis of 170 liver biopsies from adult male Africans in Durban showed that siderosis occurred in over 75% and cirrhosis in 36%. (Though it is not stated, presumably these biopsies were performed on subjects suspected of having liver disease, so these incidences do not relate to the general population). Among the cases of frank cirrhosis, 66% were portal cirrhosis, 23% postnecrotic scarring and 11% a combination of these lesions. These findings were similar to those of Higginson et al., (1953) who found that among cirrhotic livers 72% were of the

fine portal type and 26% of the coarse multilobular type.

In further work published in 1957, Gillman, Lamont, Hathorn and Canham found that in 100 liver biopsies on African subjects suspected of liver disease in Durban, 88 had siderosis. They also found a correlation between the degree of portal fibrosis and portal siderosis. The incidence of portal cirrhosis was higher in subjects with advanced hepatic siderosis. They deduced therefore, that the accumulation of iron in advanced siderosis either directly or indirectly produces liver disease.

They disagreed with Higginson et al., (1953) that it is possible to distinguish Bantu siderosis from idiopathic haemochromatosis histologically on the grounds of difference in relative distribution of iron in the reticulo-endothelial cells and parenchymal cells. They pointed out that many of Higginson's cases died of acute or chronic infections or debilitating diseases. They believed that infection can alter the relative distribution of iron between hepatic parenchymal and reticulo-endothelial cells in nutritional siderosis. They found, as had Gerritsen and Walker (1953 b), that in uncomplicated siderosis both plasma iron and unsaturated iron binding capacity are raised, therefore the percentage saturation of transferrin seldom exceeded 50%. These changes in plasma iron pattern they believed, were diagnostic of nutritional siderosis uncomplicated by infection or hepatic failure and served to differentiate it from idiopathic haemochromatosis.

In Durban also, Wainwright (1957) examined tissues from 400 consecutive African autopsies, excluding infants. Stainable iron was found in the livers of 65% (78% in adult males and 41% in adult females). Siderosis was rare under the age of 21 years. Its incidence and degree increased with age and developed later in females. This last fact was attributed to the loss of iron during the reproductiv period. It was suggested that because there is no fall in the incidence of siderosis in the later age groups siderosis alone does not affect length of life.

In common with Gillman et al., (1945) it was found that in most cases the iron pigment was first deposited in the liver cells and subsequently in the Kupffer cells. The earliest deposits were found in the periportal cells and in more advanced cases spread to involve the centrilobular cells. This author believed that siderosis and liver fibrosis were unrelated.

He noted that whenever haemosiderin was found in the liver it was also present in the spleen and in some cases the deposits in spleen were heavier than those in the liver. The distribution of iron pigment in the other organs examined was the same as that found by Higginson et al.(1953). One new fact however to which he drew attention was, that in those cases in which the pancreas contained heavy haemosiderin deposits, there was also cirrhosis of the liver.

The non-haemin iron content of 18 livers was measured and the results showed a range of from 0.062 to 3.50 g/100 g dry weight. It was noted that in seven livers the iron concentration was within the

range given by Sheldon (1935) for cases of haemochromatosis but the total liver iron in four typical cases of severe Bantu siderosis ranged between 5.5 and 8.0 grammes which is very much lower than usually found in haemochromatosis. The difference in total liver iron was ascribed to the fact that in haemochromatosis the liver is usually enlarged while in Bantu siderosis it is not.

He also estimated the non-haemin iron concentration of the livers of 30 African infants and found a range of from 0.018 to 0.666 g/100 g dry weight (average 0.150 g/100 g dry weight) and compared these with the iron content of 10 livers from European and Coloured infants in which the range was from 0.018 to 0.335 g/100g dry weight (average 0.102 g/100 g dry weight). The higher average iron content in African infants he attributed to the increased iron stores in many of the mothers. The iron concentrations found in African infants' livers were in marked contrast to the low values found by Gillman et al., (1945).

The iron concentration of the bile increased with the degree of siderosis and, according to his calculations, the average daily excretion of iron in the bile in patients with advanced siderosis would be between 4.3 and 8.6 mg.

In investigating the serum iron and total iron binding capacity of African patients and with hospital staff of all races as controls, it was found that in most cases both S.I. and T.I.B.C. values were slightly lower than normal. None of his cases showed the high T.I.B.C. values encountered by Gerritsen and Walker (1953) in their

series. S.I. and T.I.B.C. values in patients who had had liver biopsies showed that, in subjects with cirrhosis and siderosis, the percentage saturation of transferrin may be raised due to lowering of the T.I.B.C. and, in rare cases, these values may be comparable with those found in haemochromatosis.

In considering the pathogenesis of siderosis in the African he believed that it was caused by a diet rich in iron, whose absorption was enhanced by the predominantly carbohydrate nature of the diet, and possibly related to its phosphate content. It was thought that the iron found in the spleen and other parts of the reticulo-endothelial system was derived from normal haemoglobin breakdown. This iron would, in non-siderotics, be released for fresh haemoglobin synthesis but as a result of the large amounts of newly absorbed iron available for haemoglobin synthesis in siderotics, the iron in the reticulo-endothelial cells would not be utilised and thus accumulate.

This theory does not explain the difference in the degree of haemosiderin deposition between Bantu siderosis and idiopathic haemochromatosis where, in the latter, no doubt similar amounts of newly absorbed iron would be available for haemoglobin synthesis yet the accumulation of iron in the reticulo-endothelial system is rarely of the same degree as in Bantu siderosis.

In 1958, Higginson, who had previously believed that iron deposition had little effect on the liver, altered his opinion to the belief that the iron was probably the cause of fine septal fibrosis.

Bothwell and Bradlow (1960) carried out a combined histopathological and chemical study of the livers from 147 unselected autopsies on African subjects dying as a result of trauma in Johannesburg. There were 131 males and 16 females and their ages ranged from 11 to 70 years. In only 16 of their cases (10.9%) was the concentration of iron in the liver less than 0.1 g/100 g dry weight which they considered was the upper limit of normal. They confirmed the findings of Gillman et al., (1945), and Higginson et al., (1953), that usually when the concentration of iron in the liver exceeded 0.1 g/100 g dry weight it could be demonstrated histologically, and conversely when the concentration was less than this, no iron could be seen histologically.

They found that haemosiderin deposits first appeared in the parenchymal cells of the liver with but a few exceptions. In this respect they agreed with Gillman and Gillman (1945), and Wainwright (1957), but differ from Higginson et al., (1953) who found deposits first in the Kupffer cells. There was good correlation between the amount of iron seen histologically and that estimated chemically in both liver and spleen. In the spleen, iron could not be demonstrated histologically at concentrations of less than 0.15 g/100g dry weight; i.e. at an appreciably higher concentration than in the liver. A possible explanation is that these authors estimated the total iron concentration which includes haemoglobin iron. Though in most cases this last is of little importance in the liver, it can account for an appreciable amount of iron in the more vascular

spleen. Iron concentrations in spleen were fairly similar to those found in the liver but in most cases were slightly higher. A striking correlation between portal fibrosis and iron content of the liver was noted.

They considered that the iron in the liver parenchymal cells was readily explained by the high iron content of the diet which would produce a raised serum iron. They quote Gerritsen and Walker's (1953 b) figures as proof that this is common in the African. Gerritsen and Walker however found that the T.I.B.C. was also raised and the percentage saturation was approximately normal unlike the experiment of Jandl, Inman, Simmons and Allen (1959), which they also quote, where the percentage saturation was raised to produce increased uptake by liver slices. It will be shown however in the present thesis that a heavy dose of oral iron not only raises the serum iron temporarily but also raises the percentage saturation above 60 so that the above explanation is probably in fact correct.

The high splenic iron concentrations, they felt, were more difficult to explain as the spleen takes up little if any iron directly from the plasma (Elmlinger, Huff, Tobias and Lawrence 1953), but thought that impaired release of the iron derived from haemoglobin from these sites might occur due to the raised plasma iron levels.

The relationship between siderosis and diabetes in the African was investigated by Seftel, Isaacson and Bothwell (1960). Histological and chemical estimations were made of the iron content of various organs obtained from autopsies on 20 diabetic African subjects.

Four of their cases were very similar to idiopathic haemochromatosis as far as histopathological appearances were concerned, but the iron concentrations in spleen were in all cases higher than the range of values given by Sheldon (1935) for idiopathic haemochromatosis. They concluded that one of the causes of diabetes in Africans was massive iron overload.

In a further paper Seftel, Keeley, Isaacson and Bothwell (1961) discussed serum iron levels found in a random group of 100 adult African diabetic subjects. In 14 cases the serum iron was raised (mean 216, range 165-312µg/100 ml.). Liver and gastric biopsies were performed in 11 of these and 7 were found to have siderosis with portal cirrhosis. Four of the seven had epithelial deposits of iron in the gastric mucosa. They regarded all 7 cases as having fully developed haemochromatosis and when two died later, autopsy findings confirmed this view.

They considered that despite many similarities between African diabetics with haemochromatosis and idiopathic haemochromatosis nevertheless there were important differences. 1) The progress of the cirrhosis in Africans was rapid and was the principal cause of death unlike in idiopathic haemochromatosis where the progress of the cirrhosis was slow. It was suggested that toxic substances present in their alcoholic beverages might be responsible for the rapid advancement of the cirrhosis in Africans. 2) There was a relatively high incidence of porphyria in the African cases which was not seen in idiopathic haemochromatosis. 3) Though cardiac

complications were common in idiopathic haemochromatosis these did not occur in the African form.

In Cape Town the incidence of siderosis in all racial groups was investigated by Uys, van der Walt, Potgieter and Golby (1960) using material from 1200 consecutive autopsies. Stainable iron was found in the livers of 55.9% of 134 Africans, 29.1% of 518 Coloureds (Eurafricans) and of 30.1% of 548 Europeans.

The effect of cirrhosis on iron storage was examined by Bradlow, Dunn and Higginson (1961). They compared the distribution of iron in the body in 19 Africans with siderosis and cirrhosis with the distribution found in 15 Africans with siderosis and either normal portal areas or portal areas showing moderate to marked periportal fibrosis whose lobular architecture was normal.

In the group with cirrhosis the distribution of iron was widespread in the parenchymal cells of many organs and resembled that found in idiopathic haemochromatosis. On the other hand, in the cases without cirrhosis, deposits in parenchymal cells were scanty or absent.

They concluded that in Bantu siderosis deposition of iron in extrahepatic parenchymal cells depended partly on the presence of cirrhosis and partly on the degree of iron overload. They quote Schwartz (1956) who suggested that extrahepatic parenchymal deposits of iron in idiopathic haemochromatosis might be due to the high percentage saturation of transferrin and noted that Wainwright (1957) had found a frequent occurrence of high transferrin saturation in

subjects with cirrhosis while in subjects without cirrhosis the great majority had a transferrin saturation of less than 50%.

Isaacson, Seftel, Keeley and Bothwell (1961) found that out of 700 Bantu autopsies 38 (5.4%) had cirrhosis. They compared the histological and chemical iron content of various organs, from these cirrhotic subjects with similar organs from subjects with marked hepatic siderosis but without cirrhosis.

It was found that out of 24 cases of portal cirrhosis all but two had excessive hepatic deposits of iron. The average liver iron concentration was 2.31 g/100 g dry weight (range 0.07 to 8.16). The average splenic iron concentration was 2.63 g/100 g dry weight (range 0.31 to 7.36). Haemosiderin deposits were also found in the epitholial cells of the pancreas, thyroid, adrenal and myocardial fibres of the heart.

There were 14 cases of postnecrotic cirrhosis. In this group the average iron concentrations in both liver (0.38 g/100 g dry weight) and spleen (0.67 g/100 g dry weight) were lower than found in the general African population in the same age group. In contrast to the findings in portal cirrhosis, there was only one case with post-necrotic cirrhosis in which iron deposits were present in parenchymal cells other than the liver. In this case the hepatic iron concentration was 1.87 g/100 g dry weight.

In the 20 siderotic subjects without cirrhosis, iron deposits were confined to the liver, reticulo-endothelial system and lamina propria of the upper small bowel.

Because of the relatively low average liver iron concentrations in the group with postnecrotic cirrhosis it was concluded that excessive deposits of iron play no part in the genesis of this condition. They confirmed the findings of previous workers that, histologically, iron distribution in siderotic subjects with portal cirrhosis was "virtually indistinguishable" from that of idiopathic haemochromatosis except for the fact that in Bantu siderosis the splenic iron concentration was higher.

In this study, as in a previous one (Bothwell and Bradlow, 1960), they noted that the incidence of portal fibrosis increased as the hepatic iron concentration increased.

22.7% of their patients with portal cirrhosis were diabetic which, they pointed out, though much lower than the incidence found in idiopathic haemochromatosis was nevertheless ten times greater than found in non-haemochromatotic African subjects of comparable age distribution.

Bothwell and Isaacson (1962) compared the incidence of siderosis in autopsy material from 318 male and 265 female African adults. The chemical iron concentrations found in specimens of liver were compared with the degree of portal fibrosis or cirrhosis seen in histological sections of the livers.

It was found that while only 29.6% males had liver iron concentrations which were either normal or slightly raised (i.e. up to 0.19 g/100 g dry weight) there were 75.4% females who fell into this category. 37.4% males and 11.6% females had liver iron

concentrations of more than 1.0 g/100 g dry weight and were described as having severe siderosis.

Portal fibrosis or cirrhosis was very common in both male and female subjects with liver iron concentrations of more than 2.0 g/100g. It was suggested that, as fibrosis was not present in all such cases, "there are other factors which potentiate the fibrogenic effects of excessive iron deposits".

MacDonald, Becker and Pechet (1963) compared the findings in unselected autopsies on 106 Europeans and 42 Africans in Johannesburg with 84 unselected autopsies on "whites" in Boston, U.S.A.

They found that in Johannesburg 61% of the Europeans had stainable iron in the liver and the iron concentration varied from 0.203 to 0.740 mg/g wet weight. Stainable iron was found in the livers of 79% of their African subjects, with iron concentrations of between 0.102 and 21.21 mg/g wet weight. 45% of the "whites" in Boston had stainable iron in the liver. The iron concentrations varied between 0.227 and 0.518 mg/g wet weight.

They also noted the similarity in the pathological appearances between Africans with cirrhosis and siderosis, and idiopathic haemochromatosis.

In a review Bothwell (1964) drew attention to the fact that in Africans with siderosis and liver cirrhosis the percentage saturation of the circulating transferrin is raised while in Africans with siderosis but no cirrhosis the percentage saturation is about normal. He referred to the work of Jandl et al., (1959) which

showed that the uptake of iron by liver slices was greater where the percentage saturation of transferrin was high and suggested that a similar mechanism might be responsible for the widespread epithelial deposits of iron found in many African siderotic subjects with cirrhosis.

The availability of iron in African beer was studied by Bothwell, Seftel, Jacobs, Torrance and Baumslag, (1964). The average iron concentration in home-brewed African beer was 8.2 mg/100 ml beer (range 0.8-15.0 mg/100 ml) while the average iron concentration brewed by the municipal authorities was only 1.9 mg/100 ml beer (range 1.1-3.8). The mean pH for the home-brewed beer was 3.8 and the municipal beer 3.7. It was calculated that the average daily intake of iron from beer in men was at least 50 mg. These authors also showed that the mean absorption of iron from a volume of beer containing 8 mg of iron was 3.9 per cent, and from a volume of beer containing 25 mg of iron was 1.9 per cent. On the whole, African subjects were found to absorb less than European controls. This was attributed to the fact that most Africans had some degree of iron overload and this has been shown to depress absorption (Pirzio-Biroli and Finch, 1960).

Bothwell, Abrahams, Bradlow and Charlton (1965) compared the iron distribution in 13 European subjects suffering from idiopathic haemochromatosis with that in 13 Africans with advanced hepatic siderosis and portal cirrhosis. The aim of this was to ascertain whether idiopathic haemochromatosis is merely a variant of nutritional

cirrhosis occurring in subjects exposed to a high dietary intake of iron,as had been suggested by MacDonald (1963). It was found that in the liver in idiopathic haemochromatosis,iron deposits were heaviest in the parenchymal cells, moderate amounts were present in Kupffer cells and bile duct epithelium, little was seen in the phagocytes of the portal tracts. In Africans the heaviest deposits were found in the portal tract phagocytes and Kupffer cells, with moderately heavy deposits in the parenchymal cells and scanty deposits in bile duct epithelium. In the spleen very little iron was found in the patients with idiopathic haemochromatosis while the deposits were heavy in the African patients.

Because of these differences they believed "that idiopathic haemochromatosis is a metabolic entity" and is morphologically distinct from the haemochromatosis following the prolonged use of alcohol rich in iron,in subjects on a poor diet.

Seftel, Malkin, Schumaman, Abrahams, Lynch, Charlton and Bothwell (1966) drew attention to the association of osteoporosis, scurvy and siderosis in Africans in Johannesburg. They believed that osteoporosis among Africans in Johannesburg was common though its exact incidence was not known. They analysed the findings in 32 patients with severe osteoporosis. Symptoms were related to collapse of the vertebral bodies of the thoracic and lumbar spine due to the osteoporosis. Fourteen of their patients showed signs of scurvy and all consumed large quantities of African beer. Liver biopsy on eighteen of their subjects showed moderate or severe siderosis and the average

S.I. levels and percentage saturation of transferrin in their patients were higher than in controls.

In attempting to explain the pathogenesis of the condition, they pointed out that the association of osteoporosis and scurvy is well recognised, and suggested that siderosis might give rise to the scurvy due to the rapid oxidative catabolism of any absorbed ascorbic acid by the ferric iron, as had been shown to occur by Mazur, Green and Carleton (1960) as a result of in vitro studies.

SUMMARY

It has been shown that in South Africa, Bantu siderosis is common. The iron is most commonly found in the liver parenchymal cells, the reticulo-endothelial system and small bowel mucosa. In a few cases, usually associated with portal cirrhosis, there are also widespread epithelial deposits of iron and in these cases the condition is pathologically very similar to idiopathic haemochromatosis except for the greater reticulo-endothelial deposits in Bantu siderosis. It has been suggested that the epithelial deposits of iron are due to high percentage saturation of transferrin. Diabetes is frequently present in those subjects with widespread epithelial deposits.

The source of the iron in this condition is excessive consumption in the diet, especially African beer.

Most workers believe that the excessive amounts of liver iron play some part in the production of portal fibrosis and cirrhosis but believe that other factors also are involved.

Recently attention has been drawn to the association between siderosis, osteoporosis and scurvy.

Apart from confirming that this condition occurs in Rhodesia no attempt has been made to study its incidence and severity in that country in any detail.

OBJECT OF THE STUDY

Up to the present time siderosis in Rhodesian Africans has been studied only in a very superficial way, the sole investigation in Rhodesia being that of Gelfand (1955). The total number of cases examined was relatively small, viz. 103 and, as was pointed out earlier, no microscopic examination for iron was made on any tissue nor was there any attempt to assess the chemical concentration of tissue iron in this study. Higginson et al., (1953), as part of their investigation, examined an undisclosed number of livers from Rhodesian Africans but apart from noting that haemosiderin deposits were present in such livers no further details were given. The iron content of the Rhodesian African diet has not yet been investigated. Carr and Gelfand (1961) in a study of the serum iron and total iron binding capacities on Salisbury Africans showed none of the very high mean S.I. and T.I.B.C. values which were a feature of many of the Africans investigated by Gerritsen and Walker (1953) in Johannesburg. This could possibly mean that there was some difference between siderosis as it occurred in South Africa and Rhodesia, and it was felt that though there were some indications that siderosis in South African and Rhodesian Africans were probably similar, if not identical, this had been by no means well established.

The aims of the present study are:

1) to investigate in detail the incidence and degree of siderosis in Rhodesian Africans;

2) to consider whether or not the high concentrations of haemosiderin in the tissues are harmful;

3) to investigate the distribution of iron in the body of siderotic subjects and to consider any factors that might modify the distribution;

4) to measure the serum iron and total iron binding capacity values of a group of relatively healthy Africans;

5) to investigate the iron content of the Rhodesian African's diet;

6) to attempt to substantiate, by experimental and other methods, any theories formed as to the nature of the condition, its genesis and the tissue iron distribution.

SECTION II

INCIDENCE & DEGREE OF SIDEROSIS IN RHODESIAN AFRICANS

A) PRELIMINARY HISTOLOGICAL INVESTIGATION

Material & Methods

A preliminary histological study was made on tissues obtained from 200 unselected African autopsies performed by the author in the mortuary of Hararo Hospital, Salisbury in 1963. These included both autopsies carried out at the request of hospital consultants and police authorities. The only cases excluded were those in which autolytic changes were marked.

The tissues selected were liver, pancreas, heart and skin from the deltoid region. The skin specimens were taken from the deltoid region so as to be free of haemosiderin deposits due to other causes, which may be found in a number of sites (Lever, 1961). All tissues examined histologically or used for chemical estimation of iron concentration throughout all of the following investigations were fixed in neutral buffered formalin. The buffered formalin was prepared as described by Culling (1957) using sodium dihydrogen phosphate (anhydrous) (NaH_2PO_4) and disodium hydrogen phosphate (anhydrous) (Na_2HPO_4). Paraffin sections were prepared 5 microns thick and stained with haematoxylin and eosin and Lison's modification of Perl's method for iron (Pearse, 1961). Histological grading of the amounts of haemosiderin in tissues and grading of liver fibrosis in the preliminary and also subsequent investigations was based on the code devised by Bothwell and Bradlow (1960) with minor

modifications, viz.

Liver:

a) Parenchymal cells:

Grade 0 = no stainable haemosiderin granules.

Grade + = a few fine granules of haemosiderin in some or most of the liver cells, especially in the periportal regions.

Grade ++ = numerous fine granules of haemosiderin in most of the liver cells.

Grade +++ = numerous coarse and fine granules in most of the liver cells.

b) Kupffer cells:

Grade 0 = no stainable haemosiderin granules.

Grade + = occasional haemosiderin granules in some Kupffer cells.

Grade ++ = numerous fine haemosiderin granules in most Kupffer cells.

Grade +++ = numerous coarse haemosiderin granules in most Kupffer cells.

c) Portal tracts:

Grade 0 = no stainable haemosiderin granules.

Grade + = occasional haemosiderin granules in portal tract macrophages.

Grade ++ = small clumps of macrophages containing fine and coarse haemosiderin granules.

Grade +++ = heavy deposits of haemosiderin in coarse and fine granules both intra- and extra- cellular.

Spleen:

Grade 0 = no stainable haemosiderin granules.

Grade + = occasional fine granules of haemosiderin in some of the pulp macrophages.

Grade ++ = numerous fine and coarse haemosiderin granules in the pulp macrophages.

Grade +++ = numerous large masses of coarse intra- and extra-cellular haemosiderin in the splenic pulp and trabeculae.

Pancreas & Other Epithelial Tissues:

Grade 0 = no stainable haemosiderin granules.

Grade + = a few fine haemosiderin granules in some epithelial cells and/or in interstitial tissue.

Grade ++ = fine haemosiderin granules in most epithelial cells or moderate numbers of coarse granules patchily distributed in epithelial cells. Scattered coarse granules usually also found in interstitial tissue.

Grade +++ = heavy deposits of haemosiderin in fine and coarse granules in most epithelial cells and in interstitial tissue.

Heart:

Grade 0 = no stainable haemosiderin granules.

Grade + = a few fine haemosiderin granules in some myocardial fibres.

Grade ++ = many fine and a few coarse haemosiderin granules in most myocardial fibres.

Heart contd:

Grade +++ = fine and coarse granules of haemosiderin in most myocardial fibres.

Small Bowel:

Grade 0 = no stainable haemosiderin granules.

Grade + = a few fine haemosiderin granules in macrophages in the stroma of the villi.

Grade ++ = a moderate number of coarse haemosiderin granules in macrophages in the stroma of the villi.

Grade +++ = large masses of haemosiderin in coarse granules, mostly in macrophages but sometimes apparently extracellular, in the stroma of the villi and to a lesser extent scattered in the lamina propria of the mucosa.

The term "total score" of an organ used in reference to degree of siderosis in the preliminary investigation means the sums of the grades for the several sites, i.e. in the liver the total score equals the grade of the hepatic cells plus that of the Kupffer cells plus that of the portal areas.

Liver Fibrosis:

Grade 0 = portal tracts normal.

Grade + = slight thickening of portal tracts.

Grade ++ = moderate thickening of portal tracts.

Grade +++ = marked thickening of portal tracts with linkage of adjacent tracts in some areas, but without distortion of the architectural pattern.

Liver Fibrosis contd:

Grade ++++ = frank cirrhosis which was subdivided into coarse (C) and fine (F).

Results

The findings in the preliminary histological study are contained in Appendix I.

In the 61 subjects younger than 10 years, scanty deposits of haemosiderin were seen in the livers of 17 (28%) and in the pancreas of one. In no case was any stainable iron found in the heart or skin.

In the 139 subjects older than 10 years, stainable iron was found in the livers of 86 (62%). In females the incidence was 17 out of 43 (39.5%) and in males 69 out of 96 (72%). Hepatic siderosis of a total score of 4+ or more severe was found in 8 females (19%) and 39 males (41%). The frequency of occurrence of liver siderosis and its degree increased with age. The distribution of iron in the liver will be discussed in subsection (B).

Haemosiderin granules were found in the pancreas in 9 males and 6 females. In all cases where pancreatic deposits were moderate or heavy, i.e. a score of 2+ or 3+, there was also a fine cirrhosis of liver. In 6 cases with fine cirrhosis, scanty haemosiderin deposits were found in the heart and 2 of these cases had a few fine granules of haemosiderin in the skin, in proximity to the sweat glands.

These results will be discussed with the results in subsection (B).

B) COMBINED HISTOLOGICAL & CHEMICAL INVESTIGATION

Material & Methods

A more detailed investigation was then carried out in which livers and spleens were examined histologically and the iron content graded as in the preliminary investigation. Portions of fixed tissue of each organ were then used for chemical estimation of the iron. Usually this was done after 3 or 4 days of fixation and in no case was a greater interval than 10 days allowed to elapse because, even when neutral buffered formalin is used, iron tends to diffuse out of the tissue into the fixing fluid and in severely siderotic organs the fluid can easily be seen to be discoloured after a time. Samples of liver were taken from about the centre of the right lobe and those of the spleen from about the centre of the organ.

The method used for chemical estimation of the iron was that of Bothwell, Moos, and Lifschitz (1964). Using this method the haemoglobin iron concentration of the specimen is measured and subtracted from the total iron concentration to give the tissue iron concentration. This last value in liver and spleen is virtually the same as for storage iron as the only other source of iron in these tissues is the iron contained in enzymes and this is relatively very small. (Granick, 1959; Bothwell & Finch 1962; MacDonald, 1964). A short discussion on the method is contained in Appendix II. Iron concentrations are expressed as milligrammes

per gramme wet weight of tissue. In comparing the results of other authors, when these are expressed in dry weight, with the present results the former have been divided by four. The rationale of this is also discussed in Appendix II.

Samples of liver and spleen were obtained from 661 unselected autopsies on African subjects (383 males and 278 females) whose ages ranged from birth to old age. It should be noted that the ages in African subjects were usually estimates and are therefore approximate. Similar specimens were obtained from 101 Europeans (69 males and 32 females) of all ages for comparison. All autopsies on Africans were performed in the mortuary of Harare Hospital and on Europeans in the mortuary of the Salisbury European Hospital.

Results

The detailed results are contained in Appendix III.

Iron became visible histologically in Perl's stained sections, in both liver and spleen at a concentration of approximately 0.25 mg/g wet weight.

(i) Iron Distribution in Liver and Spleen.

Liver: in the liver haemosiderin deposits were usually first seen in the parenchymal cells except when the patient was suffering from a chronic infective process or renal failure. In such cases the haemosiderin first appeared in the Kupffer cells, and heavy deposits were also seen in the spleen.

Deposits in the parenchymal cells first appeared in those cells at the periphery of the lobule in the form of fine granules. As the iron concentrations increased the iron spread to involve the whole lobule, initially, in finely granular form and later the granules became progressively coarser. In very severe cases the coarse granular masses of haemosiderin obscured details of the cell structure. Regenerating cells found in livers with cirrhosis contained less iron than cells showing no evidence of proliferation. No iron was seen in the cells of any of the liver cell carcinomas in this series even in those cases with marked siderosis.

The Kupffer cells were almost invariably involved in moderate or severe degrees of siderosis and fairly frequently in the lesser degrees. As in the case of parenchymal cells haemosiderin granules were initially fine and became coarser eventually becoming an

irregular mass of haemosiderin making it difficult to see the cell.

In the portal areas usually no stainable iron was seen in early siderosis. As liver iron concentration increased the degree of haemosiderin deposition in this site, in most cases, roughly paralleled that in the hepatic and Kupffer cells but occasionally, even in the face of heavy deposits in hepatic and Kupffer cells, the deposits in portal areas were rather scanty. The haemosiderin was seen both in macrophages and apparently lying free in the interstitial tissue. Except in the presence of cirrhosis stainable iron was rarely seen in bile duct epithelium.

Spleen: in early siderosis haemosiderin was found in the pulp macrophages as fine granules. As the condition advanced the granules became coarser and in the most severe cases the haemosiderin also formed coarse irregular masses much of which was apparently extracellular. Deposits in trabeculae and capsule were inconstant. These were usually present when siderosis was marked but in some cases with heavy splenic involvement deposits in these sites were scanty. On the other hand they were sometimes quite pronounced when the pulp involvement was only moderate. Except in the most severe cases no haemosiderin was found in the lymphoid follicles.

(ii) Incidence and Degree of Siderosis.

<u>Europeans</u>

a) Children under 10 years: only 9 children under the age of 10 years came to autopsy during the period of the survey. There were two children of less than 6 months and both had stainable iron

in their hepatic cells. One child of one year and nine months, who died of bronchopneumonia following severe burns, had fairly heavy iron deposits in the Kupffer cells of the liver and in the spleen. No stainable iron was found in the livers or spleens of any of the others.

b) Subjects older than 10 years; in subjects older than 10 years stainable iron was found in the livers of 37 (40%). In males it was present in the livers of 27 (43.5%) and in females in the livers of 10 (33%). Respecting the sites of deposition of the iron; it was present in parenchymal cells only in 27 cases (29%), in the Kupffer cells only in 2 cases (2%), and in both parenchymal and Kupffer cells in 8 cases (9%). No stainable iron was found in the portal areas of any of the European subjects examined.

In Figure I the average concentrations of storage iron in the liver and spleen in each decade are compared graphically. The first decade has been omitted as the liver iron concentration normally varies considerably during this period (Ramage et al., 1933; Brückmann and Zondek, 1939) and an average value would not be very meaningful, also the numbers are too few to allow for further breakdown into narrower age groups.

Table I shows the average concentrations of storage iron and Table II the average total storage iron in European livers and spleens and the ranges of values found. The highest concentration of iron in a liver was 0.95 mg/g wet weight, found in a man of 40 years who died following a road accident. There was some evidence

FIGURE I

EUROPEANS

AVERAGE CONCENTRATION OF STORAGE IRON IN

LIVER & SPLEEN

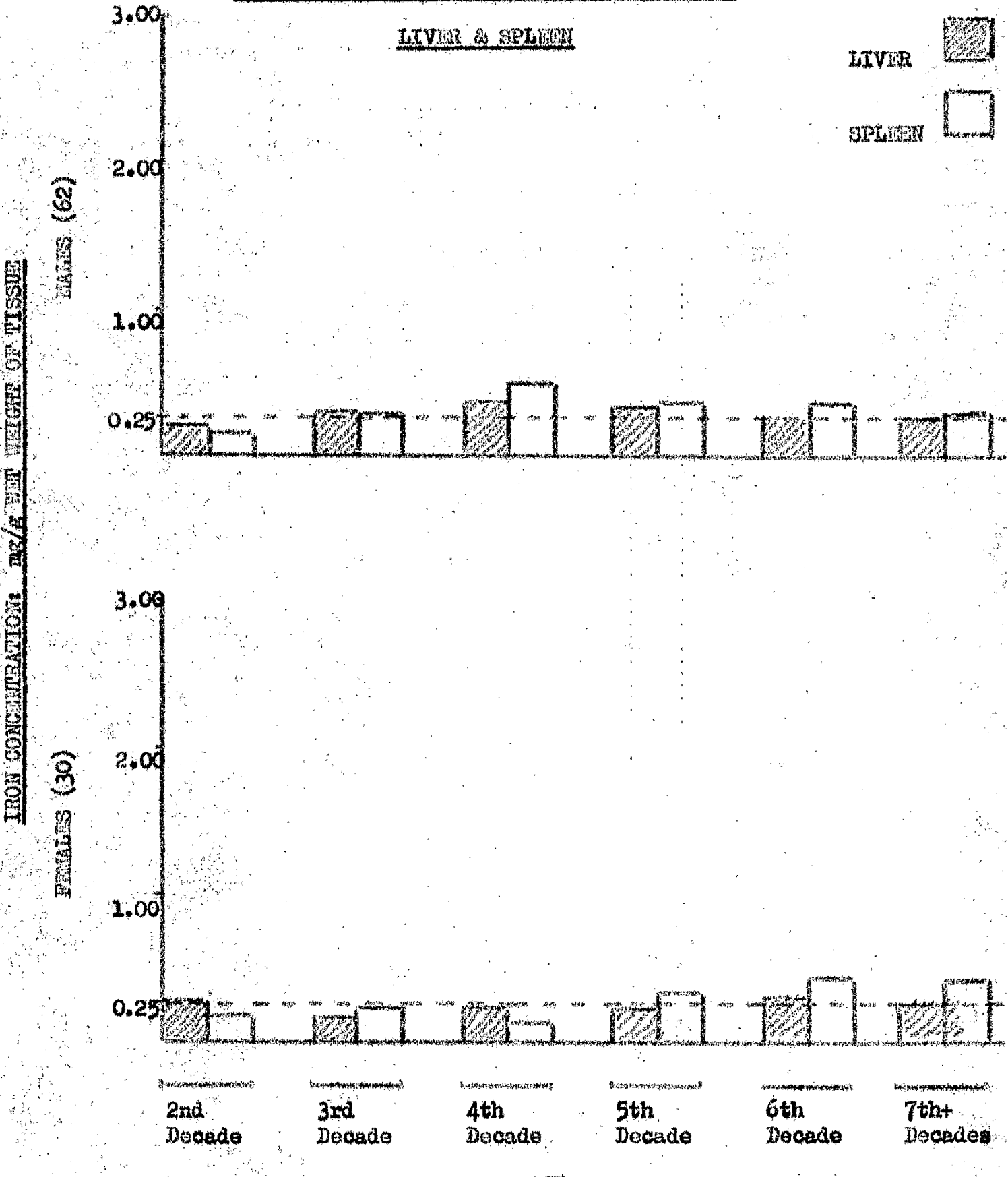


TABLE I

STORAGE IRON CONCENTRATIONS IN LIVER & SPLEEN

(mg/g wet weight of tissue)

EUROPEANS

	Age Group	Number of Cases	LIVER			SPLEEN		
			Average Concentration	Standard Deviation	Range	Average Concentration	Standard Deviation	Range
Male & Female	0-9	9	0.21	0.14	0.05-0.49	0.29	0.37	0.05-1.25

LIVER

	MALES				FEMALES			
Age Group	Number of Cases	Average Concentration	Standard Deviation	Range	Number of Cases	Average Concentration	Standard Deviation	Range
10-19	5	0.20	0.10	0.03-0.31	5	0.27	0.15	0.15-0.52
20-29	10	0.29	0.11	0.15-0.46	2	0.17	0.07	0.12-0.22
30-39	8	0.35	0.14	0.19-0.63	3	0.22	0.06	0.18-0.29
40-49	12	0.33	0.27	0.04-0.96	3	0.21	0.08	0.13-0.29
50-59	13	0.29	0.18	0.02-0.68	7	0.30	0.24	0.02-0.64
60 <	14	0.25	0.14	0.06-0.52	10	0.27	0.11	0.12-0.46

TABLE I CONTD.

STORAGE IRON CONCENTRATIONS IN LIVER & SPLEEN

(mg/g wet weight of tissue)

EUROPEANS

SPLEEN

	MALES				FEMALE			
Age Group	Number of Cases	Average Concentration	Standard Deviation	Range	Number of Cases	Average Concentration	Standard Deviation	Range
10-19	5	0.15	0.03	0.11-0.20	5	0.18	0.03	0.15-0.22
20-29	10	0.29	0.17	0.12-0.70	2	0.21	0.01	0.21-0.22
30-39	8	0.49	0.71	0.11-2.24	2	0.14	0.07	0.09-0.18
40-49	12	0.36	0.35	0.09-1.19	3	0.22	0.11	0.09-0.30
50-59	13	0.35	0.31	0.07-1.17	7	0.43	0.58	0.005-1.69
60 <	13	0.30	0.20	0.07-0.68	10	0.41	0.46	0.08-1.67

TABLE II

TOTAL STORAGE IRON IN LIVER & SPLEEN (grammes)

EUROPEANS

Male & Female

Age Group	Number of Cases	LIVER			SPLEEN		
		Average Total Iron	Standard Deviation	Range	Average Total Iron	Standard Deviation	Range
0-9	9	0.10	0.05	0.02-0.15	0.01	0.01	0.003-0.04

LIVER

	MALES				FEMALES			
Age Group	Number of Cases	Average Total Iron	Standard Deviation	Range	Number of Cases	Average Total Iron	Standard Deviation	Range
10-19	5	0.35	0.19	0.06-0.60	5	0.37	0.13	0.17-0.48
20-29	10	0.46	0.15	0.23-0.62	2	0.24	0.12	0.16-0.32
30-39	8	0.60	0.25	0.37-0.99	3	0.36	0.23	0.15-0.61
40-49	12	0.60	0.52	0.08-1.49	3	0.34	0.13	0.21-0.47
50-59	13	0.55	0.37	0.04-1.25	7	0.52	0.41	0.02-1.05
60 <	14	0.42	0.21	0.06-0.88	10	0.39	0.17	0.08-0.69

TABLE II CONTD.

TOTAL STORAGE IRON IN LIVER & SPLEEN (grammes)

EUROPEANS

SPLEEN

	MALES				FEMALES			
Age Group	Number of Cases	Average Total Iron	Standard Deviation	Range	Number of Cases	Average Total Iron	Standard Deviation	Range
10-19	5	0.03	0.01	0.01-0.04	5	0.03	0.02	0.01-0.05
20-29	10	0.05	0.03	0.03-0.11	2	0.03	0.004	0.025-0.03
30-39	8	0.09	0.14	0.01-0.44	2	0.01	0.006	0.01-0.02
40-49	12	0.04	0.03	0.01-0.10	3	0.05	0.02	0.03-0.06
50-59	13	0.06	0.05	0.01-0.19	7	0.04	0.07	0.001-0.19
60-69	13	0.04	0.03	0.01-0.07	10	0.06	0.07	0.005-0.23

that this man was a fairly heavy drinker. The highest concentration in a spleen was 2.24 mg/g wet weight, in a man of 30 years who died of renal failure secondary to chronic pyelonephritis.

Cirrhosis was present in the livers of 3 (4%) males but was not found in any of the European females. Two of the cases of cirrhosis were of the fine (portal or alcoholic) type and in both of these the parenchymal cells showed marked fatty change. In the third liver the cirrhosis was of the coarse (postnecrotic) type and no evidence of fatty change was found. Stainable iron was not present in any of these cirrhotic livers.

Africans

a) Children under 10 years: out of the total of 661 African autopsies performed, 126 were on children (61 males and 65 females) of less than 10 years of age. In view of the considerable normal variation in liver iron concentration during the first decade previously referred to with regard to European children, it was decided to subdivide this decade into smaller age groups and consider them separately from the older subjects.

Stainable iron was found in the livers of 53 (42%) and in the spleens of 78 (62%). The average storage iron concentration in the liver, the ranges found, and the incidence of stainable iron in various age groups are shown in Table III. The average storage iron concentration rose during the first two months of life then fell to a level little more than half that at birth. Apart from a slight unexplained rise in the 4 to 6 year age group it continued

TABLE III

STORAGE IRON CONCENTRATION

IN LIVERS OF 126 AFRICAN CHILDREN

(Expressed as mg/g wet weight of tissue)

Age Group	Total Cases	Average Concentration	Standard Deviation	Range	Number with Stainable Iron	Percentage with Stainable Iron
Foetus	16	0.36	0.21	0.097 - 0.79	9	56.2
-2 months	27	0.51	0.20	0.14 - 1.57	22	81.5
-2 years	35	0.20	0.23	0.04 - 1.43	8	22.4
-4 years	13	0.18	0.12	0.06 - 0.52	3	23.1
-6 years	11	0.31	0.18	0.04 - 0.71	6	54.6
-8 years	14	0.22	0.12	0.05 - 0.48	5	35.7
-10 years	10	0.15	0.17	0.08 - 0.26	0	0

at that level until the age of 8 years when there was a further slight drop. In the 10 subjects examined between 8 and 10 years of age no stainable iron was found in any of their livers.

The histological distribution of stainable iron between hepatic and Kupffer cells showed no specific pattern. Haemosiderin was seen in the hepatic cells only in 13 (10%) cases, in the Kupffer cells only in 9 (7%) cases, and in more than one site in 30 (23%) cases. In one case, scanty deposits were found in the portal areas only. The average total liver storage iron and ranges of values found are contained in Table IV.

TABLE IV

TOTAL STORAGE IRON IN LIVERS

OF 126 AFRICAN CHILDREN

(Expressed as grammes of iron)

Age Group	Number of Cases	Average Total Storage Iron	Standard Deviation	Range
Foetus	16	0.04	0.04	0.01 - 0.14
-2 months	27	0.05	0.03	0.01 - 0.15
-2 years	35	0.05	0.06	0.01 - 0.38
-4 years	13	0.07	0.04	0.02 - 0.17
-6 years	11	0.17	0.12	0.03 - 0.45
-8 years	14	0.13	0.07	0.04 - 0.33
-10 years	10	0.12	0.07	0.06 - 0.29

Table V shows the average storage iron concentrations found in the spleens of the various age groups. The ranges of values found and incidence of stainable iron seen histologically are also shown.

b) Subjects older than 10 years: in the 535 Africans of more than 10 years of age stainable iron was found in the livers of 334 (62.4%). It was present in 222 male livers (68.9% of males) and 112 female livers (52.7% of females). Table VI shows the percentage of cases in each decade in which the liver storage iron exceeded 0.25 mg/g wet weight i.e. were considered to be siderotics as defined in the introduction. It also shows the percentage of cases in which the splenic iron concentration exceeded 0.25 mg/g wet weight.

The ranges of iron concentrations in liver and spleen are contained in Table VII and ranges of total storage iron in these organs in Table VIII. The highest liver storage iron concentration was 14.13 mg/g wet weight found in a man of 80 years who died of pneumonia. The highest iron concentration found in a spleen was 20.72 mg/g wet weight and was in a female of 70 years killed in a road accident.

TABLE V

STORAGE IRON CONCENTRATION

IN SPLEENS OF 126 AFRICAN CHILDREN

(Expressed as mg/g wet weight of tissue)

Age Group	Number of Cases	Average Concentration	Standard Deviation	Range	Number with Stainable Iron	Percentage with Stainable Iron
Foetus	16	0.27	0.15	0.07 - 0.60	9	56.2
-2 months	27	0.57	0.32	0.14 - 1.23	20	74.0
-2 years	35	0.32	0.26	0.04 - 1.08	21	60.0
-4 years	13	0.31	0.35	0.08 - 1.39	7	53.8
-6 years	11	0.39	0.20	0.12 - 0.81	8	72.8
-8 years	14	0.55	0.53	0.08 - 2.06	9	64.2
-10 years	10	0.25	0.18	0.05 - 0.54	4	40.0

TABLE VI

AFRICANS

PERCENTAGE OF CASES IN EACH DECADE IN WHICH

LIVER & SPLEEN STORAGE IRON EXCEEDS 0.25 mg/g

Age Group	Liver		Spleen	
	Male	Female	Male	Female
10-19	25	23	31	45
20-29	60	32	58	44
30-39	71	59	71	54
40-49	85	54	86	71
50-59	78	71	83	74
60 <	80	81	85	84

TABLE VII

STORAGE IRON CONCENTRATIONS IN LIVER & SPLEEN

(mg/g wet weight of tissue)

AFRICANS

LIVER

	MALE				FEMALE			
Age Group	Number of Cases	Average Concentration	Standard Deviation	Range	Number of Cases	Average Concentration	Standard Deviation	Range
10-19	32	0.19	0.12	0.06-0.57	30	0.23	0.21	0.05-1.09
20-29	55	0.57	0.95	0.07-4.71	45	0.31	0.39	0.05-2.29
30-39	65	0.74	0.82	0.05-4.47	35	0.59	0.95	0.06-4.37
40-49	59	2.13	2.65	0.08-11.90	41	0.51	0.80	0.05-4.47
50-59	71	1.95	2.32	0.05-10.42	31	1.23	1.33	0.09-5.60
60 <	40	2.46	3.08	0.06-14.13	31	1.84	2.26	0.08-10.48

TABLE VII CONTD.

STORAGE IRON CONCENTRATIONS IN LIVER & SPLEEN

(mg/g wet weight of tissue)

AFRICANS

SPLEEN

	MALE				FEMALE			
Age Group	Number of Cases	Average Concentration	Standard Deviation	Range	Number of Cases	Average Concentration	Standard Deviation	Range
10-19	32	0.26	0.29	0.04-1.37	29	0.26	0.26	0.05-1.33
20-29	55	0.52	0.83	0.00-5.96	45	0.43	0.41	0.03-2.64
30-39	65	0.91	1.24	0.08-5.90	35	0.61	0.78	0.07-3.56
40-49	59	2.07	2.42	0.10-11.41	41	0.62	0.77	0.02-3.79
50-59	71	2.44	2.83	0.05-12.55	31	0.98	1.05	0.06-4.02
60 <	40	2.98	4.04	0.11-17.87	31	2.52	4.37	0.03-20.72

TABLE VII CONTD.

STORAGE IRON CONCENTRATIONS IN LIVER & SPLEEN

(mg/g wet weight of tissue)

AFRICANS

COMPARISON OF IRON CONCENTRATION IN LIVER & SPLEEN

	MALE			FEMALE		
Age Group	Number of Cases	Mean Difference	p Value	Number of Cases	Mean Difference	p Value
10-19	32	+0.075	> 0.05	30	+0.037	> 0.05
20-29	55	-0.054	> 0.05	45	+0.120	>0.05
30-39	65	+0.158	>0.05	35	+0.015	>0.05
40-49	59	+0.063	>0.05	41	+0.097	>0.05
50-59	71	+0.487	< 0.05	31	-0.249	>0.05
60 <	40	+0.542	>0.05	31	+0.676	>0.05

+ means spleen concentration greater than liver

- means spleen concentration less than liver

TABLE VIII

TOTAL STORAGE IRON IN LIVER & SPLEEN

(Expressed in Grammes)

AFRICANS

LIVER

	MALE				FEMALE			
Age Group	Number of Cases	Average Total Iron	Standard Deviation	Range	Number of Cases	Average Total Iron	Standard Deviation	Range
10-19	32	0.25	0.19	0.05-0.77	30	0.31	0.35	0.06-1.59
20-29	55	0.77	1.24	0.09-6.07	45	0.47	0.65	0.06-4.01
30-39	65	1.18	1.44	0.08-6.95	35	0.96	1.77	0.09-8.20
40-49	59	3.18	4.14	0.12-22.18	41	0.74	1.19	0.07-6.48
50-59	71	3.33	4.28	0.06-16.88	31	1.92	2.83	0.12-15.25
60 <	40	3.01	3.81	0.05-17.68	31	2.54	3.67	0.07-16.28

TABLE VIII CONTD.

TOTAL STORAGE IRON IN LIVER & SPLEEN

(Expressed in Grammes)

AFRICANS

SPLEEN

	MALE				FEMALE			
Age Group	Number of Cases	Average Total Iron	Standard Deviation	Range	Number of Cases	Average Total Iron	Standard Deviation	Range
10-19	32	0.05	0.09	0.01-0.48	29	0.04	0.04	0.01-0.14
20-29	55	0.08	0.08	0.005-0.36	45	0.08	0.09	0.01-0.39
30-39	65	0.16	0.26	0.01-1.68	35	0.11	0.14	0.01-0.77
40-49	59	0.35	0.46	0.02-2.64	41	0.09	0.08	0.003-0.29
50-59	71	0.46	0.49	0.01-2.35	31	0.15	0.15	0.01-0.60
60 <	40	0.59	1.13	0.01-6.10	31	0.28	0.46	0.01-1.86

DISCUSSION

The concentration at which iron became visible histologically in the liver, viz. 0.25 mg/g wet weight, was the same as that found by South African workers (Gillman et al., 1945; Higginson et al. 1953; Bothwell and Bradlow, 1960). In the present series iron became visible in the spleen at the same concentration, though Bothwell and Bradlow (1960) could only demonstrate iron in the spleen histologically at concentrations of 0.37 mg/g wet weight or more. The probable explanation of this anomaly is that these authors estimated the total iron concentration while in the present series the storage iron concentrations were measured. The haemoglobin iron concentration in the spleen could easily account for the difference in the two findings.

i) Iron Distribution in Liver and Spleen: this does not differ in any material way from that reported by the South African workers (Gillman et al, 1945; Higginson et al., 1953; Bothwell and Bradlow, 1960). Higginson et al.(1953) believed that iron deposits first appeared in the Kupffer cells of the liver but most other workers considered that the hepatic cells were the site of primary iron deposition (Gillman et al., 1945; Wainwright, 1957; Bothwell and Bradlow, 1960). In this study it appeared that though haemosiderin first appeared in the hepatic cells in uncomplicated cases, the presence of infection or chronic renal disease resulted in its first becoming visible in the Kupffer cells.

ii) Incidence and Degree of Siderosis:

Europeans: a) Children under 10 years. There were too few children in this group to allow for strict statistical comparison with findings elsewhere but the values found for liver iron concentration and total iron were of the same order as found by Ramage et al. (1933) in British children.

b) Subjects older than 10 years. In Table IX the frequency with which stainable iron occurs in the livers of non-African subjects in various parts of the world is compared with that in Rhodesian Europeans. It seems plain that, as the incidence in Rhodesian Europeans is lower than found in other centres with the exception of Cape Town, the factors responsible for siderosis in Rhodesian Africans are apparently confined to that section of the community. The storage iron concentrations in European livers were all within the range found in European subjects elsewhere in the world by numerous workers and summarized in a recent study (Powell, 1966). Probably those subjects with concentrations in the upper part of the range were fairly heavy drinkers as it has been shown that alcohol enhances the absorption of orally administered ferric iron (Charlton, Jacobs, Seftel and Bothwell, 1964) and that many alcoholic beverages consumed by Europeans are rich in iron (MacDonald, 1963). In none of the European cases were the very high iron concentrations found in Africans noted. Idiopathic haemochromatosis occurs in Europeans in Rhodisia (Gelfand, 1967) but its incidence is not known and no cases were seen in the course of the present study.

TABLE IX

INCIDENCE OF STAINABLE IRON IN LIVERS OF NON-AFRICAN SUBJECTS IN VARIOUS PARTS OF THE WORLD

Country & Centre	Investigators	Percentage with Stainable Iron in Liver
Ireland (Galway)	MacDonald & Pechet (1965)	66
Israel (Tel Hashomer)	MacDonald & Pechet (1965)	55
Japan (Tokyo)	MacDonald & Pechet (1965)	72
South Africa (Johannesburg)	MacDonald & Pechet (1965)	61
U.S.A. (Boston)	MacDonald & Pechet (1965)	(53 (70
U.S.A. (San Francisco)	MacDonald & Pechet (1965)	80
South Africa (Cape Town)	Uys et al (1960)	30
Rhodesia (Salisbury)	Present Series	40

As Table I and Figure I show, the average iron concentrations in European spleens roughly paralleled those in the liver in each decade and concentrations in both liver and spleen were slightly higher in males than females except in the sixth and subsequent decades when they were about the same. The lower values found in females were no doubt due to blood loss associated with menstruation and increased iron requirements during pregnancy.

In six cases the iron concentration in the spleen was greater than 1.0 mg/g wet weight. Details of these are shown in Table X. The first three illustrate the reticuloendothelial involvement in presence of infection and renal disease, referred to previously and to be discussed further in Section III. Probably in the case of the child with the burns red cell damage, which occurs in such a condition (de Gruchy, 1960), contributed to the heavy iron deposits. It should be noted however, that four female African children in this series also died of burns and in none of these were there heavy iron deposits in the reticuloendothelial system. The likely explanation of this apparent anomaly is that none of the African children survived for more than 3 days while the European child survived for 47 days.

The second three cases in Table X also show a predominantly reticuloendothelial involvement though no evidence of infection or renal disease was found at autopsy. In none of these cases was there any history of blood transfusion or of injections of iron-containing compounds.

TABLE X

EUROPEAN SUBJECTS WITH STORAGE IRON CONCENTRATIONS

IN SPLEEN OF MORE THAN 1.0 mg/g WET WEIGHT

AGE	SEX	SPLEEN		LIVER			CAUSE OF DEATH
		Iron Concentration*	Histological Iron	Iron Concentration*	Histological Iron		
					H.C.†	K.C.‡	
1.9/12	M	1.25	+++	0.27	0	++	Bronchopneumonia Severe Burns
30	M	2.24	+++	0.46	0	+++	Renal Failure Chronic Pyelonephritis
54	F	1.69	+++	0.61	+	++	Renal Failure Chronic Pyelonephritis
45	M	1.19	+++	0.26	+	0	Road Accident
50	M	1.17	+++	0.40	++	++	Road Accident
64	F	1.64	+++	0.33	+	+	Codeine Poisoning

* mg/g wet weight

† H.C. = Hepatic parenchymal cells

‡ K.C. = Kupffer cells

Africans. a) Children under 10 years.

In Table XI the liver iron concentrations in various age groups of Rhodesian African children are compared with the findings in similar groups by Ramage et al., (1933) in Britain, Brückmann and Zondek (1939) in Palestine, and with the values found in European foetuses by Wainwright (1957) in South Africa. As the results given by Ramage et al., are for total liver iron concentrations they are compared with the total iron concentrations found in the present series. The results of both Brückmann and Zondek, and Wainwright are for non-haemin iron concentrations and are thus compared with the storage (non-haemin) iron values of the present series.

In the foetus no significant difference in liver iron concentration was seen in any of the four groups compared. Between birth and two months there was no significant difference between Rhodesian African infants and the findings of Bruckmann and Zondek but the values of Ramage et al. were significantly higher. No significant difference was found in any of the groups between two months and two years but in the older age groups a slight but significantly higher average concentration was found in Rhodesian African children compared with the two others. Most of the values found for total liver iron were within the range given by Ramage et al., for British children but the average total found by these authors was higher, up till the age of two months.

The fact that average liver iron concentrations in the African foetus showed no significant difference from that found in

TABLE XI

COMPARISON BETWEEN LIVER IRON CONCENTRATIONS IN RHODESIAN AFRICAN CHILDREN WITH SIMILAR GROUPS OF EUROPEANS ELSEWHERE IN THE WORLD

(Expressed as mg/g wet weight of tissue)

	Britain (Ramage et al.)			Rhodesia			
Age Group	Number of Cases	Average total liver iron concentration	Standard Deviation	Number of Cases	Average total liver iron concentration	Standard Deviation	Value
Foetus	14	0.55	0.23	16	0.51	0.19	Not significant
0-2 months	26	0.87	0.33	27	0.57	0.33	.01 p .001
-2 years	56	0.35	0.28	35	0.24	0.23	Not significant
-10 years	23	0.18	0.09	48	0.27	0.12	.01 p .001

	Palestine (Brückman & Zonder)			Rhodesia			
Age Group	Number of Cases	Average storage iron concentration in liver	Standard Deviation	Number of Cases	Average storage iron concentration in liver	Standard Deviation	Value
Foetus	5	0.42	0.20	16	0.36	0.21	Not significant
0-2 months	10	0.47	0.21	27	0.51	0.20	Not significant
-2 years	8	0.11	0.09	35	0.20	0.23	Not significant
-8 years	4	0.065	0.13	38	0.23	0.135	.05 p .01
	South Africa (Wainwright)			Rhodesia			
Foetus	7	0.31	0.240	16	0.36	0.21	Not significant

N.B. 1. Total iron = storage iron + haemoglobin iron
2. Storage iron = non-haemin iron
3. In converting values given by other authors in dry weight into wet weight it has been assumed that the average liver contains 75% water

Europeans would appear to indicate that, despite raised iron stores as evidenced by the presence of excess storage iron in the livers of between 23 and 59% women of childbearing age (Table VI), this iron is not transmitted to the foetus to any appreciable extent. The average non-haemin liver iron concentration found by Wainwright in his 30 African foetuses in Durban was 0.375 mg/g wet weight which corresponds closely to the 0.36 mg/g found in this group in Salisbury.

It is difficult to explain why the average liver iron concentration between birth and two months found by Ramage et al. was higher than found in the present series especially as the iron values in the foetus are not significantly different in the two groups. Possibly the introduction of iron supplements into the diet of the European children might have contributed to the difference but this seems rather an inadequate explanation because of the short time available to build up these stores. Nevertheless it has been shown that absorption of ferrous iron is almost complete in the first few weeks of life (Ezekiel, 1967) so this possibility cannot be completely discounted.

The higher liver iron concentrations found in African children over the age of two years compared with non-Africans of the same age is possibly due to the high iron content of their diet, a fact which will be demonstrated in Section V. As could be expected, this would not affect children of less than two years because the average age at which mixed feeding is started in local African children is six and a half months (Buchanan, 1967).

The absence of stainable iron from the livers of children between 8 and 10 years was in marked contrast to earlier age groups in which the incidence of stainable iron was quite high. It is possibly related to greater demands for erythropoiesis at this age.

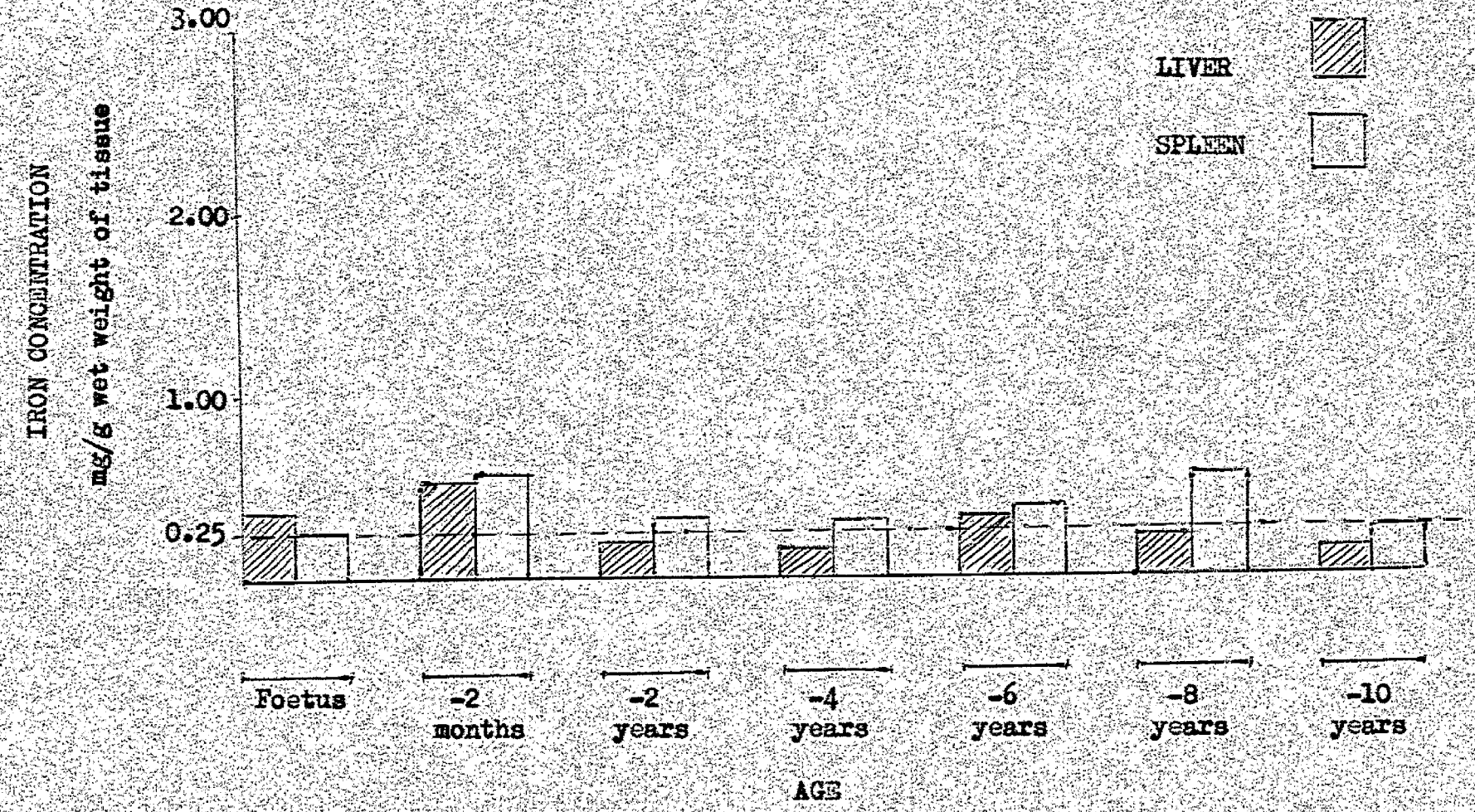
The average iron concentrations in livers and spleens of Rhodesian African children of various ages are compared in Figure II. Except in the foetus the splenic concentrations were higher than those of the liver. In the 6 to 8 year age groups the average concentration in spleen was elevated by two relatively high values, one, 2.06 mg/g wet weight in a child with peritonitis following a colostomy operation and Hirsprung's disease and the other, 1.14 mg/g wet weight in a child with Burkitt's sarcoma.

b) Subjects older than 10 years. The average iron concentrations found in both livers and spleens of Africans were dramatically higher than those found in Europeans. The highest liver iron concentration in an African (14.13 mg/g) was approximately fifteen times the highest found in a European (0.96 mg/g) and the highest splenic iron concentration in an African (20.72 mg/g) was approximately nine times the highest in a European (2.24 mg/g).

The incidence of stainable iron in the liver of all subjects over the age of ten years in Subsection (A) was 62% and in Subsection (B) was 62.4% showing a very close agreement. Similarly in males the incidence of 72% in Subsection (A) was very close to the incidence of 68.9% found in Subsection (B). The incidence in females is however somewhat different being 39.5% in Subsection (A) and 52.7%

FIGURE II

AVERAGE STORAGE IRON CONCENTRATIONS IN LIVERS & SPLEENS OF 126 AFRICAN CHILDREN



in Subsection (B). This higher incidence found in the latter might be in some measure explained by the fact that 48% of the females in this subsection were aged 40 years or more, an age group in which the incidence of siderosis is higher than in younger people, while in Subsection (A) only 44% females were over 40 years of age. It should also be remembered that as most ages given for Africans are approximate there might be an even greater difference in the age group distribution between the two subsections.

The incidence of stainable iron in the livers of Rhodesian Africans is compared with that found in other African centres in Table XII. This appears to be rather less than found by most workers in Johannesburg, about the same as in Durban, and greater than in Cape Town and Ghana. The incidence in Rhodesia also appears to be greater than in Zambia (Gadd, 1967) and Malawi (Buchanan, 1965) though no investigations have yet been carried out in either of these countries.

The highest liver iron concentrations found by some South African workers are compared with those of the present series in Table XIII. As this shows, the values found in Rhodesian Africans are very similar to those found in the South African Bantu.

The splenic storage iron concentrations ranged from negligible amounts to 20.72 mg/g wet weight (Table VII) and are very similar to those found in Johannesburg by Bothwell and Bradlow (1960) in whose series there was a range of from less than 0.38 to 13.3 mg/g wet weight (<0.15 to 5.32 g/100 g dry weight). Isaacson et al., (1961)

TABLE XII

INCIDENCE OF STAINABLE IRON IN LIVERS OF AFRICANS IN RHODESIA COMPARED WITH OTHER PARTS OF AFRICA

Country & Centre	Investigators	Percentage with Stainable Iron in Liver
South Africa		
Johannesburg	Strachan, (1929)	49
	Gillman et al., (1948)	86
	Bothwell & Bradlaw, (1960)	89
	MacDonald, (1963)	79
Durban	Wainwright, (1957)	65
Cape Town	Uys et al., (1960)	56
Ghana	Edington, (1959)	40
Rhodesia		
Salisbury	Gelfand, (1955)	65
	Present Series	62

TABLE XIII

MAXIMUM LIVER IRON CONCENTRATIONS FOUND BY SOME SOUTH AFRICAN WORKERS COMPARED WITH THE PRESENT SERIES

(Converted into mg/g wet weight)

INVESTIGATORS	MAXIMUM IRON CONCENTRATION
Gillman et al., 1945	13.6
Gillman & Gillman, 1948	12.50
Higginson et al., 1953	13.80
Wainwright, 1957	8.75
Bothwell & Bradlow, 1960	10.25 *
Isaacson et al., 1961	20.40
Present Series, Section II	14.13
Section IV	28.12

* Approximate taken from Bothwell & Bradlow's Figure I.

found the splenic iron concentration in subjects with portal cirrhosis ranged from 0.77 to 18.4 mg/g wet weight (0.31 to 7.36 g/100 g dry weight) which is also similar to that found in the present series.

Reference to Table VI shows that the incidence of siderosis in Rhodesian African males and females was roughly the same in the second decade. In males there was a rapid increase to a peak in the fifth decade and thereafter there was a slight decline. The incidence in females rose more slowly to reach the same level as males in subjects of over 60 years.

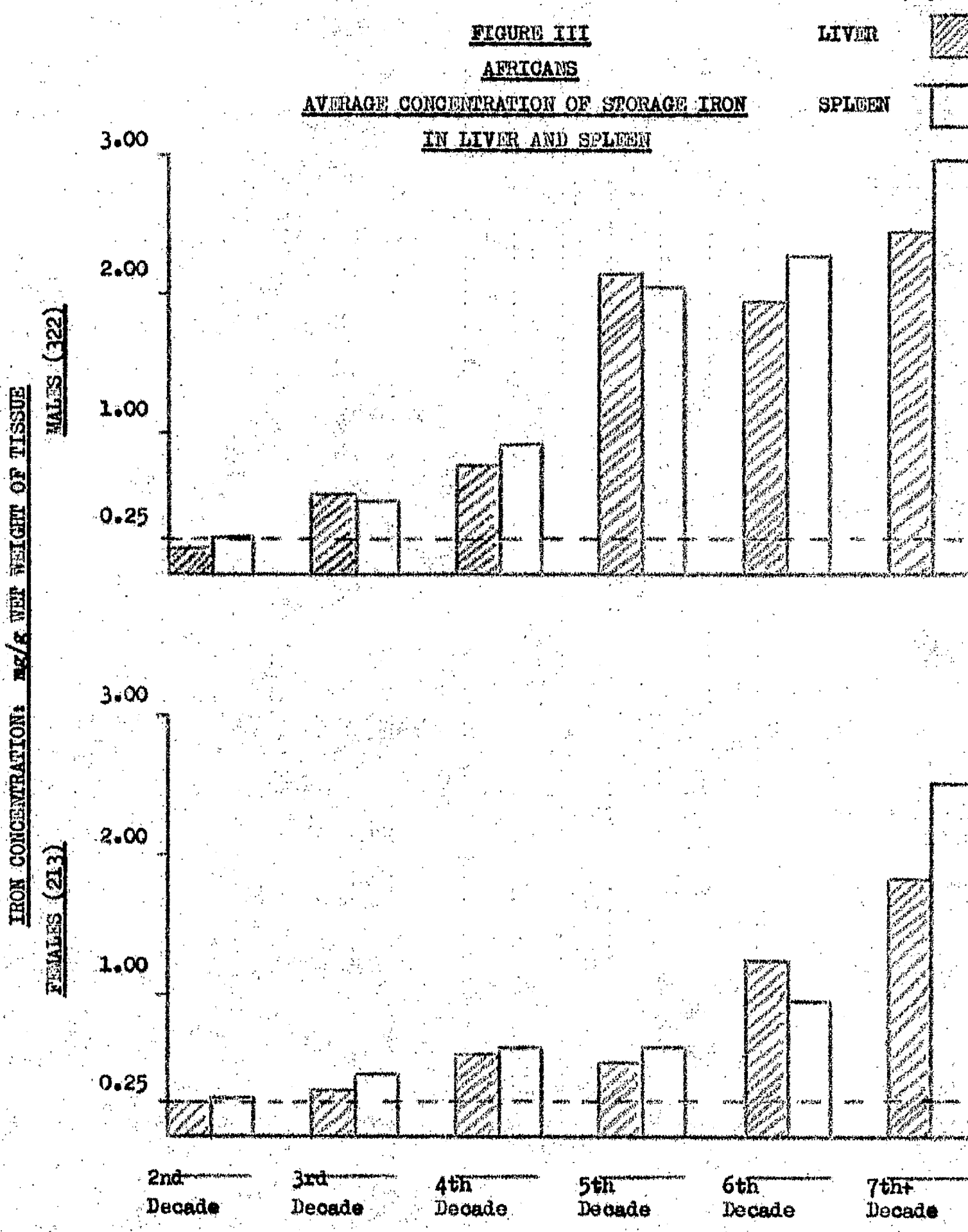
Comparison of the incidence of siderosis between livers and spleens of each sex (Table VI) shows that in males there is a close parallel between liver and spleen in all age groups. In females the spleen shows a higher percentage incidence of siderosis in the second, third and fifth decades but, rather surprisingly, there is a slightly lower incidence in the fourth decade. After the age of fifty the incidence of siderosis is about the same in the two organs.

Figure III shows the average concentrations of storage iron in the livers and spleens of both sexes. Comparison with those values found in Europeans (Figure I) shows that after the second decade the concentrations found in both liver and spleen in both sexes were much higher in Africans. Also the average concentration of iron in liver and spleen increased with age and roughly at the same rate. In Table VII it can be seen that there was no significant difference between the average liver and spleen iron concentrations in any of

FIGURE III

AFRICANS

AVERAGE CONCENTRATION OF STORAGE IRON IN LIVER AND SPLEEN



the groups of females. In males, however, the average splenic iron concentration was slightly but significantly higher in the sixth decade though not in any of the others.

In males the average concentrations were normal in the second decade, rose gradually in the third and fourth decades, then sharply in the fifth decade. A further rise was noted in the older age groups but again this was more gradual. In females also the rise in iron concentrations started in the third decade but at this stage was less pronounced than in males. There was a sudden rise in the sixth and subsequent decades but the average concentrations were still less than those found in males.

iii) Comparison with Idiopathic Haemochromatosis.

There were 72 African adults (57 males and 15 females) whose liver iron concentration was in excess of 2.6 mg/g wet weight, i.e. was within the range given by Sheldon (1935) for idiopathic haemochromatosis. In these cases however, the average liver storage iron concentration was only 4.99 mg/g wet weight which is much less than the average quoted by Sheldon which was 11.5 mg/g wet weight.

The total liver storage iron was greater than 6.85 g in 34 African adults (27 males and 7 females) and this again is within the range given by Sheldon for subjects with haemochromatosis, but the average total liver iron in the 34 cases was 11.10 g which is little more than half Sheldon's average, viz. 21.36 g.

These factors all appear to show that despite the fact that there are similarities in iron deposition between idiopathic

haemochromatosis and Bantu siderosis nevertheless there are definite differences. This point will be further discussed in Section IV in relation to iron distribution in the body of subjects with Bantu siderosis.

SUMMARY

Tissues from a total of 962 autopsies were examined in an attempt to assess fairly accurately the incidence and degree of siderosis in the Rhodesian population. In a preliminary investigation on 200 African subjects, specimens of liver, pancreas, heart and skin were examined histologically for iron. This was followed by a combined histological and chemical study of the iron content of livers and spleens of 101 Europeans and 661 Africans.

In the liver, iron deposits first appeared in the hepatic cells at the periphery of the lobules except when the patient suffered from an infection or chronic renal disease. In these cases, the iron first appeared in the Kupffer cells and there were also heavy deposits in the spleen. The iron distribution found in liver and spleen was similar to that found by South African workers.

Iron concentrations in the livers of African children under 10 years of age showed no major differences from those of British children though, between the ages of 2 and 10 years, the average concentration was slightly higher than that found in Britain. This difference was attributed to the high iron content of the African diet.

In subjects over 10 years of age stainable iron was found in the livers of 40% of Europeans and of 62% of Africans. In both races the incidence was appreciably higher in males than females. In Africans the incidence and degree of siderosis increased with

age though this was not noted in Europeans (possibly this was due to the relatively small number of European tissues examined). The average iron concentrations in both livers and spleens of Africans after the second decade were very much higher than those of Europeans.

Comparison between the incidence of siderosis in Rhodesian Europeans and the incidence in non-African subjects elsewhere in the world shows that this is rather lower in the former than in most of the other centres quoted. This is interpreted as meaning that whatever factors may be responsible for siderosis in Africans they are restricted to Africans and do not affect the European section of the community.

Concentrations of iron found in the liver and spleen of Rhodesian Africans are similar to those found in the South African Bantu.

The liver and spleen iron concentrations in severe Bantu siderosis are compared with these values in idiopathic haemochromatosis and though some features are alike there are also important differences. Further comparison between these two conditions will be found in Section IV.

SECTION III

PATHOLOGICAL EFFECTS OF IRON ON THE TISSUES

There is some controversy as to whether iron in the liver in Bantu siderosis plays any part in the production of fibrosis. Gillman et al.(1945), and Higginson et al.(1953) believed that the iron did not produce fibrosis while Strachan (1929), Gillman et al. (1957), Higginson (1958), Bothwell and Bradlow (1960), and Bothwell and Isaacson (1962), believed that it was at least partly responsible for the fibrosis,when present.

Strachan (1929), noted the frequency of tuberculosis in siderotic subjects and Gillman and Gillman (1945) suggested that the haemosiderin in the reticuloendothelial system lowered the body's resistance to tuberculosis. In this section the question whether or not large amounts of iron are harmful to the tissues is considered with reference to the findings in Rhodesian Africans.

Results

In the subjects examined in Section II (B), cirrhosis of the liver was present in 56 subjects of over 10 years of age, i.e. 10.4% The incidence in both sexes and types of cirrhosis are shown in Table XIV. Slight or moderate fatty change was found in 7 (54%) subjects with fine cirrhosis and 20 (47%) subjects with coarse cirrhosis. Severe fatty change was present in only one subject with fine cirrhosis and in none with coarse cirrhosis.

The relationship between liver iron concentration and fibrosis in this group is shown in Table XV. The term fibrosis as used in this context means all grades of fibrosis from moderate thickening of the portal tracts to cirrhosis, i.e. grades ++ to ++++ as defined in Section II. Slight thickening of the portal tracts, i.e. grade +, which is so common in the local African population with or without siderosis, has however been ignored.

Primary carcinoma of liver was present in 13 males (4.0%) and 4 females (1.9%). Only 4 (23.5%) of these cases had heavy deposits of iron in the liver and 9 (53.0%) had no stainable iron at all (See Table XVI).

Active tuberculosis was present in 30 subjects over the age of 10 years. In 16 of these the iron concentration in the liver was less than 1.00 mg/g while in 14 it was greater than 1.00 mg/g. That is, the incidence of active tuberculosis in subjects with a liver iron concentration of less than 1.00 mg/g was 4.1% and in subjects with a liver iron concentration of greater than 1.00 mg/g

TABLE XIV

CIRRHOSIS IN RHODESIAN AFRICANS, SEX INCIDENCE & TYPE

Sex	Total Number	Coarse Cirrhosis		Fine Cirrhosis		Both Types of Cirrhosis	
		Number	Percent of Total	Number	Percent of Total	Number	Percent of Total
Males	322	36	11.2	9	2.8	45	14.0
Females	213	7	3.3	4	1.9	11	5.2
Males & Females	535	43	8.0	13	2.4	56	10.4

TABLE XV

RELATIONSHIP BETWEEN CIRRHOSIS & MARKED PORTAL FIBROSIS AND LIVER IRON CONCENTRATION

(Total of 535 African Males & Females aged from 10-80 years)

STORAGE IRON CONCENTRATION mg/g wet weight	NUMBER OF CASES		PERCENTAGE WITH FIBROSIS
	TOTAL	NO. WITH FIBROSIS	
Less than 0.25	200	29	14.5
0.25 - 0.49	114	14	12.3
0.50 - 0.99	74	8	10.8
1.00 - 1.49	36	8	22.2
1.50 - 1.99	22	6	27.3
2.00 - 3.99	49	21	43.0
More than 4.00	40	26	65.0

TABLE XVI

CARCINOMA OF LIVER, CIRRHOSIS & SIDEROSIS

MALES over 10 years (322)			FEMALES over 10 years (213)		
Ref. No.	Type of Cirrhosis of Degree of Portal Fibrosis	Siderosis Total Score*	Ref. No.	Type of Cirrhosis of Degree of Portal Fibrosis	Siderosis Total Score*
BS/70/4	Coarse	0	BS/52/3	PNC	0
BS/67/5	Coarse	0	BS/49/6	PNC	8
BS/25/5	Coarse	7	BS/12/7	Portal Fibrosis +	0
BS/50/5	Coarse	1	BS/66/7	PNC	1
BS/52/6	Coarse	1			
BS/13/6	Coarse	9			
BS/76/6	Coarse	0			
BS/44/6	Coarse	0			
BS/35/6	Coarse	0			
BS/34/6	Portal Fibrosis ++	8			
BS/28/6	Coarse	1			
BS/9/6	0	0			
BS/5/6	Coarse	0			

* For explanation of this term see Section II, Page 34

was 9.5%. It was also noted that in these subjects with the higher liver iron concentrations the tuberculosis had taken a rapidly progressive form.

Between January 1963 and October 1967, among the autopsies performed on Africans in Harare Hospital mortuary, there were 20 subjects who proved to have peritonitis for which no cause could be found. Some details of these cases are given in Appendix IV. All but one of these had marked siderosis, 13 had cirrhosis and one severe bilharzial fibrosis. In six cases there was slight or moderate portal fibrosis.

DISCUSSION

It can be seen from Table XIV that the incidence of cirrhosis in African males over the age of 10 years (14%) is almost three times that in females (5%) and coarse cirrhosis is much more common than fine cirrhosis in both sexes. In Britain coarse cirrhosis is twice as common in females as males (Sherlock, 1963) while in Rhodesian Africans the position is reversed, coarse cirrhosis being about three and a half times as common in men as women. Cirrhosis in African males was three and a half times as common as in local European males in whom the incidence was 4%. These facts suggest that the cause of the cirrhosis in Rhodesian Africans is related especially to the habits or environment of men. A survey of the drinking habits of local Africans, details of which are contained in Section V, showed that men were much heavier consumers of African beer than women. It is possible that some toxic substance in the beer produces the cirrhosis though this substance is probably different from that which produces cirrhosis in European alcoholics as in the latter, fine cirrhosis and marked fatty change (at least in the early stages) are striking features (Sherlock, 1963). Also alcoholic hyaline, described by Mallory, (1911) was not seen in any of the African livers examined.

Siderosis is both more common and more severe in African men than women so this must be considered as a possible cause of cirrhosis. Table XV shows that with iron concentrations of less than 1.00 mg/g

wet weight the incidence of fibrosis in liver was not affected by iron concentration, but above that level the incidence of fibrosis increased steadily with the iron concentration. This very marked relationship between the incidence of liver fibrosis and high iron concentrations, which was also noted by South African workers (Bothwell and Bradlow, 1960; Isaacson et al., 1961; Bothwell and Isaacson, 1962), would tempt one to conclude that the iron was responsible for the fibrosis. On the other hand there were 21 livers with iron concentrations exceeding 3.00 mg/g wet weight in which there was little or no fibrosis. These cases are listed in Table XVII. Particularly striking is case BS/53/7 with a liver iron concentration of 14.13 mg/g wet weight (i.e. approximately 56 times the commonly accepted maximum normal value) whose portal areas showed minimal fibrous tissue reaction.

It will be shown in Section V that African beer is probably the main source of iron in siderotics. As was suggested earlier some toxic substance in the beer (other than iron) may produce the fibrosis and, while heavy drinkers could then be expected to have a higher incidence of liver fibrosis than more temperate individuals, they could also be expected to have heavier deposits of iron which would incidentally be ingested in the beer. Thus the iron may play no part in production of the fibrosis.

It has been shown also that absorption of iron is enhanced in subjects with cirrhosis (Conrad, Berman and Crosby 1962; Greenberg, Stromeyer, Kline, Curtis and Chalmers, 1964; Friedman, Schaefer and

TABLE XVII

SUBJECTS WITH LIVER STORAGE IRON CONCENTRATIONS EXCEEDING 3.00 mg/g WET WEIGHT AND LITTLE OR NO PORTAL FIBROSIS

MALES			FEMALES		
Ref. No.	Liver Iron Concentration (mg/g wet weight)	Portal Fibrosis	Ref. No.	Liver Iron Concentration (mg/g wet weight)	Portal Fibrosis
BS/23/5	3.82	0	BS/78/5	3.05	0
BS/22/5	3.09	0	BS/60/5	4.47	+
BS/15/5	8.82	+	BS/90/6	3.12	+
BS/43/5	3.43	+	BS/71/7	3.30	+
BS/30/5	4.08	0	BS/45/7	3.71	+
BS/29/5	4.81	0	BS/10/7	5.75	+
BS/27/5	5.89	+	BS/38/7	4.74	+
BS/5/5	4.67	+			
BS/1/5	7.20	+			
BS/74/6	4.20	+			
BS/56/6	4.18	+			
BS/8/6	4.07	0			
BS/22/7	3.82	0			
BS/53/7	14.13	+			

Schiff, 1966) so one would expect the average iron concentration in cirrhotic livers to be higher than in non-cirrhotic livers where the oral intake of iron was equal.

Another clinical observation which casts doubt on the ability of haemosiderin per se to cause fibrosis is that there is an absence of any real evidence of cirrhosis resulting from iron accumulation in transfusional siderosis (Cappell, 1950; Cappell, 1957; Oliver, 1959).

If evidence derived from human autopsy material regarding the fibrogenic potentiality of iron in liver is difficult to interpret, experimental evidence is almost equally so. Repeated animal experiments have shown that when large quantities of iron are fed in conjunction with a normal diet, though heavy deposits of iron have been found in the liver, in none of these has there been any fibrosis or cirrhosis (Polson, 1929; Hegsted, Finch and Kinney, 1952; Rather, 1956; MacDonald, 1960). Nissim (1953) however, using mice, rats, rabbits and guinea-pigs fed on a normal diet and given gross excess of iron by an intravenous route produced atrophy and degeneration of hepatic cells. There was, however, no fibrosis or nodular hyperplasia of the parenchyma. Goldberg and Smith (1960) fed rats with a diet designed to produce cirrhosis and administered large amounts of iron. As a result of their experiment they concluded "that a liver loaded with excessive quantities of iron is vulnerable to the action of toxic agents or deficient diets to a far greater degree than is the case with the normal liver". Like the other workers however, they point out that "severe iron overload per se does not appear to

induce tissue damage". Unlike Goldberg and Smith, other workers (MacDonald and Pechet, 1965, and Dunn 1967) also using rats on a similar type of diet could not demonstrate that large amounts of iron in the liver increased the rate of fibrosis. Witzleben and Chaffey (1962), experimenting with mice, demonstrated that storage iron enhanced the effects of certain hepatotoxins while it had no effect on others. Relating their results to iron storage diseases they believed that they had demonstrated "that storage iron is not inert in terms of an effect on the tissues in which it is stored".

As regards tissues other than liver, no evidence was found during this study that heavy iron deposits caused fibrosis. The pancreas, for example, in cases D 41 and D 42 (see Appendix V) contained 5.43 and 3.01 mg/g iron respectively and three pancreases in Appendix XI had iron concentrations of 3.00 mg/g or more yet none showed any evidence of fibrosis.

It has been suggested, as far as the liver is concerned, that probably the iron though not fibrogenic on its own may potentiate the fibrogenic action of some toxic substance ingested in the diet (Isaacson et al, 1961). This view agrees with that of Goldberg and Smith, and Witzleben and Chaffey quoted above, and at the present time it appears to be probably correct, but it is felt that the bulk of the evidence points to the fact that the pathogenicity of the iron is of a very low grade.

In this series there was no relationship between primary carcinoma of liver and siderosis, a fact which has also been noted

in idiopathic haemochromatosis (Sheldon, 1935).

The higher incidence of tuberculosis and its rapid progress in Rhodesian Africans with severe siderosis seem to confirm the view of Gillman and Gillman (1945) that the iron might have lowered the body's resistance to the disease. Other explanations for the activity of the disease are however possible. Firstly, heavy drinkers of African beer would be expected to have severe siderosis and the money spent on beer would not be available for the purchase of food, therefore some degree of malnutrition would probably result. This last would make the body more vulnerable to tuberculosis. Secondly, heavy drinkers are frequently careless of their health and would not seek medical attention until the disease was at an advanced stage. The mortality rate would be expected to be higher in this group than more abstemious non-drinking tuberculotics and therefore more would be seen at autopsy.

These points do not discount the possibility of siderosis potentiating the pathogenicity of tuberculosis but it is felt that no very convincing evidence has yet been produced showing that it does.

Another fact, noted by the writer which suggests that excessive iron might lower tissue resistance to disease, was the occasional occurrence of unexplained peritonitis in subjects with severe siderosis. Now it is well known that unexplained peritonitis occurs from time to time in subjects with cirrhosis (Conn, 1964; Kerr, Pearson and Read, 1963; Matz and Jurmann, 1966) but it is

considered that the role of siderosis is significant in the 20 cases being discussed in that,1) only one case of such peritonitis has been seen among the many cirrhotics examined without siderosis,and 2) six of the twenty cases had siderosis but no cirrhosis. A similar type of peritonitis has been reported in idiopathic haemochromatosis (Jones, 1962) and in idiopathic haemosiderosis (Ploem et al.,1965) but it has not been reported in any of the many South African studies on Bantu siderosis.

Conclusion:

The circumstantial evidence discussed suggests that the gross overload of iron in severe Bantu siderosis might be mildly harmful to the tissues and lower their resistance to infection.

SUMMARY

In Rhodesian Africans cirrhosis is about three times as common in men as women, and coarse cirrhosis is more common than fine cirrhosis.

The incidence of liver fibrosis increases as the average liver iron concentration rises at concentrations of more than 1.00 mg/g. Though the obvious deduction is that the iron is responsible for the fibrosis, arguments are presented to show that this relationship of liver iron concentration to fibrosis in Rhodesian Africans may be fortuitous. Experimental evidence of the effect of iron on animal livers is reviewed. It is concluded that possibly storage iron in the liver potentiates the fibrogenic action of other toxic substances but in this respect its activity is of a very low grade. Active tuberculosis is more common in severe siderotics. While it is conceeded that there might be some causal relationship between them it is also shown that factors other than iron may be responsible.

A number of cases of unexplained peritonitis have been seen in subjects with severe siderosis. It is thought that these may have resulted from lowered tissue resistance to infection caused by the massive iron stores.

SECTION IV

DISTRIBUTION OF IRON IN THE BODY OF SUBJECTS WITH BANTU SIDEROSIS

It has been shown by South African workers that in the great majority of subjects with Bantu siderosis iron deposits are confined to the liver, reticuloendothelial system and small bowel mucosa (Higginson et al., 1953; Wainwright, 1957). There are however a small proportion of cases in which there are iron deposits in many glandular tissues. Subjects with this distribution usually have an associated fine cirrhosis of liver (Bradlow et al.,1961; Isaacson et al.,1961).

Material & Methods

The body iron distribution in this study was investigated by selecting 31 male and 11 female African subjects at autopay who had heavy deposits of iron in liver and spleen, some, though not all of these, were selected from cases included in Section II (B). Blocks were taken from a large number of organs, fixed and stained as described in Section II. In addition, chemical estimations of the iron concentration were performed on the livers and spleens, and in two cases on the pancreas.

Results

The results are detailed in Appendix V.

The patterns of iron distribution fell roughly into three categories:-

1) Subjects without cirrhosis - in these, heavy iron deposits were found in the liver, reticuloendothelial system, and small bowel mucosa.

2) Subjects with fine cirrhosis, - in addition to heavy deposits in the above tissues, moderate to heavy deposits were present in the pancreas, pituitary, thyroid, adrenal and salivary glands, the choroid plexus, the heart, gastric mucosa and a number of other organs.

3) Subjects with coarse cirrhosis - the distribution was as in the second category, but epithelial deposits were only scanty or moderate.

There was one exception to this rule, case D.2. This was a man aged 60 years without cirrhosis but with a very high concentration of iron in the liver, viz. 13.4 mg/g wet weight, in whom the distribution was the same as found in subjects with fine cirrhosis.

The distribution of iron in individual tissues, when these were involved, will now be described.

LIVER: The iron distribution in this organ was described in Section II. Suffice it to say again that in cases uncomplicated by infection iron deposits first appeared in the hepatic parenchymal cells at the periphery of the lobule. When the organ was more severely involved all of the parenchymal cells contained deposits

of iron as did the Kupffer cells and portal areas. The haemosiderin in the portal areas appeared usually to be in macrophages but especially when deposits were heavy, lay free in the interstitial tissue. When fine cirrhosis was present variable deposits were seen in the bile duct epithelium.

SPLEEN: In this organ also the iron deposition was described in Section II. Thus, in brief, haemosiderin deposits were found in the pulp macrophages and in more severe cases, large extracellular masses were seen. Variable deposits were found in the capsule and trabeculae, especially in the more severe cases. The lymphoid follicles contained little or no iron.

PANCREAS: In cases where this organ was heavily involved it was dark brown in colour on naked eye inspection. Microscopically haemosiderin granules were seen in both acinar and islet cells. Deposits in the interstitial tissue were very variable being sometimes heavier than in the epithelial cells as in case D.41 and sometimes relatively slight as in D.42. Scanty deposits were sometimes found in the epithelium of the ducts. In 11 cases there was some increase in the fibrous tissue of the pancreas. This was in no case very marked and in all there was an associated cirrhosis of liver.

SALIVARY GLANDS: The submandibular glands were examined in all cases. Deposits of haemosiderin were conspicuous in the serous secreting cells and duct epithelium. Deposits in the connective tissue were variable but never very heavy.

PITUITARY: Sometimes this organ looked brownish to the naked eye. Microscopically, the anterior pituitary was seen to be more heavily involved than the posterior pituitary and the basophil cells rather more heavily than the oxyphil cells. Deposits were also found in the interstitial tissue. In the posterior pituitary iron deposition was patchy and was in the form of fine or coarse granules most of which appeared to be extracellular.

THYROID: When heavily involved this gland had, on gross examination a definite dark brown colour. Iron deposits were present both in the follicular cells and interstitial tissue.

ADRENAL: Grossly, the appearance was normal in all cases. Microscopically the gland sometimes contained fairly heavy deposits of haemosiderin which were largely confined to the zona glomerulosa though in a few cases a number of fine granules were seen in the cells of the zona fasciculata. Interstitial deposits were variable being in some cases quite heavy and were usually most prominent in the medulla.

CHOROID PLEXUS: Frequently on naked eye inspection this was dark brown in colour. Microscopically, haemosiderin was seen in the epithelial cells and in the stroma. In some cases the epithelial involvement was conspicuously patchy, some cells being heavily laden with haemosiderin and others containing little or none.

HEART: In only one case were heavy iron deposits seen in the myocardial fibres. In the remainder of those in which the heart was involved, the deposits were scanty to moderate and were very

patchy in distribution.

KIDNEYS: Iron deposits were seen in the distal convoluted tubules, and in a few cases in the loops of Henle, the glomerular tufts and interstitial tissue. Iron was never seen in the proximal convoluted tubules, so the grading given for kidney in Appendix V refers to the degree of involvement of the distal tubules only and not to the whole organ. Deposits in the glomeruli were always very scanty and took the form of an isolated granule or two apparently in the walls of the glomerular capillaries.

STOMACH: Deposits of iron in the gastric mucosa were rarely heavy and affected the epithelial cells nearest the muscularis mucosa to the greatest extent. Patchy deposits were also present in macrophages in the lamina propria of the mucosa and in the submucosa.

SMALL BOWEL: Haemosiderin deposits in small bowel were heaviest in the duodenum and decreased progressively to the terminal ileum. In all post mortem specimens the epithelium covering the villi was absent, so no opinion as to whether or not it contained iron could be formed. However biopsy specimens of duodenal mucosa, taken by a Crosby capsule from siderotic patients, showed that the epithelial cells contained little or no stainable iron. In marked contrast were the numerous coarse haemosiderin granules, mostly contained in macrophages, in the stroma of the villi. Deposits in Brunner's glands of the duodenum were uncommon and were never more than scanty.

COLON: Scattered granules of haemosiderin were seen in the lamina propria of the colonic mucosa in severe cases.

LYMPH NODES: Haemosiderin deposits were heaviest in the lymph nodes round the porta hepatis, and at the root of the small bowel mesentery. In these nodes deposits were usually massive but, even in these, the germinal centres were spared. Other lymph nodes throughout the body were involved to a greater or lesser extent in severe siderosis. Especially notable in this respect were the nodes along the internal mammary vessels and in the hylar regions of the lungs.

BONE MARROW: In almost all cases heavy iron deposits were present in the bone marrow, imparting to it a dark brown colour. Microscopically, coarse and fine granules of haemosiderin were seen both intracellularly and lying free. In many cases heavy deposits completely obscured cellular detail but it was felt that most iron deposits were in reticulum cells.

OTHER TISSUES: Iron deposits in prostate, testis, ovary and uterus were rarely more than slight and were usually confined to interstitial tissue. In a few cases fine granules were found in the glandular cells of the prostate. Deposits in the uterus were in the stroma of the basal layer of endometrium. The brain rarely contained any stainable iron but very occasionally a few coarse granules of haemosiderin were seen in the region of the lentiform nucleus. These granules were usually in the proximity of blood vessels and in most cases were in the cytoplasm of macrophage-like cells.

DISCUSSION

a) Distribution of Iron in the Liver: The periportal hepatic cells are the first to contain stainable iron, and even in the most severe cases are usually more heavily involved than the centrilobular cells. It is suggested that as the periportal cells are first to come in contact with serum, rich in iron absorbed from the bowel seconds before, it would seem reasonable to suppose that they would take up any available iron, leaving none for the centrilobular cells. When iron absorption was massive or when the peripheral cells became loaded, and possibly therefore less avid for iron, some iron would become available for the centrilobular cells.

It might be thought that this explanation for the heavy iron deposits in the peripheral cells of the liver lobule is inadequate, especially as it has also been reported in transfusional siderosis (Oliver, 1959). In this condition, the excessive iron deposits are derived from the haemoglobin of transfused red cells (Cappell, Hutchison and Jowett, 1957) and not absorbed from the bowel, so one would not expect the portal blood to be particularly rich in iron. However, in transfusional siderosis the iron-laden Kupffer cells, which are such a conspicuous feature of this condition, "are found most frequently at the periphery of the liver lobule in the portal spaces" (Oliver, 1959). Presumably the iron diffuses out of the reticuloendothelial cells into the adjacent liver parenchymal cells. A similar phenomenon occurs in experimental siderosis in which

the hepatic cells most affected are those nearest the masses of reticuloendothelial cells loaded with iron, (Polson, 1929; Cappell, 1930; Nissim, 1953) i.e. chiefly the cells at the periphery, and to a lesser extent at the centre of the lobules, but avoiding the midzonal cells.

The iron deposits in the Kupffer cells are discussed later when considering the reticuloendothelial system. The deposits in the portal areas are probably the result of migration of iron-laden Kupffer cells from the sinusoids (Cappell, 1929; Cappell, 1930; Cappell, 1957; Goldberg and Smith, 1960).

b) Comparison of Iron Distribution in Siderotic Africans with Cirrhosis and Idiopathic Haemochromatosis

In uncomplicated Bantu siderosis iron distribution in the body is quite different from that found in idiopathic haemochromatosis, because in the former, epithelial deposits are rare. On the other hand, subjects with siderosis and cirrhosis of the liver have a distribution similar in most respects to the findings of Sheldon (1935) in idiopathic haemochromatosis. Nevertheless there are differences even in these. The relatively high concentrations in spleen in Bantu siderosis compared with idiopathic haemochromatosis was noted in Section II (B). In Bantu siderosis also, iron deposition in bone marrow and in the villi of the small bowel is very much heavier than described by Sheldon in idiopathic haemochromatosis.

i) Iron Deposits in Reticuloendothelial Systems. The very much heavier deposition of iron in the reticuloendothelial system in Bantu siderosis than in idiopathic haemochromatosis is such a conspicuous

feature as to be worthy of comment. It has been shown that reticuloendothelial cells take up iron bound to transferrin only to a very limited extent (Huff, Elmlinger, Garcia, Oda, Cackrell and Lawrence 1951; Elmlinger, Huff, Tobias and Lawrence, 1953), so presumably the iron found in the reticuloendothelial cells in Bantu siderosis is derived from effete red cells. The heavy iron deposits found in these cells therefore must presumably be due either, to abnormal red cell destruction, or failure to release the iron derived from normal red cell destruction. Furthermore, to account for the difference between the two conditions the responsible factor must affect exclusively, or at least predominantly, subjects with Bantu siderosis.

Red cell fragility and life span have been shown to be normal, and there is no evidence of abnormal blood destruction in patients with idiopathic haemochromatosis (Howard and Stevens, 1917; Pollycove and Mortimer, 1961). Strachan (1929) in his work on Bantu siderosis reported that in Africans "there was a slight but definite increase in the fragility of red cells as compared with Europeans especially in subjects over 30 years of age". Gillman, Lamont, Hathorn and Canham (1957) suggested that infection and haemolysis might be responsible for the presence of the large amounts of iron in the reticuloendothelial system of Bantu siderotics.

It will be shown in Section VII that there is no evidence of increased red cell fragility or shortened erythrocyte life span in healthy Rhodesian Africans. The alternative explanation for the heavy iron deposits in the reticuloendothelial system, viz.

failure to release iron derived from the normal breakdown of erythrocytes has then to be considered. It has been shown that in various inflammatory processes the reticuloendothelial system fails to release such iron into the blood stream at a normal rate, producing a fall in serum iron (Noyes, Bothwell and Finch, 1960). This has been demonstrated clinically (Laurell, 1947; Cartwright and Wintrobe 1949) and confirmed experimentally on dogs (Cartwright, Lauriston, Jones, Merril, and Wintrobe 1946; Freireich, Miller, Emerson, and Ross, 1957). Chronic renal disease is also said to lower the serum iron (Laurell, 1947; Rath and Finch, 1949) possibly due to retention of iron by the reticuloendothelial system. Personal clinical and autopsy experience of the writer suggests that both infection and chronic renal disease, especially chronic pyelonephritis, are probably more common in Africans than Europeans. This prevalence of infection in Africans is attributed to latent or overt malnutrition, and of chronic pyelonephritis, to urinary bilharzia with its associated cystitis.

Some suggestive evidence in support of the idea that infection is the cause of the heavy iron deposits in the reticuloendothelial system is provided by a number of the cases considered in Section II (B) Appendix III. Three of the Europeans mentioned in Table IV with infection and renal disease at time of death had heavy deposits of iron in the Kupffer cells and spleen. Three others also had heavy deposits in these sites but no evidence of infection was seen at autopsy. This does not however exclude the possibility that these subjects had frequent infections during life as little medical history

was available.

Reference to Appendix III also shows, that in African subjects aged between 1 year and 19 years, 50 out of 76 who died of infection or chronic renal disease, i.e. 66%, had stainable iron in the spleen and also often in the Kupffer cells while only 15 out of 58, i.e. 26% who died due to some other cause had stainable iron in these sites. An analysis of this sort in older age groups would be difficult to interpret because of the heavy iron deposits and high incidence of stainable iron in liver and spleen of all subjects. Inspection of the appendix however shows that many people who died of infective conditions and renal failure, and who had only minor degrees of siderosis, exhibited an iron deposition which was predominantly in the Kupffer cells and spleen.

These findings appear to confirm the work of Schairer and Rechenberger (1948) and Morgan and Walters (1963) which showed that infections of various kinds produce an increase in the iron content of the spleen and Kupffer cells of the liver, but not in the liver parenchymal cells.

It would be unrealistic to suppose that no subject with idiopathic haemochromatosis ever suffered from infection or renal disease, so one would occasionally expect high iron concentrations in the reticuloendothelial system in these people. This has in fact been shown to occur, and Bernoulli (1910) and Roth (1915) have reported cases in which the iron concentrations in spleen exceeded 2.0 g/100 g dry weight.

ii) Iron Deposits in the Mucosa of the Bowel. The heavy deposits of iron in the villi of the small bowel in Bantu siderosis may result from rapid absorption of very large amounts of ingested iron. The source of this iron will be demonstrated in Section V. On the other hand, in idiopathic haemochromatosis, the excessive tissue iron is thought to result from an inborn error of metabolism, causing abnormal absorption of a normal amount of dietary iron (Sheldon 1935). Presumably in the latter case the rate of absorption would be slower, as massive amounts of iron would not be available in the lumen of the gut. Accumulation of iron in the mucosa is therefore less likely because there would be more time for absorbed iron to be transported from this site.

Wainwright (1957) observed that iron deposits in the duodenum were greater below the opening of the common bile duct that above it. This finding was not confirmed in the present series though it was sought both macroscopically and microscopically.

It is felt that the iron deposits in the bowel demonstrate that there is a fundamental difference between Bantu siderosis and idiopathic haemochromatosis. MacDonald, however, believes that idiopathic haemochromatosis is merely a variant of dietary siderosis in subjects with nutritional (alcoholic) cirrhosis (MacDonald 1961, 1963). The recent discovery by Davis, Luke and Deller (1966) that patients with idiopathic haemochromatosis have a deficiency of an iron-binding protein, present in the gastric secretions of normal people, makes MacDonald's theory less likely. Also there have been an ever

increasing number of reports of the familial incidence of haemochromatosis, and of abnormalities in iron metabolism of relatives of patients with haemochromatosis, not all of whom were alcoholics (Williams, Scheuer, and Sherlock, 1962; Powel, 1965; Ploem, Otten, Huizinga and Verloop, 1965; Beleerzak, Westerman, Lee and Doyle, 1966; Turner, 1966).

iii) Iron Deposits in other Tissues The only other tissue which showed much difference in iron deposition between Bantu siderotics with fine cirrhosis and idiopathic haemochromatosis was the heart. In this series heavy deposits of iron in the heart were rare while in idiopathic haemochromatosis they are common (Sheldon, 1935). This is probably due to the fact that the iron overload in Bantu siderosis is usually less than in idiopathic haemochromatosis (see discussion on liver iron concentrations).

c) The Effect of Cirrhosis on Distribution of Iron in the Body in Bantu Siderosis

Possible explanations as to why cirrhosis, especially fine cirrhosi should produce widespread epithelial deposits of iron,usually not seen i subjects with Bantu siderosis without cirrhosis,will now be considered.

i) High Iron Concentrations in Cirrhotics While it is true that many of the subjects examined with fine cirrhosis had very high liver iron concentrations,it is clear that epithelial deposits found in subjects with cirrhosis are not dependent on high liver iron concentrations alone. This is shown in Appendix V by two typical cases: firstly case D.22, a man of 50 years with fine cirrhosis, whose liver iron concentration was only 1.8 mg/g wet weight and in whose body widespread cipthelial deposits of iron were seen; and secondly, case D.36, a man of 60 years, without cirrhosis whose liver iron concentration was 12.1 mg/g wet weight, almost seven times as much as the previous case, with no epithelial deposits.

ii) Mechanical Shunting of Blood The presence of cirrhosis of the liver obstructs the portal circulation and causes much of the blood flow in the portal vessels to by-pass the liver by way of numerous collateral channels (McIndoe, 1928). As the pancreas is one of the epithelial tissues most heavily involved,and is in such close proximity to the liver, it would seem reasonable to suppose that some of this blood, diverted from the liver and rich with iron absorbed

from the intestine, would flow through the pancreas. If mechanical shunting of blood alone were responsible for the epithelial deposits, one would expect that part of the pancreas which first came in contact with the iron-rich diverted blood, i.e. the head, would contain heavier deposits than those parts to be perfused with blood which had already flowed through the head, i.e. the tail, very much as happens with the periportal and centrilobular hepatic cells in the liver.

In Section VII it will be shown that there is no significant difference between the iron concentration in the head and tail of the pancreas in subjects with or without cirrhosis. Because of this, it is felt that the mechanical shunting of blood in cirrhosis cannot explain the widespread epithelial deposits of iron found in cirrhotics.

iii) Degree of Saturation of Transferrin In iron storage diseases in which epithelial deposits of iron are extensive, the percentage saturation of transferrin is high; e.g. in idiopathic haemochromatosis (Finch and Finch, 1955) and in transfusional siderosis (Cappell, 1958). Epithelial deposits of iron are also common in pernicious anaemia, and in this condition too percentage saturation of transferrin is high (Laurell, 1947).

It has been suggested by Bothwell (1964) that the different patterns of iron distribution found in Bantu siderosis may be connected with the percentage saturation of circulating transferrin. Transferrin levels are lowered in cirrhosis (Laurell, 1947; MacDonald, 1964) which means that if serum iron levels were normal in these cases the percentage saturation would be increased. Also this increase in percentage saturation would be accentuated if the serum iron were

raised as has been reported in some African subjects (Gerritsen and Walker, 1953). It has been reported that when transferrin saturation was greater than 60% there was a marked increase in the uptake of iron by liver slices (Jandl, Inman, Simmons and Allen, 1959). It would seem to be possible that epithelial cells of other tissues such as pancreas, thyroid, salivary glands, etc., might also have an increased uptake when the percentage saturation was greater than 60. Experimental evidence in support of this idea will be presented in Section VII. Also it will be shown in Section VI, that in a number of subjects with Bantu siderosis who had a high percentage saturation of transferrin shortly before death, there were widespread deposits of iron in epithelial tissues found at autopsy. On the other hand, a few with normal percentage saturation values did not show these epithelial deposits.

All of these points appear to indicate that the epithelial deposits of iron depend on high percentage saturation of transferrin. In such circumstances, probably some of the iron is less firmly bound to transferrin as suggested by Katz and Jandl (1964), and enters the epithelial cells in a non-specific fashion, very much as these authors believed it did in mature red cells in their experiments. Wheby and Jones (1962) have shown that in rats, when transferrin is completely, or almost completely saturated, the liver uptake of ^{59}Fe absorbed from the gut was 90% compared with an uptake of 10% in animals with normal transferrin saturation. It is felt that probably iron deposits found in the livers of people with normal percentage saturation of

transferrin are produced in a similar fashion. This could come about in two ways, either the portal blood transferrin might reach a high degree of saturation if large quantities of iron were being rapidly absorbed, or the binding of normal amounts of absorbed iron to transferrin, though rapid, is not instantaneous and is therefore incomplete by the time the blood reaches the liver. It is probable that, even in people with normal iron stores, iron from the portal blood enters the liver cells but that as this iron is required for erythropoiesis, it is mobilized again and transported to the bone marrow so would not reach the concentration in the liver cells at which haemosiderin is produced.

Transferrin is synthesised in the liver (Bothwell and Finch, 1962) and this would explain its low levels in cirrhosis in common with other proteins synthesised in the liver. It is therefore probably not the cirrhosis itself but the resulting lowering of the transferrin and increase in its percentage saturation with iron that determines the widespread parenchymal deposits. In subjects without cirrhosis and with gross iron overload, as in case D.2, the high percentage saturation of transferrin may not be due to lowering of the blood transferrin, but to raising of the serum iron due to the inability of the sites of iron storage to control their stores, thus releasing iron into the serum in large amounts. This could also explain the high percentage transferrin saturation found in transfusional siderosis, without cirrhosis, where widespread epithelial deposits are also found (Cappell, 1930; Cappell, 1957; Cappell, 1958; Oliver, 1959).

iv) Transferrin Variants There have been 15 types of transferrin discovered with different electrophoretic mobilities, and these appear to be genetically determined (Bearn and Parker, 1964). One of these variants (D_1) is commonly found in American Negroes most of whom originated in West Africa. It is possible that other variants may be discovered in Southern Africa, and that these variants may favour deposition of iron in epithelial tissues. Up till now however, no physiological differences between the transferrin variants have been found (Bearn and Parker, 1964).

SUMMARY & CONCLUSIONS

In all subjects with Bantu siderosis iron deposits are found in the liver, reticuloendothelial system and small bowel mucosa. In addition if fine cirrhosis is present, deposits are found in many epithelial tissues. Occasional cases are seen with these widespread epithelial deposits in absence of liver cirrhosis.

The sites and degree of deposition of iron in various tissues are described. The distribution of iron in the liver is discussed in some detail, and the fact that the hepatic cells at the periphery of the lobules are first to contain stainable iron, is attributed to the fact that these cells are first to come into contact with iron-rich portal blood. The similarities between iron distribution in Bantu siderotics with fine cirrhosis and that in idiopathic haemochromatosis is noted but the differences are stressed viz. the heavier iron deposits in reticuloendothelial system and small bowel mucosa in Bantu siderosis. The former is attributed to the prevalence of infection and renal disease in Africans and the latter to the more rapid absorption of iron, which is present in the bowel in massive amounts in Africans. It is concluded that Bantu siderosis and idiopathic haemochromatosis are fundamentally different conditions.

Causes for the different iron distribution in the body of Bantu siderotics with and without cirrhosis are then considered. The high iron concentrations in the livers of cirrhotics, and mechanical shunting of portal blood to by-pass the liver, are considered as an explanation

only to be rejected. Evidence for the high degree of transferrin saturation as a cause for widespread epithelial deposits of iron is then considered, and it is concluded that this is the most probable explanation, though it is realised that at present it is impossible to exclude a variant of transferrin occurring in Africans in Southern Africa as a possible cause.

A SELECTION OF PHOTOMICROGRAPHS OF TISSUES FROM CASES WHOSE DETAILS CAN BE FOUND IN APPENDICES III & V, VOLUME II

Plates I and II are from a case referred to on Page 62. The remaining plates are from cases referred to on Page 67.

All slides were stained with Perl's stain except that seen in Plate II which was stained with H. & E.

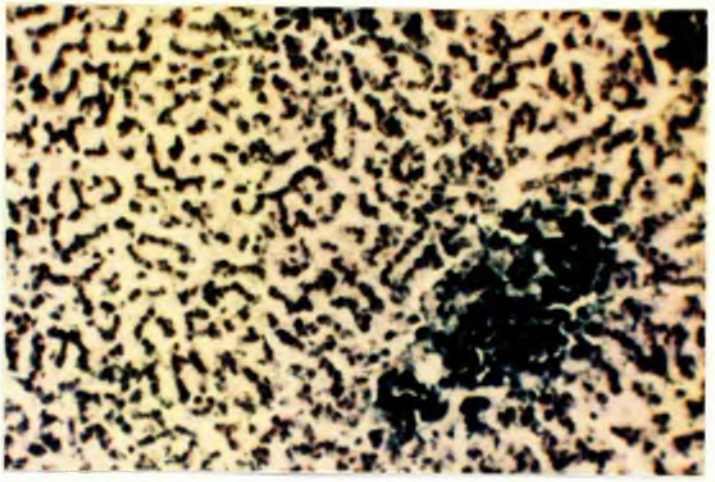


Plate I

Case BS/53/7, LIVER x 125

This shows a liver with heavy iron deposition in hepatic cells, Kupffer cells and portal areas.

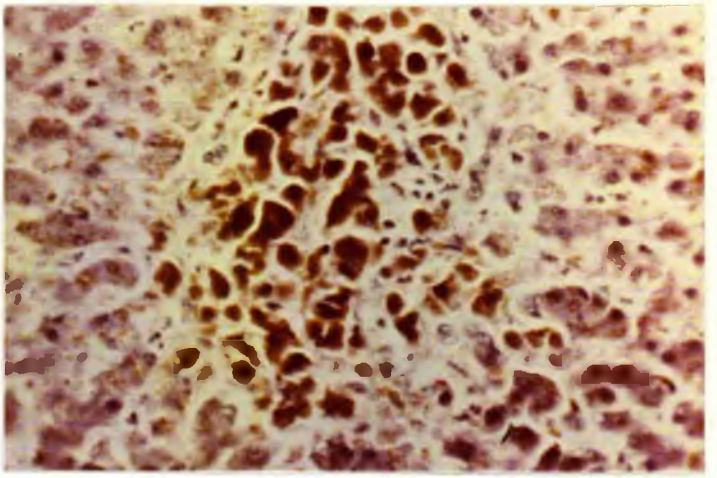


Plate II

Case BS/53/7, LIVER x 320

Massive iron deposition is seen in a portal area but there is minimal fibrous tissue reaction.

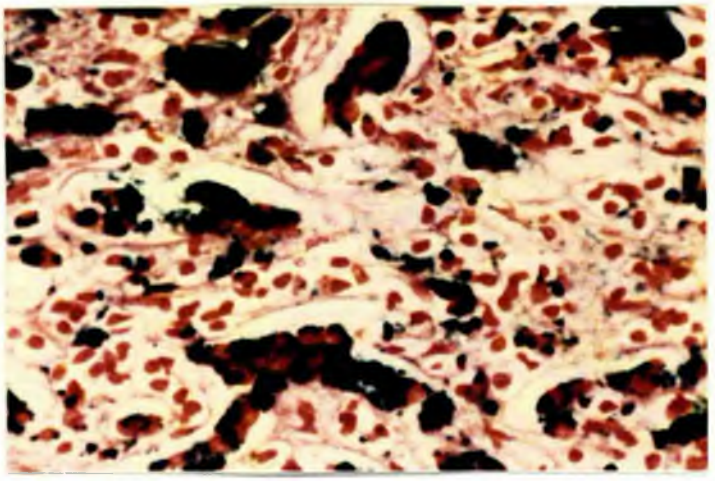


Plate III

Case D 6, LIVER x 500

Here a portal area is shown which contains bile ducts whose epithelium contains heavy iron deposits. This is normally found only in subjects with fine cirrhosis.

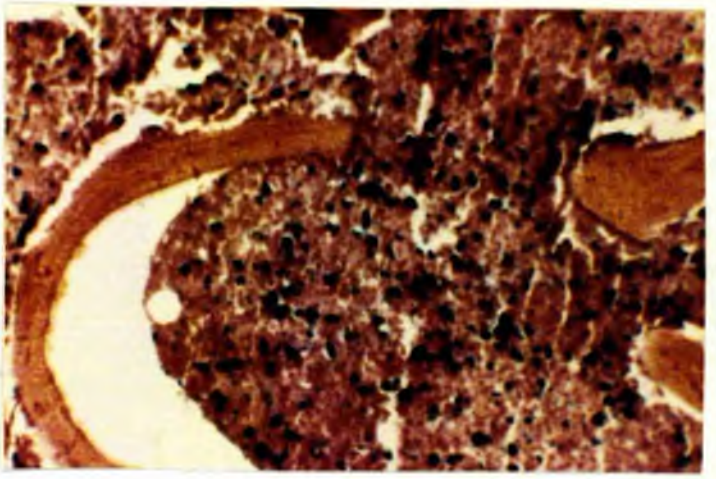


Plate IV

Case D 33, BONE MARROW x 125

This shows the heavy iron deposits so commonly found in this site in Bantu siderosis.

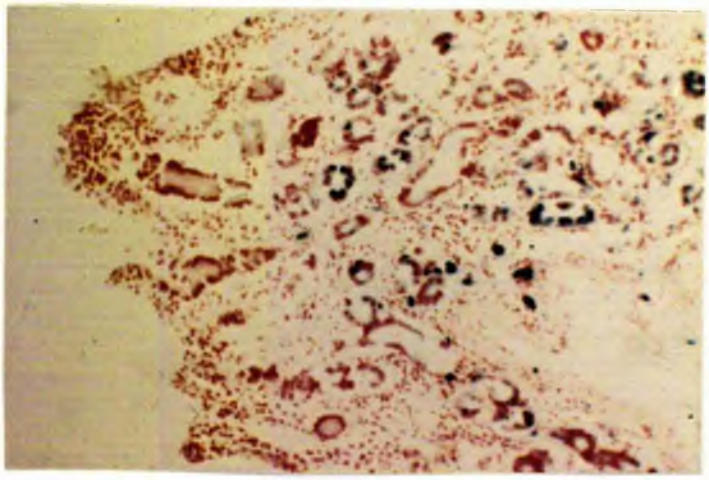


Plate V

Case D 16, GASTRIC MUCOSA x 125

Heavy iron deposits are seen in the glandular epithelial cells farthest from the lumen of the stomach.

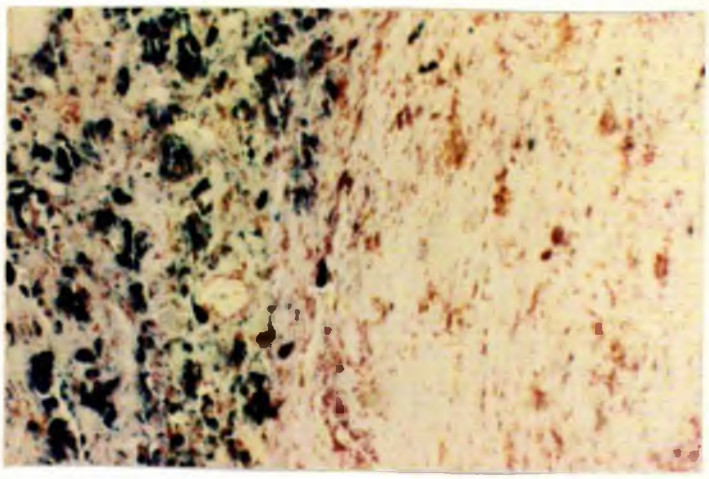


Plate VI

Case D 8, PITUITARY x 125

Deposits of iron are heavy in the glandular part of the pituitary but almost absent from the posterior part

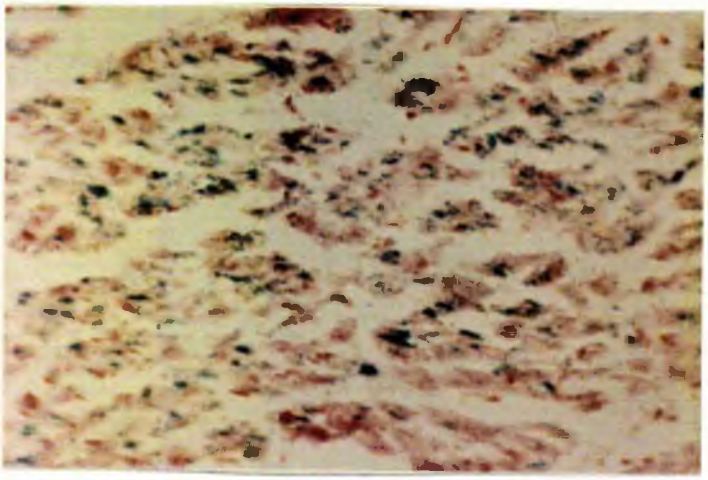


Plate VII

Case D 41, HEART x 320

This shows the heart which had the heaviest deposits of iron in this series. Deposits in the heart were usually scanty.

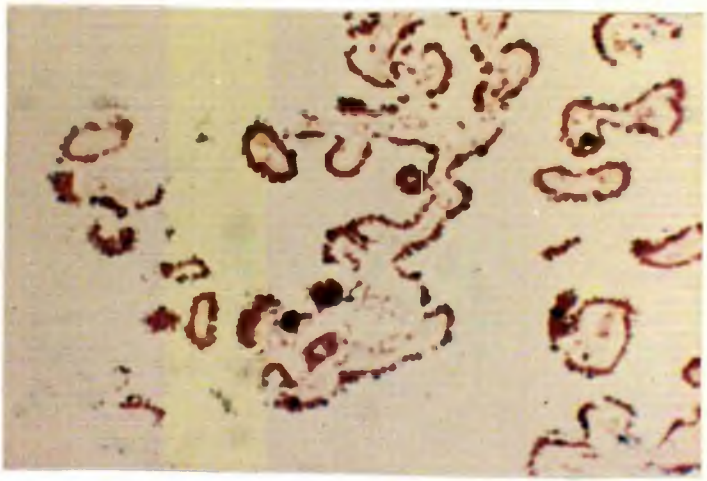


Plate VIII

Case D 33, CHOROID PLEXUS x 125

The patchy distribution of iron in th lining cells is well shown. There are also coarse clumps of haemosideri in the stroma.

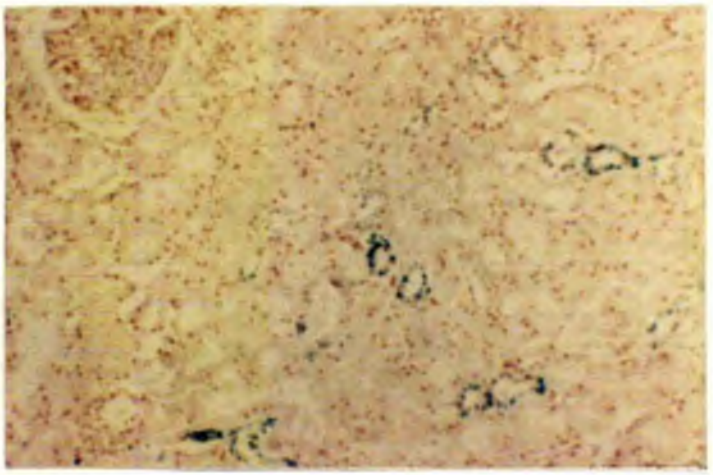


Plate IX

Case D 41, KIDNEY x 125

There are fairly heavy deposits of haemosiderin in the epithelium of the distal convoluted tubules but virtually none is present in the proximal tubules.

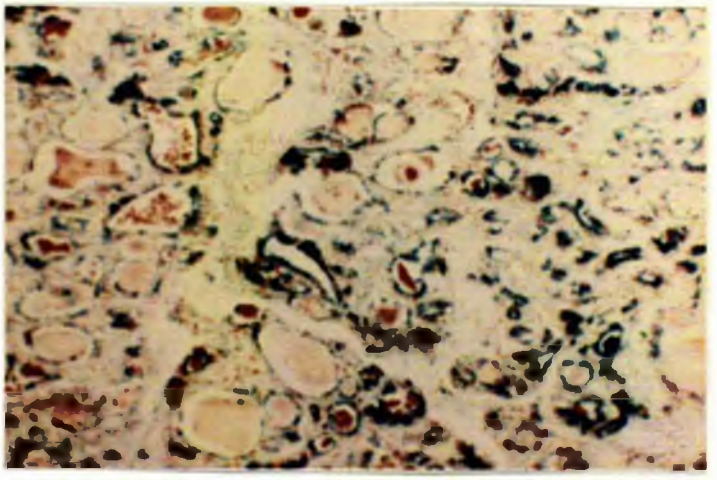


Plate X

Case D 2, THYROID x 125

This shows the heavy iron deposits in the follicular epithelium.

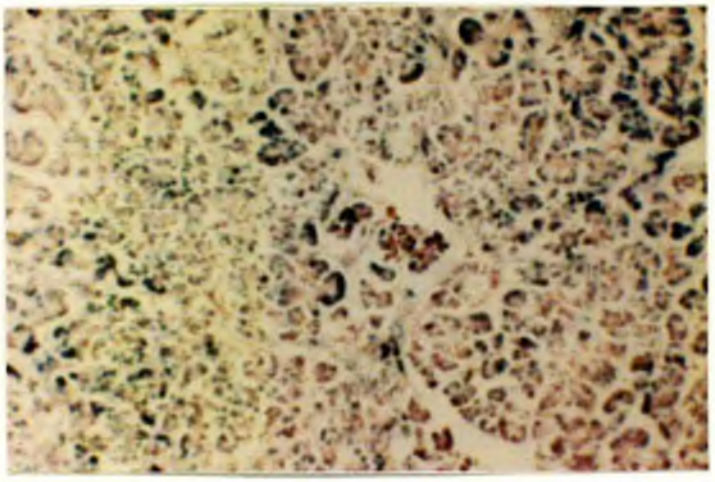


Plate XI

Case D 2, PANCREAS x 125

The heavy iron deposition in the acinar cells is shown.

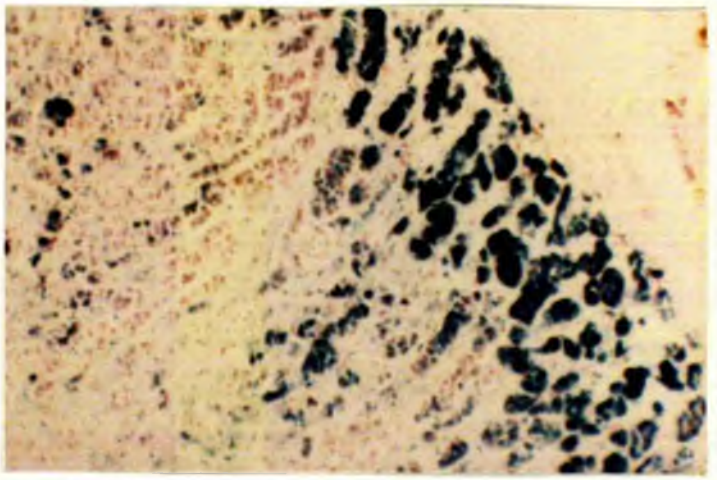


Plate XII

Case D 8, ADRENAL x 125

Iron deposits are extremely heavy in the cells of the zona glomerulosa but scanty in the zona fasciculata.

SECTION V

IRON CONTENT OF THE AFRICAN DIET

It has been shown in South Africa that African food after cooking contains large amounts of iron, which appears to be derived from the vessels in which the food was cooked (Walker and Arvidsson, 1950, 1953). Walker and Arvidsson also showed that "kaffir" beer (called in this study African beer) was also rich in iron, a fact which was later confirmed by Bothwell et al. (1964). These authors believed that this high intake of dietary iron by Africans was responsible for the prevalance of siderosis in the South African Bantu.

Carr (1956) analysed a number of cooked African foods in Rhodesia. Among the constituents measured was iron but two important sources of iron, viz. porridge and beer, were omitted from his study. It was decided therefore to carry out a further investigation into the iron content of the Rhodesian African diet to confirm Carr's findings and to include porridge and beer in the study. It was also decided to investigate the drinking habits of a representative section of the African population.

Material and Methods

Food: Thirty eight samples of cooked African food were obtained from various sources viz. a village near Salisbury, the homes of a number of African hospital staff, and the kitchen of Harare Hospital. The foods chosen were maize meal porridge, locally called "sadza", and a variety of green vegetables and beans,

called "relish" by local Africans. These foods form the bulk of the diet of both rural and urban Rhodesian Africans. A small percentage of Africans, including those who are servants in European houses and African professional and business men, tend to eat a more European type of diet.

Food samples were weighed, and dried in an oven, in aluminium containers, at 100°C to constant weight. They were then re-weighed to estimate the water content. Approximately one gramme of the dried food was weighed to the nearest milligramme, placed in a 300 ml Kjeldahl flask with antibumping granules and 5 ml of the nitric/ sulphuric acid mixture as used in the tissue digestions in Section II added. The mixture was boiled until a clear, more or less colourless fluid was produced. During the boiling process it was necessary to add small amounts of nitric acid to the mixture to remove charring as it occurred. 0.5 ml of 100 vols. hydrogen peroxide was then added and the liquid boiled for a further 10 minutes to remove any residual nitrites. This was then cooled and thioglycollic acid and ammonia added, as was done in the tissue estimations in Section II. The optical density was measured at a wave length of 540 mμ against a water blank and the iron concentration read from the graph used for the tissue iron determinations. The results were expressed as milligrammes of iron per 100 grammes dry substance.

Some idea of the amount of sadza and relish consumed by local Africans per day was obtained by questioning 20 of the junior African hospital staff (10 males and 10 females) and discussing the

matter further with an African doctor.

African Beer: Sixty samples of home-brewed African beer were obtained from the British South Africa Police. Most of these samples were obtained by the police as a result of raids on premises where the beer was being illicitly brewed within the Salisbury Municipal area. A small number of samples however were of beer which had been legally brewed on farms.

The pH of the beer was measured on a pH meter (Radiometer Model 27). Ten millilitre aliquots of the beer, which had been well shaken to ensure that the particulate matter was uniformly suspended, were then measured into 300 ml Kjeldahl flasks and treated exactly as was done with the food. The results were expressed as milligrammes of iron per 100 ml beer.

Five samples of African beer brewed by the Municipal Authorities were also analysed for iron content in the same way as a comparison.

African Drinking Habits: 185 male and 156 female African outpatients, whose blood was taken for the serum iron investigations described in Section VI, were questioned on their drinking habits with particular respect to African beer. Estimates of the volume consumed are somewhat approximate as illicit brews are drunk out of mugs, tin cans, cups etc. and it is difficult to be sure of the exact volume of any of these containers. A better idea of consumption was gained from estimates of individual expenditure on beer in a given period. One pint of home-brewed African beer in the Salisbury area cost about six pence.

Results

The iron content of the various foods analysed is shown in Appendix VI and summarised in Table XVIII. Questions to the African staff elicited that the average adult consumes between 2 and 2.5 lbs (908-1134 grammes) of sadza, and between 0.5 and 1.0 lb (227-454 grammes) of relish, in cooked form, a day. The iron concentrations of the beer samples are also contained in Appendix VI and they are summarised in Table XIX.

The average amount of beer consumed per week by each of the 341 Africans questioned is contained in Appendix VIII. Table XX shows the average consumption of beer in each decade in males and females. Also shown is the amount of iron consumed in the beer calculated using the average of 9.4 mg iron per 100 ml beer.

TABLE XVIII

IRON CONCENTRATIONS IN VARIOUS AFRICAN FOODS

COOKED & READY FOR EATING

Results are expressed as mg iron/100g dry weight

Type of Food	Source									All Sources		
	Village			Hospital Staff Homes			Hospital Kitchen					
	Mean	SD.*	Range	Mean	S.D.	Range	Mean	S.D.	Range	Mean	S.D.	Range
Sadza	7.6 (11)	2.5	4.9-12.2	5.4 (4)	1.6	5.1-7.1	5.1 (3)	0.3	4.7-5.3	6.7 (18)	2.3	4.7-12.2
Relish (Green Vegetables)	24.8 (11)	19.6	6.1-68.4	17.45 (2)		9.3-27.6				23.8 (13)	18.4	6.1-68.4
Relish (Beans)	12.6 (5)	3.8	8.5-19.2	15.2 (2)		9.8-20.6				13.3 (7)	4.7	8.5-20.6

Figures in brackets are the number of samples examined.

* S.D. = standard deviation.

TABLE XIX

IRON CONCENTRATIONS IN AFRICAN BEER

Type of Beer	Average Iron Concentration mg/100 ml	Standard Deviation	Range mg/100 ml	Average pH	Standard Deviation	Range
Home Brewed	9.4 (60)	7.1	0.5 -35.2	3.9	0.2	3.3-4.2
Brewed by Municipal Authorities	0.3 (5)		0.32-0.35	3.1		3.0-3.2

Figures in brackets are the number of samples examined.

TABLE XX

AFRICAN DRINKING HABITS - HOME-BREWED BEER

Age Group	Average Consumption Pints/Week		mg Iron Derived From Beer/Day	
	Males	Females	Males	Females
15-19	2.2	0	17.7	0
20-29	3.4	0.35	27.4	2.8
30-39	5.75	1.3	46.3	10.5
40-49	8.81	2.25	71.0	18.1
50-59	7.8	4.5	62.8	36.3
60 +	8.0	7.4	64.4	59.6
Range	0-70+	0-70	0-564+	0-564

Calculations Based on the Average Iron Content of Beer of 9.4 mg/100 ml Beer

DISCUSSION

Food: The average iron content of sadza found in this investigation (6.7 mg/100 g dry weight) was somewhat lower than that found by Walker and Arvidsson (1953) (9.7 mg/100 g dry weight). This was almost certainly due to the fact that some of the samples examined in the present study were cooked in European-type aluminium and enamel pots. These pots are rapidly replacing the older iron pots and cans which were used for cooking by Rhodesian Africans in the past. Also samples of sadza obtained from the hospital were cooked in stainless steel vessels and these would contribute little if anything to the iron content of the food. Iron pots are still used fairly frequently in rural areas and this is reflected in the higher average iron concentration in sadza samples from the village.

The average iron content of the relish (20 mg/100 g dry weight) was lower than that found by Carr (1956) (32 mg/100 g dry weight) but Carr points out that one of his samples had an extremely high iron content due to contamination with soil. If this sample is omitted, Carr's average iron content of relish is 26 mg/100 g dry weight, which is only slightly higher than found in the present study and again this may be due to the less frequent use of iron cooking pots in the last few years.

The approximate range of iron consumed in food by adult Africans per day can be calculated using the iron concentrations in Table XVIII and average values of 25% solid material in sadza, and 20% in relish.

Thus assuming a consumption of between 907 to 1134 grammes of sadza and between 227 and 454 grammes of relish a day an average adult would ingest between 11 and 55 mg iron in the sadza and between 3 and 62 mg iron in the relish, that is a total of from 14 to 97 mg of iron per day in food.

MacDonald (1964) states that less than 10% food iron is absorbed, and with increasing amounts of ingested iron the percentage absorbed decreases (Smith and Pannacciulli, 1958) but even if the iron absorption was of the order of 8% in these Africans taking the lower limit of iron viz. 14 mg and 3% in those Africans on the higher limit viz. 97 mg the amount absorbed, approximately 1-3 mg would be completely adequate for the requirements of normal males. Indeed a number consuming the upper limits of the range would be adding to their body stores. Most females also would be expected to absorb enough iron from this diet for their requirements, but some on the lower range of iron intake and with repeated pregnancies might be iron deficient.

Beer: Home-brewed beer is apparently greatly preferred to that brewed by the Municipal breweries. Most of the subjects questioned said that this was because the home brewed variety was stronger. Females start drinking later than males and only in the fifth and subsequent decades were there any heavy drinkers among the female subjects. Also even in the older age groups there was a smaller percentage of females who drank heavily compared with males.

The method of brewing the home-brewed beer and the ingredients used are essentially the same as described by Strachan (1929). The containers used are oil drums of various sizes and paraffin tins. The insides of these containers become very rusty and eventually are eroded into holes. Quite frequently the holes are patched with rusty nuts, washers and bolts because of the difficulty experienced in obtaining replacements for these drums. Much of the erosion is presumably caused by the acidity of the beer which was shown to have an average pH of 3.9. The high iron concentrations found on analysis of the beer therefore result from iron dissolved from the containers in which it is brewed.

The enormous range of iron concentrations found in the various beer samples could be due to the fact that the police discovered the samples at different stages of maturity. That is, the less mature samples, having been a shorter time in contact with the container, would contain less dissolved iron. Some support is given to this impression by the fact that samples 52 to 60 (see Appendix VI) all came from semi-rural police stations in whose areas concealment is easier and so the brews would be able to mature. In these 8 samples the iron concentration is fairly high. This could mean that normally those samples with low iron concentrations would not be drunk as they were not yet mature, and therefore the average iron concentration of 9.4 mg/100 ml beer found in this study is too low. On the other hand there is little doubt that some of the beer consumed by those questioned was from the Municipal breweries which had a very much lower iron content (0.3 mg/100 ml beer). This would tend to compensate

for any error in the following calculations on iron derived from beer, caused by using a falsely low iron concentration for home-brewed African beer. The low iron concentrations found in Municipal beer is apparently due to the fact that any metal used in the brewing process is stainless steel. The ingredients used in the brewing process are basically the same as those used in home brewing.

If it is allowed, as shown previously, that in most Africans the amount of iron derived from food supplies the average body's needs, then any extra absorbed from beer would be in excess of requirement and would be added to iron stores or excreted. Iron excretion is normally low (Dubach, Moore and Callender, 1955) and though increased excretion has been reported in subjects with increased iron stores the amount is small (Chappelle, Gabrio, Stevens and Finch 1955; McMahon, 1956), thus the bulk of the iron absorbed must be added to the iron stores.

Bothwell et al. (1964) have shown that the mean absorption of iron from a volume of African beer containing 8 mg iron was 3.9% and from a volume of beer containing 25 mg iron was 1.9%. Using these absorption rates and the amount of iron available from beer as shown in Table XX, it is possible to calculate the amount of iron added to body stores in a decade. Also, as the liver contains between one quarter and one third of the total body storage iron (Bothwell and Finch, 1962), presumably about one third of this additional storage iron will be deposited in liver, thus the amount of iron added to the liver each decade can be calculated. The calculations are

detailed in Appendix VII. Table XXI shows the average total storage iron values found in the liver in each decade (extracted from Table VIII) and compares them with the theoretical values calculated from the above data and using the values found in the second decade as a base.

There is a fairly good correlation between the actual and theoretical total liver iron stores in females, apart from the fourth decade, where the theoretical value is less than half the actual value. In males there is fair correlation up to and including the fifth decade. In the older age groups however the theoretical values are much higher than those actually found. The most obvious explanation for this discrepancy in older males is that, when body stores are increased absorption of oral iron is depressed (Bothwell, Pirzio-Biroli and Finch, 1958; Pirzio-Biroli and Finch, 1960) and thus, in older men, who commonly have severe siderosis, a smaller percentage of the ingested iron is absorbed than the percentage used in the calculation.

TABLE XXI

ACTUAL AVERAGE TOTAL STORAGE IRON IN AFRICAN LIVERS

COMPARED WITH VALUES EXPECTED FROM IRON INTAKE IN BEER

(Expressed in Grammes)

Decade	Males		Females	
	Actual Liver Storage Iron	Theoretical Liver Storage Iron	Actual Liver Storage Iron	Theoretical Liver Storage Iron
2	0.25	0.25	0.31	0.31
3	0.77	1.11	0.47	0.31
4	1.18	1.78	0.96	0.45
5	3.18	2.91	0.74	0.96
6	3.33	4.64	1.92	1.84
7+	3.01	6.17	2.54	2.72

CONCLUSION

Obviously the above calculations are very approximate as the percentage absorption of iron varies so much (Bothwell et al.,1964), and it is not certain that one third of the absorbed iron is deposited in the liver. Especially might further deposition in liver be reduced when the organ already contains heavy iron deposits. Nevertheless, it is felt that there is enough correlation between actual and theoretical total liver storage iron values to suggest most strongly that the main source of iron in Bantu siderosis is home-brewed African beer, though no doubt there is a variable contribution of iron from food.

SUMMARY

The iron content of 38 samples of those kinds of cooked food which form the bulk of Rhodesian Africans diet was estimated. Also the iron content of 60 samples of home-brewed African beer, and 5 samples of African type beer brewed by the Municipal Authorities, were estimated.

It was calculated that in most subjects the amount of iron supplied in food would be sufficient for normal body requirements. Home-brewed African beer was shown to be very rich in iron. As the body's needs for iron were adequately met from food sources, it was argued that any extra absorbed from beer would be added to body stores.

Calculations based on average beer consumption, approximate rates of iron absorption, and postulating that about one third of this extra iron is stored in the liver, produced values for the average theoretical storage iron content of the liver in each decade. These values correlate sufficiently well with the average liver storage iron actually found in Section II to make it extremely probable that home-brewed African beer is the main source of the iron found in subjects with Bantu siderosis.

Plate XII

An iron cooking pot such as was commonly used by Africans for preparing sadza. These vessels are nowadays gradually being replaced by aluminium pots

Plate XIV

A selection of iron drums used by Africans to brew beer

Plate XV

The inside of a "beer drum" showing the rusty, pitted lining which adds iron to the beer during the brewing process

Plate XVI

Samples of African beer showing its high content of particulate matter

SECTION VI

SERUM IRON STUDIES

Squires (1952) first noted high serum iron (S.I.) values in Africans while working with the inhabitants of Bechuanaland (now Botswana). This was followed shortly by the work of Gerritsen and Walker (1953 a & b) who showed in Johannesburg that the S.I. and total iron-binding capacity (T.I.B.C.) values in certain other groups of Africans were also considerably raised. The groups in which these values were raised were newly recruited labourers for the Rand gold mines from Mozambique, Angola and Nyasaland (now Malawi). They also found high values in male African out-patients in the Johannesburg Hospital.

It was later shown that hepatic siderosis was associated with raised S.I. and T.I.B.C. levels in Africans, and that with increasing degrees of siderosis there was an increase in the mean S.I. levels (Gillman et al., 1957; Higginson, Keeley, Anderson and Walker, 1957; Hathorn, Gillman, Canham and Lamont, 1960).

Carr and Gelfand (1961) carried out a small survey on the S.I. and T.I.B.C. values in Rhodesian Africans. In none of their patients did they find the high S.I. and T.I.B.C. levels reported by Gerritsen and Walker (1953, b) and indeed, in a number of other South African studies these high values were not encountered (Wainwright, 1957; Hathorn et al., 1960).

It was therefore decided to undertake a further small survey of S.I. and T.I.B.C. values in Rhodesian Africans to confirm the findings

of Carr and Gelfand. During the course of this survey a number of serum iron and total iron binding capacity estimations were performed, at the request of clinicians, on African patients suspected of having severe siderosis. Some of these patients subsequently died and autopsies were performed. The findings in these cases are discussed. The post-mortem S.I. values of a number of siderotics dying of "shock" are also discussed.

Material and Methods

The subjects chosen for the survey were African patients and normal relatives coming to the Out Patients Department of Harare Hospital. Seriously ill patients, patients with raised temperatures or other evidence of acute inflammation, and patients with anaemia due to obvious blood loss, such as females with menorrhagia or incomplete abortion, were excluded because of depression of serum iron in these conditions (Bothwell and Finch, 1962).

The commonest complaints responsible for the patients seeking medical advice were related to urinary bilharzia, backache, muscle and joint pains, vague abdominal pains and skin diseases. In female patients, as well as the above, many complained of dysmenorrhoea or infertility. Approximately 50% of the women were mothers bringing sick children and were not themselves indisposed.

Cases coming to the Out Patients Department of Harare Hospital appear to be fairly representative of the African population of Salisbury and its immediate environs. It was realised that the group of people chosen might not reflect with complete accuracy the

S.I. and T.I.B.C. values in healthy Africans but it was felt that, by exclusion of patients with conditions which were known to affect these values, the error would not be great. Also, one of the groups found to have high S.I. and T.I.B.C. values by Gerritsen and Walker (1953,b) were hospital out patients so it seemed likely that if these high values occurred in Rhodesia they also would be found in out patients.

Approximately 30 patients of each sex were taken from each decade, starting at the second decade. Due to the small numbers of older women coming to the hospital only 30 females were taken in the sixth and seventh decades together. In all, 185 males and 156 females were examined. Each was questioned about drinking habits and the results of this survey have been analysed in Section V.

20 ml of blood were withdrawn from an arm vein into a disposable polythene syringe. A small quantity was transferred to a sequestrene tube for estimation of haemoglobin and for the blood film; the remainder was placed in an iron-free glass container where the serum was allowed to separate. All specimens were taken between 8.30 a.m. and 10.00 a.m. because of the diurnal variation of serum iron levels (Bothwell and Finch, 1962).

The blood slides were stained with Leishman's stain and the haemoglobin estimated by the cyanmethaemoglobin method (Dacie and Lewis, 1966). The serum iron was estimated by the method of Bothwell and Mallet (1955) and the unsaturated iron-binding capacity by the method of Bothwell, Jacobs and Kamener (1959). Both estimations were carried out in duplicate but sometimes the second S.I. estimation

had to be done using half quantities of serum due to insufficient amounts being available. The mean of the two results was used.

Serum iron and total iron-binding capacity levels were also estimated on a number of patients suspected of having severe siderosis. Eight of these subsequently died and the iron content of the pancreas, thyroid, pituitary and adrenals was estimated histologically on sections stained by Perl's method. The iron content was graded as described in Section II. The storage iron concentration in liver was estimated on seven of these. In one further case, on whom a gastrostomy was being performed because of carcinoma of oesophagus, biopsy specimens of liver, pancreas and gastric mucosa were taken and S.I. and T.I.B.C. estimations were made.

While this survey was in progress, there were three patients who died and in whose case notes the physician had particularly stressed signs of severe shock, which did not respond to any form of therapy. At autopsy all three were found to have an extreme degree of siderosis. In these cases, during the autopsy, blood was collected into iron-free glass containers from the external iliac vein by elevating the leg and massaging the thigh towards the trunk. Serum was separated by centrifuging the blood and S.I. estimations carried out in triplicate, using 0.2 ml serum for one (case D 33), and 0.5 ml serum for the other two cases. The serum volume was made up to 4 ml with deionized water (known to be free of iron) and the S.I. estimations carried out exactly as described by Bothwell and Mallet (1955) with allowance being made for the

dilutions in calculating the results. In case S.I. values were commonly raised in post mortem blood in siderotic subjects, serum was obtained in three other cadavers with siderosis in the same way, to use as controls. In the controls there was no history of shock.

Results

Details of the age, sex, haemoglobin, S.I., T.I.B.C. and percentage saturation of transferrin values, and comments on the blood film found in each case in the survey, are shown in Appendix VIII. The average values, their ranges and standard deviations in each decade are summarised in Table XXII.

Table XXIII shows the relationship of percentage saturation of transferrin to iron distribution in certain epithelial tissues of the body in the eight autopsies and one surgical case.

In Table XXIV, the post mortem serum iron values of the three siderotic subjects who died with signs of shock are compared with these values in three siderotic subjects in whom shock was not observed clinically prior to death.

TABLE XXII

MEAN BLOOD VALUES, STANDARD DEVIATIONS AND RANGES OF 341 AFRICAN SUBJECTS

Age Group	MALES				
	No.	Serum Iron µg/100 ml	Total Iron Binding Capacity µg/100 ml	% Saturation Transferrin	Haemoglobin g/100 ml
10-19	30	Mean 105 ± 42.8	403 ± 50.7	26 ± 9.4	13.2 ± 1.34
		Range 40 - 189	323 - 568	8 - 46	11.2 - 16.0
20-29	31	Mean 126 ± 40.0	358 ± 49.1	36 ± 12.7	15.0 ± 0.93
		Range 65 - 204	266 - 462	16 - 66	12.7 - 16.8
30-39	32	Mean 110 ± 44.5	347 ± 53.9	32 ± 14.2	14.7 ± 1.59
		Range 49 - 218	246 - 456	11 - 80	10.8 - 17.2
40-49	31	Mean 112 ± 48.4	313 ± 61.5	37 ± 18.8	13.6 ± 2.08
		Range 33 - 231	223 - 447	11 - 86	6.3 - 17.2
50-59	31	Mean 109 ± 41.6	329 ± 56.1	34 ± 17.9	14.0 ± 1.54
		Range 38 - 228	213 - 434	10 - 68	10.4 - 18.6
60 +	30	Mean 111 ± 57.7	321 ± 66.4	36 ± 19.9	13.3 ± 1.34
		Range 34 - 258	196 - 445	12 - 84	10.8 - 16.0
Total	185	Mean 112	345	34	14
		Range 33 - 258	196 - 568	8 - 86	6.3 - 18.6

TABLE XXII CONTD.

MEAN BLOOD VALUES, STANDARD DEVIATIONS AND RANGES OF 341 AFRICAN SUBJECTS

Age Group	No.	FEMALES			
		Serum Iron µg/100 ml	Total Iron Binding Capacity µg/100 ml	% Saturation Transferrin	Haemoglobin g/100 ml
10-19	31	Mean 94 ± 32.8	403 ± 57.6	24 ± 8.6	11.8 ± 1.21
		Range 40 - 166	274 - 555	10 - 40	8.9 - 14.5
20-29	32	Mean 110 ± 32.0	393 ± 49.0	29 ± 9.2	12.4 ± 1.13
		Range 58 - 186	253 - 480	14 - 53	10.4 - 14.8
30-39	31	Mean 88 ± 35.8	390 ± 67.6	23 ± 10.25	12.1 ± 1.23
		Range 24 - 143	290 - 593	5 - 45	7.8 - 14.1
40-49	31	Mean 86 ± 35.7	390 ± 73.4	23 ± 10.8	11.8 ± 2.14
		Range 28 - 147	291 - 571	5 - 49	6.3 - 14.5
50-59	21	Mean 115 ± 58.2	362 ± 79.6	33 ± 18.6	12.2 ± 1.89
		Range 26 - 272	225 - 563	5 - 85	5.2 - 13.8
60 +	10	Mean 96 ± 55.6	323 ± 65.7	31 ± 21.1	12.1 ± 1.51
		Range 50 - 243	265 - 401	15 - 87	8.2 - 13.4
Total	156	Mean 97	367	26	12.1
		Range 24 - 272	225 - 593	5 - 87	5.2 - 14.8

TABLE XXIII

RELATIONSHIP BETWEEN IRON DISTRIBUTION IN THE BODY & PERCENTAGE SATURATION OF TRANSFERRIN

Reference Number	Liver Iron Concentration (mg/g wet weight)	Cirrhosis	Histological Iron				S.I. µg/100 ml	T.I.B.C. µg/100 ml	% Saturation
			Pancreas	Thyroid	Pituitary	Adrenal			
P 102/67	7.26	0	++	++	++	++	187	256	73
64/67	2.38	Fine	+++	++	++	+	218	250	87
D 21	6.38	Fine	++	+++	++	++	144	160	90
D 39	15.37	Fine	+++	+++	++	++	201	231	87
13/67 ±	+++	Fine	+++	++	++	++	214	233	92
Jacob (Biopsy * Specimens)	8.00	0	++				120	148	81
D 25	6.74	Coarse	++	0	0	0	188	538	35
D 29	11.67	0	+	+	0	0	200	401	50
D 40	4.46	0	0	0	0	0	260	530	49

± Chemical iron concentration of liver not measured

* Histological iron content of gastric mucosa + +

TABLE XXIV

POST MORTEM SERUM IRON VALUES IN SIDEROTIC SUBJECTS

Reference Number	Age	Sex	Serum Iron µg/100 ml	Histological Iron in Hepatic Cells	Liver Cirrhosis or Fibrosis	Cause of Death	Remarks
D 35	60	F	1,253	+++	Portal Fibrosis +++	Congestive Heart Failure Cardiomyopathy (Terminal Shock)	Slight centrilobular necrosis of liver cells Liver iron concentration 10.5 mg/g
212/67	50	M	3,810	+++	Portal Fibrosis ++	Irreversible Shock Bantu Siderosis	Widespread focal necrosis of liver cells. Liver iron conc. 12.9 mg/g Pancreas iron conc. 2.1 mg/g
D 33	50	M	12,300	+++	Fine Cirrhosis	Liver Failure (Terminal Shock)	Widespread focal necrosis of liver cells. Liver iron conc. 7.9 mg/g
13/67	45	M	214	+++	Coarse Cirrhosis	Pulmonary Tuberculosis	No liver cell necrosis. Liver iron concentration 8.1 mg/g
BS/74/6	50	M	165	+++	Portal Fibrosis +	Carcinoma of Oesophagus	No necrosis of liver cells. Liver iron conc. 4.20 mg/g
BS/69/6	50	F	124	+++	Portal Fibrosis +	Status Asthmaticus	No necrosis of liver cells. Liver iron conc. 2.59 mg/g

DISCUSSION

The mean serum iron level for all ages in both males and females i.e. 112 and 97 µg/100 ml respectively, was 13 µg/100 ml lower than the normal means given by Bothwell and Finch (1962). The mean value for males was also between 13 and 43 µg/100 ml lower than the mean values found in four small groups of healthy male Rhodesian adult Africans by Carr and Gelfand (1961). On the other hand, the mean values in males was almost the same as found by Wainwright in healthy African males in Durban, and the mean value for females was 22 µg/100 ml higher than found by Wainwright in his healthy female Africans.

The mean T.I.B.C. for males was slightly above the average normal and for females appreciably above the average normal given by Bothwell and Finch (1962). Except in their old-age group, the average T.I.B.C. values found in healthy Rhodesian African males by Carr and Gelfand were between 20 and 32 µg/100 ml higher than found in males of the present series. In contrast, the mean T.I.B.C. values found by Wainwright in Africans of both sexes were very much lower than found in this study. There were no cases with very high S.I. and T.I.B.C. values such as were found by Gerritsen and Walker (1953, a and b).

There were 24 males and 24 females with S.I. values of less than 60 µg/100 ml, which is the lower limit of normal (Bothwell and Finch, 1962). It is not the intention to discuss anaemia in Africans

in this study but it is probably worth noting that among the 24 males with depressed S.I. values, only 5 (i.e. 2.7% of all males) had also low haemoglobin and percentage saturation of transferrin values with an associated normal or raised T.I.B.C., so were probably suffering from a mild degree of iron deficiency anaemia. In none of these was there any appreciable hypochromasia seen in the blood films. There were 13 females (8.3%) who, as well as having low S.I. values, also had low haemoglobin and percentage saturation of transferrin, with normal or raised T.I.B.C. values. They also probably had iron deficiency anaemia. This view is strengthened by the fact that in five, the blood films showed ring staining and in a further six, there was polychromasia of the erythrocytes.

Those subjects with low S.I. but without other criteria of iron deficiency were probably suffering from some low grade inflammatory process, because S.I. has been shown to be lowered in such conditions (Noyes et al., 1960). Other causes of lowered S.I. such as rheumatoid arthritis and cancer were excluded clinically. One low grade inflammatory process which is common in Africans, judged by autopsy findings, is chronic cystitis associated with urinary bilharzia and this may well be the principal cause of the low serum irons found.

In 17 (9.2%) males and 2 (1.3%) post menopausal females the percentage saturation of transferrin was greater than 60 (range 62 - 87). These are shown in Table XXV. As the table shows, in these subjects the serum iron values were normal or moderately

TABLE XXV

SUBJECTS WITH TRANSFERRIN SATURATION OF GREATER THAN 60%

	Years of Age	g/100 ml. Hb	µg/100 ml. Serum Iron	µg/100 ml. U.I.B.C.	µg/100 ml. T.I.B.C.	% Saturation
MALES	23	14.8	211	110	321	66
	32	15.6	218	131	349	62
	35	10.8	204	52	256	80
	45	14.5	231	38	269	86
	45	14.8	194	106	300	65
	45	13.0	162	66	228	71
	45	14.1	186	65	251	74
	50	14.0	172	69	241	71
	52	14.1	210	99	309	68
	52	15.2	228	133	361	63
	55	14.8	190	103	293	65
	55	13.0	204	94	298	68
	65	13.4	206	121	327	63
	65	12.7	182	94	276	66
	65	14.5	238	110	348	68
	70	16.0	202	40	242	84
	70	14.8	258	53	311	83
FEMALES	52	13.0	272	47	319	85
	60	13.0	243	38	281	87

raised, ranging from 162 to 272 µg/100 ml while their T.I.B.C. values were normal or slightly lowered, ranging from 228 to 361 µg/100 ml. The haemoglobin values in none of these 19 cases was abnormally high and indeed in 5 males the haemoglobin was less than 13.5 g/100 ml (range 12.7 - 13.4 g/100 ml) i.e. were anaemic (de Gruchy, 1960). There is insufficient information to state with any certainty the cause of the anaemia but it may be related to protein or folic acid deficiency. In none of these 19 subjects was there any significant abnormality found in the blood film. Also none had jaundice, palpable livers, or ascites at the time they were seen and unfortunately, it was impossible to recall them for further investigation.

It is probable that these 17 men and 2 women with raised S.I. and percentage saturation values had siderosis and that most had cirrhosis, though this was not diagnosed clinically. It is also probable that the type of siderosis was that in which iron deposits are widespread in epithelial tissues, as was described in Section IV and in the following paragraphs.

The autopsy findings in the 8 cases on whom serum iron studies had been carried out during life tend to confirm the theory, referred to in Section IV, that widespread epithelial deposits of iron depend on high percentage saturation of transferrin, as Table XXIII shows. In the first 5 cases, all of whom had percentage saturation values of greater than 80, heavy deposits of iron were found in pancreas, thyroid, pituitary and adrenal. In the sixth

case, a man with carcinoma of the oesophagus who also had a percentage saturation of greater than 80, biopsy specimens showed moderate iron deposits in pancreas and gastric mucosa.

In contrast, in the last three cases in Table XXIII, the percentage saturation was less than 60 and deposits of iron were absent, or at most scanty in thyroid, pituitary and adrenal. Moderate deposits were present in the pancreas in D 25, and scanty deposits in the pancreas of D 29. In D 40 the pancreas was free of stainable iron. As was noted previously, high liver iron concentration does not determine the widespread epithelial deposits, as liver iron concentration in D 40 is approximately twice that in case 64/67, and there are no epithelial deposits in the former, while in the latter all organs examined contained stainable iron.

In idiopathic haemochromatosis a number of patients die following signs of severe shock. Two such cases were described by Desforges (1949), a further case was reported among a series of 35 cases of idiopathic haemochromatosis by Klocknor, Kark, Baker, Chapman, Kaplan and Moore (1955), and, more recently, Jones (1962) described a case of "irreversible shock in haemochromatosis". The writer has performed a number of autopsies on African subjects in whom some of the terminal signs included shock. In three of these the serum iron was estimated on the post mortem blood and was found to be very high. These three cases are compared in Table XXIV with the findings in three other siderotic subjects who died without symptoms of shock.

Two striking differences are noted between the two groups. In the three who suffered from shock, the S.I. values were enormously high while those without shock had S.I. values within normal limits or, at most, slightly raised. The other interesting finding in the three shocked cases was the presence of liver cell necrosis. The necrosis was widespread in cases 212/67 and D 33, in which S.I. values were extremely high, but was slight in case D 35. In this last case the S.I., though raised, was much less so than in the other two. It was however, not at all clear what precipitated the liver cell necrosis and no leucocytic reaction was seen in the areas of necrosis.

Shock is a common finding in acute iron poisoning in children (Thomson, 1947; Spencer, 1951; Westlin, 1966). In one series, shock was present in 7 cases out of 10, in whom the initial S.I. values were 1,000 µg/100 ml or greater (Westlin, 1966). It is suggested therefore, that the shock in Bantu siderosis, and possibly in idiopathic haemochromatosis, is a manifestation of acute iron poisoning caused by release of iron into the blood, as a result of liver cell necrosis. Small increases in S.I. are known to occur in subjects with liver cell necrosis and normal liver iron stores (Beissman, Doley, Christianson and Delp, 1954), so large increases could be expected when necrosis occurs in livers with greatly increased iron stores.

Unfortunately, with reference to the cases of shock in idiopathic haemochromatosis reported by Desforges, 1949; Kleckner et al., 1955; and Jones, 1962 the serum iron values when shock was established were

not recorded. Also, in the case reported by Jones, liver cell necrosis was not mentioned, though it is possible that a minor degree of necrosis was present but not thought worthy of comment. High terminal serum iron values have been reported in one case of idiopathic haemochromatosis (Howard, Balfour and Cullen, 1954). Howard et al in the same paper however, described a second case of idiopathic haemochromatosis in whom the serum iron values reached the extremely high level of 8,000 µg/100 ml. Not only did this patient survive the high serum iron values but is reported to have felt well and experienced "no untoward symptoms of any kind".

Thus while it is appreciated that the connection between shock and iron poisoning (as manifested by hyperferraemia) in idiopathic haemochromatosis has not yet been definitely established, the similarity between the features of shock in Bantu siderosis and shock in idiopathic haemochromatosis makes a common aetiology of the shock probable.

SUMMARY

The results of a survey of the haemoglobin, serum iron and total iron binding capacity values in 341 out patients attending Harare Hospital are presented. As far as possible clinically, patients with conditions likely to alter the S.I. values to any extent were excluded.

The mean S.I. values found were a little lower than found in Europeans. The male values were also somewhat lower than found in healthy male Rhodesian Africans (Carr and Gelfand, 1961), but the same as found in healthy male Africans in Durban (Wainwright 1957).

Serum iron values were lower than 60 µg/100 ml in 48 patients. In 18 of these, other findings suggested that the depressed S.I. was due to iron deficiency i.e. 2.7% males and 8.3% females had some degree of iron deficiency. It is suggested that the low serum iron in the remaining 30 patients was due to low grade chronic infection.

There were 19 patients with percentage saturation of transferrin values of greater than 60. It is suggested that these cases had siderosis with widespread epithelial deposits of iron. The very high S.I. and T.I.B.C. values found in certain groups of Africans by Gerritsen and Walker (1953,b) were not observed in any of the patients in this series.

Widespread epithelial deposits of iron were found at autopsy on 5 subjects in whom the ante-mortem percentage saturation of transferrin exceeded 80. In three other subjects, in whom the percentage

saturation of transferrin was less than 60 during life, little or no stainable iron was found in epithelial tissues (other than liver) at post-mortem. These findings are interpreted as being confirmation of the theory that the widespread epithelial deposits of iron, found in some cases of Bantu siderosis, depend on high percentage saturation of transferrin.

Three siderotic subjects, who died following a period of shock, were found to have extremely high post-mortem serum iron levels and in these, there was evidence of liver cell necrosis. Serum iron values in three siderotic subjects who died without signs of shock were not markedly raised and no liver cell necrosis was found. It is suggested that the shocked subjects died of acute iron poisoning, caused by release of iron into the blood from necrotic liver cells, and that this condition is similar to that reported in a few subjects with idiopathic haemochromatosis.

SECTION VII

EXPERIMENTAL WORK

In this section a number of investigations are presented in support of some of the theories propounded in previous sections.

1) In Section IV it was noted that one of the principal differences in iron distribution between Bantu siderosis and idiopathic haemochromatosis was the much heavier concentration of iron in the reticuloendothelial system of the former. It was suggested that this iron could result from either increased destruction of red cells, or failure to release iron derived from normal red cell destruction, as can occur in infection. In the first part of this section the red cell life span and red cell fragility of a number of male Africans are examined.

2) Siderotic subjects with cirrhosis, especially fine cirrhosis, frequently have widespread epithelial deposits of iron seldom found in non-cirrhotic, siderotic subjects (Section IV). Among the organs most affected by the epithelial deposits of iron is the pancreas. One suggestion put forward for the widespread epithelial deposits in these cirrhotic cases was that the blood by-passed the liver, and that iron normally deposited in hepatic cells was carried further afield to other epithelial tissues. If this explanation were correct it was felt that the head of the pancreas, which would be most affected by the diverted blood, would have heavier concentrations of iron than the tail, which would be perfused by the diverted blood to a much smaller extent, if at all. The validity of

this suggestion was tested by measuring the iron concentrations in the heads and tails of pancreas in a number of Africans with, and without cirrhosis.

3) The findings in Section V suggest that home-brewed African beer is the main source of the iron found in Bantu siderosis. It was decided to attempt to produce siderosis in guinea-pigs by feeding them African beer.

4) In an attempt to investigate further the theory that high percentage saturation of transferrin was responsible for the epithelial deposits of iron in some cases of Bantu siderosis, in vitro studies were undertaken using various human tissues.

5) It was decided also to observe the effect on the serum iron and percentage saturation of transferrin of feeding African patients an amount of iron, such as might be ingested during a heavy beer drink.

PART I

MEASUREMENT OF A) RED CELL LIFE SPAN & B) RED CELL FRAGILITY IN HEALTHY MALE AFRICANS

A) Material and Methods

The red cell survival was measured on 22 healthy male African adults. Seven of those were serving members of the Royal Rhodesian Air Force, two were members of the British South Africa Police and thirteen were laboratory assistants. Their ages ranged from 20 to 59 years.

The method used was the radioactive chromium method described by Dacie and Lewis (1966). Description of the apparatus used for counting and the details of counting methods are contained in Appendix IX.

It was originally intended to carry out this investigation on 50 males but the difficulty in getting volunteers made it necessary to be satisfied with 22. It was felt that if diminished red cell life span played any significant part in the reticuloendothelial deposits this number would be sufficient, because in Section II it has been shown that the incidence of siderosis in adult African males was over 70%. Also, as siderosis is commoner in males, it was felt that if a normal life span was found in males it would be unnecessary to repeat the investigation in females.

Results

The results are recorded in Appendix IX. These take the form of a ^{51}Cr survival curve for each patient. At the foot of each curve the $T\frac{1}{2}$ ^{51}Cr value is given. The $T\frac{1}{2}$ ^{51}Cr ranged between 25 and 33 days.

Discussion

In presenting the results no attempt has been made to correct for elution or to calculate the mean cell life span because, as Dacie and Lewis point out, whether the red cell life span is normal or not is indicated by the $T\frac{1}{2}$ ^{51}Cr.

In this series of patients the $T\frac{1}{2}$ ^{51}Cr ranged between 25 and 33 days with a mean of 29.4 days and a standard deviation of 2.34. The range given by Dacie and Lewis (with no correction for elution) is 25 to 32 days but no mean value or standard deviation is quoted.

It would appear therefore that the red cell life span in healthy Rhodesian Africans falls within the limits accepted as normal in Europeans. Consequently it can be assumed that the heavy reticulo-endothelial deposits in Bantu siderosis are not due to diminished red cell life span.

B) RED CELL OSMOTIC FRAGILITY

Material and Methods

The osmotic fragility in 50 healthy male Africans was measured. Their ages ranged from 20 to 62 years (mean 38 years). Forty two of these were staff of the Hararo Hospital, Public Health and Blair Research Laboratories, three were mortuary attendants, and five were members of the British South Africa Police.

Blood from the subjects being examined was taken from an arm vein and transferred to tubes containing heparin. The osmotic fragility was measured using the method described by Dacie and Lewis (1966). The solutions were allowed to stand for 30 minutes at room temperature. The degree of haemolysis in each tube was measured on an "Eel" colorimeter.

Results

The detailed results are to be found in Appendix X. In only one case Machipison, aged 53 years, was there a definite slight increase in osmotic fragility. There were five other cases in which the degree of haemolysis at 0.5% NaCl was between 6 and 12% (Dacie and Lewis give 5% as the upper limit at this concentration of NaCl).

Discussion

The five cases in which haemolysis at 0.5% NaCl was between 6 and 12% could be considered to be at the upper limit of normal, as in these cases, the degree of haemolysis at the other NaCl

concentrations all fell within normal limits. There was therefore only one case in the 50 examined with increased osmotic fragility and this was not marked. It is felt therefore that increased red cell fragility can play little or no part in the reticuloendothelial deposits of iron in Bantu siderosis.

This investigation fails to confirm the findings of Strachan (1929) in Johannesburg Africans that there was a slight but definite increase in the fragility of red cells in Africans.

PART II

IRON CONCENTRATION IN HEAD AND TAIL OF PANCREAS IN CIRRHOTIC AND NON-CIRRHOTIC SUBJECTS

Material and Methods

Blocks of tissue were taken at autopsy from the head and tail of pancreas in 30 cadavers. Fifteen of the subjects had cirrhosis of liver and 15 had no cirrhosis. The cases were selected to include a wide range of degrees of siderosis but were not selected on the basis of cause of death. Those cases in which autolysis was marked were excluded.

The tissues were fixed in buffered formalin and sections cut and stained with haematoxylin and eosin and by Perl's method for iron, as described in Section II. The total iron concentration in the tissues was measured chemically as was also described in Section II. The haemoglobin iron concentration was not measured, as there was no certainty that the correction factor used by Bothwell et al. (1964) for the liver haemoglobin estimations would be valid for the pancreas. Also no marked difference between the red cell content of the two sites examined was seen histologically, so it was felt that the total iron concentrations of the tissues was satisfactory for this study. The histological grading of the iron was based on the code used in Sections II and IV.

Results

The detailed results are contained in Appendix XI. No statistical difference was found between the iron concentration in the heads and that in the tails of pancreas either in cirrhotic or non-cirrhotic subjects. There was also no difference appreciable histologically, between the amount of iron in the head and tail of pancreas in any of the subjects examined.

Discussion

The fact that no significant difference in iron content was found, either chemically or histologically, between the heads and tails of the pancreases examined makes it extremely unlikely that mechanical shunting of blood in cirrhotic subjects plays any part in the widespread epithelial deposits of iron sometimes found in these subjects. It is felt, also that if shunting of blood were an important factor, there would be less difference between the degree of epithelial deposition of iron in fine and coarse cirrhotics (Section IV). Personal experience of the writer with autopsy material suggests that oesophageal varices are about as common in subjects with coarse cirrhosis as with fine cirrhosis, and so probably there is little difference in the degree of blood shunting in the two conditions.

PART III

THE EFFECT ON GUINEA PIGS OF FEEDING THEM WITH HOME-BREWED AFRICAN BEER

Material and Methods

Forty male guinea pigs were obtained from the Public Health Laboratory, Salisbury, where they are bred. No epidemic or deficiency diseases were said to have occurred among these animals in the past five years.

The animals chosen were aged between 9 months and 1 year. Their diet while in the Public Health Laboratory consisted of a) "horse-cubes" obtained from the Rhodesian Milling Co. These cubes were stated by the maker to contain bran, rolled oats, cotton seed cake, monkey nut cake, vitamins A and D, and minerals, including phosphorous, calcium, manganese, potassium iodide, cobalt, copper and ferrous sulphate. b) Lucerne c) maize meal d) water. All of these substances were administered ad lib.

Illegally brewed African beer was obtained fresh once a week from the British South Africa Police and its iron content estimated as described in Section V. It was stored in a refrigerator in plastic containers.

The guinea pigs were placed in separate cages. They were divided into two groups of 20. The first group were used as controls and and were fed on the laboratory diet described above. The second group were also given the laboratory diet and in addition were given

African beer. The amount of beer fed to each animal was calculated according to its weight, and was equivalent to that which would be consumed by a fairly heavy drinker. It was based on the assumption that a man weighing 70 kg consumed 6 pints of beer a day, and the guinea pigs were given an amount directly proportional to their weight, e.g. a 700 g guinea pig received a daily ration of 36 ml of beer.

The beer was fed to the animals twice daily, half of the daily ration being given at 9 a.m. and the other half at 4 p.m. It was administered by means of 20 ml plastic disposable syringes to ensure that each animal ingested its full ration daily. Though in most cases the beer was accepted reluctantly initially, after the first few days it was consumed readily from the nozzle of the syringe.

All of the animals were weighed at the commencement of the experiment and those on the beer supplement again, when they were sacrificed. They were sacrificed using chloroform in a glass jar. As soon as they were dead blocks of tissue were taken from liver, spleen, duodenum and pancreas. The tissues were immediately placed in buffered formalin, fixed, blocked, cut and stained with H.& E. and by Perl's method for iron as described in Section II.

The amount of iron found in the tissues on histological examination was graded as described in Section II.

One of the guinea pigs on the beer supplement (reference No. 1) choked and died on the 25th day of the experiment. It was replaced by another which was given the reference number 9 in Appendix XII. The original number 9 escaped while feeding on the 80th day of the

experiment and was not found. Four of the animals were sacrificed after 40 days on the beer supplement, three after 50 days and the remainder after 97 days. Number 9 was on the beer supplement for 72 days.

One guinea pig (No. 17) developed an abscess of jaw which discharged and healed without treatment. The time between the lesion being first noticed and its complete healing was three weeks.

Results

Details of the findings in this experiment are contained in Appendix XII. At autopsy, lesions were found in three control and two beer-fed animals which histologically resembled those produced by Pasturella pseudotuberculosis. These were present in the liver in two animals, in the spleen in one animal, and in both organs in two animals.

In none of the twenty control guinea pigs was any iron seen histologically in the liver or pancreas. In all cases slight or moderate deposits of stainable iron were found in the spleen and in one, scanty deposits were found in the villi of the duodenal mucosa.

Stainable iron was found in the livers of all the animals which had received the beer supplement. In 7 animals iron deposits were heavier in the hepatic cells than the Kupffer cells, in 6 the reverse was true, while in 7 deposits appeared to be equally heavy in both sites. Deposits were either scanty or absent in the portal areas of all these animals except one in which the deposits were moderate.

Deposits of stainable iron in spleen were moderate in 2 cases and heavy in the remainder. In the stroma of the duodenal villi moderate deposits of iron were found in 15 animals and heavy deposits in 5 animals. Stainable iron was not found in the pancreas of any of the animals on the beer supplement.

There was an average loss of weight of 150 g (range 29-385 g) in the animals being fed beer. The greatest weight loss 385 g was noted in the guinea pig which had the abscess of jaw.

Discussion

In the control guinea pigs the only tissue examined which contained stainable iron was the spleen and deposits were never more than moderate. In contrast those animals which had been given the beer supplement had not only much heavier deposits in the spleen than the control animals but they had also appreciable amounts of stainable iron in the liver and duodenal mucosa. Liver iron deposits increased with the time for which beer was fed, i.e. 6 out of 12 animals (50%) who had been on beer for more than 50 days had a "total score" (see Section II) of 4+ or more, while only 2 out of 8 (25%) who had been on beer for 50 days or less had a similar total score. This did not apply apparently to all cases as animal No. 10, which had been on beer for 97 days had a "total score" in liver of only 1+, and animals numbers 12, 14 and 20, which had been on beer for the same length of time had each "total scores" of only 2+.

There is no clear cut indication from this study whether the iron is first deposited in the hepatic or Kupffer cells of the liver.

The effect of infection on iron deposition, discussed in Section IV, is not clearly demonstrated because, though in guinea pigs numbers 2 and 17 who had an infection, iron deposits in the Kupffer cells are heavier than those in the hepatic cells, this is also seen in other animals apparently without infection, and is not seen in guinea pig number 3 with infection.

Iron deposits in duodenal mucosa apparently build up rapidly as heavy deposits were found in guinea pig number 1 which had been on beer for only 25 days. This presumably results from the rapid absorption of iron from the gut as discussed in Section IV.

The mean concentration of iron in the beer given to the animals was 16.55 mg/100 ml beer which was rather higher than the average concentration found in Section V viz. 9.4 mg/100 ml. It was however noted in Section V that the average iron concentration in beer in certain semi-rural areas is higher than the overall average. It is therefore felt that the somewhat higher than average iron concentration in the beer given to the animals does not in any way invalidate the results of the experiment.

If the results in guinea pigs in Appendix XII are compared with those in humans in Appendices I and II, it can be seen that the iron distribution in liver, spleen and duodenum is virtually the same as found in subjects with Bantu siderosis without cirrhosis. In no case did the liver deposits in the animals become as heavy as was found in Africans with severe siderosis, but if it is borne in mind that severe siderosis in Africans only begins to appear at the end

of the third decade, i.e. after about 15 years, or 1/5th of their natural life consuming beer, it is not surprising that no cases of severe siderosis were seen in guinea pigs after 97 days consuming beer which is only about 1/11th of their natural life (average life of a guinea pig is approximately 3 years) (Hendrie, 1968).

No check was kept on the amount of food taken by any animal though plenty was always available. The loss of weight noted in the animals receiving beer may have been due to depression of the appetite or to the presence of some toxic substance in the beer. No evidence of fibrosis was found in any of the livers examined (excluding those with Pasturella pseudotuberculosis). It is however felt that the duration of this experiment was too short to allow any conclusion to be reached on this issue.

The heavy deposits of iron in the reticuloendothelial system following the feeding of iron-rich beer to guinea pigs are in contrast to the results of Polson (1929). This worker found that when iron was administered orally to rabbits there was considerable accumulation of haemosiderin in the hepatic parenchymal cells but little in the Kupffer cells or spleen. One is therefore tempted to speculate whether or not some toxic substance in the beer was responsible for the iron accumulation in the reticuloendothelial system of the guinea pigs, and if indeed such a substance also did not play some part in producing the heavy reticuloendothelial deposits in Bantu siderosis. Further tests on various animals using beer and pharmaceutical preparations of iron will however be necessary before any firm conclusion can be reached.

The results of this experiment appear to confirm the findings and conclusions in Section V that African home-brewed beer can produce Bantu siderosis. In order to test the hypothesis that the beer plays a part in the production of cirrhosis in Africans a similar experiment of much longer duration would be required.

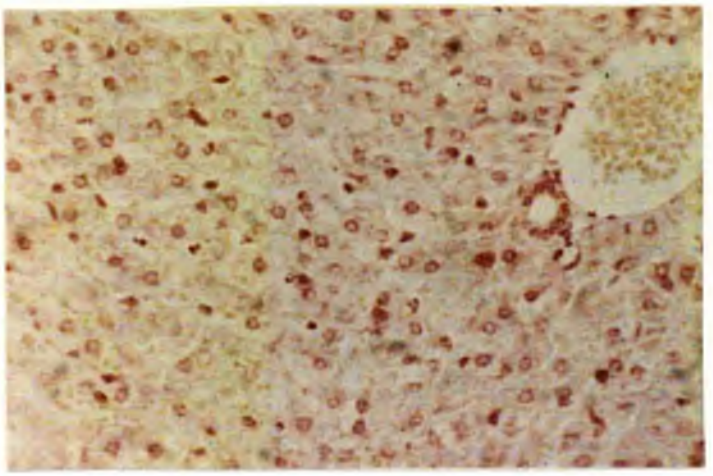


Plate XVII

**Liver of control guinea pig,
2. x 320
No stainable iron present**

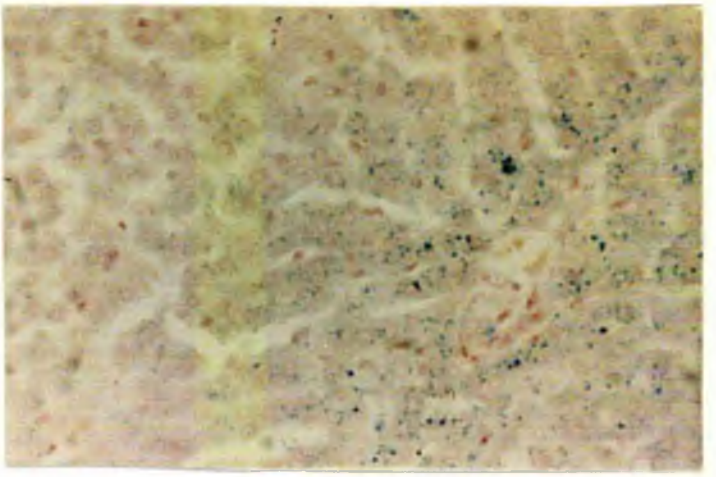


Plate XVIII

**Liver of beer-fed guinea pig,
12. x 320
Stainable iron is seen in all hepatic cells but is much heavier round the portal area**

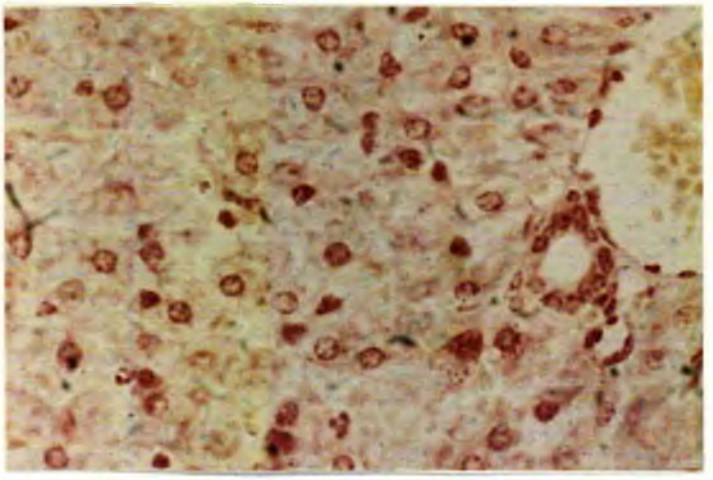


Plate XIX

**Liver of control guinea pig,
2. x 500**

Plate XX

**Liver of beer-fed guinea pig,
12. x 500
No iron is present in the Kupffer cells.**

In Plates XVII - XXIV all sections are stained by Perl's method for iron

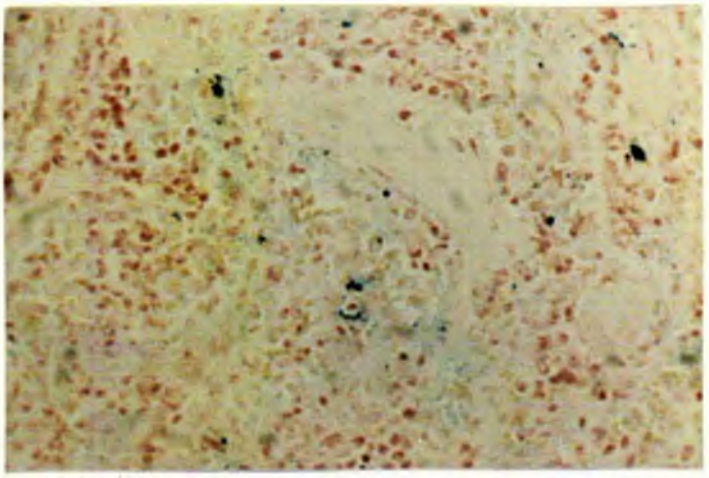


Plate XXI

Spleen of control guinea pig,
2. x 320
Little stainable iron present

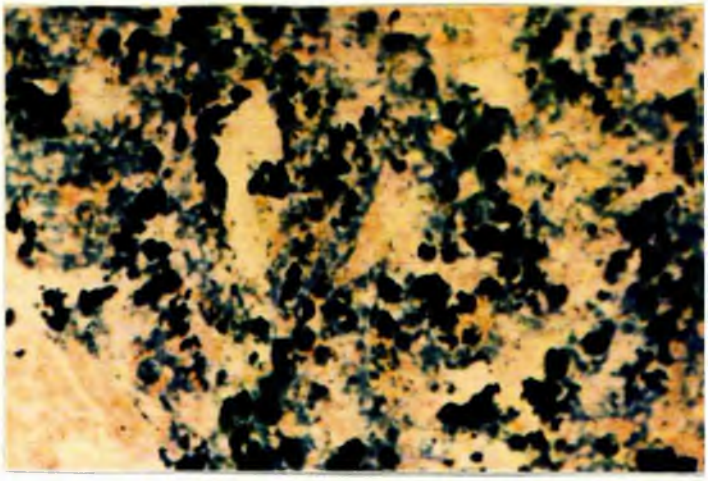


Plate XXII

Spleen of beer-fed guinea pig,
9. x 320
Deposits of stainable iron are extremely heavy

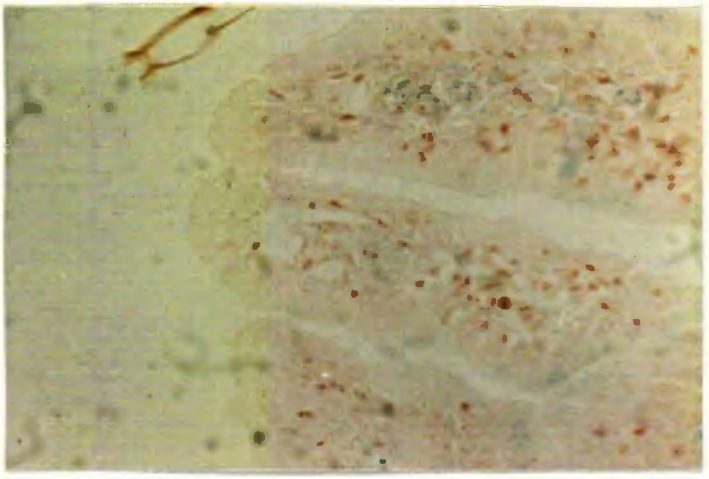


Plate XXIII

Duodenum of control guinea pig,
6. x 320
A few fine granules of stainable iron are seen in the stroma of the villi

Plate XXIV

Duodenum of beer-fed guinea pig,
7. x 320
Extremely heavy deposits of stainable iron are seen in the stroma of the villi

P A R T IV

UPTAKE OF IRON IN VITRO BY VARIOUS HUMAN TISSUES FROM TRANSFERRIN AT DIFFERENT PERCENTAGES OF SATURATION

It has been shown that when rat liver slices are incubated in an ionic solution of iron, there is passive diffusion of the iron through the cell membranes and it is bound to "some entity within the cell in such a way as to remove effectively the iron from ionic equilibria" (Saltman, Fiskin and Bellinger, 1956). Their experiments also showed that the rat liver slices could accumulate ionic iron against an apparent concentration gradient. Jandl et al. (1959) have shown that the uptake of iron bound to transferrin by rat liver slices is greater when the percentage saturation of transferrin is more than 60 than when it is less than 60. These authors showed that the accumulation of iron by liver slices was not diminished by preliminary boiling for 5 minutes which, when taken in conjunction with the findings of Saltman, Fiskin, Bellinger and Alex (1956) that this accumulation of iron is not affected by metabolic inhibitors, would seem to indicate that this is not an active metabolic process. It has been suggested by Mazur et al. (1960) and Katz and Jandl (1964) that, at high degrees of transferrin saturation iron is less firmly complexed with the protein and behaves more like ionic iron so might diffuse into the liver cells in a similar fashion to that demonstrated in the experiment of Saltman et al. (1959).

In Sections IV and VI, some evidence has been presented which suggests that high percentage saturation of transferrin is responsible for the widespread epithelial deposits of iron in certain cases of Bantu siderosis. It has also been pointed out, that in a number of other conditions in which iron overload is present and in which percentage saturation of transferrin is high there are also widespread epithelial deposits of iron (see Section IV). It was therefore decided to attempt to demonstrate more clearly that percentage saturation is important in determining iron uptake in human tissues.

Material and Methods

The principle behind the experiment was to incubate thin slices of viable human tissue, obtained at operation, in the patient's own serum. In each case the serum was so treated that in one tube the transferrin was approximately 50% saturated with iron (^{59}Fe) while in a second tube it was approximately 90% saturated. The radioactivity of the tissue slices was measured and compared with a standard and from this, the iron uptake by the tissue was calculated in μg Fe/100 mg wet weight of tissue. In order to obviate the errors due to non-specific trapping of radioactive serum in the intercellular spaces of the tissue, the serum in the second tube was so prepared with non-radioactive iron that the same amount of radioactive iron could be added to both tubes to produce approximately 50% saturation in one tube, and approximately 90% saturation in the second. This meant that the amount of radioactivity in both pieces

of tissue due to non-specific trapping of serum would be approximately the same (if allowance were made for any difference in weight), and any difference in radioactivity would presumably be due to difference in uptake of radioactive iron by the tissue cells.

Patients were unselected except in so far as it was necessary to know of the time of the operation at least one day before the test in order to prepare the serum.

The procedure was as follows: approximately 60 ml of blood was withdrawn from the patient, the serum separated, and the serum iron and unsaturated iron binding capacities estimated in the same was as described in Section VI. 8 ml of serum was measured by pipette into each of two 10 ml polythene tubes which had been previously acid-washed, well rinsed with deionized water and dried.

Two solutions of ferric ammonium citrate were made up in a similar way as that described by Bothwell et al. (1959) for use in estimation of unsaturated iron binding capacity as follows:

1) 0.3 ml of a solution of ferric chloride in dilute HCl (solution contained 5 mg of iron in 1.0 ml) was pipetted into a centrifuge tube. Ammonium hydroxide was then added, drop by drop, until a dark brown flocculant precipitate formed. After centrifugation, the supernatant solution was discarded and about 0.1 gramme of citric acid was added to the tube. Four millilitres of iron free water was then added and the tube heated with stirring in a boiling water bath. If the precipitate did not dissolve rapidly more citric acid was added. The solution was then cooled and one drop of

bromothymol blue was added. Ammonium hydroxide was then pipetted, drop by drop, into the solution until the colour changed from yellow to blue. The solution was then made up to 50 ml and the iron content of an aliquot measured in triplicate using the thioglycolic acid method.

2) About 100 μc ^{59}Fe were added to 0.3 ml of the solution of ferric chloride and the procedure then followed exactly as with solution 1.

The iron content of solution 1, in this series of experiments, was 28 μg/ml, while that of the three preparations of solution 2 was 25, 30 and 32 μg/ml. The pH of solution 1 was 8.31, and of solutions 2, were 8.84, 9.1 and 9.2.

In case there should be much difference in pH between the serum with the low saturation of transferrin and that with the high saturation which could affect the stability of the transferrin-iron system (Surgenor, Koechlin and Strong, 1949), the pH values of both were measured in 5 cases, and the results are recorded in Table XXV.

TABLE XXV

pH VALUES OF SOME SERA IN WHICH TISSUES WERE INCUBATED

Case Number	Serum with Low Percentage Saturation of Transferrin pH	Serum with High Percentage Saturation of Transferrin pH
23	7.37	7.39
24	7.45	7.48
25	7.49	7.51
26	7.35	7.36
27	7.37	7.40

The amount of iron "x" to be added to the 8 ml of serum to produce: A) approximately 50% saturation of transferrin was calculated from the formula $x = (\frac{T.I.B.C.}{2} - S.I.) \times \frac{8}{100}$ µg, and the amount of iron "y" to be added to the 8 ml of serum to produce: B) approximately 90% saturation, from the formula $y = (9/10 \times T.I.B.C. - S.I.) \times \frac{8}{100}$ µg where T.I.B.C. = total iron binding capacity of the serum and S.I. = the serum iron, both values being expressed as µg/100 ml serum. The amount of non-radioactive iron to be added to serum "B" was $y - x$ µg. The volume of iron solution to be added in each case was calculated from the amount of iron required and the iron concentration of the solution, by simple proportion.

The non-radioactive iron solution was added to serum "B" at least three hours before the radioactive solution to allow time for complete stability of the transferrin-iron system. The radioactive iron solution was then added to both serum A and B, and the sera allowed to stand at room temperature for about 18 hours before the tissue slices were added. Directly following addition of the iron solution to the serum, mixing was performed by repeated inversion of the tubes.

A tissue slicer was constructed on the principle of the Stadie-Riggs microtome (Stadie and Riggs, 1944). This was designed to cut tissue to a thickness of 0.5 mm.

At the time of operation the equipment was taken to a room adjacent to the operating theatre. As soon as the tissue had been removed from the patient it was taken immediately to the side room and slices cut with the Stadie-Riggs Microtome. The slices were

rinsed in Krebs-Ringer solution and aliquots placed in the tubes containing the serum. The tubes were taken to the laboratory and placed on a "Mathurn" rotary mixer (as used for mixing blood cell suspensions), which was housed inside an incubator. The tubes were then incubated at 37°C for 3 hours while undergoing constant mixing.

When incubation was complete the tissue slices were removed from the serum and washed in three changes of Krebs-Ringer solution. They were then dried carefully between two sheets of blotting paper. A small piece of each was placed in buffered formalin and, when fixed and blocked, sections were cut for autoradiography. The remaining pieces of each tissue were weighed to the nearest milligramme and placed in a boiling tube with 2 ml of concentrated nitric acid. The acid-tissue mixture was boiled for 15 minutes, the tube being attached to a reflux condenser to reduce loss of radioactive iron in the vapour.

The acid digest, when cool, was decanted into a 10 ml polythene tube. The digest was diluted with deionized water to a volume of 4 ml, a mark having previously been made on the tube at that level. The radioactivity of the solution was then measured in a "well" counter as described in the introduction to Appendix IX, Page 86, Volume II.

A standard was prepared by measuring a suitable amount of radioactive iron solution (previously referred to as "solution 2") into an identical polythene tube and the volume made up to 4 ml as with the acid digest.

The iron uptake of the tissue slices "U", in μg iron/100 mg wet weight of tissue, was calculated from the formula $U = \frac{A}{S} \times I \times \frac{100}{T.W.}$, where A = the number of counts per unit time of the acid digest; S = the number of counts per unit time of the standard solution; I = the amount of iron in the standard solution expressed as micrograms; and T.W. = the tissue weight in milligrammes.

Results

All attempts at autoradiography failed because three different batches of stripping film sent by air from London were found to be badly fogged on arrival in Salisbury. This was thought to be due to either rough handling or cosmic radiation during the flight.

Detailed results of the tissue digests are contained in Appendix XIII and are summarised in Table XXVI.

The uptake of ^{59}Fe from transferrin approximately 90% saturated is greater than that from transferrin approximately 50% saturated in all tissues and all samples. This difference is statistically significant when all samples of all the tissues are considered together. It is also significant when liver and thyroid are considered individually but not significant in striated muscle. No significant difference in uptake is noted between liver and thyroid.

There is considerable variation in uptake of ^{59}Fe not only between one tissue and another but between different samples of the same tissue. The specimen of liver, reference number 26, is considered separately from the other liver specimens as the patient

TABLE XXVI

UPTAKE OF ^{59}Fe BY SLICES OF VARIOUS HUMAN TISSUES FROM TRANSFERRIN AT DIFFERENT PERCENTAGES OF SATURATION

μg Fe/100 mg wet weight of tissue

Ref. No.	Tissue	Uptake of Iron From Transferrin Approx. 50% Saturated	Uptake of Iron From Transferrin Approx. 90% Saturated	Difference
1	Liver +	0.005	0.017	0.012
2	Liver	0.030	0.108	0.078
3	Liver	0.061	0.234	0.173
4	Liver	0.018	0.157	0.139
5	Liver	0.022	0.171	0.149
6	Liver	0.013	0.188	0.175
7	Thyroid	0.017	0.175	0.158
8	Thyroid	0.000	0.085	0.085
9	Thyroid	0.028	0.183	0.155
10	Thyroid	0.031	0.080	0.049
11	Thyroid	0.022	0.023	0.001
12	Thyroid	0.059	0.181	0.122
13	Thyroid	0.008	0.023	0.015
14	Thyroid	0.026	0.241	0.215
15	Thyroid	0.010	0.103	0.093
16	Pancreas	0.024	0.105	0.081
17	Pancreas	0.017	0.560	0.543
18	Parotid Gland	0.024	0.076	0.052
19	Parotid Gland	0.011	0.200	0.189
20	Myocardium	0.012	0.073	0.061
21	Smooth Muscle	0.053	0.060	0.007
22	Striated Muscle	0.015	0.068	0.053
23	Striated Muscle	0.019	0.030	0.011
24	Striated Muscle	0.038	0.050	0.012
25	Striated Musole	0.085	0.112	0.027
26	Liver	0.038	2.350	2.312
27	Spleen	0.005	0.015	0.010

+ Stainable iron in hepatic cells +++

1) The overall mean difference = 0.106
   t = 4.7 which is significant at 0.001
2) The mean difference for liver (exoluding case No. 26) = 0.121
   t = 4.6 which is significant at 0.01
3) The mean difference for thyroid = 0.099
   t = 4.2 which is significant at 0.01
4) The mean difference for striated muscle = 0.026
   t = 2.7 which is not significant

There is no significant difference between the uptake of liver and thyroid

was suffering from severe iron deficiency anaemia.

Discussion

It appears from this experiment that human tissues take up iron from serum to a greater extent when the transferrin is almost completely saturated than when it is only half saturated, thus confirming the findings of Jandl et al, (1959) on rat liver slices. This difference in uptake is significant in liver and thyroid but not in striated muscle. It is possible however, that a small, but significant, difference might be found if a larger number of samples of striated muscle had been examined. There appears to be little doubt that the difference in uptake in liver and thyroid is greater than in striated muscle. This concept is strengthened by the fact that the uptake by liver number 3 was 0.173 µg iron/100 mg tissue and from striated muscle number 24, from the same patient, was 0.012 µg iron/100 mg tissue.

Only small differences in uptake were noted in the single samples of myocardium, smooth muscle, and spleen examined. The small difference in uptake in spleen number 27 (0.010 µg Fe/100 mg tissue) is specially significant when it is compared with that of the liver (No. 2) of the same patient (0.078 µg Fe/100 mg tissue). The difference in uptake in pancreas and parotid gland was also fairly considerable but there were too few samples to allow statistical comparison with other tissues.

The variation in difference of uptake between samples of the same tissue is striking. This value in liver sample number 1 was

very low being only 0.012 µg Fe/100 mg tissue while in liver number 26 it was 2.312 µg Fe/100 mg tissue. One possible explanation for this is that in sample number 1 stainable iron +++ was found in the hepatic cells, while in sample number 26, the patient was suffering from severe iron deficiency anaemia and, of course, had no stainable iron in the hepatic cells. This could mean that the rate of uptake of available iron from serum by hepatic cells depended, at least partly, on the amount of iron in the cells. If this were so it would agree well with the findings of Saltman et al. (1956) that the uptake of iron by rat liver cells was initially rapid but later became slower and finally there was no further uptake (presumably when the cells were loaded with iron).

There was also considerable variation in difference of uptake between samples of thyroid tissue. In sample number 11 the difference was negligible while in sample number 14 it was 0.215 µg Fe/100 mg tissue. This variation may be in some way related to activity of the thyroid follicles, as it was noted that in sample number 11 the follicles were very large and distended with colloid while in sample number 14 the follicles were small and fairly normal looking.

It cannot be stated from this experiment how much of the uptake of ^{59}Fe by the tissues in transferrin approximately half saturated is due to non-specific trapping of the serum in the tissue, and how much is due to uptake by the tissue cells.

The findings in this experiment appear to confirm the theory that the uptake of iron by human tissues from serum is much greater

when there is a high percentage saturation of transferrin than when the percentage saturation is normal. It also shows that this difference in uptake is very much greater in epithelial tissues such as liver, thyroid, pancreas and salivary gland than in connective tissues such as striated muscle, smooth muscle, myocardium and spleen.

PART V

THE EFFECT OF A LARGE DOSE OF ORAL IRON ON SERUM IRON LEVELS

It has been shown in Part IV of this Section that human liver, and a number of other epithelial tissues, readily take up iron from serum when there is a high percentage saturation of transferrin. It is easy therefore to understand the widespread epithelial distribution of iron in subjects with a more or less constant high iron saturation of transferrin. There are however instances in which small iron deposits are seen in epithelial tissues of patients whose livers are not cirrhotic (see Appendix V) and whose percentage saturation of transferrin is normal (see Case D 29, Table XXIII).

Iron tolerance tests have shown that S.I. levels rise to a maximum about 2 to 3 hours after ingesting iron (Wiltink, Ybema, Leijnse and Gerbrandy, 1966) then gradually fall to the original level. There is a period, when the S.I. is raised, that the transferrin is almost completely saturated and it was felt, that if such a transient period of high percentage saturation of transferrin occurred in Africans after drinking home-brewed beer, this might account for the small epithelial deposits of iron found in the cases referred to above.

It was thought that some useful information might be gained with reference to this point, by giving a number of patients an oral dose of iron approximately equal in amount to that which might be consumed during a heavy beer drink. It would have been ideal to have given

the iron in the form of beer but this was impracticable so a pharmaceutical preparation of iron was substituted.

Material and Methods

It has been shown in Section V that the average iron content of home-brewed African beer is 9.4 mg/100 ml. Questions to African staff and patients indicate that commonly at a "beer drink" eight pints of beer are consumed and frequently there is much more taken. If the iron content of the beer is 9.4 mg/100 ml, eight pints would contain 451 mg of iron. An attempt to find the effect of this amount of iron on the serum iron and percentage saturation of transferrin was made by giving patients seven tablets of ferrous sulphate Co., B.P.C., which contained approximately 441 mg iron (each tablet contained approximately 63 mg iron). In order to simulate the acidity of the beer (average pH 3.9) $\frac{1}{2}$ ounce Gentian and acid mixture B.P.C. was administered with the tablets.

Blood was taken from each patient between 8.15 a.m. and 8.45 a.m. and the S.I., U.I.B.C. and haemoglobin values estimated as in Section VI. The PCV was also measured. As soon as the blood had been withdrawn, the patient was given the tablets of ferrous sulphate and gentian acid mixture. In all cases the tablets and mixture were consumed in the presence of the writer. Further blood samples were taken after exactly 2 hours and after 24 hours, for repeat estimation of serum iron and U.I.B.C. Five patients with cirrhosis refused to have the third sample of blood taken.

The patients chosen were 18 who had been diagnosed clinically as having cirrhosis, and 12 who clinically did not have cirrhosis and whose liver function tests were normal. In 4 of the cirrhotic patients the diagnosis was confirmed by biopsy and in all of the remaining cirrhotic cases a nodular liver was felt. Unfortunately clinicians in charge of these cases did not consider that liver biopsies were justifiable in all subjects, so it is possible that some had also primary carcinoma of liver.

Results

The results are contained in Table XXVII.

There were two non-cirrhotic, and twelve cirrhotic subjects with haemoglobin values of less than 13.5 G/100 ml, i.e. were suffering from anaemia according to de Gruchy's definition (de Gruchy, 1960).

In all subjects without cirrhosis, the serum iron 2 hours after taking the oral iron compound was substantially raised and the transferrin was more than 60% saturated. Also in all cases both S.I. and percentage saturation had returned to approximately the initial level after 24 hours.

Results in the cirrhotic subjects were much more variable. In a number, the increase in S.I. two hours following the dose of iron was slight, and in one patient (No. 17) it was actually slightly lower than the first value. Also in most cirrhotic subjects, the serum iron after 24 hours was rather higher than that found before the iron was given.

TABLE XXVII

EFFECT OF A LARGE ORAL DOSE OF IRON ON SERUM IRON LEVELS & PERCENTAGE SATURATION OF TRANSFERRIN

NON-CIRRHOTICS

Ref. No.	Hb. g/100 ml	PCV	Before Administration of Iron			2 Hours after Administration of Iron			24 Hours after Administration of Iron		
			S.I. µg/100 ml	T.I.B.C. µg/100 ml	% Sat'n.	S.I. µg/100 ml	T.I.B.C. µg/100 ml	% Sat'n.	S.I. µg/100 ml	T.I.B.C. µg/100 ml	% Sat'n.
1*	14.3	43	260	530	49.0	347	529	65.7	270	528	51.1
2*	15.9	47	188	533	35.3	534	561	95.0	189	530	35.7
3*	15.1	42	200	397	50.4	330	416	79.3	191	401	47.6
4	14.7	42	74	219	33.8	191	212	90.1	70	222	31.5
5	12.9	40	76	279	27.2	204	289	71.3	71	282	25.2
6	16.5	47	168	395	42.5	301	387	77.8	160	394	40.6
7	13.6	48	105	318	33.0	298	344	86.6	101	324	31.2
8	16.5	45	148	383	38.7	262	377	69.5	148	390	37.9
9	15.1	44	147	402	36.6	347	409	84.9	146	410	35.6
10	15.6	49	196	434	45.2	365	440	83.0	203	425	47.8
11	12.9	41	80	315	25.4	225	304	74.0	82	308	26.6
12	14.1	47	171	415	41.2	370	408	90.8	120	413	29.1

* Biopsy showed moderately heavy deposits of stainable iron in the liver. There was no liver fibrosis.

TABLE XXVII CONTD.

EFFECT OF A LARGE ORAL DOSE OF IRON ON SERUM IRON LEVELS & PERCENTAGE SATURATION OF TRANSFERRIN

CIRRHOTICS

Ref. No.	Hb. g/100 ml	PCV	Before Administration of Iron			2 Hours after Administration of Iron			24 Hours after Administration of Iron		
			S.I. µg/100 ml	T.I.B.C. µg/100 ml	% Sat'n.	S.I. µg/100 ml	T.I.B.C. µg/100 ml	% Sat'n.	S.I. µg/100 ml	T.I.B.C. µg/100 ml	% Sat'n.
1	12.1	40	33	377	8.8	249	382	65.2	80	380	21.1
2*	10.0	32	121	143	84.6	129	144	89.6			
3	14.7	48	66	225	29.4	129	215	60.0	91	217	41.9
4	11.9	38	18	149	12.1	73	146	50.0			
5	14.7	42	67	309	21.6	210	305	68.8	87	310	28.1
6	11.3	34	137	407	33.4	173	412	42.0			
7	15.3	43	84	255	33.0	128	250	51.2	88	257	34.3
8	8.8	27	123	254	48.5	146	247	59.2	135	263	51.3
9*	10.8	29	144	160	90.1	148	160	92.4			
10	6.8	27	18	470	3.8	60	474	12.6			
11	9.4	34	68	354	19.2	72	361	19.9	92	368	25.0
12	17.8	46	170	346	49.2	306	344	89.0	228	334	68
13	9.2	29	23	238	9.7	133	233	57.1	68	230	32.5
14	9.6	44	114	237	48.1	142	248	57.2	114	257	44.4
15	8.9	33	79	240	32.9	168	254	66.2	90	227	39.6
16+	12.6	42	47	185	25.4	83	200	41.4	50	196	25.5
17*	13.9	52	201	230	87.4	195	228	85.6	180	216	83.4
18	16.2	45	205	294	69.7	260	302	86.1	238	301	79.1

* Liver biopsy showed moderate deposits of stainable iron and a fine cirrhosis.

+ Liver biopsy showed no stainable iron. There was a coarse cirrhosis.

Discussion

The results appear to indicate that, in subjects without cirrhosis of liver, an oral dose of iron, such as would be ingested at a heavy "beer drink", gives rise to a transient increase in serum iron and high percentage saturation of transferrin. Some of this iron, being loosely bound to transferrin, would be deposited in epithelial cells (Jandl et al., 1959; and Part IV of this Section) during the period of high transferrin saturation. The amount of iron deposited in epithelial tissues would then depend on the frequency of heavy "beer drinks". That is, a regular heavy drinker would be expected to have stainable iron deposits in epithelial cells even though he did not have fine cirrhosis and had normal transferrin saturation levels between beer drinks. Possibly Cases D 2; D 32; and D 38 in Appendix V are typical examples of this but unfortunately neither the S.I. and T.I.B.C. values nor the drinking habits were known in these cases.

In subjects with cirrhosis of liver the results are more difficult to interpret. Only 5 cases, among those with an initially normal transferrin level, showed such an increase in serum iron as to cause the transferrin to be more than 60% saturated. There were 4 cases whose transferrin saturation was greater than 60% before the iron was given, and in none of those did the oral iron produce much of a rise in serum iron. This may have been due to the fact that these subjects had siderosis, and iron overload depresses absorption of iron from the gut (Bothwell et al 1958; Pirzio-Biroli and Finch 1960).

In three cases in which the S.I. was less than 25 µg/100 ml and the haemoglobin low, there was a fairly marked rise in serum iron, but not to such an extent as to cause more than 60% saturation of transferrin. It seems probable that these people were suffering from some degree of iron deficiency, and that much of the absorbed iron was deposited in the liver cells, thus did not enter the systemic circulation in sufficient amounts to saturate the transferrin. This suggested explanation receives some confirmation from the results in Part IV where it was seen that slices of liver from a patient suffering from iron deficiency anaemia took up abnormally large amounts of iron from highly saturated transferrin. A similar explanation might apply in Case 16, though the iron deficiency was probably not so severe as in the other 3 cases as the initial serum iron value was higher.

It is perhaps rather surprising that there was not a greater response to the oral iron by the serum iron in the cirrhotic patients, because it has been shown that there is increased absorption of iron in patients with cirrhosis (Conrad et al., 1962; Greenberg et al.1964; Friedman et al.1966). It is possible however, the effect that this increased absorption would normally have on the serum iron has been modified in the cases examined by the complicating factors of iron deficiency, or iron overload referred to above.

The results of this experiment show that following the oral intake of a large dose of iron in non-cirrhotic subjects there is a transient period during which the transferrin is highly saturated.

It is suggested that this transient period of transferrin saturation occurs in Africans following a "beer drink" and that this explains the, usually slight, epithelial deposits of iron in siderotic subjects without cirrhosis.

CONCLUSIONS

The results of the experimental work recorded in this Section seem to indicate that:

1) as the red cell life span and red cell fragility are normal in healthy male Africans, these factors cannot account for the heavy iron deposits found in the reticuloendothelial system in subjects with Bantu siderosis. It is probable then that the reticuloendothelial deposits are a result of infection as suggested in Section IV, or possibly some toxic substance in the home-brewed beer, as suggested by the guinea pig experiments, or a combination of both of these factors.

2) the absence of any significant difference in iron concentration between the heads and tails of pancreas in cirrhotic subjects makes it unlikely that diversion of blood, from the cirrhotic liver through anastomotic channels, is responsible for epithelial deposits of iron in siderotic subjects with cirrhosis.

3) because home-brewed African beer, when fed to guinea pigs, produces a siderosis in which the body iron distribution is the same as in Bantu siderosis, the beer is probably the source of the iron found in African siderotics thus confirming previous evidence to this effect.

4) because, in vitro, a) human tissues take up iron from transferrin that is almost completely saturated much more readily then from transferrin that is only half saturated b) the iron

uptake by epithelial tissues is greater than by connective tissues,
c) the iron uptake by thyroid tissue is not significantly different from that of liver; therefore

i) the widespread distribution of stainable iron in epithelial tissues found in idiopathic haemochromatosis, transfusional siderosis, certain cases of Bantu siderosis etc. is probably due to the high percentage saturation of transferrin found in these conditions.

ii) the heavier deposits of iron in epithelial than connective tissues is explained.

iii) the liver is the main organ in the body for iron storage largely, if not entirely, by virtue of its proximity to the iron inflow and not through any special ability to store iron.

5) the transient rise in serum iron and percentage saturation of transferrin, found after a large oral dose of iron, may occur in Africans following the consumption of a large amount of home-brewed beer and thus account for the variable deposits of stainable iron in a number of epithelial tissues in subjects whose transferrin saturation levels are normal between beer drinks.

SECTION VIII

GENERAL DISCUSSION & CONCLUSIONS

Comparison Between Bantu Siderosis in Rhodesia and South Africa

Evidence produced in Sections II and IV demonstrate conclusively that there are no significant differences between the Bantu siderosis found in the Africans of Rhodesia and South Africa. The incidence and degree of the siderosis is about the same in the two countries, as is the iron distribution in the bodies of cirrhotic and non-cirrhotic subjects.

In comparing the condition in Rhodesia and South Africa a number of small points are perhaps worth noting.

1) In South Africa there have been a number of reports of generalised osteoporosis with collapse of lumbar vertebrae occurring in Africans with severe siderosis (Walker, Strydom, Reynolds and Grobbelaar, 1955; Seftel et al.,1966). This does not appear to occur in Rhodesia, though one case has recently been reported from Zambia (Lowenthal, Siddorn, Patel and Fine, 1967). Seftel et al. (1966) showed that, in their group of patients in Johannesburg, this vertebral collapse in siderotics was associated with scurvy. Personal experience of the writer indicates that overt scurvy is rare amongst Africans in Salisbury, which could explain the absence of this form of osteoporosis in Rhodesia.

2) A number of cases of "idiopathic" peritonitis have been seen at autopsy in Rhodesia in subjects with severe siderosis (see Section III). No such cases have yet been reported from South Africa.

3) Three cases of terminal shock occurring in patients who, at autopsy, were found to have severe siderosis are reported in Section VI. These cases have been likened to the shock found occasionally in patients with idiopathic haemochromatosis (Desforges, 1949; Klecknor et al., 1955; Jones, 1962). One further case (D 8, Page 67, Volume II) who probably died of shock was that of a middle aged African who died shortly after a road accident. Autopsy showed that he had severe siderosis but his injuries were relatively trivial viz. moderate contusion of one thigh and simple fracture of the tibia, and there was no histological evidence of fat emboli in the brain. Such cases of shock causing death are said to occur commonly in idiopathic haemochromatosis following even relatively minor surgical procedures (Finch and Finch, 1955).

No cases of shock in subjects with Bantu siderosis have been reported from South Africa.

The Actiology of Bantu Siderosis

In South Africa it has been demonstrated beyond reasonable doubt that Bantu siderosis results from the ingestion of enormous amounts of iron in food and alcoholic beverages (Walker and Arvidsson, 1950; Walker, 1951; Walker and Arvidsson, 1953; Bothwell et al. 1964).

Results of analysis of the iron content of the Rhodesian African diet and a survey of drinking habits also show that there is a sufficiently great amount of iron consumed in cooked food and African beer to account for the degree of siderosis found. The home-brewed beer when fed to guinea pigs produced a siderosis with an iron distribution similar to that found in Bantu siderosis.

Comparison Between Bantu Siderosis and Idiopathic Haemochromatosis

In South Africa it has been said that some subjects with Bantu siderosis and cirrhosis "develop pathological findings which are virtually indistinguishable from those in idiopathic haemochromatosis" (Isaacson et al, 1961). Similar cases are also seen in Rhodesia. It is felt however, that though the iron distribution is very similar to that found in idiopathic haemochromatosis, the conditions can be distinguished in that iron deposition in the spleen and upper small bowel mucosa is almost always much heavier in Bantu siderosis than the average given for haemochromatosis by Sheldon (1935). A further similarity between these cases of Bantu siderosis with cirrhosis and idiopathic haemochromatosis is the frequency with which diabetes is seen in both (Sheldon, 1935; Seftel et al., 1960). Examples of diabetes in siderotics were seen in the course of this work, such as Case D 22, Page 67, Appendix V.

It is suggested that the greater incidence of infection in subjects with Bantu siderosis as compared with that in idiopathic haemochromatosis, is responsible for the heavy iron deposits in the reticuloendothelial system of the former (Section IV). The heavy deposits in the small bowel mucosa in Bantu siderosis are attributed to the absorption of the massive amounts of iron from the lumen of the gut. In idiopathic haemochromatosis there is increased absorption of normal dietary iron (Finch and Finch, 1955) and therefore less likelihood of a local buildup of iron in the bowel mucosa.

The Probable Cause of the Widespread Epithelial Deposits of Iron in Some Cases of Bantu Siderosis

One striking feature of Bantu siderosis is the widespread epithelial deposits of iron in some cases, and its absence in others with an equal or even greater degree of hepatic siderosis. This distribution of iron is usually, but not invariably, associated with fine cirrhosis of liver.

It was suggested by Bothwell (1964) that these epithelial deposits of iron, other than in the liver, were caused by a high degree of saturation of circulating transferrin. Evidence has been presented in this work which appears to confirm Bothwell's thesis. This view is however not shared by MacDonald, Friend, Pechet, Pechet and Appelbaum (1967), who state that the degree of saturation of the serum iron binding protein is not important in determining the characteristic distribution of iron in experimental haemochromatosis. It is contended by MacDonald and his co-workers (MacDonald, Jones and Pechet, 1965) that deposits of iron in epithelial tissues of subjects with idiopathic haemochromatosis could be due to folic acid deficiency and they produced experimental evidence on rats in support of this view.

If folic acid deficiency is responsible for the extra-hepatic epithelial deposits of iron, as suggested by MacDonald et al. (1965), it is difficult to explain why Dellor, Kimber and Ibbotson (1965) found reduced levels of folic acid activity in only one out of eight cases of idiopathic haemochromatosis, a condition in which extra-

tissues used for the experiment were folic acid deficient (which is possible in Africans because of poor diet (Howard, 1967)), the folic acid deficiency theory would still be compatible with the results. Indeed a variable degree of folic acid deficiency in the patients chosen for the experiment may have been responsible for the variability in iron uptake found in different specimens of the same tissue.

It is felt therefore that though most of the evidence produced in this work indicates that high percentage saturation of transferrin is the most important single factor in producing extra-hepatic epithelial deposition of iron in siderotic subjects, the importance of the role of folic acid deficiency in this respect has not yet been clarified. Possibly both factors are important.

Is Siderosis Harmful?

South African workers have demonstrated that, in subjects with Bantu siderosis, the incidence of portal fibrosis increases as the concentration of iron in the liver rises (Bothwell and Bradlow, 1960; Isaacson et al., 1961). In Section III it was shown that a similar relationship existed between liver fibrosis and degree of siderosis in Rhodesian Africans.

It would be wrong to conclude that iron is responsible for the fibrosis on the strength of this evidence alone, as there are livers which contain massive deposits of iron with minimal portal fibrosis. Such a case is BS/53/7 (Page 62, Volume II). A portal area in the liver of this case is illustrated in Plate II, Page II2. Furthermore, as the main source of the iron in Bantu siderosis is home-brewed African beer, those subjects with severe siderosis must consume large quantities of beer. If the beer contained some toxic substance which produced cirrhosis, the amount of this ingested would approximately parallel the amount of iron ingested, and so the extent of liver fibrosis would parallel the degree of iron overload even though the iron was completely inert. The existence of such a toxic substance in beer is at present hypothetical but a substance of this type is a more likely cause of liver fibrosis than iron, as most of the experimental work on animals (quoted in Section III) has shown that haemosiderin does not provoke a fibrous tissue reaction in liver.

It is concluded that if iron is fibrogenic at all, it is so only to a very minor extent. This does not mean that severe

siderosis is completely harmless because it has been shown that "idiopathic" peritonitis and irreversible shock occasionally occur in these cases. Also South African workers have demonstrated osteoporosis and vertebral collapse in subjects with severe siderosis and scurvy.

Haemofuscin

In this study the pigment haemofuscin (lipofuscin) has been ignored as it is, at the present day, widely believed to be of little importance in iron metabolism (Sheldon, 1935; Finch and Finch 1955; Beutler, Fairbanks and Fahey, 1963; MacDonald, 1964).

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BANTU SIDEROSIS IN RHODESIA

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CONTENTS

APPENDIX I

HISTOLOGICAL GRADING OF IRON

IN LIVER, PANCREAS AND HEART OF 200 AFRICANS

Type of Cirrhosis: C. = Coarse
F. = Fine

No.	Sex	Age	LIVER IRON			LIVER FIBROSIS		Pancreatic Iron	Heart Iron	Cause of Death
			Hepatic cells	Kuppfer cells	Portal areas	Portal Fibrosis	Type of cirrhosis			
1	M	2/52	-	-	-	-	-	-	-	Viral pneumonia
2	F	1	-	-	-	-	-	-	-	Malaria
3	M	1	-	-	-	-	-	-	-	Kwashiorkor/Broncho-pneumonia.
4	M	1	-	-	-	-	-	-	-	Kwashiorkor/Gastro-enteritis.
5	F	1	-	+	-	-	-	-	-	Bronchopneumonia
6	F	1	-	-	-	-	-	-	-	Bronchopneumonia
7	F	1	-	+	-	-	-	-	-	Kwashiorkor/Broncho-pneumonia
8	F	1	-	-	-	-	-	-	-	Bronchopneumonia
9	M	1	-	-	-	-	-	-	-	Bronchopneumonia
10	M	1	-	-	-	-	-	-	-	Gastro-enteritis
11	F	1 3/12	-	-	-	+	-	-	-	Bronchopneumonia
12	M	1½	-	-	-	-	-	-	-	Kwashiorkor/Broncho-pneumonia.
13	M	1½	-	-	-	-	-	-	-	Kwashiorkor/Viral pneu-monia.
14	F	1½	-	-	-	-	-	-	-	Gastro-enteritis.
15	M	1½	-	+	-	-	-	-	-	Bronchopneumonia
16	F	1½	-	-	-	-	-	-	-	Meningitis.
17	F	1½	-	-	-	-	-	-	-	Kwashiorkor/Broncho-pneumonia.
18	M	1½	-	-	-	-	-	-	-	Bronchopneumonia
19	F	1 7/12	+	-	-	+	-	+	+	Kwashiorkor/Broncho-pneumonia
20	M	1 9/12	-	-	-	-	-	-	-	Bronchopneumonia
21	F	2	-	-	-	-	-	-	-	Kwashiorkor/Broncho-pneumonia
22	F	1	+	-	-	-	-	-	-	Gastro-enteritis
23	M	2	-	-	-	-	-	-	-	Bronchopneumonia
24	M	2	-	-	-	-	-	-	-	Bronchopneumonia
25	M	2	-	-	-	+	-	-	-	Bronchopneumonia

No.	Sex	Age	LIVER IRON			LIVER FIBROSIS		Pancreatic Iron	Heart Iron	Cause of Death
			Hepatic cells	Kuppfer cells	Portal areas	Portal Fibrosis	Type of cirrhosis			
26	F	2	-	+	-	-	-	-	-	Bronchopneumonia
27	M	2	-	-	-	-	-	-	-	Bronchopneumonia
28	M	2	-	-	-	-	-	-	-	Bronchopneumonia
29	M	2	-	-	-	-	-	-	-	Bronchopneumonia
30	M	2	-	-	-	-	-	-	-	Road accident
31	M	2½	-	-	-	-	-	-	-	Kwashiorkor/Broncho-pneumonia.
32	F	$2\frac{10}{12}$	-	-	-	-	-	-	-	Strangulated interaal hernia
33	M	3	-	-	-	-	-	-	-	Drowning
34	M	3	-	-	-	-	-	-	-	Cerebral malaria
35	M	3	-	-	-	-	-	-	-	Kwashiorkor/Broncho-pneumonia.
36	F	3	-	-	-	-	-	-	-	Bronchopneumonia
37	M	3	-	-	-	-	-	-	-	Acute enteritis
38	M	3	+	-	-	-	-	-	-	Bronchopneumonia
39	M	3	-	-	-	-	-	-	-	Kwashiorkor/Broncho-pneumonia
40	M	3	-	-	-	+	-	-	-	Gastro-enteritis
41	M	3	+	+	-	-	-	-	-	Kwashiorkor/Broncho-pneumonia
42	M	3	-	-	-	-	-	-	-	Bronchopneumonia
43	M	3	-	-	-	-	-	-	-	Broncho-pneumonia
44	F	3	-	-	-	-	-	-	-	Fractured skull
45	F	3	+	-	-	-	-	-	-	Bronchopneumonia
46	M	3½	-	-	-	-	-	-	-	Kwashiorkor/Broncho-pneumonia
47	F	4	+	-	-	+	-	-	-	Purulent Meningitis
48	F	4	-	+	-	-	-	-	-	Road accident
49	M	4	-	+	-	+	-	-	-	Bronchopneumonia
50	F	4½	+	++	-	+	-	-	-	Mitral incompetence
51	F	4½	-	-	-	-	-	-	-	Road accident
52	M	5	-	-	-	+	-	-	-	Medullo Blastoma

No.	Sex	Age	LIVER IRON			LIVER FIBROSIS		Pancreatic Iron	Heart Iron	Cause of Death
			Hepatic cells	Kupffer cells	Portal areas	Portal Fibrosis	Type of cirrhosis			
53	M	5	-	-	-	-	-	-	-	Extensive burns
54	M	5	-	-	-	-	-	-	-	Bronchopneumonia
55	F	5	-	-	-	-	-	-	-	Bronchopneumonia
56	M	5	-	-	-	-	-	-	-	Bronchopneumonia
57	F	5	+	-	-	-	-	-	-	Gastro-enteritis
58	F	6	+	++	-	-	-	-	-	Acute peritonitis
59	F	6	+	-	-	-	-	-	-	Gastro-enteritis
60	M	8	-	+	-	++	-	-	-	Hydronephrosis
61	M	8	-	-	-	-	-	-	-	Drowning
62	M	10	-	-	-	-	-	-	-	Acute massive necrosis of liver
63	F	12	-	-	-	-	-	-	-	Lobar pneumonia
64	M	14	-	-	-	-	-	-	-	Traumatic intra-cranial haemorrhage
65	M	14	-	-	-	-	-	-	-	Electrocution
66	F	15	-	-	-	+++	-	-	-	Cor Pulmonale
67	M	20	+	-	-	-	-	-	-	Road accident
68	M	20	+	-	-	+	-	-	-	Meningococcal meningitis
69	F	20	-	-	-	-	-	-	-	Road accident
70	F	20	+	++	-	-	-	-	-	Miliary tuberculosis
71	F	20	+	+	-	+	-	-	-	Uraemia-ureteric stenosis
72	M	20	++	-	-	-	-	-	-	Pericarditis
73	F	22	-	-	-	-	-	-	-	Severe epistaxis
74	M	22	+	-	-	-	-	-	-	Traumatic intra-cranial haemorrhage
75	M	23	+	-	-	++	-	-	-	Road accident
76	M	24	+	-	-	-	-	-	-	Fracture cervical vertebra
77	M	25	-	-	-	-	-	-	-	Traumatic brain damage
78	F	25	-	+++	-	-	-	-	-	Haemorrhage following caesarian section

No.	Sex	Age	LIVER IRON			LIVER FIBROSIS		Pancreatic Iron	Heart Iron	Cause of Death
			Hepatic cells	Kuppfer cells	Portal areas	Portal Fibrosis	Type of cirrhosis			
79	F	25	++	+	-	-	-	-	-	Thrombocytopaenic purpura
80	M	25	-	-	-	-	-	-	-	Empyema
81	M	25	+++	+++	+++	+++	-	-	-	Road accident
82	M	25	+	-	-	-	-	-	-	Algid Malaria
83	F	25	-	-	-	-	-	-	-	Status epilepticus
84	M	25	-	-	-	+	-	-	-	Road accident
85	F	25	-	+	-	-	-	-	-	Eclampsia
86	F	25	-	-	-	-	-	-	-	Ruptured ectopic pregnancy
87	F	27	-	-	-	-	-	-	-	Ruptured ectopic pregnancy
88	F	26	-	-	-	-	-	-	-	Hypertension, Cerebral haemorrhage
89	F	27	-	-	-	-	-	-	-	Lobar pneumonia
90	M	25	+	-	-	-	-	-	-	Status epilepticus
91	M	30	-	-	-	-	-	-	-	Road accident
92	M	30	++	+++	+	-	-	-	-	Road accident
93	M	30	-	-	-	-	-	-	-	Traumatic brain damage
94	M	35	+	+	-	+	-	-	-	Extensive burns
95	F	30	-	-	-	-	-	-	-	Traumatic intra-cranial haemorrhage
96	M	30	++	+	-	+	-	-	-	Asphyxia due to inhalation of vomit.
97	M	30	+++	+++	+++	++++	C	+	-	Hypertensive cardiac failure
98	F	30	-	-	-	-	-	-	-	Ruptured uterus
99	M	30	++	+	-	-	-	-	-	Status asthmaticus
100	F	30	-	-	-	-	-	-	-	Asphyxia due to inhalation of vomit.
101	F	30	-	-	-	-	-	-	-	Road accident
102	F	30	-	-	-	-	-	-	-	Tetanus
103	M	30	++	-	+	+	-	-	-	Volvulus of small bowel
104	F	34	-	-	-	-	-	-	-	Ruptured ectopic pregnancy
105	M	35	++	+++	+	-	-	-	-	Hypertensive heart failure
106	M	35	+++	+++	+++	-	-	-	-	Multiple injuries

No.	Sex	Age	LIVER IRON			LIVER FIBROSIS		Pancreatic Iron	Heart Iron	Cause of Death
			Hepatic cells	Kuppfer cells	Portal areas	Portal Fibrosis	Type of cirrhosis			
107	M	35	+	-	-	-	-	-	-	Extensive burns
108	M	35	+++	+++	+++	+	-	-	-	Road accident
109	M	35	+	-	-	-	-	-	-	Fractured skull
110	M	35	-	-	-	-	-	-	-	Road accident
111	M	35	+	+	-	+	-	-	-	Road accident
112	M	35	+++	+++	+++	-	-	-	-	Tuberculous meningitis
113	M	35	-	-	-	+	-	-	-	Bronchopneumonia/malaria
114	M	35	-	-	-	-	-	-	-	Road accident
115	M	35	++	+++	+++	++++	F	+++	+	Acute peritonitis
116	M	35	-	-	-	-	-	-	-	Road accident
117	M	35	-	-	-	+	-	-	-	Status asthmaticus
118	M	35	+++	+++	+++	+	-	-	-	Road accident
119	F	35	-	+	-	+	-	-	-	Lobar pneumonia
120	M	35	-	-	-	-	-	-	-	Fractured skull
121	M	35	++	++	+	+	-	-	-	Road accident
122	F	35	-	-	-	-	-	-	-	Congestive heart failure. Hypertension.
123	F	35	-	-	-	-	-	-	-	Extensive burns
124	M	35	+	-	-	-	-	-	-	Road accident
125	F	35	-	-	-	-	-	-	-	Uraemia, chronic pyelo-nephritis.
126	M	36	+++	+++	+++	-	-	-	-	Pemphigus vulgaris
127	F	40	++	+	+	+	-	-	-	Ruptured ectopic pregnanc
128	M	40	-	-	-	-	-	-	-	Road accident
129	M	40	+++	+++	+++	-	-	-	-	Bacillary dysentery
130	F	40	+	+	-	-	-	-	-	Carcinoma of liver
131	M	40	-	-	-	-	-	-	-	Pulmonary tuberculosis
132	F	40	+	+	-	-	-	-	-	Road accident
133	M	40	++	+++	+	+	-	-	-	Cerebral haemorrhage. Hypertension.
134	M	40	+++	+++	+++	-	-	-	-	Hodgkin's disease

No.	Sex	Age	LIVER IRON			LIVER FIBROSIS		Pancreatic Iron	Heart Iron	Cause of Death
			Hepatic cells	Kupffer cells	Portal areas	Portal Fibrosis	Type of cirrhosis			
135	M	40	++	+++	+++	-	-	-	-	Toxaemia - old spinal injury with paraplegia
136	M	40	+++	+++	+++	+	-	-	-	Bronchopneumonia
137	M	40	+	-	-	+	-	-	-	Road accident
138	M	40	+	-	-	-	-	-	-	Road accident
139	M	40	++	+	-	+	-	-	-	Road accident
140	M	40	++	-	-	-	-	-	-	Malaria
141	M	40	-	-	-	-	-	-	-	Perforated duodenal ulcer
142	M	40	-	-	-	-	-	-	-	Haemorrhage from bowel
143	F	40	+++	+++	+++	++	-	+	-	Asphyxia due to inhalation of vomit.
144	M	40	-	-	-	-	-	-	-	Road accident
145	M	40	+++	+++	++	+	-	-	-	Road accident
146	F	40	+++	+++	++	+	-	-	-	Carcinoma of bladder
147	F	40	+	-	-	+	-	-	-	Status asthmaticus
148	M	40	++	+++	+++	+	-	-	-	Peritonitis
149	F	40	-	-	-	-	-	-	-	Acute peritonitis, salpingitis.
150	M	40	-	-	-	+	-	-	-	Road accident
151	M	40	++	+++	+++	-	-	-	-	Road accident
152	M	40	+++	+++	+++	+	-	+	-	Bronchial carcinoma
153	M	44	+	-	-	++	-	-	-	Acute pancreatitis
154	M	45	-	-	-	-	-	-	-	Carcinoma of liver
155	M	45	++	-	-	+	-	-	-	Road accident
156	M	45	-	-	-	-	-	-	-	Fractured ribs / bronchopneumonia.
157	M	45	+	++	-	+	-	-	-	Traumatic intracranial haemorrhage
158	M	45	++	+	+++	+	-	-	-	Extensive burns
159	M	45	++	+++	+	-	-	-	-	Fractured cervical vertebrae
160	M	45	+	-	-	-	-	-	-	Road accident
161	M	45	+	-	-	++	-	-	-	Cerebral malaria

No.	Sex	Age	LIVER IRON			LIVER FIBROSIS		Pancreatic Iron	Heart Iron	Cause of Death
			Hepatic cells	Kuppfer cells	Portal areas	Portal Fibrosis	Type of cirrhosis			
162	M	50	-	-	-	-	-	-	-	Carcinoma of liver
163	M	50	+++	++	++	++++	F	++	-	Ruptured oesophageal varices.
164	M	50	+++	+++	+++	++++	F	++	+	Carcinoma of liver
165	F	50	+++	+++	+++	++++	F	+++	+	Acute pulmonary oedema
166	M	50	+++	++	+++	+	-	+	-	Road accident
167	M	50	-	-	-	-	-	-	-	Road accident
168	M	50	++	++	-	+	-	-	-	Road accident
169	M	50	+	+	++	+++	C	-	-	Carcinoma of bronchus
170	M	50	+	+	+	+	-	-	-	Road accident
171	M	50	+++	+++	+++	++++	F	++	+	Purulent pericarditis
172	F	50	+++	+++	+++	++++	F	+++	-	Carcinoma of ovary
173	M	50	+++	+++	+++	+	-	+	-	Road accident
174	M	50	++	+	-	-	-	-	-	Carcinoma of oesophagus
175	M	50	+	-	-	-	-	-	-	Road accident
176	M	50	++	+	+	-	-	-	-	Carcinoma of oesophagus
177	M	55	++	+++	+++	++	-	-	-	Road accident
178	F	55	-	-	-	-	-	-	-	Isolated myocarditis
179	M	55	+++	+++	+++	++	-	+	-	Hypoglycaemic coma
180	F	55	-	-	-	+	-	-	-	Intra-cranial haemorrhage Hypertension
181	M	55	-	-	-	+	-	-	-	Suicidal hanging
182	M	60	++	++	+	-	-	-	-	Ruptured aortic aneurysm
183	F	60	++	+++	+++	++	-	+	-	Road accident
184	M	60	-	-	-	+	-	-	-	Cor pulmonale
185	M	60	+	-	-	+	-	-	-	Carcinoma of oesophagus
186	F	60	++	+++	++	++++	F	++	+	Acute peritonitis
187	F	60	-	-	-	-	-	-	-	Congestive cardiac failure.
188	F	60	-	-	-	-	-	-	-	Hypertensive heart failure.
189	F	65	-	-	-	-	-	-	-	Road accident

No.	Sex	Age	LIVER IRON			LIVER FIBROSIS		Pancreatic Iron	Heart Iron	Cause of Death
			Hepatic cells	Kuppfer cells	Portal areas	Portal fibrosis	Type of cirrhosis			
190	M	65	+++	+++	+++	+	-	-	-	Toxic myocarditis
191	M	65	+++	+	+	-	-	-	-	Carcinoma of prostate. Bronchopneumonia
192	M	65	++	+++	+	+	-	-	-	Road accident
193	F	65	-	-	-	-	-	-	-	Extensive burns
194	M	65	+++	++	+++	-	-	-	-	Anaesthetic death
195	M	65	+++	++	++	-	-	-	-	Uraemia
196	F	65	+++	+++	+++	++++	F	+++	+	Ruptured oesophageal varices.
197	M	65	+++	++	++	+	-	-	-	Pulmonary embolus
198	M	70	+	-	-	-	-	-	-	Hypertensive cardiac failure
199	M	70	+	-	-	+	-	-	-	Carcinoma of lung
200	F	80	-	-	-	+	-	-	-	Carcinoma of bladder

APPENDIX II

TISSUE STORAGE IRON

TISSUE STORAGE IRON

The method used to estimate storage iron in tissues was that of Bothwell et al., (1964). All reagents used were "Analar" grade. Before use all glassware was rinsed in concentrated hydrochloric acid, rinsed six times in tap water (iron content very low) and lastly rinsed in deionized water. The fixed tissue, having been dried on blotting paper as described, was weighed on a "Mettler" balance, Type H.15 and the weight to the nearest milligramme recorded. Optical densities of the solutions were measured using a Hilger "Uvispek" spectrophotometer.

In the early stages of the study estimations were carried out in duplicate but owing to the complete absence of technical help (none being available even to help with cleaning the glassware) this had to be abandoned. However, as Bothwell was interested in the iron content of Rhodesian African livers, 214 specimens were sent to him. He made his results available in due course and comparison with my results showed the correlation to be good.

Furthermore some idea of the accuracy of the method was obtained by estimating several samples taken from different parts of the right lobe of the same liver and analysing the results obtained for a) storage iron, b) haemoglobin iron

Results a) Storage iron concentration

Specimen	Storage Iron mg/g wet weight	Number of samples
Liver No. 1	0.204, 0.184, 0.212, 0.198, 0.194, 0.235, 0.202 0.237, 0.230, 0.248, 0.213, 0.163	12
Liver No. 2	0.951, 1.085, 1.039, 1.003, 0.995, 1.063	6
Liver No. 3	0.108, 0.095, 0.113, 0.103, 0.121, 0.132, 0.184	7
Liver No. 4	0.223, 0.178, 0.155, 0.130	4
Liver No. 5	2.993, 3.095, 3.646, 3.425	4

These specimens with respect to the storage iron gave the following means and standard deviations.

Specimen	Mean	Standard Deviation
Liver No. 1	0.228	0.027
Liver No. 2	1.090	0.049
Liver No. 3	0.122	0.030
Liver No. 4	0.174	0.044
Liver No. 5	3.290	0.365

The high standard deviation found in liver No. 5 may have been partly due to a genuine variation in iron concentration as this liver was cirrhotic and histologically a variation in iron concentration in pseudolobules is commonly seen in cirrhosis.

b) Haemoglobin iron concentration.

Specimen	Haemoglobin Iron mg/g wet weight	Number of samples
Liver No. 1	0.017, 0.018, 0.016, 0.020, 0.022, 0.014, 0.018 0.015, 0.016, 0.018, 0.022, 0.017	12
Liver No. 2	0.064, 0.064, 0.064, 0.068, 0.066, 0.076	6
Liver No. 3	0.051, 0.048, 0.054, 0.053, 0.057, 0.053, 0.057	7
Liver No. 4	0.013, 0.020, 0.017, 0.017	4
Liver No. 5	0.007, 0.005, 0.004, 0.005	4

Specimen	Mean	Standard Deviation
Liver No. 1	0.018	0.0025
Liver No. 2	0.067	0.0045
Liver No. 3	0.053	0.0031
Liver No. 4	0.017	0.0029
Liver No. 5	0.005	0.0013

As the results show in both storage iron and haemoglobin iron the values for the standard deviation tend to increase with the mean. This fact detracts from the value of an overall measure of the error. However, with storage iron the calculated value for the overall standard deviation is 0.105 giving a coefficient of variation of 15%, the overall mean being 0.708. In respect to haemoglobin iron the overall mean is 0.0325 and standard deviation 0.0032 giving a coefficient of variation of 10%.

In view of the fact that many workers have expressed their results as weight of iron per unit dry weight of tissue the water content of a small series of livers was measured in an attempt to find a factor to convert dry weight into wet weight.

An aliquot of the fixed liver was dried on blotting paper as described by Bothwell et al., It was weighed, the placed in a small crucible and dried in a hot air oven at 100°C to constant weight and re-weighed. The water content is shown below.

Specimen	% Water
Liver No. 1	78.2
Liver No. 2	77.2
Liver No. 3	77.6
Liver No. 4	77.1
Liver No. 5	74.6
Liver No. 6	74.4
Liver No. 7	70.1
Liver No. 8	72.6
Liver No. 9	71.8
Liver No. 10	73.0
Mean	74.7

The average water content found by Brückman and Zondek (1939) in their series of livers (49) was 76.2%. It was considered therefore that an approximate average water content of liver was 75% and that to convert dry weight into wet weight the former value could be divided by four, to give a fairly good approximate value for the latter.

Several points were noted about the method after the first few estimations had been carried out:

1) if the heat applied during the course of the wet oxidation were not intense enough even if applied for the prescribed time, the colour developed on adding the reagents was brownish-orange instead of violet. This was probably due to the presence of nitrous acid (Swank & Mellon, 1938).

2) When liver specimens were very fatty, as in children with kwashiorkor, the liquid after the wet oxidation was often yellow in colour. This has been attributed by Monier-Williams (1950) to action of the nitric acid on fat and he suggests the addition of ammonium oxalate to the solution, which should be then further heated. Because of this, in this study when fatty livers were being estimated, after the addition of the hydrogen peroxide and heating a millilitre of a saturated solution of ammonium oxalate was added and heating continued for a further ten minutes. In most cases a colourless solution resulted but in a few there was still a slight yellow tinge. This did not appear to affect the development of a pure violet colour when the reagents were added.

3) It was found that if the liver specimens were deeply bile stained the formula for finding the corrected value for pyridine haemochromagen failed. This was because the optical density at 470 mμ was greater than 2.7 x the optical density at 540 mμ. In the few cases that this happened, the haemoglobin iron concentration was estimated histologically after comparing with several similar sections where

the (chemical) concentration of haemoglobin iron was known.

4) The haem iron content of the spleen was estimated in the same way as the liver except that the pyridine haemochromagen reading at 540 mμ was used uncorrected on the graph since the amount of blood is high in relation to other pigments (Bothwell, 1964).

EUROPEAN MALES

Total Cases 69

APPENDIX III

IRON CONCENTRATIONS IN LIVERS AND SPLEENS OF 101 EUROPEAN AND 661 AFRICAN SUBJECTS

Ref. No.	Age	LIVER								SPLEEN				Cause of Death
			Chemical Iron Estimations		Histological Iron Estimations						Chemical Iron Estimations			
		Weight (grammes)	Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hepatic cells	Kupffer cells	Portal areas	Portal Fibrosis	Cirrhosis	Weight (grammes)	Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hist. Iron	
E73	18 days	127	0.339	0.043	+	-	-	-	-	9	0.380	0.003	+	Fallot's tetralogy
E 54	4/12	218	0.487	0.106	++	-	-	-	-	29	0.187	0.005	-	Viral pneumonia
E 72	1 9/12	567	0.270	0.153	-	++	-	-	-	28	1.248	0.035	+++	Bronchopneumonia - severe burns.
E 83	5	708	0.066	0.047	-	-	-	-	-	57	0.045	0.003	-	Drowning
E 85	5	964	0.103	0.099	-	-	-	-	-	85	0.140	0.012	-	Laryngo-tracheo-bronchitis
E 89	5	737	0.193	0.142	-	-	-	-	-	57	0.175	0.010	-	Drowning
E 91	5	666	0.207	0.138	-	-	-	-	-	76	0.188	0.014	-	Road accident
E 9	16	1700	0.033	0.056	-	-	-	-	-	283	0.122	0.034	-	Struck by lightning
E 24	18	1843	0.232	0.427	-	-	-	-	-	283	0.155	0.044	-	Carbon monoxide poisoning
E 71	18	1955	0.307	0.600	-	-	-	-	-	261	0.161	0.042	-	Road accident
E 94	18	1785	0.182	0.325	-	-	-	-	-	142	0.198	0.028	-	Road accident
E 74	19	1318	0.255	0.336	+	-	-	-	-	99	0.106	0.011	-	Road accident
E 11	20	1361	0.457	0.623	++	-	-	-	-	92	0.278	0.026	+	Road accident
E 14	20	1361	0.366	0.498	-	-	-	-	-	170	0.178	0.030	-	Road accident
E 45	20	1573	0.205	0.322	-	-	-	-	-	198	0.126	0.025	-	Road accident
E 44	21	2069	0.243	0.503	-	-	-	-	-	156	0.703	0.110	++	Road accident
E 21	21	1587	0.174	0.276	-	-	-	-	-	226	0.123	0.028	-	Status epilepticus
E 62	21	1675	0.232	0.388	-	-	-	-	-	156	0.347	0.054	-	Road accident
E 51	23	2025	0.300	0.615	+	-	-	-	-	212	0.302	0.064	+	Road accident
E 79	23	1630	0.353	0.575	+	-	-	-	-	212	0.340	0.072	+	Gunshot wound of head
E 18	25	1516	0.146	0.226	-	-	-	-	-	255	0.149	0.038	-	Road accident
E 93	28	1305	0.447	0.583	++	-	-	-	-	241	0.319	0.077	+	Barbiturate poisoning
E 15	30	2069	0.463	0.959	-	+++	-	-	-	198	2.237	0.442	+++	Renal failure
E 58	32	1289	0.306	0.510	+	-	-	-	-	184	0.308	0.057	+	Road accident
E 39	32	1968	0.294	0.578	-	-	-	-	-	325	0.144	0.047	-	Coronary occlusion
E 26	35	1616	0.318	0.514	+	-	-	-	-	99	0.111	0.011	-	Jumped from a three-storey building
E 17	35	1601	0.228	0.365	-	-	-	+	-	85	0.261	0.022	+	Gunshot wound of head
E 80	36	1672	0.306	0.504	+	-	-	-	-	204	0.137	0.028	-	Carbon monoxide poisoning
E 20	36	2211	0.185	0.409	-	-	-	++++	Coarse	170	0.421	0.071	+	Road accident
E 82	39	1539	0.634	0.986	++	-	-	-	-	91	0.318	0.029	+	Road accident

Ref. No.	Age	LIVER								SPLEEN				Cause of Death
			Chemical Iron Estimations		Histological Iron Estimation						Chemical Iron Estimations			
		Weight (grammes)	Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hepatic cells	Kupffer cells	Portal areas	Portal Fibrosis	Cirrhosis	Weight (grammes)	Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hist. Iron	
E 61	40	1672	0.049	0.082	-	-	-	-	-	92	0.094	0.009	-	Carbon monoxide poisoning
E 25	40	1531	0.603	0.924	++	-	-	-	-	89	0.902	0.080	++	Road accident
E 7	40	1559	0.955	1.489	++	-	-	-	-	113	0.553	0.063	++	Road accident
E 66	41	1474	0.238	0.352	-	-	-	-	-	105	0.166	0.017	-	Coronary occlusion
E 37	42	1389	0.242	0.336	-	-	-	-	-	89	0.167	0.015	-	Carbon monoxide poisoning
E 68	45	2579	0.040	0.103	-	-	-	++++	Fine	269	0.278	0.075	+	Road accident (liver showed marked fatty change.)
E 92	45	2685	0.531	1.425	+	-	-	-	-	113	0.313	0.035	+	Gunshot wound of head
E 64	45	1672	0.262	0.444	+	-	-	-	-	85	1.191	0.101	+++	Road accident
E 86	46	2253	0.485	1.091	+	-	-	-	-	226	0.259	0.058	+	Coronary occlusion
E 35	46	1332	0.123	0.164	-	-	-	-	-	57	0.143	0.008	-	Apoplexy
E 76	49	1531	0.091	0.139	-	-	-	-	-	113	0.107	0.012	-	Cor pulmonale
E 16	49	1984	0.350	0.695	-	-	-	-	-	226	0.185	0.042	-	Coronary occlusion
E 43	50	2750	0.402	1.102	++	++	-	-	-	85	1.166	0.099	+++	Road accident (liver showed advanced fatty change)
E 31	50	2097	0.320	0.670	+	-	-	-	-	283	0.390	0.110	+	Coronary occlusion
E 19	50	1417	0.491	0.696	+	+	-	-	-	142	0.338	0.048	+	Carbon monoxide poisoning
E 30	50	1785	0.020	0.036	-	-	-	-	-	79	0.119	0.009	-	Coronary occlusion
E 12	52	1361	0.083	0.113	-	-	-	-	-	312	0.073	0.023	-	Coronary occlusion
E 10	53	2126	0.281	0.597	+	-	-	-	-	170	0.252	0.043	+	Coronary occlusion
E 59	54	1729	0.174	0.301	-	-	-	-	-	212	0.088	0.019	-	Status asthmaticus
E 8	55	3373	0.239	0.806	-	-	-	++++	Fine	255	0.744	0.190	++	Drowning (liver showed advanced fatty change.)
E 41	56	1843	0.677	1.249	++	-	-	-	-	127	0.408	0.052	+	Coronary occlusion
E 81	58	1814	0.169	0.306	-	-	-	-	-	113	0.089	0.010	-	Peritonitis - ruptured appendix
E 60	58	2041	0.128	0.262	-	-	-	-	-	255	0.118	0.030	-	Coronary occlusion
E 53	59	1843	0.303	0.559	-	-	-	-	-	184	0.335	0.062	+	Coronary occlusion
E 38	59	1105	0.459	0.506	++	-	-	-	-	43	0.449	0.019	++	Road accident
E 32	61	1545	0.334	0.515	+	-	-	-	-	99	0.075	0.007	-	Coronary occlusion
E 87	61	2120	0.128	0.272	+	-	-	-	-	110	0.131	0.014	+	Hypertensive heart failure
E 77	61	1049	0.061	0.064	-	-	-	-	-	113	0.072	0.008	-	Cor pulmonale

Ref. No.	Age	LIVER								SPLEEN				Cause of Death
			Chemical Iron Estimations		Histological Iron Estimations			Portal Fibrosis	Cirrhosis		Chemical Iron Estimations			
		Weight (grammes)	Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hepatic cells	Kupffer cells	Portal areas			Weight (grammes)	Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hist. Iron	
E 55	61	2551	0.343	0.875	+	+	-	-	-	283	0.237	0.067	+	Fractured cervical vertebr
E 47	63	2041	0.273	0.558	-	-	-	-	-	283	0.214	0.060	-	Coronary occlusion
E 88	67	1587	0.366	0.580	+	-	-	-	-	85	0.543	0.046	+	Rupture of aneurysm of abdominal aorta
E 42	68	2296	0.123	0.282	-	-	-	-	-	226	0.132	0.030	-	Pulmonary embolus
E 3	68	1276	0.074	0.094	-	-	-	-	-	Old Splenectomy				Cor pulmonale
E 75	71	1559	0.243	0.378	-	-	-	++	-	241	0.154	0.037	-	Road accident
E 23	74	1729	0.125	0.216	-	-	-	+	-	241	0.263	0.063	+	Coronary occlusion
E 70	75	2154	0.188	0.404	-	-	-	-	-	156	0.442	0.069	+	Road accident
E 57	76	2126	0.278	0.591	-	-	-	-	-	198	0.330	0.065	+	Coronary occlusion
E 69	83	921	0.519	0.478	+	+	-	+	-	99	0.677	0.067	++	Hypertensive heart failure
E 50	85	1162	0.455	0.530	+	+	-	-	-	127	0.578	0.073	++	Coronary occlusion

EUROPEAN FEMALES

Total Cases 32

Ref. No.	Age	LIVER								SPLEEN				Cause of Death
			Chemical Iron Estimations		Histological Iron Estimations						Chemical Iron Estimations			
		Weight (grammes)	Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hepatic cells	Kupffer cells	Portal areas	Portal Fibrosis	Cirrhosis	Weight (grammes)	Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hist. Iron	
E 5	1 2/12	396	0.051	0.020	-	-	-	-	-	57	0.047	0.003	-	Asphyxia - diphtheria
E 4	9	737	0.163	0.120	-	-	-	-	-	113	0.154	0.017	-	Road accident
E 28	10	1049	0.160	0.168	-	-	-	-	-	156	0.221	0.034	-	Anaesthetic death
E 98	16	1559	0.250	0.390	-	-	-	-	-	85	0.177	0.015	-	Road accident
E 101	17	1771	0.273	0.483	+	-	-	-	-	203	0.179	0.031	-	Poisoning by insecticide
E 27	17	2325	0.152	0.357	-	-	-	-	-	142	0.147	0.021	-	Road accident
E 63	18	878	0.524	0.459	++	-	-	-	-	71	0.186	0.013	-	Road accident
E 90	25	1300	0.123	0.160	-	-	-	-	-	150	0.206	0.031	-	status epilepticus
E 52	28	1531	0.216	0.321	-	-	-	-	-	113	0.220	0.025	-	Intracranial haemorrhage fell from roof.
E 65	31	2098	0.290	0.608	-	+	-	-	-		Old splenectomy			Road accident
E 96	36	1729	0.183	0.316	-	-	-	-	-	170	0.092	0.016	-	Budd-Chiari syndrome
E 46	38	765	0.197	0.150	-	-	-	-	-	71	0.181	0.013	-	Barbiturate poisoning
E 34	44	1785	0.194	0.346	-	-	-	-	-	212	0.295	0.062	-	Pulmonary embolus
E 99	44	1615	0.128	0.207	-	-	-	-	-	368	0.092	0.034	-	Coronary occlusion
E 6	47	1601	0.293	0.470	+	-	-	-	-	198	0.264	0.052	+	Coronary occlusion
E 95	50	1190	0.636	0.758	++	+	-	-	-	42	0.479	0.020	++	Salicylate poisoning
E 40	51	1049	0.167	0.175	-	-	-	-	-	142	0.171	0.024	-	Road accident
E 36	54	1559	0.610	0.950	+	++	-	-	-	113	1.692	0.191	+++	Renal failure - chronic pyelonephritis.
E 33	54	1587	0.015	0.024	-	-	-	-	-	142	0.005	0.001	-	Strychnine poisoning
E 97	55	1899	0.284	0.545	+	-	-	-	-	190	0.185	0.035	-	Coronary occlusion
E 49	56	3997	0.263	1.050	-	-	-	-	-	99	0.185	0.018	-	Ruptured dissecting aneurysm of thoracic aor
E 84	58	1559	0.104	0.162	-	-	-	-	-	79	0.277	0.022	+	Road accident
E 78	60	1460	0.250	0.365	-	-	-	-	-	93	0.090	0.008	-	Road accident
E 48	60	1432	0.378	0.546	+	-	-	-	-	113	0.323	0.036	+	Coronary occlusion
E 22	62	2608	0.141	0.368	-	-	-	-	-	170	0.506	0.086	+	Road accident
E 13	64	1164	0.327	0.380	+	+	-	-	-	142	1.636	0.232	+++	Codein poisoning
E 1	68	1531	0.363	0.556	-	-	-	-	-	227	0.127	0.029	-	Coronary occlusion

Ref. No.	Age	LIVER								SPLEEN				Cause of Death
			Chemical Iron Estimations		Histological Iron Estimations			Portal Fibrosis	Cirrhosis		Chemical Iron Estimations		Hist. Iron	
		Weight (grammes)	Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hepatic cells	Kupffer cells	Portal areas			Weight (grammes)	Iron Concentration (mg/g wet wt)	Total Iron (grammes)		
E 2	70	1418	0.185	0.262	-	-	-	-	-	142	0.306	0.043	+	Coronary occlusion
E 100	72	1559	0.251	0.390	-	-	-	-	-	184	0.180	0.033	-	Coronary occlusion
E 56	72	1502	0.462	0.694	+	-	-	-	-	142	0.587	0.083	++	Road accident
E 67	75	694	0.124	0.084	-	-	-	-	-	64	0.079	0.005	-	Road accident
E 29	84	1219	0.199	0.243	-	-	-	+	-	170	0.290	0.049	-	Coronary occlusion

AFRICAN MALES

FIRST DECADE

Total Cases 61

TOTAL LIVER IRON* CONCENTRATION

MALE AFRICANS : FIRST DECADE

These are the values used in Section II in comparison with the values of Ramage et al., 1933

* i.e. storage iron plus haemoglobin iron

Ref. No.	Iron Concentration (mg/g. wet weight)	Ref. No.	Iron Concentration (mg/g. wet weight)	Ref. No.	Iron Concentration (mg/g. wet weight)	Ref. No.	Iron Concentration (mg/g. wet weight)
BS/28/1	0.586	BS/63/1	0.290	BS/44/1	1.450	BS/93/1	0.426
BS/56/1	0.367	BS/68/1	0.467	BS/77/1	0.230	BS/96/1	0.509
BS/104/1	0.640	BS/64/1	1.580	BS/30/1	0.267	BS/75/1	0.264
BS/71/1	0.362	BS/76/1	0.538	BS/24/1	0.316	BS/65/1	0.236
BS/25/1	0.259	BS/1/1	0.420	BS/51/1	0.143	BS/34/1	0.252
BS/23/1	0.329	BS/78/1	0.354	BS/73/1	0.149	BS/100/1	0.234
BS/13/1	0.528	BS/7/1	0.311	BS/14/1	0.063	BS/82/1	0.215
BS/107/1	0.346	BS/26/1	0.183	BS/83/1	0.140	BS/21/1	0.398
BS/110/1	0.189	BS/74/1	0.241	BS/57/1	0.322	BS/6/1	0.129
BS/102/1	0.422	BS/55/1	0.111	BS/89/1	0.214	BS/122/1	0.266
BS/109/1	0.682	BS/27/1	0.158	BS/87/1	0.381	BS/5/1	0.093
BS/81/1	0.339	BS/69/1	0.151	BS/4/1	0.467	BS/95/1	0.198
BS/62/1	0.576	BS/42/1	0.079	BS/80/1	0.794	BS/120/1	0.157
BS/108/1	0.563	BS/49/1	0.133	BS/84/1	0.226	BS/125/1	0.224
BS/12/1	0.754	BS/60/1	0.152	BS/85/1	0.093	BS/123/1	0.336
BS/105/1	1.508						

Ref. No.	Age	LIVER								SPLEEN				Cause of Death
			Chemical Iron Estimations		Histological Iron Estimations						Chemical Iron Estimations			
		Weight (grammes)	Iron Concentration (mg/g wet wt)	Total Iron (grammes).	Hepatic cells	Kupffer cells	Portal areas	Portal Fibrosis	Cirrhosis	Weight (grammes)	Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hist. Iron	
BS/28/1	S.B *	92	0.334	0.031	+	-	-	-	-	4	0.122	0.001	-	Atelectasis: Birth wt.4 lbs.
BS/56/1	S.B	90	0.097	0.009	-	-	-	-	-	7	0.103	0.001	-	Atelectasis: Birth wt.4 lbs.
BS/104/1	S.B	74	0.586	0.043	+	-	+	-	-	6	0.349	0.002	+	Atelectasis: Birth wt.4 lbs.8oz
BS/71/1	S.B	210	0.226	0.047	-	-	-	-	-	16	0.223	0.004	+	Atelectasis: Birth wt.9 lbs.1oz.
BS/25/1	S.B	116	0.213	0.025	-	-	-	-	-	8	0.209	0.002	+	Atelectasis.
BS/23/1	S.B	85	0.120	0.011	-	-	-	-	-	8	0.094	0.001	-	Prematurity.
BS/13/1	S.B	78	0.427	0.033	+	+	-	-	-	10	0.066	0.001	-	Hydrops foetalis
BS/107/1	A few hours	72	0.223	0.016	-	-	-	-	-	4	0.190	0.001	-	Hydrops foetalis: Bth.wt.5 lbs.3ozs
BS/110/1	1 day	72	0.142	0.010	-	-	-	-	-	3	0.181	0.0005	-	Prematurity: Birth wt.3 lbs. 11oz
BS/102/1	1 day	46	0.297	0.014	+	++	+	-	-	5	0.140	0.001	-	Prematurity: Birth wt.3 lbs. 11oz
BS/109/1	1 day	82	0.610	0.050	+	+	+	-	-	8	0.213	0.002	-	Atelectasis: Birth wt.4 lbs.8 oz.
BS/81/1	1 day	152	0.260	0.040	-	-	-	-	-	18	0.357	0.007	+	Intracranial haemorrhage: Birth weight: 5 lbs. 8 ozs.
BS/62/1	1 day	114	0.493	0.056	+	+	-	-	-	10	0.808	0.008	++	Atelectasis: Bth.wt.6 lbs.9 oz
BS/108/1	2 dys.	100	0.454	0.045	+	-	-	-	-	8	0.418	0.003	++	Neonatal pneumonia: Birth weight 5 lbs. 6 ozs.
BS/12/1	4 dys.	168	0.686	0.115	+	+	-	-	-	14	0.470	0.007	+	Intracranial haemorrhage
BS/105/1	7 dys.	104	1.408	0.146	++	+	-	-	-	20	0.948	0.019	++	Hydranencephaly: Bth.wt. 7 lb. 2ozs
BS/63/1	7 dys.	204	0.256	0.052	+	-	-	-	-	16	0.378	0.006	+	Bronchopneumonia: Bth.wt.10 lb.1oz.
BS/60/1	2/52	146	0.445	0.065	+	+	-	-	-	6	0.948	0.006	++	Bronchopneumonia.
BS/64/1	3/52	44	1.547	0.068	++	++	+	-	-	4	0.758	0.003	++	Bronchopneumonia (premature infant)
BS/76/1	3/52	132	0.477	0.063	+	-	-	-	-	4	1.057	0.004	++	Myelomeningocoel-suppurative meningitis
BS/1/1	6/52	248	0.370	0.092	-	-	+	-	-	22	0.634	0.014	++	Viral pneumonia.
BS/78/1	2/12	148	0.318	0.046	-	+	-	-	-	10	1.226	0.012	++	Purulent meningitis.
BS/7/1	3/12	138	0.269	0.037	-	-	-	-	-	6	0.488	0.003	+	Bronchopneumonia
BS/26/1	6/12	310	0.129	0.040	-	-	-	-	-	26	0.074	0.002	-	Bronchopneumonia
BS/74/1	8/12	354	0.180	0.064	-	-	-	-	-	34	0.083	0.003	-	Empyema
BS/55/1	9/12	228	0.076	0.017	-	-	-	-	-	26	0.076	0.002	-	Bronchopneumonia
BS/27/1	10/12	342	0.097	0.003	-	-	-	-	-	26	0.123	0.003	-	Bronchopneumonia
BS/69/1	11/12	250	0.100	0.025	-	-	-	-	-	28	0.136	0.004	-	Bacillary dysentery
BS/42/1	1	212	0.039	0.008	-	-	-	-	-	18	0.285	0.005	+	Bronchopneumonia-kwashiorkor
BS/49/1	1	322	0.083	0.027	-	-	-	-	-	34	0.039	0.001	-	Bronchopneumonia
BS/60/1	1 2/12	358	0.110	0.039	-	-	-	-	-	44	0.133	0.006	-	Asphyxia- inhalation of vomit

* S.B. = Stillbirth

Ref. No.	Age	LIVER							SPLEEN				Cause of Death
			Chemical Iron Estimations		Histological Iron Estimations					Chemical Iron Estimations			
		Weight (grammes)	Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hepatic cells	Kupffer cells	Portal areas.	Portal Fibrosis	Cirrhosis	Weight (grammes)	Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hist. Iron	
BS/44/1	1 5/12	264	1.432	0.378	++	++	+	-	-	16	0.503	0.008	+	Bronchopneumonia
BS/77/1	1 6/12	536	0.120	0.064	-	-	-	-	-	52	0.149	0.008	-	Anaesthetic death.
BS/30/1	1 9/12	236	0.240	0.057	-	+	-	-	-	38	0.226	0.009	+	Miliary tuberculosis
BS/24/1	2	410	0.297	0.122	+	++	-	+	-	24	1.075	0.026	+++	Bronchopneumonia - kwashiorkor
BS/51/1	2	356	0.120	0.043	-	-	-	-	-	32	0.160	0.005	-	Gastroenteritis.
BS/73/1	2½	338	0.127	0.043	-	-	-	-	-	20	0.243	0.005	+	Bronchopneumonia.
BS/14/1	2 8/12	460	0.059	0.027	-	-	-	-	-	34	0.165	0.006	+	Bronchopneumonia.
BS/83/1	3	273	0.106	0.029	-	-	-	-	-	16	0.170	0.003	-	Bronchopneumonia.
BS/57/1	3	474	0.224	0.106	-	-	-	-	-	28	0.265	0.007	+	Bronchopneumonia.
BS/89/1	4	478	0.183	0.087	-	-	-	-	-	52	0.080	0.004	-	Road accident.
BS/87/1	5	552	0.355	0.196	+	-	-	-	-	42	0.336	0.014	+	Septicaemia.
BS/4/1	6	539	0.464	0.250	+	+	-	-	-	22	0.548	0.012	+	Massive hepatic necrosis and fatty change.
BS/80/1	6	634	0.706	0.448	+	++	-	-	-	82	0.386	0.032	+	Nephrotic syndrome-bronchopneumonia.
BS/84/1	6	342	0.158	0.054	+	-	-	-	-	22	0.806	0.018	++	Bronchopneumonia
BS/85/1	6	734	0.037	0.027	-	-	-	+	-	52	0.122	0.006	-	Road accident
BS/93/1	6½	332	0.318	0.106	+	++	-	-	-	11	2.060	0.023	+++	Peritonitis following operation to relieve bowel obstruction
BS/96/1	7	352	0.476	0.128	++	+	-	-	-	34	0.684	0.023	++	Idiopathic megacolon
BS/75/1	7	514	0.169	0.087	-	-	-	-	-	60	0.623	0.037	+	Bronchopneumonia - pellagra
BS/65/1	7	518	0.204	0.106	-	-	-	-	-	30	0.530	0.016	++	Bacillary dysentery
BS/34/1	7	706	0.167	0.118	-	-	-	-	-	39	0.173	0.007	-	Septicaemia.
BS/100/1	8	672	0.195	0.131	-	-	-	-	-	48	0.328	0.016	+	Bronchopneumonia
BS/82/1	8	632	0.149	0.094	-	-	-	-	-	42	0.815	0.034	+	Bronchopneumonia (malnutrition)
BS/21/1	8	880	0.378	0.333	+	+	-	-	-	130	1.136	0.148	++	Burkitt's sarcoma
BS/6/1	8	624	0.092	0.087	-	-	-	-	-	26	0.363	0.009	-	Bronchopneumonia-kwashiorkor-Malarial pigment in red pulp of spleen.
BS/122/1	8¼	504	0.202	0.103	-	-	-	-	-	49	0.515	0.025	+	Miliary tuberculosis.
BS/5/1	8½	751	0.084	0.063	-	-	-	-	-	71	0.048	0.003	-	Bronchopneumonai - kwashiorkor
BS/95/1	9	850	0.140	0.119	-	-	-	-	-	71	0.190	0.013	-	Meningococcal meningitis.
BS/120/1	9	816	0.097	0.079	-	-	-	+	-	172	0.110	0.019	-	Asphyxia -inhalation of food.
BS/125/1	9	1060	0.176	0.187	-	-	-	-	-	60	0.543	0.033	++	Bronchopneumonia.
BS/123/1	9½	1128	0.259	0.292	-	-	-	-	-	84	0.231	0.019	-	Rheumatic carditis.

AFRICAN FEMALES

FIRST DECADE

Total Cases 65

TOTAL LIVER IRON* CONCENTRATION

FEMALE AFRICANS : FIRST DECADE

These are the values used in Section II in comparison with the values of Kanage et al., 1983.

* i.e. storage iron plus haemoglobin iron

Ref. No.	Iron Concentration (mg/g. wet weight)	Ref. No.	Iron Concentration (mg/g. wet weight)	Ref. No.	Iron Concentration (mg/g. wet weight)	Ref. No.	Iron Concentration (mg/g. wet weight)
BS/79/1	0.551	BS/17/1	1.001	BS/61/1	0.151	BS/70/1	0.227
BS/112/1	0.270	BS/19/1	0.562	BS/9/1	0.214	BS/119/1	0.456
BS/101/1	0.384	BS/22/1	0.458	BS/2/1	0.086	BS/43/1	0.300
BS/83/1	0.464	BS/11/1	0.256	BS/3/1	0.129	BS/67/1	0.437
BS/90/1	0.966	BS/53/1	0.415	BS/29/1	0.123	BS/94/1	0.272
BS/111/1	0.432	BS/58/1	0.202	BS/41/1	0.306	BS/117/1	0.296
BS/106/1	0.820	BS/45/1	0.307	BS/54/1	0.149	BS/115/1	0.323
BS/113/1	0.470	BS/39/1	0.205	BS/40/1	0.161	BS/32/1	0.075
BS/114/1	0.563	BS/59/1	0.131	BS/8/1	0.386	BS/118/1	0.326
BS/103/1	0.631	BS/52/1	0.099	BS/16/1	0.245	BS/99/1	0.157
BS/91/1	0.594	BS/48/1	0.143	BS/35/1	0.128	BS/92/1	0.329
BS/72/1	0.640	BS/46/1	0.251	BS/38/1	0.262	BS/98/1	0.407
BS/116/1	0.647	BS/33/1	0.081	BS/37/1	0.222	BS/121/1	0.216
BS/36/1	0.478	BS/20/1	0.375	BS/66/1	0.106	BS/97/1	0.236
BS/31/1	0.344	BS/18/1	0.121	BS/50/1	0.535	BS/124/1	0.204
BS/47/1	0.248	BS/15/1	0.242	BS/10/1	0.319	BS/126/1	0.097
BS/86/1	0.601						

Ref. No.	Age	LIVER							SPLEEN				Cause of Death
			Chemical Iron Estimations		Histological Iron Estimations					Chemical Iron Estimations			
		Weight (grammes)	Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hepatic cells	Kupffer cells	Portal areas	Portal Fibrosis	Cirrhosis	Weight (grammes)	Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hist. Iron	
BS/79/1	Still birth	42	0.453	0.019	+	-	-	-	-	3	0.167	0.0005	-	Prematurity:Birth wt.1 lb.13oz
BS/112/1	Still birth	46	0.205	0.009	-	-	-	-	-	5	0.112	0.0005	-	Prematurity: Birth weight : 2 lb. 8 ozs.
BS/101/1	Still birth	74	0.188	0.014	-	-	-	-	-	6	0.224	0.001	-	Prematurity: Birth weight: 3lbs. 6 ozs.
BS/88/1	Still birth	128	0.252	0.032	-	-	-	-	-	18	0.247	0.004	+	Delay in labour: Birth weight 5 lb. 0 oz.
BS/90/1	Still birth	172	0.791	0.136	+	+	++	-	-	12	0.506	0.006	++	Delay in labour: Birth weight 5 lb. 6 oz.
BS/111/1	Still birth	118	0.369	0.044	+	+	-	-	-	5	0.490	0.002	++	Atelectasis : Birth weight 6 lb. 0 oz.
BS/106/1	Still birth	188	0.720	0.135	+	-	+	-	-	9	0.301	0.003	+	Atelectasis : Birth weight 6 lb. 2 oz.
BS/113/1	Still birth	122	0.414	0.051	+	+	+	-	-	8	0.602	0.005	++	Tear of tentorium cerebelli: Birth weight 6 lb. 12 oz.
BS/114/1	Still birth	140	0.396	0.055	+	-	-	-	-	10	0.445	0.004	++	Delay in labour : Birth weight 7 lb. 2 oz.
BS/103/1	1 day	56	0.556	0.031	+	+	-	-	-	6	0.344	0.002	+	Atelectasis: Bth.wt.4lb.3oz.
BS/91/1	1 day	110	0.541	0.060	+	+	-	-	-	6	0.754	0.005	++	Atelectasis:Bth.wt.6lb.11oz.
BS/72/1	1 day	148	0.501	0.074	+	+	-	-	-	8	0.534	0.004	+	Neonatal pneumonia
BS/116/1	2 days	78	0.585	0.046	++	+	-	-	-	6	0.421	0.002	+	Multiple atresia of small bowel: Bth.wt. 4 lb. 1 oz.
BS/36/1	4 days	75	0.451	0.034	+	+	-	-	-	2	0.157	0.0003	-	Encephalocoel-bronchopneumonia
BS/31/1	5 days	84	0.278	0.023	+	-	-	-	-	5	0.752	0.004	+	Bronchopneumonia
BS/47/1	6 days	91	0.159	0.014	-	-	-	-	-	4	0.223	0.001	-	Bronchopneumonia
BS/86/1	7 days	138	0.499	0.069	+	-	-	-	-	12	0.593	0.007	+	Neonatal pneumonia
BS/17/1	8 days	78	0.958	0.075	+	++	-	-	-	5	0.701	0.004	++	Neonatal pneumonia
BS/19/1	3/52	122	0.524	0.064	+	++	-	-	-	4	1.210	0.005	+++	Bronchopneumonia
BS/22/1	6/52	138	0.411	0.057	+	+	-	-	-	20	0.692	0.014	+	Bronchopneumonia
BS/11/1	2/12	96	0.217	0.021	-	-	-	-	-	7	0.143	0.001	-	Bronchopneumonia
BS/53/1	4/12	286	0.361	0.103	+	-	-	-	-	30	0.250	0.008	+	Gastroenteritis
BS/58/1	8/12	280	0.160	0.045	-	-	-	-	-	23	0.222	0.005	-	Bronchopneumonia
BS/45/1	9/12	196	0.290	0.057	-	+	-	-	-	9	0.940	0.008	++	Bronchopneumonia
BS/39/1	10/12	260	0.181	0.047	-	-	-	-	-	24	0.431	0.010	+	Bronchopneumonia

Ref. No.	Age	LIVER							SPLEEN				Cause of Death
		Weight (grammes)	Chemical Iron Estimations		Histological Iron Estimations			Cirrhosis	Weight (grammes)	Chemical Iron Estimations		Hist. Iron	
			Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hepatic cells	Kupffer cells	Portal areas	Portal Fibrosis		Iron Concentration (mg/g wet wt)	Total Iron (grammes)		
BS/59/1	11/12	300	0.118	0.035	-	-	-	-	-	18	0.232	0.004	+	Bronchopneumonia
BS/52/1	1	246	0.061	0.015	-	-	-	-	-	30	0.250	0.008	+	Bronchopneumonia
BS/48/1	1	262	0.095	0.025	-	-	-	-	-	16	0.181	0.003	+	Bronchopneumonia
BS/46/1	1	360	0.227	0.082	-	-	-	-	-	30	0.362	0.011	+	Bronchopneumonia
BS/33/1	1	248	0.063	0.016	-	-	-	-	-	20	0.217	0.004	+	Gastroenteritis & kwashiorkor
BS/20/1	1	224	0.339	0.076	+	-	-	-	-	27	0.109	0.003	-	Bronchopneumonia
BS/18/1	1	138	0.087	0.012	-	-	-	-	-	8	0.163	0.001	-	Bronchopneumonia
BS/15/1	1	344	0.234	0.080	+	+	-	-	-	24	0.421	0.010	+	Bronchopneumonia
BS/61/1	1 2/12	288	0.119	0.034	-	-	-	-	-	32	0.331	0.011	+	Bronchopneumonia
BS/9/1	1 3/12	268	0.196	0.053	-	-	-	-	-	18	0.843	0.015	++	Suppurative meningitis
BS/2/1	1 5/12	298	0.084	0.025	-	-	-	-	-	29	0.439	0.013	++	Bronchopneumonia-kwashiorkor
BS/3/1	1 6/12	444	0.062	0.028	-	-	-	-	-	36	0.048	0.002	-	Bronchopneumonia
BS/29/1	1 6/12	278	0.096	0.027	-	-	-	-	-	34	0.208	0.007	+	Tuberculous meningitis
BS/41/1	1 6/12	302	0.273	0.082	-	+	-	-	-	30	0.872	0.026	++	Bronchopneumonia - gross malnutrition.
BS/54/1	1 6/12	348	0.103	0.036	-	-	-	-	-	10	0.208	0.002	+	Bronchopneumonia
BS/40/1	2	284	0.129	0.037	-	-	-	-	-	15	0.547	0.008	++	Gastroenteritis-kwashiorkor
BS/8/1	2	266	0.351	0.093	-	-	-	-	-	18	0.195	0.004	-	Bronchopneumonia
BS/16/1	2½	425	0.205	0.069	-	-	-	-	-	26	0.139	0.004	-	Kwashiorkor-gastroenteritis
BS/35/1	2½	362	0.072	0.026	-	-	-	-	-	27	0.088	0.002	-	Severe burns
BS/38/1	2½	386	0.242	0.093	-	+	-	-	-	30	0.594	0.018	+	Bronchopneumonia
BS/37/1	3	642	0.110	0.071	-	-	-	-	-	86	0.168	0.014	-	Bronchopneumonia
BS/66/1	3	274	0.080	0.022	-	-	-	-	-	34	0.332	0.011	+	Bronchopneumonia
BS/50/1	4	320	0.515	0.165	-	++	-	-	-	20	1.390	0.028	+++	Medullablastoma -hydrocephalus
BS/10/1	4	464	0.270	0.125	-	+	-	-	-	32	0.219	0.007	+	Extensive burns
BS/70/1	4	396	0.182	0.072	-	-	-	-	-	36	0.137	0.005	-	Extensive burns
BS/119/1	5	536	0.415	0.222	-	++	-	-	-	30	0.402	0.012	+	Bronchopneumonia
BS/43/1	5	212	0.251	0.053	-	-	-	+	-	18	0.177	0.003	-	Road accident
BS/67/1	5	534	0.365	0.195	+	-	-	-	-	68	0.485	0.033	+	Bronchopneumonia - severe malnutrition
BS/94/1	5½	544	0.233	0.125	-	-	-	-	-	30	0.330	0.010	+	Bronchopneumonia

Ref. No.	Age	LIVER								SPLEEN				Cause of Death
			Chemical Iron Estimations		Histological Iron Estimations						Chemical Iron Estimations			
		Weight (grammes)	Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hepatic cells	Kupffer cells	Portal areas	Portal Fibrosis	Cirrhosis	Weight (grammes)	Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hist. Iron	
BS/117/1	6	490	0.245	0.120	-	-	-	-	-	40	0.524	0.021	+	Bronchopneumonia
BS/115/1	6	583	0.222	0.129	-	-	-	-	-	160	0.185	0.030	-	Malaria
BS/32/1	7	726	0.049	0.036	-	-	-	++++	coarse	104	0.079	0.008	-	Bronchopneumonia
BS/118/1	7	706	0.260	0.183	-	+	-	-	-	65	0.207	0.013	+	Lobar pneumonia and pericarditis
BS/99/1	7	711	0.093	0.066	-	-	-	-	-	40	0.087	0.003	-	Astrocytoma of cerebellum
BS/92/1	8	774	0.235	0.182	-	-	-	-	-	58	0.124	0.007	-	Severe burns
BS/98/1	8	496	0.355	0.176	+	+	+	-	-	24	0.455	0.011	++	Aseptic meningitis
BS/121/1	8½	676	0.144	0.097	-	-	-	-	-	105	0.328	0.034	+	Bronchopneumonia
BS/97/1	9	515	0.131	0.067	-	-	-	-	-	41	0.094	0.004	-	Lobar pneumonia
BS/124/1	9	854	0.176	0.150	-	-	-	-	-	166	0.356	0.059	+	Enteric fever
BS/126/1	9	718	0.084	0.060	-	-	-	-	-	60	0.082	0.005	-	Rheumatic carditis

AFRICAN MALES

SECOND DECADE

Total Cases 32

Ref. No.	Age	LIVER							SPLEEN				Cause of Death
		Weight (grammes)	Chemical Iron Estimations		Histological Iron Estimations			Portal Fibrosis	Cirrhosis	Weight (grammes)	Chemical Iron Estimations		Hist. Iron	
			Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hepatic cells	Kupffer cells	Portal areas				Iron Concentration (mg/g wet wt)	Total Iron (grammes)		
BS/13/2	11½	1049	0.210	0.220	-	-	-	-	-	56	0.283	0.016	+	Acute rheumatic carditis
BS/47/2	12	1236	0.070	0.087	-	-	-	-	-	90	0.066	0.006	-	Gunshot wound of head
BS/42/2	12	1196	0.076	0.091	-	-	-	++	-	130	0.220	0.029	-	Bronchopneumonia
BS/32/2	12	864	0.154	0.133	-	-	-	-	-	88	0.119	0.010	-	Road accident
BS/20/2	12	862	0.061	0.053	-	-	-	+++	bilharzial fibrosis	110	0.072	0.008	-	Road accident
BS/19/2	12	942	0.127	0.120	-	-	-	-	-	82	0.063	0.005	-	Drowning
BS/12/2	12	860	0.304	0.261	-	-	-	-	-	102	0.395	0.040	+	Enteric fever
BS/9/2	12	1144	0.077	0.088	-	-	-	-	-	86	0.111	0.010	-	Septicaemia
BS/1/2	12	1216	0.066	0.080	-	-	-	+	-	96	0.131	0.013	-	Drowning
BS/31/2	13	992	0.075	0.074	-	-	-	-	-	71	0.099	0.007	-	Road accident
BS/23/2	13	988	0.188	0.168	-	-	-	-	-	62	0.217	0.013	-	Drowning
BS/57/2	14	805	0.122	0.098	-	-	-	++++	coarse	426	0.110	0.047	-	Liver failure
BS/56/2	14	1252	0.057	0.072	-	-	-	-	-	85	0.095	0.009	-	Drowning
BS/24/2	14	1008	0.057	0.057	-	-	-	+	-	390	0.054	0.021	-	Anaemia - onyalai
BS/2/2	14	1060	0.110	0.116	-	-	-	-	-	96	0.051	0.005	-	Stab wound of lung
BS/18/2	15	1114	0.182	0.203	-	-	-	-	-	96	1.365	0.131	++	Hypertensive heart failure
BS/15/2	15	2154	0.280	0.603	+	-	-	+++	-	1574	0.304	0.478	+	Infectious hepatitis
BS/3/2	15	1526	0.295	0.450	-	+	-	-	-	202	0.444	0.090	+	Severe anaemia
BS/40/2	16	1952	0.174	0.341	-	-	-	-	-	217	0.216	0.047	-	Carbon monoxide poisoning
BS/8/2	17	1838	0.086	0.158	-	-	-	-	-	176	0.043	0.008	-	Road accident
BS/54/2	18	1626	0.259	0.421	-	-	-	-	-	328	0.246	0.081	-	Stab wound of heart
BS/52/2	18	1384	0.309	0.428	-	-	-	-	-	154	0.237	0.036	-	Road accident
BS/38/2	18	1136	0.220	0.250	-	-	-	-	-	68	0.154	0.010	-	Road accident
BS/27/2	18	1602	0.480	0.769	+	-	-	-	-	150	0.405	0.060	+	Traumatic cerebral haemorrhage
BS/26/2	18	1770	0.209	0.370	-	-	-	+	-	238	1.130	0.269	+++	Nephrotic syndrome
BS/14/2	18	1200	0.299	0.359	-	-	-	-	-	118	0.158	0.019	-	Road accident
BS/55/2	19	1287	0.565	0.727	+	+	-	-	-	80	0.541	0.043	+	Traumatic brain damage
BS/39/2	19	1500	0.186	0.279	-	-	-	-	-	126	0.189	0.024	-	Carbon monoxide poisoning
BS/37/2	19	1265	0.089	0.112	-	-	-	-	-	214	0.108	0.023	-	Electrocution
BS/21/2	19	1000	0.237	0.237	-	-	-	+++	-	134	0.376	0.050	+	Severe trauma-extensive bruising of back muscles.
BS/6/2	19	1606	0.243	0.390	-	-	-	-	-	204	0.141	0.029	-	Cardiomyopathy
BS/4/2	19	1336	0.159	0.212	-	-	-	-	-	140	0.270	0.038	-	Extensive burns.

AFRICAN FEMALES

SECOND DECADE

Total Cases 30

Ref. No.	Age	LIVER								SPLEEN				Cause of Death
			Chemical Iron Estimations		Histological Iron Estimations						Chemical Iron Estimations			
		Weight (grammes)	Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hepatic cells	Kupffer cells	Portal areas	Portal Fibrosis	Cirrhosis	Weight (grammes)	Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hist. Iron	
BS/48/2	10	1011	0.201	0.204	-	-	-	-	-	66	0.172	0.011	-	Acute rheumatic carditis
BS/29/2	10	1342	0.175	0.235	-	-	-	-	-	82	0.096	0.008	-	Acute rheumatic carditis
BS/11/2	10	1252	0.106	0.133	-	-	-	-	-	498	0.075	0.033	-	Massive hepatic necrosis
BS/58/2	10½	918	0.115	0.106	-	-	-	-	-	56	0.108	0.006	-	Drowning
BS/61/2	11	1103	0.194	0.214	-	-	-	-	-	67	0.118	0.008	-	Acute rheumatic carditis
BS/60/2	11	909	0.223	0.203	-	-	-	-	-	81	0.243	0.020	-	Pulmonary collapse following repair atrial septal defect in heart
BS/41/2	11	736	0.150	0.110	-	-	-	-	-	62	0.363	0.022	+	Lobar pneumonia
BS/53/2	12	807	0.079	0.064	-	-	-	-	-	112	0.137	0.015	-	Hypertensive heart failure - chronic pyelonephritis.
BS/43/2	12	1040	0.248	0.258	-	-	-	-	-	210	0.399	0.084	+	Bronchopneumonia
BS/36/2	12	965	0.209	0.202	-	-	-	+	-	170	0.302	0.051	+	Tuberculous meningitis
BS/35/2	12	1920	0.717	1.377	++	++	-	-	-		Old Splenectomy			Osteomyelitis & sickle cell anaemia
BS/30/2	12	1118	0.053	0.059	-	-	-	++	Bilharzial fibrosis	168	0.079	0.013	-	Pulmonary tuberculosis
BS/28/2	12	1525	0.080	0.122	-	-	-	+	-	152	0.063	0.013	-	Cardiomyopathy with tricuspid incompetence.
BS/46/2	13	800	0.185	0.148	-	-	-	-	-	202	0.100	0.020	-	Retroperitoneal sarcoma
BS/34/2	13	1250	0.062	0.078	-	-	-	+	-	198	0.054	0.011	-	Congestive heart failure-mitral incompetence
BS/45/2	14	1351	0.210	0.252	-	+	-	-	-	516	0.262	0.135	+	Myeloid leukaemia
BS/25/2	14	1062	0.282	0.299	-	+	-	-	-	56	0.423	0.024	+	Asphyxia following partial thyroidectomy
BS/22/2	15	1516	0.111	0.168	-	-	-	-	-	92	0.285	0.026	+	Cerebral abscess
BS/5/2	15	1360	0.105	0.143	-	-	-	-	-	118	0.066	0.008	-	Drowning
BS/7/2	16	1630	0.294	0.481	+	-	-	+	-	154	0.355	0.055	+	Drowning
BS/59/2	17	2320	0.216	0.501	-	+	-	-	-	220	0.352	0.077	+	Malaria
BS/10/2	17	958	0.285	0.273	+	-	-	-	-	76	0.321	0.024	+	Road accident
BS/62/2	18	1734	0.340	0.590	+	-	-	+	-	332	0.258	0.086	-	Pulmonary collapse following hysterectomy for ruptured uterus.
BS/44/2	18	1462	1.087	1.588	++	+	-	+	-	148	0.296	0.044	+	Viral pneumonia - anaemia
BS/16/2	18	1512	0.434	0.656	++	-	-	-	-	104	1.329	0.138	+++	Encephalitis
BS/51/2	19	1038	0.198	0.206	-	-	-	-	-	98	0.772	0.076	++	Tuberculous bronchopneumonia
BS/50/2	19	1574	0.085	0.134	-	-	-	-	-	78	0.189	0.015	-	Acute rheumatic carditis
BS/49/2	19	1052	0.072	0.076	-	-	-	-	-	134	0.060	0.008	-	Suicidal hanging
BS/33/2	19	1815	0.127	0.231	-	-	-	-	-	227	0.142	0.032	-	Cardiomyopathy
BS/17/2	19	1604	0.108	0.173	-	-	-	+	-	254	0.090	0.023	-	Ruptured uterus

AFRICAN MALES

THIRD DECADE

Total Cases 55

Ref. No.	Age	LIVER							SPLEEN				Cause of Death
			Chemical Iron Estimations		Histological Iron Estimations					Chemical Iron Estimations			
		Weight (grammes)	Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hepatic cells	Kupffer cells	Portal areas	Portal Fibrosis	Cirrhosis	Weight (grammes)	Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hist. Iron	
BS/74/3	20	746	0.223	0.167	-	-	-	-	-	86	0.262	0.022	+	Road accident
BS/50/3	20	1189	0.099	0.118	-	-	-	-	-	171	0.181	0.031	-	Traumatic intracranial haemorrhage
BS/39/3	20	958	0.645	0.619	+	++	-	-	-	56	0.573	0.032	++	Brain damage - head injury
BS/36/3	21	1878	0.313	0.588	+	+	-	-	-	398	0.783	0.322	+++	Hodgkin's disease.
BS/35/3	21	1733	0.377	0.655	+	-	-	-	-	104	0.345	0.036	+	Coronary occlusion
BS/41/3	22	1428	0.244	0.348	-	-	-	++	-	122	0.172	0.021	-	Brain abscess
BS/40/3	23	1250	0.328	0.410	+	-	-	+	-	94	0.493	0.046	+	Traumatic intracranial haemorrhage
BS/20/3	23	1278	0.235	0.300	-	-	-	+	-	152	0.192	0.029	-	Road accident
BS/15/3	23	1354	0.282	0.382	+	-	-	-	-	198	0.130	0.026	-	Road accident
BS/58/3	24	1253	0.988	1.238	++	-	-	-	-	52	0.924	0.048	++	Road accident
BS/37/3	24	1254	0.409	0.513	+	+	+	++++	Coarse	194	1.185	0.230	+++	Renal failure - chronic pyelonephritis
BS/21/3	24	602	0.223	0.134	-	+	-	++++	Coarse	520	0.147	0.076	-	Suppurative meningitis
BS/17/3	24	1226	0.191	0.234	-	-	-	-	-	69	0.915	0.063	++	Road accident
BS/78/3	25	1342	0.606	0.814	+	-	-	-	-	170	0.313	0.053	+	Road accident
BS/75/3	25	1582	0.728	1.152	++	-	-	-	-	240	0.334	0.080	+	Multiple injuries- mine accident
BS/73/3	25	1484	0.168	0.250	-	-	-	-	-	254	0.019	0.005	-	Suicidal hanging
BS/70/3	25	1517	0.506	0.768	++	+	-	-	-	156	0.490	0.076	++	Status epilepticus
BS/69/3	25	1690	0.758	1.280	++	++	+	+	-	234	1.041	0.244	++	Carbon monoxide poisoning
BS/66/3	25	1340	0.160	0.214	-	-	-	-	-	162	0.205	0.033	-	Bowel obstruction
BS/65/3	25	1026	0.212	0.218	-	-	-	-	-	138	0.270	0.037	+	Traumatic intracranial haemorrhage
BS/64/3	25	1562	0.101	0.158	-	-	-	-	-	148	0.181	0.027	-	Pulmonary tuberculosis
BS/63/3	25	1486	0.693	1.030	++	-	-	-	-	90	1.800	0.162	+++	Brain damage - murder
BS/62/3	25	1422	0.457	0.650	+	-	+	-	-	118	0.331	0.039	+	Road accident
BS/61/3	25	1505	0.703	1.058	+	++	-	-	-	128	0.780	0.100	++	Cerebellar haemorrhage
BS/51/3	25	1848	0.168	0.310	-	-	-	+++	Bilhazial Fibrosis	843	0.080	0.067	-	Haemorrhage from oesophageal varices
BS/49/3	25	1284	0.963	1.281	++	+	+	-	-	115	0.510	0.059	++	Brain damage - murder
BS/48/3	25	1356	0.069	0.094	-	-	-	+	-	196	0.169	0.033	-	Road accident
BS/44/3	25	1476	0.303	0.447	-	-	-	+	-	121	0.537	0.065	++	Road accident - much bilharzial pigment in kupffer cells and portal areas.
BS/43/3	25	1407	0.278	0.391	-	-	-	-	-	144	0.361	0.052	+	Suicidal hanging
BS/42/3	25	1064	0.283	0.301	+	-	-	+	-	178	0.212	0.037	-	Road accident
BS/38/3	25	1704	0.068	0.116	-	-	-	-	-	160	0.129	0.021	-	Road accident

Ref. No.	Age	LIVER							SPLEEN				Cause of Death
			Chemical Iron Estimations		Histological Iron Estimations					Chemical Iron Estimations			
		Weight (grammes)	Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hepatic cells	Kupffer cells	Portal areas	Portal Fibrosis	Cirrhosis	Weight (grammes)	Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hist. Iron	
BS/26/3	25	1046	0.279	0.292	-	-	-	+	-	78	0.191	0.015	-	Road accident
BS/18/3	25	1750	0.308	0.539	+	-	-	-	-	118	0.317	0.037	+	Traumatic intracranial haemorhage
BS/14/3	25	1822	0.173	0.351	-	-	-	-	-	280	0.180	0.050	-	Stab wound of heart
BS/6/3	25	1678	0.301	0.505	+	-	-	+	-	216	0.210	0.045	-	Asphyxia during electro-convulsive therapy.
BS/4/3	25	1530	0.136	0.208	-	-	-	++	-	242	0.020	0.005	-	Brain damage - murder
BS/79/3	26	1202	0.393	0.473	+	-	-	-	-	119	0.269	0.032	+	Road accident
BS/67/3	26	1440	0.199	0.287	-	-	-	+	-	164	0.148	0.022	-	Dislocated neck - fell from roof
BS/39/3	26	1214	0.162	0.197	-	-	-	-	-	146	0.150	0.022	-	Road accident
BS/31/3	26	1362	0.237	0.323	-	-	-	-	-	72	0.150	0.011	-	Traumatic intracranial haemorhage
BS/7/3	26	1106	4.706	5.205	++	+++	+++	++++	Coarse	410	0.510	0.209	+	Road accident
BS/1/3	27	1862	0.354	0.659	-	-	-	-	-	236	0.475	0.112	+	Road accident
BS/55/3	28	1972	0.822	1.621	+	++	++	-	-	374	0.959	0.359	++	Septicaemia following lower limb amputation for severe injury
BS/47/3	28	1276	3.784	4.828	+++	+++	+++	++	-	147	1.396	0.205	+++	Road accident
BS/33/3	28	1334	4.552	6.072	+++	++	+++	+	-	134	0.666	0.089	++	Refractory anaemia
BS/27/3	28	1440	0.209	0.301	-	+	-	-	-	200	0.343	0.068	+	Status epilepticus
BS/19/3	28	1404	0.182	0.256	-	-	-	-	-	342	0.227	0.078	-	Pulmonary tuberculosis
BS/16/3	28	1020	0.190	0.194	-	-	-	-	-	140	0.119	0.017	-	Congestive cardiac failure
BS/3/3	28	1562	0.438	0.684	-	+	-	-	-	202	0.427	0.086	+	Viral encephalitis
BS/72/3	29	2000	0.526	1.052	+	-	-	-	-	216	0.455	0.099	+	Stab wound of abdomen
BS/71/3	29	1260	0.547	0.689	+	++	+	+	-	60	5.964	0.358	+++	Stab wound of brain
BS/57/3	29	1258	0.389	0.489	+	-	-	-	-	111	0.538	0.060	++	Road accident
BS/25/3	29	1152	0.344	0.396	+	-	-	++	-	106	0.591	0.063	++	Road accident
BS/23/3	29	1028	0.094	0.097	-	-	-	++	-	128	0.087	0.011	-	Road accident
BS/9/3	29	1604	0.468	0.751	+	-	-	+	-	238	0.148	0.035	-	Anaesthetic death

AFRICAN FEMALES

THIRD DECADE

Total Cases 45

Ref. No.	Age	LIVER							SPLEEN				Cause of Death
			Chemical Iron Estimations		Histological Iron Estimations					Chemical Iron Estimations			
		Weight (grammes)	Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hepatic cells	Kupffer cells	Portal areas	Portal Fibrosis	Cirrhosis	Weight (grammes)	Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hist. Iron	
BS/100/3	20	1186	0.209	0.248	-	-	-	-	-	122	0.362	0.044	+	Diabetic coma
BS/22/3	21	1252	0.134	0.168	-	-	-	-	-	66	0.372	0.025	+	Encephelitis
BS/91/3	21	1423	0.121	0.172	-	-	-	-	-	344	0.115	0.040	-	Pulmonary embolus following salpingectomy for ectopic pregnancy
BS/90/3	21	1860	0.174	0.324	-	-	-	-	-	301	0.178	0.054	-	Post abortion septicaemia
BS/94/3	22	1600	0.150	0.240	-	-	-	-	-	148	0.170	0.025	-	Ruptured uterus - peritonitis.
BS/84/3	22	1490	0.065	0.097	-	-	-	+	-	103	0.070	0.007	-	Eclampsia
BS/83/3	22	1200	0.050	0.064	-	-	-	-	-	170	0.031	0.005	-	Brain abscess: 8½ months pregnant
BS/97/3	23	1910	0.177	0.338	-	+	-	-	-	218	0.327	0.071	+	Bilateral renal cortical necrosis: septic abortion
BS/32/3	24	1242	0.149	0.185	-	-	-	-	-	104	0.139	0.014	-	Bacillary dysentery
BS/29/3	24	1500	0.162	0.243	-	-	-	-	-	368	0.220	0.081	+	Septicaemia following repair of ruptured uterus.
BS/11/3	24	1238	0.081	0.100	-	-	-	-	-	144	0.075	0.011	-	Shock following caesarian section
BS/8/3	24	1778	0.686	1.219	+	+	-	-	-	1655	0.236	0.391	+	Thrombosis of mesenteric vein.
BS/85/3	25	1286	0.916	1.178	+	+++	-	-	-	88	2.638	0.232	+++	Renal failure : acute pyelonephritis with abscess formation.
BS/86/3	25	856	0.478	0.409	+	-	-	-	-	138	0.237	0.033	+	Ruptured ectopic pregnancy
BS/87/3	25	1342	0.262	0.352	-	+	-	-	-	152	0.288	0.044	+	Pulmonary tuberculosis
BS/88/3	25	1330	0.173	0.230	-	-	-	+	-	160	0.369	0.059	+	Pulmonary tuberculosis
BS/89/3	25	1746	2.294	4.005	+++	++	+	-	-	122	1.156	0.141	++	Lobar pneumonia
BS/92/3	25	1618	0.346	0.567	-	+	-	-	-	90	1.051	0.095	++	Renal failure - septic abortion
BS/93/3	25	964	0.094	0.090	-	-	-	+	-	189	0.114	0.022	-	Bronchopneumonia: mitral stenosis
BS/95/3	25	1042	0.119	0.124	-	-	-	-	-	88	0.229	0.020	-	Pulmonary oedema following left salpingectomy for ectopic pregnancy
BS/96/3	25	1630	1.213	1.977	++	++	+	-	-	110	1.026	0.113	++	Puerperal pyaemia.
BS/81/3	25	1220	0.163	0.199	-	-	-	++	-	294	0.237	0.070	-	Post partum haemorrhage
BS/80/3	25	1634	0.213	0.348	-	+	-	-	-	110	0.373	0.041	+	Renal failure - chronic pyelonephritis.
BS/76/3	25	1300	0.093	0.121	-	-	-	-	-	192	0.082	0.016	-	Post partum haemorrhage
BS/68/3	25	1903	0.122	0.232	-	-	-	+	-	210	0.150	0.031	-	Carbon monoxide poisoning
BS/56/3	25	1778	0.371	0.660	-	+	-	-	-	144	0.746	0.098	++	Renal failure: chronic pyelonephritis
BS/54/3	25	1518	0.087	0.132	-	-	-	-	-	180	0.109	0.020	-	Chorionepithelioma
BS/53/3	25	1279	0.127	0.162	-	-	-	-	-	118	0.161	0.019	-	Lobar pneumonia

Ref. No.	Age	LIVER								SPLEEN				Cause of Death
		Weight (grammes)	Chemical Iron Estimations		Histological Iron Estimations			Portal Fibrosis	Cirrhosis	Weight (grammes)	Chemical Iron Estimations		Hist. Iron	
			Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hepatic cells	Kupffer cells	Portal areas				Iron Concentration (mg/g wet wt)	Total Iron (grammes)		
BS/52/3	25	1400	0.174	0.244	-	-	-	++++	Coarse	362	0.259	0.094	+	Carcinoma of liver
BS/45/3	25	1076	0.412	0.443	+	-	-	+	-	64	0.331	0.021	+	Brain injury - murder
BS/34/3	25	2148	0.344	0.739	-	+	-	+	-	302	1.237	0.374	++	Renal failure: chronic pyelonephritis.
BS/30/3	25	1690	0.179	0.302	-	-	-	-	-	218	0.134	0.029	-	Peritonitis - ruptured uterus
BS/12/3	25	2370	0.349	0.827	+	-	-	+	-	196	0.382	0.075	+	Inhalation of vomit - drunk
BS/77/3	26	1342	0.126	0.169	-	-	-	-	-	226	0.219	0.049	-	Infectious hepatitis
BS/60/3	26	1093	0.151	0.165	-	-	-	-	-	129	0.181	0.023	-	Traumatic intracranial haemorrhage
BS/28/3	26	1340	0.275	0.369	+	-	-	-	-	142	0.197	0.028	-	Suicidal hanging
BS/24/3	26	1252	0.546	0.684	+	+	-	-	-	132	0.845	0.112	++	Refractory anaemia
BS/2/3	26	1544	0.166	0.256	-	-	-	-	-	122	0.174	0.021	-	Peritonitis - ruptured uterus
BS/82/3	27	2140	0.137	0.293	-	-	-	-	-	264	0.156	0.041	-	Chorionepithelioma
BS/10/3	27	1234	0.092	0.114	-	-	-	-	-	149	0.378	0.056	+	Abortion - haemorrhage
BS/98/3	28	2315	0.375	0.868	+	-	-	-	-	220	0.334	0.073	+	Puerperal septicaemia
BS/46/3	28	1538	0.173	0.266	-	-	-	-	-	260	0.205	0.053	+	Postpartum haemorrhage
BS/13/3	28	1386	0.240	0.333	-	-	-	+	-	115	0.234	0.027	+	Haemorrhage following caesarian section
BS/99/3	29	848	0.835	0.708	+	+++	++	-	-	178	1.899	0.338	+++	Chronic pyelonephritis: miliary tuberculosis
BS/5/3	29	1324	0.161	0.213	-	-	-	-	-	141	0.940	0.133	++	Peritonitis: chronic pyelonephritis

AFRICAN MALES

FOURTH DECADE

Total Cases 65

Ref. No.	Age	LIVER								SPLEEN				Cause of Death
		Weight (grammes)	Chemical Iron Estimations		Histological Iron Estimations			Portal Fibrosis	Cirrhosis	Weight (grammes)	Chemical Iron Estimations		Hist. Iron	
			Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hepatic cells	Kupffer cells	Portal areas				Iron Concentration (mg/g wet wt)	Total Iron (grammes)		
BS/77/4	30	1609	0.165	0.235	-	-	-	-	-	146	0.142	0.021	-	Suicidal hanging
BS/74/4	30	1438	0.910	1.309	+	++	+	-	-	165	3.787	0.625	+++	Lymphatic leukaemia
BS/73/4	30	1334	0.265	0.354	-	-	-	++++	Coarse	213	0.336	0.072	+	Lobar pneumonia and empyema
BS/71/4	30	1274	0.718	0.915	+	++	+	-	-	143	0.484	0.069	++	Head injury (murder)
BS/68/4	30	1997	0.376	0.751	+	-	-	-	-	313	0.190	0.059	-	Mitral incompetence - rheumatic carditis.
BS/66/4	30	1346	0.833	1.121	++	++	+	-	-	59	1.833	0.108	+++	Road accident
BS/65/4	30	2214	0.599	1.326	++	+	-	-	-	335	0.463	0.155	++	Stab wound of abdomen: peritonitis
BS/63/4	30	1960	0.585	1.147	+	-	-	-	-	126	0.368	0.046	+	Drowning
BS/62/4	30	1286	1.319	1.696	++	++	++	-	-	133	1.844	0.245	+++	Road accident
BS/54/4	30	1509	1.282	1.935	++	++	+	-	-	72	1.025	0.074	++	Stab wound of heart
BS/53/4	30	1184	0.131	0.155	-	-	-	+++	Bilharzial Fibrosis	244	0.153	0.037	-	Uraemia: chronic pyelonephritis
BS/51/4	30	1624	0.112	0.182	-	-	-	-	-	152	0.176	0.027	-	Syphilitic aortic incompetence
BS/50/4	30	982	0.152	0.149	-	-	-	+	-	100	0.196	0.020	-	Road accident
BS/40/4	30	1820	0.271	0.493	-	+	-	-	-	151	0.359	0.054	+	Fractured spine
BS/35/4	30	1558	0.169	0.269	-	-	-	-	-	260	0.156	0.041	-	Struck by lightning
BS/34/4	30	1932	0.831	1.605	++	++	+	-	-	240	0.277	0.066	+	Peritonitis: traumatic rupture of small bowel.
BS/31/4	30	1263	1.954	2.468	+++	+	+	+	-	120	1.020	0.122	++	Road accident
BS/18/4	30	1802	1.334	2.404	++	+++	++	+	-	210	5.518	1.159	+++	Road accident
BS/23/4	31	1674	1.515	2.536	+++	+	-	-	-	202	0.978	0.198	++	Traumatic intracranial haemorrhage (murder).
BS/19/4	31	1152	0.854	0.984	+	++	-	-	-	182	0.477	0.087	+	Cor pulmonale
BS/39/4	32	1634	1.349	2.204	+++	+++	++	-	-	257	0.860	0.221	++	Food poisoning
BS/25/4	32	1242	0.249	0.309	-	-	-	+	-	198	0.181	0.036	-	Road accident
BS/10/4	33	1568	0.456	0.725	+	+	-	-	-	114	0.680	0.078	+	Brain tumour (ependymoma)
BS/76/4	35	1728	1.127	1.947	++	+++	++	-	-	187	0.843	0.158	++	Brain damage (murder)
BS/72/4	35	1580	1.522	2.405	+++	+	+	-	-	292	0.645	0.188	++	Asphyxia - mining accident
BS/70/4	35	1178	0.071	0.084	-	-	-	++++	Coarse	490	0.219	0.107	-	Carcinoma of liver
BS/67/4	35	1525	0.505	0.770	+	+	-	-	-	199	0.309	0.061	+	Suicidal hanging
BS/64/4	35	1205	1.045	1.259	++	+	+	-	-	148	0.731	0.108	++	Brain damage (murder)
BS/61/4	35	1265	0.166	0.210	-	-	-	+	-	139	0.100	0.014	-	Road accident

Ref. No.	Age	LIVER							SPLEEN				Cause of Death
			Chemical Iron Estimations		Histological Iron Estimations					Chemical Iron Estimations			
		Weight (grammes)	Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hepatic cells	Kupffer cells	Portal areas	Portal Fibrosis	Cirrhosis	Weight (grammes)	Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hist. Iron	
BS/59/4	35	1720	0.123	0.212	-	-	-	-	-	142	0.124	0.018	-	Brain damage (murder)
BS/58/4	35	1616	0.808	1.304	+	++	+	+	-	132	0.733	0.097	++	Road accident
BS/56/4	35	1917	0.396	0.759	-	-	-	+	-	369	0.394	0.145	-	Cerebral malaria: malarial pigment++ in liver and spleen
BS/49/4	35	1456	0.267	0.389	-	+	-	+	-	126	0.654	0.082	++	Bilateral pyelonephrosis
BS/47/4	35	1854	0.303	0.562	-	+	-	+	-	240	0.287	0.069	+	Bilateral hydronephrosis: bilharzial stenosis of ureters.
BS/44/4	35	1390	0.136	0.153	-	-	-	-	-	111	0.121	0.013	-	Road accident
BS/42/4	35	1268	3.210	4.070	+++	++	++	++	-	130	1.656	0.215	+++	Lobar pneumonia
BS/38/4	35	1404	0.790	1.109	+	-	++	++	-	242	0.400	0.097	+	Status epilepticus
BS/37/4	35	1804	0.702	1.266	+	++	++	-	-	82	2.050	0.168	+++	Brain damage (murder)
BS/36/4	35	1238	0.301	0.373	+	-	-	+	-	174	0.281	0.049	+	Multiple injuries - mine accident
BS/33/4	35	1726	0.108	0.186	-	-	-	+	-	198	0.300	0.059	+	Septicaemia
BS/32/4	35	1306	0.714	0.932	+	-	-	-	-	88	0.619	0.054	+	Road accident
BS/28/4	35	1100	0.391	0.430	+	-	+	-	-	126	0.197	0.025	-	Suicidal hanging
BS/27/4	35	1574	0.317	0.499	+	-	-	-	-	148	0.117	0.017	-	Suicidal hanging
BS/26/4	35	1618	0.053	0.086	-	-	-	+	-	234	0.090	0.021	-	Acute alcoholic intoxication
BS/24/4	35	1386	0.244	0.338	-	-	-	-	-	218	0.899	0.196	++	Renal failure: chronic pyelonephritis
BS/22/4	35	1200	0.731	0.877	+	+	-	+	-	142	0.646	0.092	+	Road accident
BS/16/4	35	2190	2.991	6.550	++	+++	+++	+++	Fine	109	5.167	0.563	+++	Road accident
BS/15/4	35	1902	1.376	2.617	++	+	-	-	-	118	2.402	0.283	+++	Road accident
BS/14/4	35	1245	0.540	0.672	+	-	-	+	-	138	0.548	0.076	+	Bronchopneumonia
BS/12/4	35	1574	0.444	0.699	+	-	-	-	-	176	1.369	0.241	++	Uraemia: chronic pyelonephritis
BS/11/4	35	1802	0.283	0.510	-	+	-	-	-	320	0.543	0.174	+	Renal failure: chronic pyelonephritis
BS/8/4	35	950	0.518	0.492	+	+	-	-	-	162	0.882	0.160	++	Road accident
BS/6/4	35	1116	0.555	0.619	+	-	-	-	-	164	0.508	0.082	+	Road accident
BS/4/4	35	1720	0.411	0.707	+	+	-	-	-	158	0.338	0.053	+	Road accident
BS/3/4	35	1168	0.447	0.522	+	+	-	-	-	125	1.165	0.148	++	Brain damage - assault
BS/30/4	37	1198	0.155	0.186	-	-	-	-	-	154	0.894	0.138	++	Tuberculous meningitis
BS/9/4	37	1402	0.125	0.175	-	-	-	-	-	158	0.196	0.031	-	Status epilepticus
BS/1/4	37	1774	1.050	1.863	++	+++	++	++++	Coarse	512	0.355	0.182	+	Road accident
BS/48/4	38	1210	0.905	1.095	++	++	+	+	-	125	0.937	0.117	+++	Road accident

Ref. No.	Age	LIVER								SPLEEN				Cause of Death
		Weight (grammes)	Chemical Iron Estimations		Histological Iron Estimations			Portal Fibrosis	Cirrhosis	Weight (grammes)	Chemical Iron Estimations		Hist. Iron	
			Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hepatic cells	Kupffer cells	Portal areas				Iron Concentration (mg/g wet wt)	Total Iron (grammes)		
BS/29/4	38	1818	0.236	0.429	-	+	-	-	-	124	0.154	0.019	-	Road accident
BS/13/4	38	1014	0.180	0.183	-	-	-	-	-	124	0.176	0.022	-	Suicidal hanging
BS/80/4	39	2364	2.941	6.950	+++	+++	+++	-	-	284	5.904	1.680	+++	Inhalation of vomit (drunk)
BS/79/4	39	1240	0.162	0.201	-	-	-	-	-	166	0.096	0.016	-	Status asthmaticus
BS/75/4	39	1314	0.133	0.175	-	-	-	-	-	175	0.083	0.014	-	Suicidal hanging
BS/2/4	39	1526	4.468	6.818	++	+++	+++	+++	-	166	3.054	0.507	+++	Road accident

AFRICAN FEMALES

FOURTH DECADE

Total Cases 35

Ref. No.	Age	LIVER								SPLEEN				Cause of Death
		Weight (grammes)	Chemical Iron Estimations		Histological Iron Estimations			Portal Fibrosis	Cirrhosis	Weight (grammes)	Chemical Iron Estimations		Hist. Iron	
			Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hepatic cells	Kupffer cells	Portal areas				Iron Concentration (mg/g wet wt)	Total Iron (grammes)		
BS/91/4	30	1292	0.136	0.176	-	-	-	-	-	142	0.208	0.028	-	Suppurative meningitis
BS/90/4	30	1903	0.146	0.278	-	-	-	-	-	288	0.311	0.090	+	Extensive burns - suicide
BS/85/4	30	1876	4.368	8.200	+++	+++	+++	++	-	503	1.533	0.772	++	Suppurative meningitis
BS/60/4	30	1946	0.454	0.884	-	++	-	-	-	140	0.546	0.077	++	Chronic pyelonephritis
BS/43/4	30	1276	0.073	0.093	-	-	-	++	-	196	0.099	0.019	-	Congestive cardiac failure - mitral incompetence.
BS/5/4	31	1256	0.178	0.224	-	-	-	-	-	210	0.396	0.083	+	Chronic pyelonephritis: renal failure.
BS/21/4	32	1680	0.062	0.104	-	-	-	-	-	138	0.134	0.018	-	Haemorrhage from vaginal tear following parturition
BS/20/4	33	2028	0.545	1.105	+	+	++	++++	Coarse	434	0.273	0.118	+	Inhalation of vomit - drunk
BS/94/4	35	1524	0.324	0.494	-	+	-	-	-	196	0.344	0.067	+	Asphyxia - obstructed airway - thyroidectomy
BS/93/4	35	984	0.125	0.123	-	-	-	-	-	86	0.099	0.009	-	Ruptured ectopic pregnancy
BS/92/4	35	2289	0.262	0.600	-	-	-	-	-	559	0.399	0.240	+	Septicaemia - severe burns
BS/89/4	35	1250	0.147	0.184	-	-	-	++	-	135	0.161	0.022	-	Acute peritonitis - tubo-ovarian abscess.
BS/88/4	35	1127	2.351	2.642	+++	++	++	+	-	132	1.458	0.192	+++	Pulmonary tuberculosis.
BS/87/4	35	1310	0.416	0.545	+	-	-	+	-	134	0.066	0.009	-	Carcinoma of cervix
BS/86/4	35	2135	3.332	7.110	+++	+++	+++	+	-	148	1.677	0.248	+++	Lobar pneumonia.
BS/84/4	35	1630	0.302	0.436	-	+	-	-	-	126	0.740	0.093	++	Chorionepithelioma
BS/83/4	35	1222	0.514	0.629	-	+++	-	-	-	168	1.625	0.273	++	Pyonephrosis: carcinoma bladder
BS/82/4	35	1192	0.443	0.528	-	++	-	+	-	172	0.949	0.163	++	Renal failure: renal vein thrombosis. Lobar pneumonia.
BS/81/4	35	1013	0.847	0.860	++	+++	-	-	-	104	0.874	0.091	++	Renal failure: pyonephrosis, ureteric stenosis.
BS/78/4	35	2262	0.090	0.204	-	-	-	-	-	2198	0.071	0.156	-	Myeloid leukaemia.
BS/69/4	35	1995	0.362	0.722	-	++	-	-	-	136	0.338	0.046	+	Peritonitis following caesarian section.
BS/57/4	35	1360	0.069	0.094	-	-	-	+	-	192	0.075	0.013	-	Chorionepithelioma
BS/55/4	35	1272	1.684	2.142	++	+++	++	-	-	128	1.213	0.155	+++	Ruptured uterus: obstructed labour.
BS/52/4	35	1444	0.281	0.406	-	-	-	-	-	118	0.204	0.024	-	Viral pneumonia.
BS/46/4	35	2390	0.219	0.523	-	-	-	-	-	236	0.324	0.076	+	Puerperal septicaemia

Ref. No.	Age	LIVER								SPLEEN				Cause of Death
			Chemical Iron Estimations		Histological Iron Estimations			Portal Fibrosis	Cirrhosis		Chemical Iron Estimations		Hist. Iron	
		Weight (grammes)	Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hepatic cells	Kupffer cells	Portal areas			Weight (grammes)	Iron Concentration (mg/g wet wt)	Total Iron (Grammes)		
BS/45/4	35	1528	0.115	0.175	-	-	-	-	-	160	0.076	0.012	-	Road accident
BS/41/4	35	1230	1.144	1.407	++	++	+++	++++	Fine	115	2.399	0.276	+++	Liver failure : portal cirrhosis
BS/7/4	36	1270	0.128	0.163	-	-	-	-	-	204	0.384	0.078	+	Renal failure: chronic pyelonephritis.
BS/99/4	37	1426	0.302	0.431	+	-	-	-	-	110	0.181	0.020	-	Pleomorphic salivary adenocarcinoma invading brain.
BS/100/4	38	1230	0.124	0.153	-	-	-	+	-	175	0.110	0.019	-	Carcinoma of cervix.
BS/98/4	38	1260	0.069	0.087	-	-	-	+	-	150	0.087	0.013	-	Road accident
BS/97/4	38	1098	0.255	0.280	+	-	-	-	-	100	0.119	0.012	-	Status epilepticus
BS/96/4	38	2380	0.308	0.733	+	-	-	-	-	360	0.163	0.059	-	Carcinoma of cervix
BS/95/4	39	1340	0.473	0.634	-	++	++	-	-	70	3.564	0.249	+++	Chorionepithelioma
BS/17/4	39	1118	0.091	0.102	-	-	-	-	-	142	0.077	0.011	-	Ruptured uterus - obstructed labour.

AFRICAN MALES

FIFTH DECADE

Total Cases 59

Ref. No.	Age	LIVER							SPLEEN				Cause of Death
			Chemical Iron Estimations		Histological Iron Estimations					Chemical Iron Estimations			
		Weight (grammes)	Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hepatic cells	Kupffer cells	Portal areas	Portal Fibrosis	Cirrhosis	Weight (grammes)	Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hist. Iron	
BS/81/5	40	1412	0.327	0.462	-	+	-	-	-	106	0.488	0.052	+	Road accident
BS/77/5	40	1480	0.325	0.481	-	-	-	-	-	120	0.249	0.030	-	Road accident
BS/76/5	40	1438	0.536	0.771	+	++	-	-	-	132	1.145	0.151	+++	Renal failure: chronic pyelonephritis
BS/75/5	40	1715	0.347	0.595	+	-	-	-	-	206	0.720	0.148	++	Paraplegia: terminal bronchopneumonia
BS/74/5	40	1414	0.863	1.220	++	+	+	-	-	252	2.150	0.542	+++	Road accident
BS/70/5	40	1532	1.144	1.753	++	+++	++	+	-	214	1.807	0.387	+++	Tuberculous meningitis: miliary tuberculosis
BS/65/5	40	1470	2.121	3.118	+++	+++	+++	-	-	160	0.853	0.136	+++	Hypertensive heart failure
BS/61/5	40	1410	0.703	0.991	+	++	-	-	-	130	0.426	0.055	+	Suicidal hanging
BS/57/5	40	1048	0.773	0.815	++	-	-	-	-	68	0.379	0.026	+	Bowel obstruction: volvulus of pelvic colon.
BS/55/5	40	1603	1.037	1.662	++	+++	++	++	-	173	2.404	0.436	+++	Road accident
BS/52/5	40	1512	0.189	0.286	+	+	-	+	-	178	1.278	0.227	++	Road accident
BS/36/5	40	1232	0.433	0.533	-	+++	++	+	-	148	2.901	0.429	+++	Diabetic coma
BS/35/5	40	1326	1.667	2.190	++	+++	+	++++	Coarse	150	2.811	0.422	+++	Traumatic brain injury
BS/33/5	40	1274	0.896	1.142	++	+	-	-	-	90	1.501	0.135	+++	Road accident
BS/23/5	40	1552	3.820	5.929	+++	++	++	-	-	178	2.197	0.381	+++	Road accident
BS/26/5	41	1710	0.178	0.304	-	-	-	-	-	144	0.196	0.028	-	Road accident
BS/9/5	41	1450	0.323	0.468	-	+	-	-	-	126	0.452	0.057	+	Renal failure: bilateral hydronephrosis
BS/45/5	42	1264	0.108	0.137	-	-	-	++	-	258	0.104	0.027	+	Renal failure: chronic pyelonephritis
BS/22/5	42	1100	3.092	3.401	++	+++	+++	-	-	148	3.843	0.569	+++	Traumatic intracranial haemorrhage
BS/20/5	43	1352	0.227	0.307	+	-	-	++	-	122	0.267	0.033	+	Carcinoma of oesophagus
BS/15/5	43	1558	8.815	13.734	++	+++	+++	+	-	298	8.844	2.635	+++	Acute alcoholic intoxication
BS/40/5	44	1650	1.790	2.954	++	+++	+++	+	-	194	2.484	0.487	+++	Road accident
BS/16/5	44	1742	3.451	6.002	++	+++	++	+	-	90	3.184	0.287	+++	Road accident
BS/73/5	45	1924	0.284	0.546	-	+	-	-	-	230	0.242	0.056	+	Apoplexy - hypertension
BS/72/5	45	952	0.973	0.926	++	++	+	-	-	80	1.641	0.131	+++	Ulceration of oesophagus into aorta (traumatic)
BS/71/5	45	1500	1.058	1.587	++	+	-	-	-	108	0.931	0.101	++	Traumatic intracranial haemorrhage
BS/67/5	45	1495	0.077	0.115	-	-	-	++++	Coarse	721	0.350	0.252	-	Carcinoma of liver
BS/62/5	45	1630	1.010	1.646	++	++	+	-	-	166	0.662	0.110	++	Carcinoma of bladder
BS/56/5	45	1120	1.654	1.842	+++	++	+++	++++	Coarse	1266	0.349	0.442	+	Haemorrhage from oesophageal varices
BS/53/5	45	1370	1.815	2.487	+++	++	++	+	-	102	1.772	0.181	+++	Bowel obstruction: volvulus of sigmoid colon
BS/51/5	45	2774	2.560	7.101	+	+++	+++	++++	Fine	364	1.621	0.590	+++	Road accident

Ref. No.	Age	LIVER							SPLEEN				Cause of Death
			Chemical Iron Estimations		Histological Iron Estimations					Chemical Iron Estimations			
		Weight (grammes)	Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hepatic cells	Kupffer cells	Portal areas	Portal Fibrosis	Cirrhosis	Weight (grammes)	Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hist. Iron	
BS/49/5	45	1900	0.868	1.649	++	+	-	-	-	162	1.023	0.166	++	Lung abscess
BS/48/5	45	1500	1.446	2.169	++	+++	+++	++	-	84	2.522	0.212	+++	Road accident
BS/43/5	45	1700	3.428	5.828	+++	++	+++	+	-	200	1.649	0.330	+++	Arsenical poisoning
BS/34/5	45	1518	2.151	3.265	++	+++	+++	+	-	142	1.734	0.246	+++	Syphilitic aortitis: coronary occlusion
BS/32/5	45	1325	11.095	14.701	+++	+++	+++	++++	Fine	442	2.924	1.292	+++	Tuberculous peritonitis
BS/30/5	45	1152	4.081	4.701	+++	+++	+++	-	-	110	4.593	0.505	+++	Suicidal hanging
BS/29/5	45	1572	4.814	7.568	+++	+++	+++	-	-	120	4.390	0.527	+++	Road accident
BS/27/5	45	1506	5.890	9.342	++	+++	+++	+	-	79	10.960	0.866	+++	Road accident
BS/25/5	45	2060	0.708	1.458	++	+++	++	++++	Coarse	518	0.174	0.090	-	Ruptured oesophageal varices and carcinoma of liver.
BS/19/5	45	1672	2.199	3.677	++	+++	+++	++	-	206	2.170	0.447	+++	Bacillary dysentery
BS/18/5	45	1916	2.634	5.047	+++	++	+++	+	-	602	1.956	1.178	+++	Miliary tuberculosis
BS/2/5	45	1772	0.506	0.897	+	-	-	-	-	154	0.524	0.081	+	Road accident
BS/39/5	46	1446	1.321	1.910	+	+++	+	+	-	138	2.719	0.375	+++	Renal failure: chronic pyelonephritis
BS/38/5	46	1358	0.164	0.223	-	-	-	-	-	176	0.102	0.018	-	Road accident
BS/28/5	46	1150	0.111	0.128	-	-	-	+	-	132	0.154	0.020	-	Ruptured aortic aneurysm
BS/13/5	46	1702	1.679	2.958	++	+	-	-	-	103	0.649	0.067	++	Carcinoma of oesophagus
BS/8/5	46	1368	1.270	1.737	++	++	-	+	-	194	0.987	0.191	++	Lobar pneumonia
BS/7/5	46	1183	2.799	3.311	++	++	-	+	-	98	2.064	0.202	++	Road accident
BS/46/5	47	1414	0.287	0.406	+	+	-	++	-	176	0.813	0.142	++	Renal failure: chronic pyelonephritis
BS/10/5	47	1094	0.443	0.485	+	-	-	-	-	84	0.263	0.022	+	Brain damage (murder)
BS/50/5	48	3068	0.246	0.755	-	+	-	++++	Coarse	655	0.252	0.165	+	Haemorrhage from oesophageal varices: carcinoma of liver
BS/31/5	48	1408	0.613	0.863	+	+	-	-	-	164	1.225	0.202	++	Road accident
BS/21/5	48	1024	7.866	8.055	++	+++	+++	++	-	178	11.409	2.031	+++	Pulmonary tuberculosis
BS/4/5	48	1520	0.133	0.202	-	-	-	-	-	180	0.184	0.033	+	Asphyxia: inhalation of dust - mine accident.
BS/17/5	49	956	2.582	2.484	++	+++	+	-	-	112	2.040	0.228	+++	Lobar pneumonia
BS/14/5	49	1064	11.898	22.178	+++	+++	+++	++++	Coarse	106	5.137	0.545	+++	Road accident
BS/5/5	49	1118	4.669	5.220	+++	++	+	+	-	126	4.460	0.562	+++	Congestive cardiac failure: mitral stenosis.
BS/1/5	49	1534	7.202	10.970	++	+++	+++	+	-	82	6.648	0.545	+++	? Irreversible shock in Bantu siderosis.

AFRICAN FEMALES

FIFTH DECADE

Total Cases 41

Ref. No.	Age	LIVER							SPLEEN				Cause of Death
			Chemical Iron Estimations		Histological Iron Estimations					Chemical Iron Estimations			
		Weight (grammes)	Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hepatic cells	Kupffer cells	Portal areas.	Portal Fibrosis	Cirrhosis	Weight (grammes)	Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hist. Iron	
BS/82/5	40	974	0.081	0.089	-	-	-	-	-	61	0.084	0.005	-	Congestive heart failure : cardio-myopathy.
BS/98/5	40	2004	0.296	0.592	+	+	-	-	-	214	0.916	0.196	++	Pulmonary embolus: pelvic sepsis
BS/94/5	40	1381	1.058	1.461	++	++	-	+	-	110	0.419	0.046	+	Megaloblastic anaemia
BS/93/5	40	1378	0.476	0.656	+	++	-	-	-	184	0.658	0.121	++	Pyaemia: pelvic sepsis
BS/91/5	40	1040	0.258	0.268	-	-	-	+++	Bilharzial Fibrosis.	584	0.056	0.033	-	Haemorrhage from ruptured oesophageal varices.
BS/90/5	40	973	0.531	0.517	++	+	+	++++	Coarse	240	1.075	0.253	++	Haemorrhage from ruptured oesophageal varices.
BS/89/5	40	1404	0.114	0.160	-	-	-	+	-	156	0.091	0.014	-	Apoplexy - hypertension
BS/88/5	40	1304	0.491	0.640	+	+	-	+	-	40	0.407	0.016	+	Refractory anaemia
BS/87/5	40	1075	0.948	1.020	++	++	+	-	-	195	0.713	0.139	++	Chorionepithelioma
BS/86/5	40	1592	0.142	0.226	-	-	-	-	-	208	0.145	0.030	-	Haemorrhage from tear of cervix uteri during delivery.
BS/85/5	40	1771	0.254	0.449	-	-	-	-	-	174	0.434	0.076	+	Congestive heart failure : endomyocardial fibrosis.
BS/80/5	40	1030	0.102	0.105	-	-	-	-	-	160	0.118	0.019	-	Lobar pneumonia
BS/79/5	40	1812	0.109	0.198	-	-	-	-	-	420	0.169	0.071	-	Septicaemia
BS/78/5	40	1598	3.046	4.868	+++	+++	+++	-	-	76	3.792	0.294	+++	Bacillary dysentery
BS/69/5	40	1363	0.219	0.298	-	-	-	-	-	204	0.290	0.059	-	Toxaemia : paraplegia : Pott's disease of spine.
BS/68/5	40	1920	0.397	0.762	-	+	-	-	-	252	1.009	0.254	+++	Renal failure: chronic pyelo-nephritis
BS/66/5	40	1550	0.425	0.659	-	+	-	+	-	70	0.843	0.058	+	Renal failure: chronic pyelo-nephritis.
BS/64/5	40	1920	0.236	0.453	-	+	-	-	-	326	0.107	0.035	-	Renal tubular necrosis
BS/60/5	40	1452	4.465	6.483	+++	+++	+++	+	-	64	3.514	0.225	+++	Road accident
BS/59/5	40	1463	0.644	0.942	++	++	++	++++	Fine	184	1.032	0.190	+++	Lobar pneumonia
BS/54/5	40	1320	0.149	0.197	-	-	-	-	-	73	0.367	0.027	+	Status asthmaticus
BS/47/5	40	1152	0.537	0.619	+	+	-	-	-	212	1.169	0.248	++	Renal failure: chronic pyelo-nephritis.
BS/37/5	41	1436	0.797	1.144	+	++	-	+	-	172	0.639	0.110	++	Haemorrhage into large bowel: chronic pyelonephritis.
BS/42/5	42	1720	0.546	0.939	-	++	-	-	-	160	0.768	0.123	++	Renal failure: carcinoma of bladder
BS/24/5	42	1940	0.183	0.355	-	-	-	-	-	244	0.289	0.071	+	Peritonitis: ruptured uterus

Ref. No.	Age	LIVER							SPLEEN				Cause of Death
		Weight (grammes)	Chemical Iron Estimations		Histological Iron Estimations			Portal Fibrosis	Cirrhosis	Weight (grammes)	Chemical Iron Estimations		Hist. Iron	
			Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hepatic cells	Kupffer cells	Portal areas				Iron Concentration (mg/g wet wt)	Total Iron (grammes)		
BS/12/5	42	1352	0.158	0.214	-	-	-	-	-	148	0.559	0.083	+	Pyonephrosis - bilateral
BS/100/5	45	1635	0.141	0.231	-	-	-	-	-	107	0.328	0.035	+	Road accident
BS/99/5	45	1588	0.097	0.154	-	-	-	-	-	255	0.052	0.013	-	Puerperal septicaemia
BS/97/5	45	1451	0.626	0.908	+	+++	+	-	-	378	0.550	0.208	++	Tumour of lymphoid tissue - probably lymphosarcoma
BS/96/5	45	1549	0.191	0.296	-	-	-	+++	-	148	0.455	0.068	++	Brain damage - murder.
BS/95/5	45	1712	0.283	0.484	-	+	-	-	-	166	0.332	0.055	+	Postoperative inhalation of blood - asphyxia.
BS/92/5	45	1440	0.778	1.120	++	++	-	-	-	108	0.406	0.044	+	Tuberculous bronchopneumonia
BS/84/5	45	1520	0.230	0.350	-	-	-	-	-	116	0.148	0.017	-	Apoplexy - hypertension
BS/83/5	45	1031	0.149	0.154	-	-	-	-	-	54	0.449	0.024	+	Bronchopneumonia.
BS/63/5	45	1781	0.279	0.497	-	+	-	+	-	112	0.580	0.065	++	Diabetic coma
BS/44/5	45	984	0.564	0.555	+	-	-	-	-	106	0.328	0.035	+	Brain damage - murder.
BS/41/5	45	1518	0.094	0.143	-	-	-	+	-	226	0.186	0.042	-	Road accident
BS/3/5	45	1224	0.170	0.208	-	-	-	+	-	152	0.236	0.036	+	Anaesthetic death.
BS/11/5	46	1298	0.051	0.066	-	-	-	-	-	136	0.023	0.003	-	Haemorrhage: criminal abortion
BS/50/5	48	2736	0.103	0.282	-	-	-	-	-	82	0.523	0.043	++	Carcinoma head of pancreas
BS/6/5	48	1008	0.381	0.384	+	-	-	-	-	92	1.163	0.107	+++	Apoplexy - hypertension.

AFRICAN MALES

SIXTH DECADE

Total Cases 71

Ref. No.	Age	LIVER								SPLEEN				Cause of Death
		Weight (grammes)	Chemical Iron Estimations		Histological Iron Estimations			Portal Fibrosis	Cirrhosis	Weight (grammes)	Chemical Iron Estimations		Hist. Iron	
			Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hepatic cells	Kupffer cells	Portal areas				Iron Concentration (mg/g wet wt)	Total Iron (grammes)		
BS/77/6	50	1412	0.872	1.231	+	++	++	-	-	164	1.149	0.188	++	Lobar pneumonia
BS/78/6	50	1472	0.215	0.316	-	++	-	-	-	176	0.996	0.175	++	L. Heart failure: chronic pyelonephritis: pulmonary tuberculosis
BS/79/6	50	1758	5.661	9.950	+++	++	+++	++	-	180	5.339	0.961	+++	Tuberculous meningitis: miliary tuberculosis.
BS/80/6	50	1446	3.429	4.960	+++	+++	+++	++++	Coarse	310	3.473	1.076	+++	Haemorrhage: oesophageal varices
BS/81/6	50	1942	1.101	2.140	++	+++	+	-	-	300	1.155	0.346	++	Lymphosarcoma
BS/74/6	50	1536	4.195	6.444	+++	+++	+++	+	-	290	8.093	2.347	+++	Carcinoma of oesophagus
BS/73/6	50	1188	1.794	2.131	++	+++	++	+	-	99	8.841	0.875	+++	Carcinoma of oesophagus
BS/70/6	50	1622	3.278	5.317	+++	++	+++	++++	Fine	370	0.915	0.339	++	Apoplexy - hypertension
BS/68/6	50	1196	0.464	0.555	++	+	-	-	-	115	0.733	0.084	++	Carcinoma of bronchus
BS/66/6	50	1803	4.050	7.302	+++	+++	+++	++++	Coarse	323	3.318	1.072	+++	Carcinoma of oesophagus
BS/65/6	50	1503	0.282	0.424	-	+	-	-	-	218	0.619	0.135	++	Carcinoma of stomach.
BS/62/6	50	1236	0.216	0.267	-	-	-	++	-	326	0.141	0.046	-	Road accident
BS/57/6	50	2742	0.230	0.631	-	-	-	+	-	120	0.435	0.052	+	Carcinoma of bronchus
BS/56/6	50	1651	4.182	6.904	+++	+++	+++	+	-	172	5.825	1.002	+++	Myocardial infarction: syphilitic aortitis.
BS/53/6	50	1046	0.326	0.341	+	-	-	-	-	30	0.474	0.014	+	Lobar pneumonia: Pott's disease of spine: Paraplegia.
BS/52/6	50	1722	0.368	0.634	+	-	-	++++	Coarse	434	0.252	0.011	+	Carcinoma of liver.
BS/51/6	50	2290	0.165	0.378	-	-	-	-	-	370	0.096	0.036	-	Hypertensive heart failure: chronic pyelonephritis.
BS/47/6	50	1543	1.304	2.012	++	+++	+	-	-	91	1.537	0.140	+++	Amoebic dysentery
BS/45/6	50	1198	0.515	0.617	+	++	++	++	-	150	3.707	0.556	+++	Pulmonary tuberculosis
BS/42/6	50	1090	0.057	0.062	-	-	-	++	-	112	0.053	0.006	-	Pulmonary tuberculosis
BS/33/6	50	1270	2.143	2.722	++	+++	++	++	-	150	2.006	0.301	+++	Miliary tuberculosis
BS/30/6	50	2588	3.229	8.357	+++	+++	+++	++	-	410	1.780	0.730	+++	"Mushroom" poisoning
BS/29/6	50	1268	1.490	1.889	++	+++	+	-	-	262	3.296	0.864	+++	Hodgkin's disease
BS/16/6	51	1454	1.049	1.525	++	-	+	-	-	128	1.020	0.131	++	Suppurative meningitis
BS/14/6	51	1598	1.801	2.878	++	+++	++	+	-	142	1.582	0.225	+++	Lobar pneumonia
BS/6/6	51	2174	0.361	0.785	-	+	-	-	-	564	1.435	0.809	++	Bilateral pyonephrosis.
BS/41/6	52	1868	0.250	0.467	-	+	-	+	-	178	0.143	0.025	-	Meningioma
BS/38/6	52	842	0.487	0.410	++	+++	-	++++	Fine	102	0.441	0.045	++	Liver failure.

Ref. No.	Age	LIVER								SPLEEN				Cause of Death
			Chemical Iron Estimations		Histological Iron Estimations						Chemical Iron Estimations		Hist. Iron	
		Weight (grammes)	Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hepatic cells	Kupffer cells	Portal areas	Portal Fibrosis	Cirrhosis	Weight (grammes)	Iron Concentration (mg/g wet wt)	Total Iron (grammes)		
BS/36/6	52	1006	0.565	0.568	++	+	+	+	-	70	0.600	0.042	++	Inhalation of vomit : drunk
BS/27/6	52	1124	0.229	0.257	+	-	-	+	-	138	0.260	0.036	+	Carcinoma of bronchus
BS/18/6	52	1472	5.733	9.419	++	+++	+++	++++	Coarse	94	9.501	0.443	+++	Lobar pneumonia
BS/13/6	52	1620	10.418	16.877	+++	+++	+++	++++	Coarse	162	8.361	1.354	+++	Carcinoma of liver.
BS/11/6	52	1098	0.441	0.484	+	+	-	-	-	128	2.012	0.258	+++	Carcinoma of oesophagus
BS/76/6	55	2215	0.145	0.321	-	-	-	++++	Coarse	272	0.218	0.059	-	Carcinoma of liver.
BS/75/6	55	2282	6.385	14.566	+++	+++	+++	++++	Fine	242	5.405	1.308	+++	Diabetic coma
BS/72/6	55	1686	1.699	2.865	+++	+++	+++	++	-	89	7.451	0.663	+++	Bronchopneumonia: old brain injury.
BS/67/6	55	1666	0.749	1.248	++	+	++	-	-	110	0.629	0.069	++	Amoebic abscess of liver
BS/64/6	55	1506	0.745	1.122	++	++	+++	-	-	130	0.931	0.121	++	Carcinoma of oesophagus
BS/61/6	55	1118	0.974	1.089	++	-	-	-	-	86	0.996	0.086	++	Carcinoma of oesophagus
BS/60/6	55	1590	0.723	1.150	+	+	-	+	-	70	2.213	0.155	+++	Lobar pneumonia: chronic pyelonephritis
BS/59/6	55	1862	1.818	3.385	++	++	+++	++++	Coarse	460	1.670	0.768	+++	Pulmonary tuberculosis
BS/58/6	55	1042	0.866	0.902	++	++	++	-	-	80	1.397	0.112	+++	Carcinoma of bronchus
BS/54/6	55	2775	4.629	12.845	++	+++	+++	++++	Fine	380	3.467	1.237	+++	Peritonitis: severe Bantu siderosis.
BS/50/6	55	1290	0.088	0.114	-	-	-	-	-	124	0.108	0.013	-	Hypertensive heart failure
BS/48/6	55	1084	0.870	0.943	++	++	-	+	-	60	0.767	0.046	++	Peritonitis: volvulus of sigmoid colon.
BS/46/6	55	1070	2.607	2.789	+++	+++	+++	+	-	100	1.216	0.122	+++	Carcinoma of stomach
BS/44/6	55	3709	0.239	0.349	-	-	-	++++	Coarse	135	0.162	0.022	-	Carcinoma of liver.
BS/43/6	55	1284	2.302	2.956	+++	++	+++	++	-	162	2.561	0.415	+++	Tuberculous meningitis: miliary tuberculosis
BS/40/6	55	1582	3.938	6.230	++	+++	+++	++++	Coarse	126	12.552	1.582	+++	Brain abscess
BS/39/6	55	1250	0.118	0.148	-	-	-	-	-	146	0.129	0.019	-	Road accident
BS/37/6	55	1010	3.005	3.035	+++	+++	+++	++++	Fine	312	2.313	0.722	+++	Haemorrhage: rupture of oesophageal varices
BS/35/6	55	1638	0.049	0.080	-	-	-	++++	Coarse	684	0.050	0.034	-	Carcinoma of liver
BS/34/6	55	1790	2.935	5.250	++	+++	+++	++	-	204	1.333	0.272	+++	Carcinoma of liver
BS/31/6	55	1478	0.359	0.531	+	-	-	+	-	274	0.622	0.170	+	Fractured spine: terminal bronchopneumonia
BS/28/6	55	3182	0.340	1.055	-	+	-	+++	Coarse	252	0.349	0.088	+	Carcinoma of liver

Ref. No.	Age	LIVER								SPLEEN				Cause of Death
		Weight (grammes)	Chemical Iron Estimations		Histological Iron Estimations			Portal Fibrosis	Cirrhosis	Weight (grammes)	Chemical Iron Estimations		Hist. Iron	
			Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hepatic cells	Kupffer cells	Portal areas				Iron Concentration (mg/g wet wt)	Total Iron (grammes)		
BS/25/6	55	1612	0.153	0.247	-	-	-	+	-	182	0.162	0.029	-	Apoplexy - hypertension
BS/23/6	55	1568	0.417	0.654	-	+	-	-	-	468	0.987	0.462	++	Hypertensive cardiac failure: chronic pyelonephritis.
BS/21/6	55	1714	9.205	15.777	+++	+++	+++	++++	Coarse	174	5.825	1.014	+++	Liver failure.
BS/19/6	55	1132	0.682	0.772	+	+++	++	-	-	132	3.616	0.477	+++	Carcinoma of pancreas
BS/17/6	55	1376	0.202	0.278	-	-	-	+	-	114	0.941	0.107	++	Apoplexy: hypertension. Chronic pyelonephritis.
BS/15/6	55	1182	2.830	3.345	++	+++	+++	+	-	106	4.069	0.431	+++	Pulmonary tuberculosis.
BS/12/6	55	1276	0.261	0.333	-	+	-	-	-	100	0.658	0.066	+	Carcinoma of lung
BS/9/6	55	2134	0.181	0.386	-	-	-	-	-	84	0.120	0.010	-	Carcinoma of liver.
BS/7/6	55	1854	7.460	15.830	++	+++	+++	++++	Coarse	304	3.232	0.983	+++	Liver failure.
BS/4/6	55	1874	7.589	14.223	++	+++	+++	++++	Coarse	202	11.017	2.225	+++	Suicidal hanging.
BS/8/6	56	1792	4.070	7.293	++	+++	+++	-	-	156	4.390	0.685	+++	Lober pneumonia.
BS/5/6	56	3488	0.245	0.872	-	-	-	++++	Coarse	364	0.130	0.047	-	Carcinoma of liver.
BS/26/6	58	1230	0.862	1.060	++	+	++	++	-	214	2.196	0.470	+++	Renal failure: chronic pyelonephritis.
BS/24/6	58	1434	2.345	3.363	++	+++	+++	++++	Coarse	428	4.392	1.880	+++	Carcinoma of lung.
BS/22/6	58	1376	1.124	1.547	++	++	+	-	-	118	1.126	0.133	+++	Suicidal hanging.
BS/1/6	58	1850	4.341	8.031	++	+++	+++	++++	Coarse	196	4.012	0.786	+++	Status asthmaticus.

AFRICAN FEMALES

SIXTH DECADE

Total Cases 31

Ref. No.	Age	LIVER							SPLEEN				Cause of Death
		Weight (grammes)	Chemical Iron Estimations		Histological Iron Estimations			Portal Fibrosis	Cirrhosis	Weight (grammes)	Chemical Iron Estimations		Hist. Iron	
			Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hepatic cells	Kupffer cells	Portal areas				Iron Concentration (mg/g wet wt)	Total Iron (grammes)		
BS/102/6	50	2654	0.186	0.494	-	-	-	-	-	556	0.199	0.110	-	Carcinoma of kidney
BS/100/6	50	1360	2.643	3.594	++	++	++	-	-	158	1.163	0.184	++	Bacillary dysentery
BS/97/6	50	1140	4.112	4.688	+++	+++	+++	++	-	75	1.448	0.109	+++	Thrombosis L. middle cerebral artery.
BS/96/6	50	2184	1.525	3.331	++	+++	+++	++	-	294	1.103	0.324	++	Carcinoma of head of pancreas: suppurative cholangitis.
BS/95/6	50	1134	1.073	1.217	++	+++	++	+	-	80	4.020	0.322	+++	Renal tubular necrosis.
BS/92/6	50	920	0.437	0.402	+	+	-	-	-	68	0.521	0.035	++	Road accident
BS/91/6	50	994	1.744	1.734	++	+++	+	-	-	102	1.640	0.167	+++	Severe burns: chronic pyelo-nephritis.
BS/89/6	50	1176	0.280	0.329	+	-	-	+++	Bilharzial Fibrosis Coarse	293	0.372	0.109	+	Lobar pneumonia
BS/88/6	50	1342	1.365	1.832	+++	++	++	++++		332	0.710	0.236	++	Acute peritonitis: pericarditis
BS/87/6	50	1182	2.276	2.690	+++	++	++	+	-	80	0.764	0.061	++	Lobar pneumonia.
BS/86/6	50	1880	0.490	0.921	++	++	+	+	-	156	0.278	0.043	+	Hypertensive heart failure.
BS/83/6	50	632	0.342	0.216	+	+	+	++	-	100	0.870	0.087	++	Tuberculosis of lumbar spine: paraplegia.
BS/82/6	50	1370	0.126	0.176	-	-	-	-	-	125	0.165	0.021	-	Bronchopneumonia
BS/55/6	50	1512	0.094	0.142	-	-	-	-	-	260	0.172	0.045	-	Carcinoma of cervix.
BS/63/6	50	1566	0.242	0.379	-	-	-	-	-	116	0.318	0.037	+	Subarachnoid (spontaneous) haemorrhage.
BS/69/6	50	1108	2.590	2.870	+++	+++	+++	+	-	86	0.798	0.068	++	Status asthmaticus.
BS/71/6	50	1540	0.204	0.314	-	-	-	-	-	230	0.179	0.041	-	Pulmonary tuberculosis.
BS/101/6	51	1020	0.131	0.134	-	-	-	-	-	85	0.132	0.011	-	Brain damage: murder.
BS/3/6	52	1538	1.675	2.576	++	++	+	+	-	186	2.528	0.470	+++	Carcinoma of thyroid.
BS/32/6	52	1364	0.168	0.229	-	-	-	-	-	114	0.349	0.040	+	Renal failure: chronic pyelo-nephritis.
BS/93/6	54	2030	0.376	0.762	+	-	-	-	-	386	0.240	0.093	+	Road accident
BS/99/6	55	1258	0.267	0.336	+	-	-	+++	Bilharzial Fibrosis	184	0.502	0.092	+	Chronic dislocation of jaw: malnutrition: bilharzial fibrosis of liver.
BS/98/6	55	1138	0.198	0.225	-	-	-	-	-	100	0.178	0.018	-	Suicidal hanging
BS/94/6	55	1237	0.347	0.429	-	++	-	+	-	140	0.284	0.040	+	Carcinoma of lung.
BS/90/6	55	1324	3.118	4.128	+++	++	++	+	-	86	3.014	0.259	+++	Strangulated umbilical hernia: peritonitis.
BS/85/6	55	1314	1.373	1.804	++	+	+	-	-	119	0.402	0.048	+	Carcinoma of lung.

Ref. No.	Age	LIVER								SPLEEN				Cause of Death
			Chemical Iron Estimations		Histological Iron Estimations						Chemical Iron Estimations		Hist. Iron	
		Weight (grammes)	Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hepatic cells	Kupffer cells	Portal areas	Portal Fibrosis	Cirrhosis	Weight (grammes)	Iron Concentration (mg/g wet wt)	Total Iron (grammes)		
BS/84/6	55	1313	1.533	2.018	+++	++	++	++	-	131	1.389	0.182	+++	Acute arsenical poisoning
BS/10/6	55	1294	0.092	0.119	-	-	-	-	-	126	0.056	0.007	-	Tuberculosis of brain
BS/20/6	55	1486	2.150	3.195	++	+++	+++	-	-	102	3.567	0.364	+++	Drowning
BS/49/6	55	2184	1.346	2.940	++	+++	+++	++++	Coarse	286	2.091	0.598	+++	Carcinoma of liver
BS/2/6	58	2724	5.598	15.248	++	+++	+++	++++	Fine	444	0.935	0.415	+++	Acute peritonitis.

AFRICAN MALES

SEVENTH AND EIGHTH DECADE

Total Cases 40

Ref. No.	Age	LIVER								SPLEEN				Cause of Death
		Weight (grammes)	Chemical Iron Estimations		Histological Iron Estimations			Portal Fibrosis	Cirrhosis	Weight (grammes)	Chemical Iron Estimations		Hist. Iron	
			Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hepatic cells	Kupffer cells	Portal areas				Iron Concentration (mg/g wet wt)	Total Iron (grammes)		
BS/60/7	60	1477	2.631	3.890	++	+++	+++	++++	Coarse	255	7.634	2.021	+++	Lobar pneumonia: chronic pyelonephritis.
BS/55/7	60	1266	0.104	0.132	-	-	-	-	-	54	0.120	0.006	-	Hypertensive heart failure.
BS/49/7	60	1692	0.441	0.746	+	+	-	-	-	98	0.658	0.064	++	Carcinoma of oesophagus.
BS/48/7	60	1192	0.135	0.161	-	-	-	++++	Coarse	622	0.210	0.130	-	Liver failure
BS/44/7	60	1328	0.249	0.331	+	-	-	-	-	274	0.162	0.044	-	Road accident.
BS/43/7	60	1942	2.988	5.800	+++	++	++	+	-	280	0.881	0.247	++	Acute peritonitis.
BS/42/7	60	1136	8.776	9.970	+++	+++	+++	++	-	106	11.243	1.192	+++	Cor pulmonale: chronic vesicular emphysema.
BS/36/7	60	1323	8.122	10.745	+++	+++	+++	++++	Coarse	122	6.941	0.847	+++	Carcinoma of lung.
BS/26/7	60	1760	2.166	3.812	++	+++	+++	+	-	174	2.835	0.493	+++	Tuberculous meningitis.
BS/23/7	60	1420	0.704	1.000	+	++	+	++	-	318	1.126	0.358	++	Haemorrhage from nose following trauma.
BS/24/7	62	1706	3.353	5.710	+++	+++	+++	++	-	306	1.180	0.361	++	Viral encephalitis
BS/3/7	62	1446	0.157	0.227	-	-	-	+	-	154	0.245	0.038	+	Carcinoma of lung.
BS/4/7	64	1700	3.577	6.071	++	+++	+++	++	-	128	2.525	0.323	+++	Carcinoma of oesophagus
BS/59/7	65	1198	0.812	0.972	++	++	-	+	-	100	0.692	0.069	++	Renal failure: prostatic hyperplasia: hydronephrosis.
BS/56/7	65	2266	7.803	17.682	++	+++	+++	++++	Coarse	440	6.988	3.075	+++	Lobar pneumonia
BS/51/7	65	1340	0.267	0.358	+	-	-	+	-	72	1.044	0.075	++	Bronchopneumonia: pellagra
BS/47/7	65	1716	0.338	0.577	-	+	-	-	-	140	0.968	0.135	++	Lobar pneumonia: pellagra
BS/46/7	65	1238	0.284	0.351	+	+	-	-	-	68	0.633	0.043	+	Renal failure: hydronephrosis: prostatic hyperplasia.
BS/41/7	65	1357	0.237	0.322	-	-	-	+	-	100	0.440	0.044	+	Lobar pneumonia
BS/37/7	65	1348	0.256	0.345	-	-	-	-	-	160	0.286	0.046	+	Acute pulmonary oedema following prostatectomy.
BS/32/7	65	1184	6.018	7.125	+++	+++	+++	++++	Coarse	280	1.949	0.546	+++	Cerebral thrombosis.
BS/29/7	65	1450	1.869	2.710	++	+++	++	+	-	192	4.132	0.793	+++	Suicidal hanging
BS/18/7	65	1012	2.540	2.570	++	++	+++	++	-	92	3.509	0.323	+++	Chronic pyelonephritis: hypertensive heart failure.
BS/27/7	66	2222	1.948	4.328	+++	++	+++	-	-	242	3.747	0.907	+++	Renal failure: hydronephrosis: prostatic hyperplasia.
BS/19/7	68	902	0.463	0.418	+	+	-	++++	Coarse	308	1.197	0.369	++	Pulmonary tuberculosis
BS/8/7	68	1226	1.635	2.005	++	+++	+++	+	-	184	2.357	0.434	++	Pulmonary tuberculosis.
BS/58/7	70	1378	8.360	0.030	++	+++	+++	++++	Fine	342	17.865	6.100	+++	Liver failure.

Ref. No.	Age	LIVER							SPLEEN				Cause of Death
		Weight (grammes)	Chemical Iron Estimations		Histological Iron Estimations			Portal Fibrosis	Cirrhosis	Weight (grammes)	Chemical Iron Estimations		Hist. Iron	
			Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hepatic cells	Kupffer cells	Portal areas				Iron Concentration (mg/g wet wt)	Total Iron (grammes)		
BS/57/7	70	951	1.456	1.385	+++	++	++	-	-	170	2.248	0.382	+++	Subacute peritonitis with numerous abscesses.
BS/50/7	70	1081	1.803	1.949	+++	++	+++	-	-	68	2.541	0.173	+++	Lobar pneumonia: pellagra.
BS/34/7	70	1610	2.701	4.149	+++	+++	+++	-	-	165	2.441	0.403	+++	Septicaemia.
BS/30/7	70	1140	2.619	2.986	++	+++	+++	++	-	134	3.558	0.477	+++	Carcinoma of lung: pellagra
BS/17/7	70	665	0.107	0.071	-	-	-	+	-	102	0.395	0.040	+	Volvulus of pelvic colon.
BS/7/7	70	1204	0.232	0.279	-	-	-	+	-	202	0.116	0.023	-	Apoplexy: essential hypertension.
BS/22/7	71	1494	3.823	5.712	+++	++	++	-	-	94	2.226	0.209	+++	Renal failure: chronic pyelonephritis.
BS/16/7	71	1352	1.088	1.471	++	++	+++	+	-	152	1.623	0.247	+++	Pulmonary tuberculosis.
BS/5/7	71	1022	0.368	0.376	+	-	-	+	-	56	0.812	0.045	++	Extensive burns
BS/33/7	72	804	0.061	0.049	-	-	-	+	-	112	0.106	0.012	-	Volvulus of sigmoid colon.
BS/25/7	75	872	0.498	0.474	+	-	-	+	-	44	0.421	0.019	+	Suicidal hanging.
BS/13/7	75	880	3.305	2.908	++	+++	+++	++	-	156	5.660	0.883	+++	Pulmonary tuberculosis.
BS/53/7	80	710	14.130	10.020	+++	+++	+++	+	-	104	15.651	1.628	+++	Acute bronchopneumonia.

AFRICAN FEMALES

SEVENTH AND EIGHTH DECADE

Total Cases 31

Ref. No.	Age	LIVER								SPLEEN				Cause of Death
			Chemical Iron Estimations		Histological Iron Estimations						Chemical Iron Estimations		Hist. Iron	
		Weight (grammes)	Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hepatic cells	Kupffer cells	Portal areas	Portal Fibrosis	Cirrhosis	Weight (grammes)	Iron Concentration (mg/g wet wt)	Total Iron (grammes)		
BS/71/7	60	1290	3.296	4.250	+++	+++	++	+	-	70	2.211	0.155	+++	Apoplexy: essential hypertension.
BS/70/7	60	1516	0.184	0.279	-	-	-	-	-	190	0.265	0.050	+	Haemorrhage into site of operation: removal of meningioma.
BS/69/7	60	710	1.568	1.111	++	+	++	+	-	46	2.412	0.111	+++	Carcinoma of rectum; peritonitis
BS/68/7	60	1690	1.351	2.285	++	++	++	++	-	298	0.667	0.199	++	Congestive heart failure: mitral stenosis.
BS/67/7	60	1634	2.517	4.102	+++	+++	++	++	-	124	2.096	0.260	+++	Lobar pneumonia.
BS/64/7	60	1555	10.484	16.280	+++	+++	+++	+++	-	150	12.430	1.860	+++	Mitral incompetence.
BS/35/7	60	1654	0.579	0.958	+	+++	+	-	-	647	0.467	0.320	++	Pulmonary and miliary tuberculosis.
BS/52/7	60	1564	0.409	0.640	+	-	-	+	-	183	0.823	0.151	+	Anaesthetic death.
BS/54/7	60	766	1.400	1.071	+++	++	+++	-	-	65	1.910	0.124	+++	Hypertensive cardiac failure.
BS/45/7	60+	654	3.710	2.425	+++	++	+++	+	-	70	5.998	0.419	+++	Severe malnutrition and anaemia
BS/20/7	61	1884	1.209	2.278	++	++	+	-	-	194	0.715	0.139	++	Inhalation of vomit: drunk
BS/11/7	62	1166	1.471	1.715	++	++	++	+	-	102	1.299	0.132	++	Bacillary dysentery.
BS/12/7	62	1962	0.228	0.447	-	-	-	+	-	174	0.270	0.047	+	Carcinoma of liver.
BS/21/7	62	958	4.468	4.280	+++	+++	+++	++	-	100	8.903	0.890	+++	Status asthmaticus.
BS/31/7	62	1398	0.805	1.125	+	++	-	-	-	116	0.033	0.005	-	Chronic pyelonephritis.
BS/66/7	65	1165	0.233	0.271	+	-	-	++++	Coarse	310	0.115	0.036	-	Haemorrhage from oesophageal varices: liver carcinoma.
BS/63/7	65	1335	0.226	0.302	-	-	-	-	-	56	0.261	0.015	+	Lobar pneumonia.
BS/1/7	65	962	0.395	0.380	+	-	-	++	-	122	0.304	0.037	+	Suppurative meningitis: lobar pneumonia.
BS/2/7	65	948	0.228	0.216	-	-	-	-	-	82	0.207	0.017	-	Carcinoma of pancreas.
BS/6/7	65	1896	4.472	8.116	+++	+++	+++	++++	Coarse	200	3.642	0.728	+++	Lobar pneumonia.
BS/10/7	65	1602	5.749	11.230	+++	+++	+++	+	-	100	3.304	0.330	+++	Inhalation of vomit: drunk
BS/40/7	65	864	2.590	2.283	+++	++	+	-	-	36	1.932	0.070	+++	Lobar pneumonia.
BS/61/7	65	1152	0.266	0.306	-	-	-	+	-	83	0.283	0.023	-	Amoebic dysentery and amoebic abscess of liver
BS/15/7	68	474	1.274	0.604	++	++	++	++++	Fine	78	1.271	0.099	+++	Malnutrition: bronchopneumonia
BS/65/7	70	1310	0.825	1.081	++	-	-	+	-	308	0.726	0.224	++	Tuberculous bronchopneumonia
BS/9/7	70	1252	0.251	0.314	+	-	-	-	-	100	0.099	0.010	-	Drowning

Ref. No.	Age	LIVER								SPLEEN				Cause of Death
		Weight (grammes)	Chemical Iron Estimations		Histological Iron Estimations			Portal Fibrosis	Cirrhosis	Weight (grammes)	Chemical Iron Estimations		Hist. Iron	
			Iron Concentration (mg/g wet wt)	Total Iron (grammes)	Hepatic cells	Kupffer cells	Portal areas				Iron Concentration (mg/g wet wt)	Total Iron (grammes)		
BS/38/7	70	1638	4.737	7.759	+++	+++	+++	+	-	86	20.719	1.782	+++	Road Accident
BS/62/7	70	857	0.082	0.070	-	-	-	-	-	140	0.119	0.017	-	Diabetic coma
BS/14/7	80	720	1.031	0.742	++	++	++	+	-	68	3.022	0.205	+++	Acute bronchopneumonia
BS/28/7	80	936	0.336	0.314	+	-	-	-	-	142	0.281	0.040	+	Endomyocardial fibrosis.
BS/39/7	80	1902	0.799	1.520	+	++	++	-	-	66	1.349	0.089	+++	Septicaemia.

APPENDIX IV

AFRICAN SUBJECTS FOUND TO HAVE ACUTE PERITONITIS AT AUTOPSY YET IN WHOM NO CAUSE OF THE PERITONITIS COULD BE DEMONSTRATED

Reference No.	Sex	Age	Portal Fibrosis	Cirrhosis (Type)	Histological Liver Iron			Cause of Death
					H.C	K.C	P.A	
H/PM/13/63	M	60	++++	Coarse	+++	+++	+++	Peritonitis and liver failure.
PM/28/63	M	40	++++	Fine	+++	+++	+++	Peritonitis and liver failure.
PM/127/63	M	40	+++	Bilharzial Fibrosis	++	+++	+++	Peritonitis.
PM/139/63	F	60	++++	Fine	++	+++	++	Acute peritonitis.
PM/157/63	M	35	++++	Fine	++	+++	+++	Acute peritonitis
H/PM/349/63	F	50	++++	Coarse	+++	+++	+++	Acute peritonitis and carcinoma of ovary.
H/PM/658/63	M	35	++++	Fine	++	++	+++	Acute peritonitis
P. 571/64	F	58	++++	Coarse	++	+++	+++	Acute peritonitis
PM/42/64	M	55	++++	Fine	+++	+++	+++	Acute peritonitis and diabetes.
PM/108/64	F	50	++	-	+++	+++	+++	Acute peritonitis
P.109/64	M	50	++++	Fine	+++	+++	+++	Haemorrhage from oesophageal varices & peritonitis.
P.285/64	F	70	++	-	+++	+++	+++	Peritonitis/chronic pyelonephritis.
97/65	M	55	++++	Coarse	++	+++	+++	Acute peritonitis.
405/65	F	50	++++	Coarse	+++	++	++	Acute peritonitis. Acute pericarditis.
207/66	M	45	++	-	+++	+++	+++	Acute peritonitis.
363/66	M	42	++++	Coarse	-	-	-	Acute peritonitis.
189/65	M	60	+	-	+++	++	++	Acute peritonitis
13/67	M	45	++++	Fine	+++	+++	+++	Acute peritonitis. Pulmonary tuberculosis.
205/67	M	45	+	-	++	+	+	Acute peritonitis.
401/67	M	50	++	-	+++	+++	+++	Acute peritonitis.

APPENDIX V

DISTRIBUTION OF IRON IN VARIOUS BODY ORGANS IN SUBJECTS WITH VARYING DEGREES OF IRON OVERLOAD

Reference No.	Age	Liver Weight (grammes)	Spleen Weight (grammes)	Chemically Estimated Iron: Liver Concentration (mg/g wet wt)	Chemically Estimated Iron: Spleen Concentration (mg/g wet wt)	Chemically Estimated Iron: Liver Total Iron (grammes)	Chemically Estimated Iron: Spleen Total Iron (grammes)	Histologically Estimated Iron: Level: Cirrhosis	Level: Portal Fibrosis	Level: Hepatic cells	Level: Kupffer cells	Level: Portal areas	Other Organs: Spleen	Pancreas	Heart	Pituitary	Thyroid	Adrenal	Prostate	Testis	Kidney	Sub-Mand Salivary Gland	Brain	Choroid Plexus	Sternal Marrow	Stomach	Small Bowel	Colon	Lymph Node #	Cause of Death
D. 41*	55	1579	382	28.129	36.642	37.900	12.900	Fine	++++	+++	+++	+++	+++	+++	+++	+++	+++	+++	++	++	+++	+++	+	+++	+++	+++	+++	++	+++	Rupture of oesophageal varices
D. 39	50	2335	232	15.374	32.708	32.685	2.821	Fine	++++	+++	+++	+++	+++	+++	+	++	+++	++	+	+++	++	++	-	+++	+++	++	+++	+	+++	Liver failure.
D. 2	60	1255	145	13.390	5.670	16.804	0.709	-	++	+++	+++	+++	+++	+++	++	++	+++	++	++	-	++	++	-	+++	+++	++	+	-	+++	Cardiac failure. Amoebic dysentery.
D. 26	60	1699	80	12.046	16.070	12.860	1.282	-	+++	+++	+++	+++	+++	+	-	-	-	-	-	-	-	-	-	+	+++	+	+++	++	+++	Carcinoma of oesophagus.
D. 8 (BS/14/5)	49	1864	146	11.098	5.127	12.178	0.545	Fine	++++	+++	+++	+++	+++	++	++	+++	+++	+++	+	+++	++	++	-	+++	+++	+	+	-	+++	Road accident: injuries relatively slight ? cerebral shock
D. 29	55	1483	346	11.669	5.281	17.293	1.616	-	+++	+++	+++	+++	+++	+	-	-	+	-	-	-	+	-	-	-	+++	+	+++	++	+++	Bronchopneumonia
D. 6 (BS/32/5)	45	1325	482	13.095	2.924	14.701	1.292	Fine	++++	+++	+++	+++	+++	+++	++	++	+++	+++	+	+	++	+	-	+++	+++	+	+++	+	+++	Tuberculous peritonitis.
D. 30	65	1254	180	10.986	15.233	14.655	2.742	Coarse	+++	+++	+++	+++	+++	++	+	-	++	+	-	-	++	+	+	+	+++	+	-	-	+++	Pneumonia, bronchiectasis, emphysema
D 12 (BS/12/6)	52	1620	162	10.418	8.361	16.877	1.354	Coarse	++++	+++	+++	+++	+++	+++	+	+	+	+	-	-	+	+	-	+	+++	++	++	-	+++	Carcinoma of liver
D 14 (BS/31/6)	55	1714	174	9.805	5.825	16.777	1.014	Coarse	++++	+++	+++	+++	+++	++	-	+	++	++	-	-	-	+	-	++	+++	++	+	-	+++	Liver failure
D. 33	60	3106	798	8.134	3.678	25.915	3.075	-	++	+++	+++	+++	+++	++	+	+	++	+	-	-	+	-	+	+++	+++	++	++	++	+++	Miliary tuberculosis
D. 35	50	1932	221	7.890	5.525	15.243	1.503	Fine	++++	+++	+++	+++	+++	++	+	++	++	++	+	+	++	+	+	+++	+++	++	+	-	+++	Liver failure
D 10 (BS/21/5)	48	1024	138	7.866	11.409	8.055	2.031	-	++	++	+++	+++	+++	+	-	-	-	+	-	-	+	-	-	+	+++	+	+++	+	+++	Pulmonary tuberculosis
D 16 (BS/56/7)	65	2366	440	7.808	6.980	17.682	3.075	Coarse	+++	++	+++	+++	+++	++	-	+	++	+	-	-	+	-	-	+	+++	+	+++	++	+++	Lobar pneumonia
D. 7 (BS/7/6)	55	1854	264	7.460	3.232	15.830	0.982	Fine	++++	++	+++	+++	+++	+++	++	++	+++	++	+	-	++	++	-	++	++	++	++	-	+++	Hepatic failure.
D. 25	50	1380	154	6.737	1.476	9.297	0.331	Coarse	+++	+++	+++	+++	+++	++	-	-	-	-	-	-	-	-	-	+	+++	+	+	+	+++	Lymphosarcoma
D 21 (BS/75/6)	55	2382	241	6.383	5.405	14.566	1.300	Fine	++++	+++	+++	+++	+++	++	++	++	+++	++	++	++	++	++	-	+++	+++	++	+	-	+++	Diabetic coma
D. 28	50	2474	324	6.080	3.522	15.042	1.141	-	++	+++	+++	+++	+++	++	+	+	+	+	-	-	+	-	-	+	++	++	+++	-	+++	Renal failure. Papillary necrosis. Pyonic kidney.
D. 24	50	1938	404	5.830	8.646	10.814	4.185	-	+++	++	++	+++	+++	+++	+	-	-	-	-	-	-	-	-	+	+++	+	+	+	+++	Carcinoma of lung
D. 34	60	1779	965	4.938	1.060	0.738	0.820	-	++	+++	+++	+++	+++	-	-	-	-	-	-	-	-	-	-	+	+++	+	+++	+	+++	Miliary tuberculosis
D. 1	60	1450	190	4.760	5.220	6.900	0.993	-	+	++	+++	+++	+++	+	-	-	-	-	-	-	-	-	-	+	+++	-	+++	+	+++	Bronchiectasis and lung abscess
D. 3	65	1374	135	4.703	7.050	6.470	0.001	-	+	++	+++	+++	+++	+	-	-	-	-	-	-	-	-	-	+	+++	+	+++	-		Lobar pneumonia
D. 40	60	1020	131	4.460	1.351	4.600	0.204	-	-	+++	+++	+++	+++	-	-	-	-	-	-	-	-	-	-	+	+	-	+	-	++	Starvation
D. 20(BS/70/6)	50	1822	270	3.278	0.915	5.317	0.339	Fine	++++	+++	+++	+++	++	++	+	+	++	++	+	+	+	++	-	++	++	++	+	+	+++	Apoplexy. Hypertension
D 15 (BS/37/6)	55	1010	122	3.005	2.313	3.035	0.282	Fine	++++	+++	+++	+++	+++	++	++	++	++	+	-	+	+	-	++	+++	++	++	+	+	+++	Ruptured oesophageal varices
D 17 (BS/60/7)	60	1477	255	2.651	7.634	3.886	1.947	Coarse	++++	++	+++	+++	+++	++	-	+	+	+	-	+	-	-	-	+	+++	+	+++	-	+++	Lobar pneumonia
D 18 (BS/33/6)	50	1270	150	2.143	2.006	2.722	0.301	-	++	++	+++	+++	+++	+	-	-	+	-	-	-	-	-	-	-	+++	++	+++	+	++	Miliary tuberculosis
D. 22	50	1168	134	1.814	0.754	2.119	0.169	Fine	++++	++	+++	+++	++	++	+	++	++	+	-	+	++	++	-	+++	++	++	+	+	++	Haemorrhage from oesophageal varices.
D 13 (BS/25/5)	40	1526	150	1.667	2.811	2.190	0.422	Coarse	+++	++	+++	+	+++	-	-	-	-	-	-	-	-	-	-	-	++	-	+	-		Traumatic brain injury.
D. 4 (BS/1/4)	37	1774	582	1.080	0.355	1.863	0.182	Coarse	++++	++	+++	++	+	-	-	-	-	-	-	-	-	-	-	-	+	-	+	-	++	Road accident
D 11 (BS/28/5)	45	3060	338	0.708	0.174	1.458	0.090	Coarse	++++	++	+++	++	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	++	Haemorrhage - oesophageal varices and liver cell carcinoma

* D.41 Pancreas storage iron 5.43 mg/g

Parts hepatic

Reference No.	Age	Liver Weight (grammes)	Spleen Weight (grammes)	Chemically Estimated Iron				HISTOLOGICALLY ESTIMATED IRON																			Cause of Death
								LIVER					OTHER ORGANS															
				Liver Concentration (mg/g wet wt)	Spleen Concentration (mg/g wet wt)	Liver Total Iron (grammes)	Spleen Total Iron (grammes)	Cirrhosis	Portal Fibrosis	Hepatic cells	Kupffer cells	Portal areas	Spleen	Pancreas	Heart	Fat	Salivary	Thyroid	Adrenal	Ileum	Ovary	Kidney	Submaxillary Gland	Brain	Choroid Plexus	Sternal Marrow	Stomach	Small Bowel	Colon	Lymph Nodes #	
D. 42*	70	1047	301	10.325	1.954	11.010	0.588	Fine	++++	+++	+++	+++	+++	+++	+		+	+++	++	-	+	++	++	-	++	++	++	+	-	+++	Salmiac of sigmoid colon
D. 35 (H/64/T)	60	1555	150	10.484	12.459	16.302	1.860	-	+++	+++	+++	+++	+++	++	-		+	+	+	-	-	+	-	-	+	+	+	+++	+	+++	Atypical leucocytosis - Cardiomyopathy
D. 5 (H/2/6)	38	2724	444	5.398	0,935	15.248	0.415	Fine	++++	++	+++	+++	++	+++	++		+++	+++	+++	-	+	+++	++	-	+++	+++	+++	+	-	+++	Acute Peritonitis
D. 9 (H/6/T)	65	1896	290	4.472	2,642	8.479	0.722	Coarse	++++	+++	+++	+++	+++	++	-		+	+	+	-	-	+	+	-	+	++	+	++	-	+++	Lobar pneumonia
D. 23 (H/65/4)	38	1276	368	5.368	1.323	8,200	0.772	-	++	+++	+++	+++	++	+	-		+	-	-		+	+	-	-	-	+++	+	+++	-	+++	Purulent meningitis
D. 28 (H/67/4)	58	1102	80	3.276	0.764	3,600	0,061	-	+	+++	+++	++	++	++	-		-	-	-	-	-	-	-	+	-	++	-	+	-	+++	Lobar pneumonia + drunk
D. 21 (H/41/1)	20	994	162	1.746	1.569	1.734	0,167	-	+	+++	+++	++	++	++	-		-	-	-	-	-	-	-	+	-	++	-	++	+	++	Severe burns + chronic pyelonephritis
D. 19 (H/8/6)	35	2164	206	1.346	2,891	2.940	0,398	Coarse	++++	++	+++	+++	+++	+	-		-	-	-		+	-	-	-	+	++	+	+++	+	+++	Carcinoma of liver.
D. 17 (H/41/4)	35	1230	115	1.144	3,399	1.407	0.276	Fine	++++	++	++	+++	+++	++	-		-	+	-	+	+	+	-	+	+	++	++	+	-	+++	Liver failure.
D. 37 (H/65/T)	70	1210	305	8,025	0.724	1,401	0,214	-	+	++	-	-	++	-	-		-	-	-	-	-	-	-	-	-	-	+	++	-	-	Miliary tuberculosis - Malnutrition
D. 31 (H/30/5)	40	1463	184	0,644	1.022	0,742	0,190	Fine	++++	++	++	++	+++	+++	-		+	++	+			+	-	-	++	+	+	+	-	++	Lobar pneumonia

* D. 42 Pancreas storage iron 3.006 mg/g

\# Porta hepatis

APPENDIX VI

IRON CONTENT OF VARIOUS AFRICAN FOODS, HOME BREWED AFRICAN BEER AND MUNICIPAL BREWED BEER

S A D Z A

VILLAGE			HOSPITAL STAFF HOMES			HOSPITAL KITCHEN		
Sample Number	% Solid Material	Iron Concentration mg/100g dry weight	Sample Number	% Solid Material	Iron Concentration mg/100g dry weight	Sample Number	% Solid Material	Iron Concentration mg/100g dry weight
1	26.8	8.9	12	27.5	3.4	16	31.1	4.7
2	23.7	10.3	13	25.6	7.1	17	29.6	5.2
3	24.4	12.2	14	23.8	5.9	18	30.3	5.3
4	30.0	5.3	15	27.4	5.1			
5	26.4	6.9						
6	23.2	4.9						
7	21.9	5.8						
8	32.2	4.5						
9	25.6	8.3						
10	27.8	8.0						
11	22.8	8.7						
S.D.	± 3.1	± 2.5	S.D.	± 1.7	± 1.6	S.D.	± 0.8	± 0.5
Average	25.9	7.6	Average	26.1	5.4	Average	30.3	5.1
Range	21.9-32.2	4.9 - 12.2	Range	23.8-27.5	5.1 - 7.1	Range	29.6-30.3	4.7 - 5.3

RELISH

VILLAGE						HOSPITAL STAFF HOMES					
GREEN VEGETABLES			BEANS			GREEN VEGETABLES			BEANS		
Sample Number	% Solid Material	Iron Concentration mg/100g dry weight	Sample Number	% Solid Material	Iron Concentration mg/100g dry weight	Sample Number	% Solid Material	Iron Concentration mg/100g dry weight	Sample Number	% Solid Material	Iron Concentration mg/100g dry weight
1	19.1	12.3	12	33.7	8.5	17	15.6	9.3	19	33.1	20.6
2	21.0	68.4	13	34.8	11.7	18	22.0	27.6	20	32.0	9.8
3	22.7	9.6	14	32.9	19.2						
4	12.2	14.0	15	35.1	11.3						
5	17.1	51.4	16	32.1	12.1						
6	15.3	12.9									
7	25.4	6.1									
8	12.6	29.5									
9	19.2	16.2									
10	15.1	18.9									
11	23.3	33.7									
S.D.	± 4.6	± 19.6	S.D.	± 1.8	± 3.8						
Average	18.4	24.8	Average	33.7	12.6	Average	18.8	17.45	Average	32.5	15.2
Range	12.2-25.4	6.1 - 68.4	Range	32.1-35.1	8.5 - 19.2	Range	15.6-22.0	9.3 - 27.6	Range	32.0-33.1	9.8 - 20.6

HOME BREWED BEER

Sample Number	Iron Concentration mg./100 ml. beer	p H	Sample Number	Iron Concentration mg./100 ml. beer	p H
1	21.8	3.8	31	12.6	4.0
2	15.5	3.8	32	5.7	4.0
3	14.1	3.7	33	5.0	3.9
4	11.4	3.9	34	9.1	4.2
5	1.8	3.9	35	9.3	3.8
6	7.0	3.6	36	9.2	4.1
7	3.0	4.0	37	9.1	4.0
8	17.6	3.7	38	14.4	3.9
9	3.1	3.9	39	35.2	3.8
10	11.4	4.2	40	15.7	3.9
11	13.9	3.8	41	12.8	3.9
12	11.7	3.9	42	4.4	3.8
13	0.5	3.3	43	1.3	4.0
14	6.9	3.6	44	9.1	3.7
15	8.2	3.7	45	16.8	3.9
16	0.7	3.3	46	1.9	3.9
17	6.3	4.1	47	6.5	3.7
18	7.1	3.6	48	4.1	3.9
19	10.6	4.2	49	3.2	4.0
20	3.0	3.8	50	3.7	4.0
21	1.7	4.1	51	16.1	3.6
22	5.8	3.9	52	12.0	3.8
23	2.8	4.0	53	12.1	3.9
24	7.8	4.0	54	10.7	4.0
25	9.4	4.0	55	9.3	3.9

Sample Number	Iron Concentration mg./100 ml. beer	p H	Sample Number	Iron Concentration mg./100 ml. beer	p H
26	8.8	3.9	56	4.1	4.0
27	4.3	4.0	57	15.5	3.7
28	1.2	3.8	58	25.1	3.6
29	4.2	3.8	59	16.2	3.9
30	2.7	3.9	60	32.4	3.9

Average pH	3.9	Average Iron Concentration	9.4 mg/100 ml.
Standard Deviation	0.2	Standard Deviation	7.1 mg/100 ml.
Range	3.3 - 4.2	Range	0.5 - 35.2 mg/100 ml.

MUNICIPAL BREWED BEER

Sample Number	Iron Concentration mg./100 ml. beer	p H
1	0.32	3.1
2	0.35	3.0
3	0.34	3.2
4	0.33	3.1
5	0.33	3.0
Average	0.33	3.1
Range	0.32 - 0.35	3.0-3.2

APPENDIX VII

THEORETICAL AMOUNTS OF IRON ADDED TO LIVER IN EACH DECADE DERIVED FROM BEER (TABLE XXI)

With amounts of beer containing less than 25 mg. iron the absorption rate is considered to be 4% and with amounts of beer containing 25 mg. or more the absorption rate is considered to be 2% (Bothwell et al., 1964). The liver is said to contain about one third of the total body storage iron (Bothwell & Finch, 1962) so it is assumed that one third of the absorbed iron is stored in liver.

MALES

Age Group	Iron Added to Liver
15 - 25	$\frac{17.7 \times 4 \times 365 \times 10}{100 \times 3}$ mg. = 0.861 G
25 - 35	$\frac{27.4 \times 2 \times 365 \times 10}{100 \times 3}$ mg. = 0.667 G
35 - 45	$\frac{46.3 \times 2 \times 365 \times 10}{100 \times 3}$ mg. = 1.127 G
45 - 55	$\frac{71.0 \times 2 \times 365 \times 10}{100 \times 3}$ mg. = 1.728 G
55 - 65	$\frac{62.8 \times 2 \times 365 \times 10}{100 \times 3}$ mg. = 1.528 G

FEMALES

Age Group	Iron Added to Liver
15 - 25	0
25 - 35	$\frac{2.8 \times 4 \times 365 \times 10}{100 \times 3}$ mg. = 0.136 G
35 - 45	$\frac{10.5 \times 4 \times 365 \times 10}{100 \times 3}$ mg. = 0.511 G
45 - 55	$\frac{18.1 \times 4 \times 365 \times 10}{100 \times 3}$ mg. = 0.882 G
55 - 65	$\frac{36.3 \times 2 \times 365 \times 10}{100 \times 3}$ mg. = 0.883 G

APPENDIX VIII

SERUM IRON ESTIMATIONS

MALES - SECOND DECADE

Ref. No.	Age	Serum Iron µg/100 ml.	U.I.B.C. µg/100 ml.	T.I.B.C. µg/100 ml.	% Saturation	Haemoglobin g./100 ml	Blood Film	DRINKING HABITS Pints of African Beer consumed per week
SI/1/2	15	60	292	352	17	11.6	-	6
SI/2/2	14	112	218	330	34	12.3	-	-
SI/3/2	15	134	317	451	30	14.1	-	-
SI/4/2	18	188	269	457	41	-	-	1
SI/5/2	15	119	284	403	30	13.4	-	-
SI/6/2	19	66	257	323	20	15.2	-	4
SI/7/2	10	94	241	335	28	12.3	-	-
SI/8/2	12	88	333	421	21	12.3	-	-
SI/9/2	10	69	321	390	18	12.7	-	-
SI/10/2	12	94	322	416	23	11.2	-	-
SI/11/2	16	96	297	393	24	14.1	-	-
SI/12/2	14	100	324	424	24	16.0	-	-
SI/13/2	10	130	259	389	33	12.7	-	-
SI/14/2	10	139	205	344	40	11.5	-	-
SI/15/2	14	56	334	390	14	12.3	-	-
SI/16/2	15	96	310	406	24	12.3	-	-
SI/17/2	10	66	328	394	17	11.9	-	-
SI/18/2	19	40	299	339	12	15.6	-	6
SI/19/2	10	189	289	478	40	12.3	-	-
SI/20/2	10	83	350	433	19	13.0	-	-
SI/21/2	11	37	427	464	8	12.3	-	-
SI/22/2	18	96	278	374	26	14.8	-	-
SI/23/2	15	188	380	568	33	14.1	-	-
SI/24/2	18	136	258	394	35	12.3	-	6
SI/25/2	16	120	278	398	30	14.8	-	-
SI/26/2	14	125	244	369	34	12.7	-	-
SI/27/2	12	72	309	381	19	-	-	-
SI/28/2	14	63	358	421	15	11.9	-	-
SI/29/2	19	182	210	392	46	14.5	-	8
SI/30/2	18	106	354	460	23	14.5	-	-

FEMALES - SECOND DECADE

Ref. No.	Age	Serum Iron µg/100 ml	U.I.B.C. µg/100 ml	T.I.B.C. µg/100 ml	% Saturation	Haemoglobin g/100 ml.	Blood Film	DRINKING HABITS Pints of African Beer consumed per week.
SI/34/2	10	108	307	415	26	12.7	-	-
SI/35/2	13	153	230	383	40	11.2	-	-
SI/36/2	17	99	299	398	25	14.5	-	-
SI/37/2	19	82	312	394	21	11.5	-	-
SI/38/2	16	67	376	443	15	11.2	-	-
SI/39/2	19	114	367	481	24	12.7	-	-
SI/40/2	19	88	445	533	16	11.9	Red cells show moderate polychromasia	-
SI/41/2	19	86	280	366	23	11.9	Red cells show slight polychromasia	-
SI/42/2	18	77	283	360	21	10.8	Red cells show marked polychromasia	-
SI/43/2	11	104	271	375	28	11.9	-	-
SI/44/2	14	98	253	351	28	13.0	-	-
SI/45/2	17	124	328	452	27	14.1	-	-
SI/46/2	12	72	355	427	17	12.3	-	-
SI/47/2	15	78	301	379	21	12.3	-	-
SI/48/2	16	135	420	555	24	12.3	-	-
SI/49/2	13	78	398	476	16	11.5	-	-
SI/50/2	11	60	286	346	17	12.7	-	-
SI/51/2	14	95	311	406	23	12.7	-	-
SI/52/2	11	94	319	413	23	10.8	-	-
SI/53/2	10	110	251	361	30	10.4	-	-
SI/54/2	15	44	392	436	10	10.4	Red cells show moderate polychromasia	-
SI/55/2	19	146	216	362	40	11.5	-	-
SI/56/2	14	42	299	341	12	11.5	Red cells show slight polychromasia	+
SI/57/2	14	166	257	423	39	13.0	-	-
SI/58/2	13	44	399	443	10	9.3	Red cells show moderate ring staining	-
SI/59/2	15	40	328	368	11	11.9	-	-
SI/60/2	14	83	261	344	24	13.0	-	-
SI/61/2	19	82	312	394	21	12.7	-	-
SI/62/2	17	108	309	417	26	8.9	Red cells show marked polychromasia	-
SI/63/2	16	96	178	274	35	11.5	-	-
SI/64/2	13	148	234	382	39	11.2	Red cells show slight polychromasia	-

MALES - THIRD DECADE

Ref. No.	Age	Serum Iron µg/100 ml	U.I.B.C. µg/100 ml	T.I.B.C. µg/100 ml.	% Saturation	Haemoglobin g/100 ml.	Blood Film	DRINKING HABITS Pints of African Beer consumed per week
SI/1/3	21	125	320	445	28	16.4	-	2
SI/2/3	25	139	228	367	38	14.9	-	-
SI/3/3	25	78	258	336	23	14.8	-	-
SI/4/3	21	65	310	375	17	16.0	-	-
SI/5/3	26	160	206	366	44	16.8	Red cells are slightly hyperchromatic	1
SI/6/3	28	126	226	352	36	14.1	-	36
SI/7/3	27	118	171	289	41	-	-	4
SI/8/3	21	108	158	266	41	15.6	-	1
SI/9/3	23	71	228	299	24	15.6	-	-
SI/10/3	25	115	291	406	28	14.5	-	-
SI/11/3	26	106	259	365	29	14.8	-	2
SI/12/3	28	109	231	340	32	15.6	-	4
SI/13/3	27	112	222	334	34	14.5	-	-
SI/14/3	29	179	178	357	50	15.6	-	6
SI/15/3	21	145	148	293	50	15.2	-	1
SI/16/3	25	204	158	362	56	15.2	-	2
SI/17/3	23	211	110	321	66	14.8	-	4
SI/18/3	26	186	150	336	55	14.8	-	2
SI/19/3	23	94	368	462	20	13.4	-	-
SI/21/3	25	105	250	355	30	15.6	-	-
SI/22/3	23	99	272	371	27	13.0	-	8
SI/23/3	21	111	310	421	26	14.1	-	1
SI/24/3	28	114	263	377	30	15.2	-	1
SI/25/3	21	123	161	284	43	15.6	-	3
SI/26/3	26	176	257	433	41	15.2	-	2
SI/27/3	21	160	179	339	47	15.2	-	1
SI/28/3	26	74	262	336	22	-	-	8
SI/29/3	26	163	163	326	50	14.5	-	-
SI/30/3	25	163	168	331	49	-	-	8
SI/31/3	23	70	376	446	16	-	-	8
SI/32/3	28	89	300	389	23	12.7	-	-

FEMALES - THIRD DECADE

Ref. No.	Age	Serum Iron µg/100 ml	U.I.B.C. µg/100 ml.	T.I.B.C. µg/100 ml.	% Saturation	Haemoglobin g/100 ml.	Blood Film	DRINKING HABITS Pints of African Beer consumed per week.
SI/34/3	23	146	216	362	40	10.4	Red cells show slight anisocytosis and polychromasia	-
SI/35/3	23	142	217	359	40	12.7	-	-
SI/36/3	29	102	334	436	23	12.3	-	-
SI/37/3	21	77	333	410	19	13.0	-	An occasional bottle European beer
SI/38/3	25	99	353	452	22	14.1	-	-
SI/39/3	22	81	294	375	22	-	-	-
SI/40/3	21	143	292	435	33	10.8	-	-
SI/41/3	22	70	228	298	24	11.5	-	-
SI/42/3	24	117	233	350	33	12.3	-	-
SI/43/3	28	117	307	424	28	14.8	-	-
SI/44/3	23	116	280	396	29	13.8	-	-
SI/45/3	28	152	227	379	40	11.2	Red cells show slight polychromasia	6
SI/46/3	23	148	188	336	44	12.7	-	-
SI/47/3	23	79	366	445	18	13.0	-	-
SI/48/3	28	105	238	343	31	12.7	-	-
SI/49/3	25	83	310	393	21	11.5	-	-
SI/50/3	20	126	231	357	35	12.3	-	2
SI/51/3	20	111	285	396	28	14.1	-	-
SI/52/3	25	77	299	376	20	12.3	-	-
SI/53/3	21	130	282	412	32	12.3	Red cells show slight polychromasia	1
SI/54/3	26	77	329	406	19	12.3	-	2
SI/55/3	24	186	166	352	53	12.3	-	-
SI/56/3	23	105	283	388	27	11.5	-	-
SI/57/3	25	86	298	384	22	13.8	-	-
SI/58/3	22	165	294	459	36	13.4	Red cells show slight polychromasia	-
SI/59/3	25	150	263	413	36	13.8	-	-
SI/60/3	24	64	408	472	14	10.4	Red cells show slight polychromasia	-
SI/61/3	26	84	354	438	19	11.2	Red cells show slight polychromasia	-
SI/62/3	25	113	281	394	29	12.7	-	-
SI/63/3	27	109	371	480	23	12.3	-	-
SI/64/3	23	95	158	253	38	10.8	Red cells show slight polychromasia	-
SI/65/3	22	58	331	389	15	11.9	-	-

MALES - FOURTH DECADE

Ref. No.	Age	Serum Iron µg/100 ml	U.I.B.C. µg/100 ml.	T.I.B.C. µg/100 ml.	% Saturation	Haemoglobin g/100 ml.	Blood Film	DRINKING HABITS Pints of African Beer consumed per week
SI/1/4	36	110	166	276	40	13.3	-	2
SI/2/4	30	63	183	246	26	13.8	-	-
SI/3/4	32	97	238	335	29	16.0	-	-
SI/4/4	33	111	205	316	35	14.8	-	-
SI/5/4	33	74	265	339	22	13.8	A few target cells seen among the red cells	-
SI/6/4	35	142	175	317	45	14.5	-	-
SI/7/4	30	96	207	303	32	15.6	-	-
SI/8/4	32	156	180	336	46	11.9	-	2
SI/9/4	31	135	210	345	39	15.2	-	1
SI/10/4	38	77	267	344	22	17.2	-	-
SI/11/4	35	118	266	384	31	15.2	-	6
SI/12/4	32	111	337	448	25	16.4	-	-
SI/13/4	32	218	131	349	62	15.6	-	14
SI/14/4	35	100	297	397	25	13.8	-	-
SI/15/4	31	109	256	365	30	16.0	-	6
SI/16/4	35	53	239	292	18	14.8	-	14
SI/17/4	35	204	52	256	80	10.8	-	2
SI/18/4	34	53	313	366	14	16.4	-	4
SI/19/4	30	103	270	373	28	15.2	-	8
SI/20/4	35	86	261	347	25	14.1	-	-
SI/21/4	35	67	177	344	28	13.8	-	6
SI/22/4	30	121	213	334	36	16.4	-	-
SI/23/4	36	91	270	361	25	14.8	-	-
SI/24/4	39	49	275	324	15	16.0	-	6
SI/25/4	30	33	256	289	11	11.2	-	2
SI/26/4	30	145	283	428	34	14.1	-	64
SI/27/4	32	82	324	406	20	13.0	-	-
SI/28/4	31	100	327	427	23	13.8	-	-
SI/29/4	31	197	173	370	53	13.0	-	20
SI/30/4	33	110	242	352	31	17.2	-	-
SI/31/4	35	132	324	456	29	15.6	-	6
SI/32/4	35	163	199	362	45	16.0	-	21

FEMALES - FOURTH DECADE

Ref. No.	Age	Serum Iron µg/100 ml	U.I.B.C. µg/100 ml.	T.I.B.C. µg./100 ml.	% Saturation	Haemoglobin g/100 ml.	Blood Film	DRINKING HABITS Pints of African Beer consumed per week
SI/34/4	31	94	305	399	23	11.5	Red cells show slight polychromasia	3
SI/35/4	35	136	194	330	41	12.7	Red cells show slight polychromasia	4
SI/36/4	30	59	470	529	11	10.8	-	-
SI/37/4	35	135	239	374	36	12.3	-	-
SI/38/4	35	131	234	365	36	12.3	-	5
SI/39/4	37	105	309	414	25	14.1	-	-
SI/40/4	31	78	234	312	25	11.9	Red cells show a few target cells and slight anisocytosis	-
SI/41/4	32	125	346	471	27	11.2	Red cells show slight polychromasia	-
SI/42/4	30	55	271	326	17	11.9	-	4
SI/43/4	31	83	285	368	23	11.5	-	-
SI/44/4	37	52	424	476	11	11.5	Red cells show slight polychromasia	-
SI/45/4	35	113	269	392	30	14.1	-	-
SI/46/4	32	112	253	365	31	12.3	-	-
SI/47/4	35	70	303	373	19	13.0	Red cells show slight polychromasia	8
SI/48/4	39	130	250	380	34	13.0	-	-
SI/49/4	30	140	282	422	33	13.8	-	2
SI/50/4	30	40	553	593	7	9.3	Red cells show slight ring staining, polychromasia and anisocytosis	2
SI/51/4	35	107	234	341	31	13.0	-	8
SI/52/4	35	81	327	408	20	12.7	-	-
SI/53/4	35	67	278	345	19	12.3	-	-
SI/54/4	35	121	248	369	33	12.7	-	-
SI/55/4	30	44	246	290	15	11.9	-	-
SI/56/4	33	24	457	481	5	10.0	Red cells show moderate polychromasia and a few target cells	-
SI/57/4	30	33	297	330	10	12.7	-	-
SI/58/4	35	143	172	315	45	13.0	-	-
SI/59/4	38	90	314	404	22	12.3	-	-
SI/60/4	31	74	248	322	23	12.3	Red cells show slight polychromasia	4
SI/61/4	35	75	274	349	21	12.7	-	-
SI/62/4	35	28	387	415	7	7.8	Red cells show moderate polychromasia	-
SI/63/4	35	111	350	461	24	12.7	-	-
SI/65/4	35	64	331	395	16	12.7	-	-

MALES - FIFTH DECADE

Ref. No.	Age	Serum Iron µg/100 ml	U.I.B.C. µg./100 ml.	T.I.B.C. µg./100 ml.	% Saturation	Haemoglobin g./100 ml	Blood Film	DRINKING HABITS: Pints of African Beer consumed per week
SI/1/5	48	130	279	409	32	14.5	-	-
SI/2/5	42	164	144	308	53	15.1	-	4
SI/3/5	45	126	190	316	40	14.8	-	-
SI/4/5	45	231	38	269	86	14.6	-	36
SI/5/5	48	51	192	243	21	14.1	-	3
SI/6/5	40	148	209	357	41	14.8	-	60+
SI/7/5	44	99	274	373	27	15.2	-	-
SI/8/5	45	41	182	223	18	8.6	Red cells show slight anisocytosis	5
SI/9/5	47	167	185	352	47	13.4	-	6
SI/10/5	48	69	255	324	21	12.3	-	6
SI/11/5	47	167	112	279	60	14.8	-	5
SI/12/5	48	77	239	316	24	16.0	-	14
SI/13/5	47	105	309	414	25	14.5	-	-
SI/14/5	45	98	189	287	34	15.6	-	8
SI/15/5	47	153	133	286	54	12.7	-	15
SI/16/5	42	104	229	333	31	13.0	-	-
SI/17/5	48	120	169	289	42	12.7	-	-
SI/18/5	45	194	106	300	65	14.8	-	12
SI/19/5	45	89	239	328	27	14.1	-	-
SI/20/5	48	33	225	258	13	6.3	Red cells show polychromasia and anisocytosis, a few target cells	70
SI/21/5	47	85	197	282	30	13.0	-	8
SI/22/5	41	117	146	263	45	14.5	-	-
SI/23/5	48	51	396	447	11	13.4	-	-
SI/24/5	47	72	248	320	22	13.0	-	-
SI/25/5	45	162	66	228	71	13.0	-	6
SI/26/5	49	115	248	363	32	17.2	-	8
SI/27/5	45	186	65	251	74	14.1	-	4
SI/28/5	48	98	195	293	33	14.1	-	1
SI/29/5	45	69	287	356	19	13.8	-	-
SI/30/5	41	101	241	342	30		-	2
SI/31/5	41	68	252	320	21	11.5	-	-

FEMALES - FIFTH DECADE

Ref. No.	Age	Serum Iron µg/100 ml	U.I.B.C. µg./100 ml.	T.I.B.C. µg./100 ml.	% Saturation	Haemoglobin g./100 ml	Blood Film	DRINKING HABITS Pints of African Beer consumed per week
SI/34/5	48	97	287	384	25	11.5	-	36
SI/35/5	40	75	296	371	20	13.4	-	-
SI/36/5	40	57	304	361	16	13.0	-	-
SI/37/5	40	90	237	327	27	13.8	-	-
SI/38/5	45	28	533	561	5		-	-
SI/39/5	41	128	189	317	40	11.5	Red cells show moderate polychromasia	6
SI/40/5	44	129	245	374	34	13.0	-	-
SI/41/5	48	95	293	388	24	13.8	-	-
SI/42/5	40	68	241	309	22	12.3	Red cells show slight polychromasia	6
SI/43/5	41	83	298	381	22	13.4	-	1
SI/44/5	47	86	293	379	23	13.0	-	-
SI/45/5	42	30	461	494	6	8.9	Red cells show moderate polychromasia, slight anisocytosis	-
SI/46/5	45	112	270	382	29	13.0	-	-
SI/47/5	42	144	164	308	47	14.5	-	16
SI/48/5	40	39	454	493	8	8.6	Red cells show marked polychromasia	-
SI/49/5	40	28	309	337	8	7.4	Red cells show moderate ring staining, target cells, polychromasia	-
SI/50/5	40	92	333	425	22	11.2	-	-
SI/51/5	45	87	336	423	21	12.3	-	-
SI/52/5	47	43	367	410	10	12.7	-	2
SI/53/5	40	70	347	417	17	6.3	Red cells show moderate ring staining, target cells, polychromasia	-
SI/54/5	48	67	224	291	23	10.8	Red cells show slight polychromasia	-
SI/55/5	40	60	511	571	10	11.2	Red cells show slight polychromasia	-
SI/56/5	40	112	207	319	35	14.1	-	-
SI/57/5	44	35	396	431	8	7.8	Red cells show slight ring staining and anisocytosis	-
SI/58/5	45	141	227	368	38	13.8	-	-
SI/59/5	40	128	211	339	38	11.5	-	-
SI/60/5	43	126	314	440	29	12.3	-	-
SI/61/5	45	98	300	398	25	11.9	Red cells show slight polychromasia	2
SI/62/5	41	88	214	302	29	13.0	-	1
SI/63/5	48	67	420	487	14	11.9	-	-
SI/64/5	45	147	152	299	49	14.1	-	-

MALES - SIXTH DECADE

Ref. No.	Age	Serum Iron µg/100 ml	U.I.B.C. µg./100 ml.	T.I.B.C. µg./100 ml.	% Saturation	Haemoglobin g./100 ml	Blood Film	DRINKING HABITS Pints of African Beer consumed per week
SI/1/6	55	121	206	327	37	14.5	-	-
SI/2/6	55	69	221	290	24	13.0	-	14
SI/3/6	55	150	173	323	46	14.8	-	5
SI/4/6	50	117	278	395	30	17.2	-	-
SI/5/6	51	153	230	383	40	13.0	-	-
SI/6/6	55	190	103	293	65	14.8	-	8
SI/7/6	52	86	236	322	27	13.0	-	-
SI/8/6	54	75	242	317	24	13.8	-	14
SI/9/6	58	56	311	367	15	14.1	-	-
SI/10/6	52	210	99	309	68	14.1	-	1
SI/11/6	55	74	351	425	17	15.6	-	-
SI/12/6	50	172	69	241	71		-	-
SI/13/6	50	94	246	340	28	13.4	-	-
SI/14/6	52	42	229	271	16	12.7	-	4
SI/15/6	55	204	94	298	68	13.0	-	4
SI/16/6	50	95	304	399	24	13.0	-	3
SI/17/6	50	43	196	239	18		-	2
SI/18/6	51	38	357	395	10	10.4	Red cells show moderate polychromasia	1
SI/19/6	52	228	133	361	63	15.2	-	8
SI/20/6	55	114	99	213	54	11.9	Red cells show slight polychromasia	20
SI/21/6	54	57	219	276	21		-	6
SI/22/6	50	89	300	389	23	14.1	-	4
SI/23/6	56	129	259	388	33	14.8	-	8
SI/24/6	55	55	379	434	13	12.7	Red cells show slight polychromasia and anisocytosis	-
SI/25/6	50	125	190	315	40	13.0	-	4
SI/26/6	55	54	220	274	20	18.6	Red cells show moderate polychromasia	1
SI/27/6	51	137	143	280	49	14.1	-	2
SI/28/6	55	110	202	312	35	12.7	Red cells show a few target cells	70
SI/29/6	50	100	219	319	31	14.5	Red cells show slight polychromasia	60
SI/30/6	55	74	300	374	20	14.5	-	1
SI/31/6	55	112	208	320	35	14.1	-	2

FEMALES - SIXTH DECADE

Ref. No.	Age	Serum Iron µg/100 ml	U.I.B.C. µg./100 ml.	T.I.B.C. µg./100 ml.	% Saturation	Haemoglobin g/100 ml.	Blood Film	DRINKING HABITS Pints of African Beer consumed per week
SI/34/6	50	132	263	395	32	12.7	-	-
SI/35/6	55	116	276	392	30	13.0	-	-
SI/36/6	50	124	272	396	31	12.7	-	2
SI/37/6	50	26	537	563	5	5.2	Red cells show marked ring staining, anisocytosis, moderate polychromasia.	8
SI/38/6	55	75	251	326	23	13.8	Red cells show slight polychromasia	-
SI/39/6	52	124	147	271	46	13.4	-	4
SI/40/6	50	27	267	294	9	11.2	Red cells show slight polychromasia	-
SI/41/6	55	166	182	348	48	13.4	-	70 +
SI/42/6	55	203	224	427	48	13.0	-	-
SI/43/6	50	100	295	395	25	13.8	-	-
SI/44/6	55	166	141	307	54	13.8	-	-
SI/45/6	50	81	323	404	20	13.0	-	-
SI/46/6	52	272	47	319	85	-	-	5
SI/47/6	55	129	177	306	42	11.5	-	-
SI/48/6	52	70	166	236	30	12.7	-	-
SI/49/6	55	120	334	454	26	-	-	-
SI/50/6	50	57	396	453	13	11.9	-	1
SI/51/6	50	117	108	225	52	9.7	-	-
SI/52/6	58	92	302	394	23	13.0	-	-
SI/53/6	50	55	303	358	15	11.2	-	2
SI/54/6	52	152	172	324	47	12.7	-	2

MALES - SEVENTH DECADE AND OLDER

Ref. No.	Age	Serum Iron µg/100 ml	U.I.B.C. µg./100 ml.	T.I.B.C. µg./100 ml.	% Saturation	Haemoglobin g./100 ml	Blood Film	DRINKING HABITS Pints of African Beer consumed per week
SI/1/7	70	202	40	242	84	16.0		-
SI/2/7	65	206	121	327	63	13.4	Red cells show moderate number of target cells	4
SI/3/7	60	140	236	376	37	11.2	-	-
SI/4/7	70	258	53	311	83	14.8	-	8
SI/5/7	65	79	309	388	20	14.1	-	-
SI/6/7	65	182	94	276	66	12.7	Red cells show a few target cells	50
SI/7/7	65	81	179	260	34	11.2	-	-
SI/8/7	65	114	200	314	36	13.0	-	-
SI/9/7	85	100	236	336	30	13.4	-	4
SI/10/7	65	49	320	369	13	12.3	-	-
SI/11/7	60	102	343	445	23	13.8	-	-
SI/12/7	65	238	110	348	68	14.5	-	4
SI/13/7	60	51	310	361	14	12.7	-	-
SI/14/7	60	133	224	357	37	12.7	-	-
SI/15/7	60	118	210	328	36	14.1	-	8
SI/16/7	70	79	242	321	25	11.9	Red cells show slight polychromasia	8
SI/17/7	60	123	265	388	32	14.1	-	1
SI/19/7	70	112	306	418	27	15.2	-	70 +
SI/20/7	60	48	352	400	12	14.3	-	-
SI/21/7	65	131	212	343	38	14.1	Red cells show moderate polychromasia	3
SI/22/7	65	96	145	241	40	-	-	-
SI/23/7	60	47	149	196	24	11.5	-	2
SI/24/7	60	35	221	256	14	10.8	Red cells show slight polychromasia	-
SI/25/7	65	100	254	354	28	14.1	-	6
SI/26/7	70	73	308	381	19	15.2	-	-
SI/27/7	60	133	100	233	57	11.9	-	2
SI/28/7	65	71	233	304	23	11.9	Red cells show slight polychromasia	-
SI/29/7	70	91	183	274	33	14.8	-	70
SI/30/7	80	115	162	277	41	13.0	Red cells show slight polychromasia	-
SI/31/7	65	34	259	293	12	12.7	-	1

FEMALES - SEVENTH DECADE AND OLDER

Ref. No.	Age	Serum Iron µg/100 ml	U.I.B.C. µg./100 ml.	T.I.B.C. µg/100 ml.	% Saturation	Haemoglobin g./100 ml	Blood Film	DRINKING HABITS: Pints of African Beer consumed per week
SI/34/7	60	100	227	327	31	13.0	-	-
SI/35/7	60	60	341	401	15	11.2	Red cells show slight polychromasia	-
SI/36/7	65	99	259	358	28	13.0	-	60
SI/37/7	70	60	207	265	22	8.2	Red cells show moderate polychromasia	-
SI/38/7	65	67	286	353	19	12.3	-	2
SI/39/7	60	70	240	310	23	13.4	-	-
SI/40/7	64	66	245	311	21	12.7	Red cells show slight polychromasia	2
SI/41/7	65	75	283	358	21	11.8	-	-
SI/42/7	60	243	38	281	87	13.0	-	5
SI/43/7	87	120	145	265	45	12.3	-	5

APPENDIX IX

Red cell survival measured in 22 African males using the Radioactive-Chromium method as described by Dacie and Lewis in "Practical Haematology", 3rd Edition, 1966, Churchill, London.

The T½ ^{51}Cr in normal subjects using this method was found by the authors to range between 25 and 32 days.

In the following tables the measurements have not been corrected for elution.

The ^{51}Cr used for labelling the red cells was in the form of $Na_2\ ^{51}Cr\ O_4$ and was obtained from the Radiochemical Centre, Amersham, Buckinghamshire, England. Counting of samples was carried out in a "well" counter using a sodium iodide crystal activated with thallium. The counts were made on an Echo scaler plus timer. Each sample was counted for a 100 seconds three times and an average of the three counts used for the calculation.

Approximately 100 µc of $Na_2\ ^{51}Cr\ O_4$ were added to each sample of cells.

Case 1. Occupation. Laboratory Technical Assistant.
Age. 38 years. Hb. 15.1 G%.
P.C.V. 47%. Reticulocyte Count 0.2%.

Day.	^{51}Cr. Survival.
1	98
2	93
3	85
8	77
12	76
17	69
21	59
25	56
30	51
43	32

$T_{\frac{1}{2}}$ ^{51}Cr 34 days.

Case 2. Occupation. Laboratory Technical Assistant.
Age. 21 years. Hb. 14.6 G%.
P.C.V. 45%. Reticulocyte Count 0.2%.

Day.	^{51}Cr. Survival.
1	96
3	95
5	92
7	82
13	68
17	60
21	50
29	40
35	40

$T_{\frac{1}{2}}$ ^{51}Cr. 20 days.

Case 3. Occupation. Police Constable.
Age. 29 years. Hb. 15.6 G%.
P.C.V. 46%. Reticulocyte Count 0.1%.

Days.	^{51}Cr. Survival.
1	97
2	95
3	90
7	85
12	72
16	71
20	68
23	56
28	52
35	46

$T_{\frac{1}{2}}$ ^{51}Cr. = 30 days.

Case 4. Occupation. Odd Job Man.
Age. 38 years. Hb. 14.7 G%.
P.C.V. 46%. Reticulocyte Count 0.6%.

Day.	^{51}Cr. Survival.
1	90
2	93
3	90
7	80
13	68
17	62
22	56
28	48
32	45
35	41

$T_{\frac{1}{2}}$ ^{51}Cr. = 27 days.

Case 5. Occupation. Police Sergeant.
Age. 29 years. Hb. 14.9 G%. P.C.V. 45%. Reticulocyte Count 0.2%.

Day.	% ^{51}Cr. Survival.
1	92
2	91
3	85
7	78
11	71
16	69
19	63
22	56
26	57
29	51
32	49

T½ ^{51}Cr. = 30 days.

Case 6. Occupation. Mortuary Attendant.
Age. 59 years. Hb. 14.1 G%. P.C.V. 42%. Reticulocyte Count 1.0%.

Day.	% ^{51}Cr. Survival.
1	94
2	89
3	86
7	76
11	66
15	63
19	56
23	54
28	47
33	40

T½ ^{51}Cr. = 25 days.

Case 7. Occupation. Mortuary Attendant.
Age. 58 years. Hb. 14.6 G%. P.C.V. 48%. Reticulocyte Count 0.2%.

Day.	% ^{51}Cr. Survival.
1	92
2	89
3	84
7	78
11	70
15	67
19	60
23	54
28	48
33	46

T½ ^{51}Cr. = 27 days.

Case 8. Occupation. Laboratory Servant.
Age. 26 years. Hb. 14.1 G%. P.C.V. 43%. Reticulocyte Count 1.0%.

Day.	% ^{51}Cr. Survival.
1	96
2	88
3	85
9	73
13	70
17	60
22	55
26	51
31	45

T½ ^{51}Cr. = 27 days.

Case 9. Occupation. Laboratory Servant.
Age. 48 years. Hb. 15.4 G%. P.C.V. 46%. Reticulocyte Count 0.2%

Day.	% ^{51}Cr Survival.
1	96
2	90
3	85
9	77
13	71
17	62
22	54
26	52
31	44

T½ ^{51}Cr = 27 days.

Case 10. Occupation. Laboratory Servant.
Age. 42 years. Hb. 17.0 G%. P.C.V. 55%. Reticulocyte Count 0.3%.

Day.	% ^{51}Cr Survival.
1	97
2	97
3	94
7	85
12	71
17	70
21	66
26	58
30	52
33	49

T½ ^{51}Cr = 32 days.

Case 11. Occupation. Laboratory Servant.
Age. 27 years. Hb. 14.8 G%. P.C.V. 48%. Reticulocyte Count 1.0%.

Day.	% ^{51}Cr Survival.
1	98
2	94
3	89
7	83
11	77
17	67
21	65
26	56
30	54
33	43

T½ ^{51}Cr = 32 days.

Case 12. Occupation. Flight Sergeant.
Age. 34 years. Hb. 17.7 G%. P.C.V. 50%. Reticulocyte Count 0.2%.

Day.	% ^{51}Cr Survival.
1	99
2	95
3	94
7	88
12	81
16	75
21	67
26	59
30	54
34	48

T½ ^{51}Cr = 33 days.

Case 13. Occupation. S.A.C. R.R.A.F.
Age. 25 years. Hb. 17.4 G%. P.C.V. 48%. Reticulocyte Count 0.1%.

Day.	% ^{51}Cr Survival.
1	98
2	96
3	92
7	87
12	76
16	71
21	62
26	56
30	51
34	45

T½ ^{51}Cr = 31 days.

Case 14. Occupation. S.A.C. R.R.A.F.
Age. 23 years. Hb. 17.4 G%. P.C.V. 50%. Reticulocyte Count 0.2%.

Day.	% ^{51}Cr Survival.
1	94
2	92
3	90
7	85
13	77
16	73
21	66
30	52
34	46

T½ ^{51}Cr = 32 days.

Case 15. Occupation. S.A.C. R.R.A.F.
Age. 25 years. Hb. 15.5 G% P.C.V. 50%. Reticulocyte Count 0.5%.

Day.	% ^{51}Cr Survival.
1	97
2	94
3	93
7	87
12	79
21	64
26	56
30	50
34	46

T½ ^{51}Cr = 30 days.

Case 16. Occupation. Corporal R.R.A.F.
Age. 30 years. Hb. 15.9 G%. P.C.V. 51%. Reticulocyte Count 0.2%.

Day.	% ^{51}Cr Survival.
1	96
2	94
3	92
6	86
10	78
15	70
20	64
24	56
28	53
31	50

T½ ^{51}Cr = 31 days.

Case 17. Occupation. A.C. R.R.A.F.
Age. 20 years. Hb. 14.9 G%. P.C.V. 46%. Reticulocyte Count 0.1%.

Day.	% ^{51}Cr Survival.
1	100
2	99
3	96
6	89
10	83
15	76
20	65
24	63
28	54
31	52
34	47

T½ ^{51}Cr = 32 days.

Case 18. Occupation. A.C. R.R.A.F.
Age. 22 years. Hb. 15.2 G%. P.C.V. 46%. Reticulocyte Count 0.5%.

Day.	% ^{51}Cr Survival.
1	99
2	96
3	95
6	89
10	81
15	67
20	61
24	54
28	47
31	43

T½ ^{51}Cr = 26 days.

Case 19. Occupation. Laboratory Technical Assistant.
Age. 25 years. Hb. 14.9%. P.C.V. 45%. Reticulocyte Count 0.2%.

Day.	% ^{51}Cr Survival.
1	99
2	98
3	92
7	84
11	74
15	67
18	61
23	56
31	47

T½ ^{51}Cr = 28 days.

Case 20. Occupation. Laboratory Technical Assistant.
Age. 26 years. Hb. 15.1G%. P.C.V. 46%. Reticulocyte Count 0.3%.

Day.	% ^{51}Cr Survival.
1	98
2	95
3	93
7	89
11	81
15	73
18	65
23	58
31	48

T½ ^{51}Cr = 29 days.

Case 21. Occupation. Laboratory Technical Assistant.
Age. 35 years. Hb. 14.7 G%.
P.C.V. 45%. Reticulocyte Count 0.5%.

Day.	% ^{51}Cr Survival
1	99
2	98
3	95
7	87
11	77
15	74
18	66
23	58
31	50

T½ ^{51}Cr = 31 days.

Case 22. Occupation. Laboratory Assistant.
Age. 45 years. Hb. 14.8 G%.
P.C.V. 44%. Reticulocyte Count 0.2%

Day.	% ^{51}Cr Survival.
1	95
2	94
3	90
7	82
11	75
15	69
18	64
23	55
31	45

T½ ^{51}Cr = 27 days.

A P P E N D I X X

OSMOTIC FRAGILITY OF RED CELLS IN 50 AFRICANS

METHOD USED WAS THAT DESCRIBED BY DACIE AND LEWIS,
"PRACTICAL HAEMATOLOGY". THIRD EDITION. 1963,
CHURCHILL, LONDON

(TUBES WERE ALLOWED TO STAND FOR 30 MINUTES AT ROOM TEMPERATURE).

Percent NaCl.	COLIN Aged 56	CAMPANIA Aged 59	SERG Aged 29	RUBEN Aged 38	CONSTABLE Aged 28	DYSON Aged 56	CHISIMA Aged 30	PHINEAS Aged 56	CHADRICK Aged 42	DEK Aged 50	PATRICK B. Aged 24	LUCAS Aged 27	MATEMA Aged 40	JOHANNES T. Aged 26	STEPHEN C. Aged 48	MUSUMOJI, Sgt. Aged 27	NELSON Aged 60	SAMUEL Aged 45	WALINDA Aged 35	WILLIAM Aged 25	WILFRED Aged 26	ARTHUR Aged 62	SILAS Aged 21	SEBASTIAN Aged 20	SGT. MUBAY Aged 30
0.85%	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
0.75%	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
0.65%	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
0.60%	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
0.55%	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
0.50%	-	-	3%	-	2%	-	-	1%	7%	4%	-	-	2%	4%	3.5%	3%	5%	1%	2%	6%	-	-	6%	3%	-
0.45%	-	5%	29%	7%	17%	7%	14%	12.5%	19%	9%	11%	1%	23%	29%	38%	30%	18%	13%	33%	57%	12%	4%	48%	24%	5%
0.40%	5%	42%	92%	66%	77%	70%	80%	87%	78%	63%	89%	11%	87%	79%	91%	89%	76%	70%	95%	91%	72%	33%	88.5%	85%	71%
0.35%	35%	87%	96%	95%	95%	94%	98%	97%	87%	96%	94%	42%	93%	94%	98%	92%	94%	88%	99%	94%	93%	79%	90%	98%	94%
0.30%	77%	97%	98%	96%	96%	95%	98%	99%	95%	96%	95%	76%	98%	98%	98%	92%	96%	88%	99%	95%	94%	88%	99%	99%	96%
0.20%	99%	99%	99%	97%	100%	97%	99%	100%	97%	98%	95%	98%	99%	98%	98%	94%	98%	91%	100%	95%	96%	98%	100%	100%	96%
0.10%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%
	PERCENT HAEMOLYSIS																								
P.C.V. %	48	54	45	48	42	44	52	42	55	42	48	48	48	43	45	47	47	45	46	44	47	49	47	45	50
Retics %	0.2	2.0	0.2	0.6	0.5	0.1	0.2	1.0	0.3	1.0	1.5	1.2	0.5	1.0	0.2	1.0	1.0	1.0	1.8	1.5	0.5	1.5	1.0	2.0	1.2
Hb in grammes %	13.0	14.1	14.9	13.7	15.6	13.4	15.6	13.75	17.0	14.8	15.6	14.8	16.2	14.1	15.95	15.25	13.75	15.95	14.8	14.5	14.8	14.8	14.8	14.5	16.3

Percent NaCl	SAM Aged 50	WITNESS Aged 34	ABERINO Aged 34	CHIBATA Aged 60	JIMIR Aged 46	CLAUDIO Aged 23	LAWRENCE Aged 48	DAVID Aged 28	NICHES Aged 29	CHASAMBO Aged 46	MACOE Aged 32	SOSAYI Aged 31	SWEET Aged 42	OLIVER Aged 42	HERBERT Aged 41	MACHIPISON Aged 53	MUBAYIWA Aged 55	MUDSON Aged 50	GRAVOS Aged 30	MICHAEL Aged 28	TIMOTHY Aged 41	CONST. MORGAN Aged 25	FREDRICK Aged 25	ALLEN Aged 21	PETER Aged 30
0.85%	- -	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
0.75%	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
0.65%	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
0.60%	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
0.55%	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	2%	-	-	-	-	-	-	-	-	-
0.50%	1%	-	3%	3%	-	-	3%	4%	-	-	-	-	-	2.5%	12%	22%	6%	-	1%	3.5%	-	-	-	2%	-
0.45%	8%	7%	49.5%	29.5%	11%	12%	41%	31%	5%	9.5%	-	11%	6%	23%	69%	90%	33%	9%	13%	37%	25%	2%	5%	20%	4%
0.40%	66%	69%	92%	88%	66%	82%	84%	81%	42%	63%	12%	80%	40%	78%	88%	97%	97%	59%	73%	91%	29%	41%	60%	72%	50%
0.35%	100%	94%	98%	96%	93%	100%	94%	94%	98%	90%	64%	93%	87%	93%	95%	97%	92%	78%	98%	98%	68%	88%	99%	82%	92%
0.30%	100%	95%	100%	96%	98%	100%	94%	96%	94%	90%	91%	95%	97%	96%	99%	98%	92%	96%	98%	98%	87%	88%	99%	86%	93%
0.20%	100%	95%	100%	98%	99%	100%	95%	98%	95%	90%	95%	98%	97%	99%	99%	100%	92%	99%	98%	99%	89%	91%	99%	89%	97%
0.10%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%
										PERCENT HAEMOLYSIS															
P.C.V. %	44	49	43	44	43	49	42	53	53	45	47	49	42	51	52	48	47	48	46	50	48	51	48	47	46
Retics %	1.0	5.0	1.8	1.0	1.0	2.0	1.8	1.6	1.5	2.0	1.6	2.0	0.5	1.0	0.5	1.5	1.5	1.0	2.5	0.5	3.0	0.5	1.5	1.0	0.8
Hb in grammes %	14.8	15.2	15.2	14.1	13.7	15.6	13.7	15.6	16.3	14.1	13.7	15.6	11.5	15.6	15.6	15.2	15.2	15.2	14.5	15.2	14.5	15.6	14.5	13.7	14.1

APPENDIX XI

IRON CONCENTRATIONS IN HEAD AND TAIL OF PANCREAS COMPARED

IRON CONCENTRATIONS IN HEAD AND TAIL OF PANCREAS COMPARED

NON-CIRRHOTICS					CIRRHOTICS				
Pancreas Ref. No.	Site	Iron Concentration mg/g. Wet Weight	Difference (Head minus Tail)	Histological Iron	Pancreas Ref. No.	Site	Iron Concentration mg/g. Wet Weight	Difference (Head minus Tail)	Histological Iron
1	Head	0.126	- 142	-	16	Head	1.900	- 1.107	+++
	Tail	0.268		-		Tail	2.107		+++
2	Head	0.110	+ 009	-	17	Head	5.200	- 477	+++
	Tail	0.101		-		Tail	5.677		+++
3	Head	0.080	+ 001	-	18	Head	2.952	- .054	+++
	Tail	0.079		-		Tail	3.006		+++
4	Head	0.091	- 004	-	19	Head	4.256	+ .006	+++
	Tail	0.095		-		Tail	4.250		+++
5	Head	0.272	- 010	+	20	Head	0.904	- .003	++
	Tail	0.282		+		Tail	0.907		++
6	Head	0.083	- 026	-	21	Head	0.084	+ .002	-
	Tail	0.109		-		Tail	0.082		-
7	Head	0.119	+ 033	-	22	Head	2.441	+ .621	+++
	Tail	0.086		-		Tail	1.820		+++
8	Head	0.112	- 075	-	23	Head	0.470	- .125	++
	Tail	0.187		-		Tail	0.595		++
9	Head	0.067	- 004	-	24	Head	1.142	+ .374	+++
	Tail	0.071		-		Tail	0.768		+++
10	Head	0.366	+ 002	+	25	Head	0.076	- .010	-
	Tail	0.364		+		Tail	0.086		-
11	Head	0.155	- 016	-	26	Head	0.133	+ .047	-
	Tail	0.171		-		Tail	0.086		-
12	Head	0.127	- 026	-	27	Head	0.703	- .142	++
	Tail	0.153		-		Tail	0.845		++

NON-CIRRHOTICS				
Pancreas Ref. No.	Site	Iron Concentration mg/g. Wet Weight	Difference (Head minus Tail)	Histological Iron
13	Head	0.120	+ 009	-
	Tail	0.111		-
14	Head	0.113	+ 017	-
	Tail	0.096		-
15	Head	0.047	- 029	-
	Tail	0.076		-
		Total Difference - 0.261		
		Mean Difference - 0.0174		
		t = 1.58 which is not significant		

CIRRHOTICS				
Pancreas Ref. No.	Site	Iron Concentration mg/g. Wet Weight	Difference (Head minus Tail)	Histological Iron
28	Head	1.306	+ .124	+++
	Tail	1.283		+++
29	Head	0.696	+ .010	++
	Tail	0.686		++
30	Head	1.944	+ 0.122	+++
	Tail	1.820		+++
		Total Difference - 0.621		
		Mean Difference - 0.041		
		t = 0.41 which is not significant		

APPENDIX XII

EFFECT ON GUINEA PIGS OF FEEDING THEM WITH AFRICAN BEER

The African beer used in this experiment was obtained from the police in the same manner as the samples analysed in Section V. The iron content was also estimated as described in Section V. Beer samples were obtained fresh every week.

Beer Sample No.	Iron Content (mg Iron/100 ml. Beer)
1	15.5
2	25.1
3	16.25
4	32.4
5	16.15
6	12.00
7	12.10
8	10.70
9	9.30
10	7.20
11	20.10
12	22.0
13	16.8
14	16.1
Mean Concentration	16.55

CONTROL GUINEA-PIGS

Guinea-Pig Number	Weight (grammes)	HISTOLOGICAL IRON						REMARKS
		LIVER			SPLEEN	DUODENUM	PANCREAS	
		H.C.	K.C.	P.A.				
1	822	-	-	-	++	-	-	
2	606	-	-	-	+	-	-	
3	553	-	-	-	++	-	-	
4	630	-	-	-	++	-	-	
5	648	-	-	-	+	-	-	
6	746	-	-	-	+	+	-	Several large areas of necrosis in liver. Lesions like Pasturella pseudotuberculosis.
7	555	-	-	-	++	-	-	Several small areas of necrosis in liver and spleen. Lesions like Pasturella pseudotuberculosis.
8	620	-	-	-	+	-	-	
9	636	-	-	-	++	-	-	
10	400	-	-	-	+	-	-	
11	653	-	-	-	+	-	-	
12	680	-	-	-	++	-	-	Necrotic areas in spleen - lesions like Pasturella pseudotuberculosis.
13	876	-	-	-	+	-	-	
14	882	-	-	-	++	-	-	
15	794	-	-	-	+	-	-	
16	634	-	-	-	+	-	-	
17	706	-	-	-	+	-	-	
18	710	-	-	-	++	-	-	
19	642	-	-	-	+	-	-	
20	672	-	-	-	++	-	-	

GUINEA-PIGS FED ON AFRICAN BEER

Guinea-Pig Number	Number of Days on Beer	Weight When Beer Commenced (grammes)	Weight at Time of Death (grammes)	HISTOLOGICAL IRON						Volume of Beer fed per Day (ml.)	REMARKS
				LIVER			SPLEEN	DUODENUM	PANCREAS		
				H.C	K.C.	P.A					
1	25	838	800	+	-	-	++	+++	-	42	Choked and inhaled beer.
2	40	721	543	+	++	+	+++	++	-	36	Areas of necrosis in liver and spleen - lesions like Pasturella pseudotuberculosis
3	40	641	597	+	+	+	+++	++	-	32	Areas of necrosis in liver - lesions like Pasturella pseudotuberculosis
4	40	623	513	+	+	-	+++	++	-	30	Hb. 16.0 G % P.C.V. 51 %
5	40	595	511	+	+	+	+++	++	-	30	
6	50	579	490	+	-	+	++	++	-	28	
7	50	601	507	+	+	-	+++	+++	-	30	
8	50	792	680	+	++	+	+++	++	-	40	
9	72	876	658	+	++	+	+++	++	-	44	Replaced No. 1
10	97	744	546	+	-	-	+++	++	-	36	
11	97	662	458	+	++	++	+++	++	-	33	
12	97	716	600	++	-	-	+++	++	-	35	
13	97	874	646	++	++	+	+++	++	-	44	
14	97	662	545	+	+	-	+++	+++	-	33	
15	97	848	575	++	+	-	+++	++	-	42	
16	97	766	574	++	+++	-	+++	++	-	38	
17	97	936	551	++	+++	+	+++	+++	-	46	Had an abscess of jaw which discharged and healed (duration 3 weeks).
18	97	918	741	++	+	+	+++	++	-	46	
19	97	804	596	++	+	-	+++	++	-	40	
20	97	434	405	+	+	-	+++	+++	-	22	

APPENDIX XIII

UPTAKE OF ^{59}Fe BY SLICES OF VARIOUS HUMAN TISSUES FROM TRANSFERRIN AT DIFFERENT PERCENTAGES OF SATURATION

Case No. 1 Sex M Age: 55 years

Nature of Operation: Oesophagectomy - carcinoma of oesophagus

Tissue Examined Liver

S.I. 111 µg/100 ml.

U.I.B.C. 138 µg/100 ml.

T.I.B.C. 249 µg/100 ml.

% Saturation: 44.5

Tube No.	Vol. of Serum	^{59}Fe Soln. 25µg ^{59}Fe/ml	Fe Soln. 28µg Fe/ml	^{59}Fe	Fe	Total Iron Added.	% Saturation
1	8 ml.	0.11 ml.		2.75 µg		2.75 µg	58.2
2	8 ml.	0.11 ml.	0.24 ml.	2.75 µg	6.72µg	9.47 µg	92.1

Tube No.	Tissue Weight	Counts/100 Seconds		Tissue Uptake of ^{59}Feµg/100mg Tissue
		4 ml. Tissue Solution	4 ml. Standard Soln. Containing 1.25µg ^{59}Fe	
1	77 µg	191	62,738	0.005
2	100 µg	866		0.017

REMARKS: T.I.B.C. low probably because of the malignancy and malnutrition.

Case No. 2 Sex M Age: 26 years

Nature of Operation: Splenectomy - mobile but otherwise normal spleen.

Tissue Examined: Liver

S.I.: 110 µg/100 ml.

U.I.B.C.: 288 µg/100 ml.

T.I.B.C.: 398 µg/100 ml.

% Saturation: 27.6

Tube No.	Vol. of Serum	^{59}Fe Soln. 30µg ^{59}Fe/ml	Fe Soln. 28µg Fe/ml	^{59}Fe	Fe	Total Iron Added	% Saturation
1	8 ml.	0.25 ml.		7.5 µg		7.5 µg	51.5
2	8 ml.	0.25 ml.	0.50 ml.	7.5 µg	14µg	21.5 µg	95.3

Tube No.	Tissue Weight	Counts/100 seconds		Tissue Uptake of ^{59}Feµg/100 mg Tissue
		4 ml. Tissue Solution	4 ml. Standard Soln. Containing 1.5µg ^{59}Fe	
1	10 mg	23	11,473	0.030
2	44 mg	364		0.108

REMARKS: Patient complained of vague abdominal pain - mass found in upper abdomen ? tumour. At laparotomy mass proved to be a very mobile spleen slightly enlarged (255 G) but otherwise normal. See also case No. 27

Case No. 3 Sex F Age 35 years

Nature of Operation Laparotomy - chronic gastric ulcer.

Tissue Examined Liver

S.I. 20 µg/100 ml.

U.I.B.C. 512 µg/100 ml.

T.I.B.C. 532 µg/100 ml.

% Saturation 3.8

Tube No	Vol. of Serum	^{59}Fe Soln. 30µg ^{59}Fe/ml	Fe Soln 28µg Fe/ml	^{59}Fe	Fe	Total Iron Added	% Saturation
1	8 ml.	0.65 ml.		19.3µg		19.5µg	49.6
2	8 ml.	0.65 ml.	0.65 ml	19.5µg	18.2µg	37.7µg	92.3

Tube No	Tissue Weight	Counts/100 Seconds		Tissue Uptake of ^{59}Feµg/100 mg Tissue
		4 ml. Tissue Solution	4 ml. Standard Soln. Containing 1.5 µg ^{59}Fe	
1	47 mg	587	28,810	0.061
2	35 mg	1576		0.234

REMARKS: Patient was suffering from slight iron deficiency anaemia.

Case No. 4 Sex F Age: 49 years

Nature of Operation Laparotomy ? carcinoma of stomach.

Tissue Examined Liver

S.I. 42 µg/100 ml.

U.I.B.C. 220 µg/100 ml.

T.I.B.C. 262 µg/100 ml.

% Saturation 16

Tube No	Vol. of Serum	^{59}Fe Soln. 30µg ^{59}Fe/ml	Fe Soln 28µg Fe/ml	^{59}Fe	Fe	Total Iron Added	% Saturation
1	8 ml	0.25 ml		7.5µg		7.5µg	51.9
2	8 ml	0.25 ml	0.30 ml	7.5µg	8.4µg	15.9µg	92.0

Tube No	Tissue Weight	Counts/100 Seconds		Tissue Uptake of ^{59}Feµg/100 mg Tissue
		4 ml. Tissue Solution	4 ml. Standard Soln. Containing 1.5 µg ^{59}Fe	
1	124 mg	447	29,847	0.018
2	108 mg	3375		0.157

Case No. 5 Sex F Age: 45 years

Nature of Operation: Laparotomy - bowel obstruction - adhesions old peritonitis.

Tissue Examined: Liver

S.I. 79 µg/100 ml.

U.I.B.C. 294 µg/100 ml.

T.I.B.C. 373 µg/100 ml.

% Saturation 21.2

Tube No.	Vol of Serum	^{59}Fe Soln. 30µg ^{59}Fe/ml.	Fe Soln. 28µg Fe/ml.	^{59}Fe	Fe	Total Iron Added	% Saturation
1	8 ml	0.29 ml		8.7 µg		8.7 µg	50.4
2	8 ml	0.29 ml	0.46 ml	8.7 µg	12.88 µg	21.58 µg	93.6

Tube No.	Tissue Weight	Counts/100 Seconds		Tissue Uptake of ^{59}Fe µg/100 mg Tissue
		4 ml. Tissue Solution	4 ml. Standard Soln. Containing 1.5 µg ^{59}Fe	
1	76 mg	186	16,638	0.022
2	100 mg	1892		0.171

Case No. 6 Sex M Age: 62 years

Nature of Operation: Oesophagectomy for carcinoma of oesophagus

Tissue Examined: Liver

S.I. 32 µg/100 ml.

U.I.B.C. 224 µg/100 ml.

T.I.B.C. 256 µg/100 ml.

% Saturation 12.5

Tube No.	Vol. of Serum	^{59}Fe Soln. 30µg ^{59}Fe/ml.	Fe Soln. 28µg Fe/ml.	^{59}Fe	Fe	Total Iron Added	% Saturation
1	8 ml	0.25 ml.		7.5 µg		7.5 µg	49.2
2	8 ml	0.25 ml.	0.32 ml.	7.5 µg	9.0 µg	16.5 µg	93.0

Tube No.	Tissue Weight	Counts/100 Seconds		Tissue Uptake of ^{59}Fe µg/100 mg Tissue
		4 ml. Tissue Solution	4 ml. Standard Soln. Containing 1.5 µg ^{59}Fe	
1	102 mg	194	21,923	0.013
2	75 mg	2064		0.188

REMARKS: See also Case No. 17

Case No. 7 Sex F Age: 45 years

Nature of Operation: Partial thyroidectomy - colloid goitre

Tissue Examined: Thyroid

S.I. 75 µg/100 ml.

U.I.B.C. 198 µg/100 ml.

T.I.B.C. 273 µg/100 ml.

% Saturation 27.5

Tube No.	Vol. of Serum	^{59}Fe Soln. 30µg ^{59}Fe/ml	Fe Soln. 28µg Fe/ml.	^{59}Fe	Fe	Total Iron Added	% Satu atio
1	8 ml	0.15 ml		4.5 µg		4.5 µg	48.0
2	8 ml.	0.15 ml.	0.35 ml.	4.5 µg	9.8 µg	14.3 µg	93.0

Tube No.	Tissue Weight	Counts/100 Seconds		Tissue Uptake of ^{59}Fe µg/100 mg Tissue
		4 ml. Tissue Solution	4 ml. Standard Soln. Containing 1.5µg ^{59}Fe	
1	90 mg	428	38,241	0.017
2	19 mg	847		0.175

REMARKS: Follicles are moderately large and distended with colloid

Case No. 8 Sex F Age: 31 years

Nature of Operation: Partial thyroidectomy - colloid goitre

Tissue Examined: Thyroid

S.I.: 123 µg/100 ml

U.I.B.C.: 208 µg/100 ml

T.I.B.C.: 331 µg/100 ml.

% Saturation: 37.2

Tube No.	Vol. of Serum	^{59}Fe Soln. 30µg ^{59}Fe/ml.	Fe Soln. 28µg Fe/ml.	^{59}Fe	Fe	Total Iron Added	% Saturation
1	8 ml.	0.15 ml		4.5 µg		4.5 µg	54.5
2	8 ml.	0.15 ml	0.35 ml	4.5 µg	9.8 µg	14.3 µg	92.3

Tube No.	Tissue Weight	Counts/100 Seconds		Tissue Uptake of ^{59}Fe µg/100 mg Tissue
		4 ml. Tissue Solution	4 ml. Standard Soln. Containing 1.5µg ^{59}Fe	
1	27 mg	2	15,879	0.000
2	91 mg	814		0.085

Case No. 9 Sex F Age: 21 yrs

Nature of Operation: Partial thyroidectomy

Tissue Examined: Thyroid

S.I.: 40 µg/100 ml

U.I.B.C.: 349 µg/100 ml

T.I.B.C.: 389 µg/100 ml.

% Saturation: 10.3

Tube No.	Vol. of Serum	^{59}Fe Soln. 30µg ^{59}Fe/ml.	Fe Soln. 28µg Fe/ml.	^{59}Fe	Fe	Total Iron Added	% Saturation
1	7 ml.	0.36 ml		10.8 µg		10.8 µg	49.8
2	7 ml.	0.36 ml	0.44 ml.	10.8 µg	12.32µg	23.12µg	95.1

Tube No.	Tissue Weight	Counts/100 Seconds		Tissue Uptake of ^{59}Fe µg/100 mg Tissue
		4 ml. Tissue Solution	4 ml. Standard Soln. Containing 1.5 µg ^{59}Fe	
1	58 mg	144	13,292	0.028
2	106 mg	1722		0.183

Case No. 10 Sex F Age 27 years

Nature of Operation: Partial thyroidectomy

Tissue Examined: Thyroid

S.I.: 99 µg/100 ml.

U.I.B.C.: 278 µg/100 ml.

T.I.B.C.: 377 µg/100 ml.

% Saturation: 26.3

Tube No.	Vol. of Serum	^{59}Fe Soln. 30µg ^{59}Fe/ml.	Fe Soln. 28µg Fe/ml	^{59}Fe	Fe	Total Iron Added	% Saturation
1	8 ml.	0.24 ml		7.2µg		7.2 µg	50.1
2	8 ml.	0.24 ml.	0.50 ml.	7.2µg	14 µg	21.2 µg	96.3

Tube No.	Tissue Weight	Counts/100 Seconds		Tissue Uptake of ^{59}Feµg/100 mg Tissue
		4 ml. Tissue Solution	4 ml. Standard Soln. Containing 1.5 µg ^{59}Fe	
1	175 mg	817	22,790	0.031
2	113 mg	1380		0.080

Case No. 11 Sex F Age 17 years

Nature of Operation Partial thyroidectomy : colloid goitre

Tissue Examined Thyroid

S.I. 133 µg/100 ml.

U.I.B.C. 341 µg/100 ml.

T.I.B.C. 474 µg/100 ml.

% Saturation 28.1

Tube No.	Vol. of Serum	^{59}Fe Soln. 30µg ^{59}Fe/ml.	Fe Soln. 28µg Fe/ml.	^{59}Fe	Fe	Total Iron Added	% Saturation
1	8 ml	0.28 ml		8.4 µg		8.4 µg	50.2
2	8 ml	0.28 ml.	0.55 ml	8.4 µg	15.4 µg	23.8 µg.	90.3

Tube No.	Tissue Weight	Counts/100 Seconds		Tissue Uptake of ^{59}Fe µg/100 mg Tissue
		4 ml. Tissue Solution	4 ml. Standard Soln. Containing 1.5 µg ^{59}Fe	
1	118 mg	364	20,588	0.022
2	123 mg	394		0.023

REMARKS: Thyroid follicles were very large and distended with colloid

Case No. 12 Sex F Age: 29 years

Nature of Operation: Partial thyroidectomy - colloid goitre

Tissue Examined: Thyroid

S.I.: 44 µg/100 ml.

U.I.B.C.: 351 µg/100 ml.

T.I.B.C.: 395 µg/100 ml.

% Saturation: 11.1

Tube No.	Vol. of Serum	^{59}Fe Soln. 30µg ^{59}Fe/ml.	Fe Soln. 28µg Fe/ml	^{59}Fe	Fe	Total Iron Added	% Saturation
1	8 ml	0.4 ml		12 µg		12 µg	49.1
2	8 ml	0.4 ml	0.5 ml	12 µg	14 µg	26 µg	93.4

Tube No.	Tissue Weight	Counts/100 Seconds		Tissue Uptake of ^{59}Feµg/100 mg Tissue
		4 ml. Tissue Solution	4 ml. Standard Soln. Containing 1.5 µg ^{59}Fe.	
1	123 mg	845	17,234	0.059
2	116 mg	2393		0.181

REMARKS: Follicles of goitre are small

Case No. 13 Sex F Age 20 years

Nature of Operation Partial thyroidectomy - colloid goitre.

Tissue examined Thyroid

S.I. 121 µg/100 ml

U.I.B.C. 204 µg/100 ml.

T.I.B.C. 325 µg/100 ml.

% Saturation 37.2

Tube No.	Vol. of Serum	^{59}Fe Soln. 30µg ^{59}Fe/ml.	Fe Soln. 28 µg Fe/ml.	^{59}Fe	Fe	Total Iron Added	% Saturation
1	8 ml	0.15 ml		4.5 µg		4.5 µg	54.5
2	8 ml	0.15 ml	0.35 ml	4.5 µg	9.8 µg	14.3 µg	92.3

Tube No.	Tissue Weight	Counts/100 Seconds		Tissue Uptake of ^{59}Fe µg/100 mg Tissue
		4 ml. Tissue Solution	4 ml. Standard Soln. Containing 1.5 µg ^{59}Fe	
1	103 mg	89	15, 879	0.008
2	104 mg	282		0.023

Case No. 14 Sex F Age: 14 years

Nature of Operation: Partial thyroidectomy - colloid goitre.

Tissue Examined: Thyroid

S.I.: 126 µg/100 ml.

U.I.B.C.: 296 µg/100 ml.

T.I.B.C.: 422 µg/100 ml.

% Saturation: 29.9

Tube No.	Vol. of Serum	^{59}Fe Soln. 30µg ^{59}Fe/ml.	Fe Soln. 28µg Fe/ml	^{59}Fe	Fe	Total Iron Added	% Saturation
1	8 ml.	0.23 ml.		6.9 µg		6.9 µg	50.2
2	8 ml.	0.23 ml.	0.53 ml.	6.9 µg	14.8 µg	21.74µg	94.3

Tube No.	Tissue Weight	Counts/100 Seconds		Tissue Uptake of ^{59}Feµg/100 mg Tissue
		4 ml. Tissue Solution	4 ml. Standard Soln. Containing 1.5 µg ^{59}Fe	
1	84 mg	245	16,638	0.026
2	80 mg	2141		0.241

REMARKS: Histologically follicles are small.

Case No. 15 Sex F Age: 42 years

Nature of Operation: Partial thyroidectomy - colloid goitre

Tissue Examined: Thyroid

S.I.: 117 µg/100 ml.

U.I.B.C.: 224 µg/100 ml

T.I.B.C.: 341 µg/100 ml.

% Saturation: 34.3

Tube No.	Vol. of Serum	^{59}Fe Soln. 30µg ^{59}Fe/ml.	Fe Soln 28 µg/Fe/ml.	^{59}Fe	Fe	Total Iron Added	% Saturation
1	8 ml.	0.14 ml		4.2 µg		4.2 µg	49.6
2	8 ml.	0.14 ml.	0.43 ml.	4.2 µg	12.04 µg	16.26µg	93.8

Tube No.	Tissue Weight	Counts/100 Seconds: 4 ml. Tissue Solution	Counts/100 Seconds: 4 ml. Standard Soln. Containing 1.5 µg ^{59}Fe	Tissue Uptake of ^{59}Fe µg/100 mg Tissue
1	50 mg	49	14,199	0.010
2	90 mg	874		0.103

REMARKS: Tissue taken appeared to be normal thyroid.

Case No. 16 Sex F Age: 35 years

Nature of Operation Laparotomy - partial gastrectomy

Tissue Examined Pancreas

S.I. 60 µg/100 ml

U.I.B.C. 266 µg/100 ml.

T.I.B.C. 326 µg/100 ml.

% Saturation

Tube No.	Vol. of Serum	^{59}Fe Soln. 30µg ^{59}Fe/ml.	Fe Soln. 28µg Fe/ml.	^{59}Fe	Fe	Total Iron Added	% Saturation
1	9 ml	0.3 ml		9.0 µg		9 µg	49.1
2	8 ml	0.3 ml	0.3 ml	9.0 µg	8.4 µg	17.4 µg	85.3

Tube No.	Tissue Weight	Counts/100 Seconds		Tissue Uptake of ^{59}Fe µg/100 mg Tissue
		4 ml. Tissue Solution	4 ml. Standard Soln. Containing 3.2 µg ^{59}Fe	
1	73 mg	228	41, 557	0.024
2	51 mg	701		0.105

REMARKS: There was a slight delay in tissue collection.

Case No. 17 Sex M Age: 62 years

Nature of Operation Oesophagectomy for carcinoma of oesophagus

Tissue Examined Pancreas

S.I. 32 µg/100 ml.

U.I.B.C. 224 µg/100 ml.

T.I.B.C. 256 µg/100 ml.

% Saturation 12.5

Tube No.	Vol. of Serum	^{59}Fe Soln. 30µg ^{59}Fe/ml	Fe Soln. 28 Fe/ml µg	^{59}Fe	Fe	Total Iron Added	% Saturation
1	8 ml	0.25 ml		7.5 µg		7.5 µg	49.2
2	8 ml	0.25 ml	0.32 ml	7.5 µg	9.0 µg	16.5 µg	93.0

Tube No.	Tissue Weight	Counts/100 Seconds		Tissue Uptake of ^{59}Fe µg/100 mg Tissue
		4 ml. Tissue Solution	4 ml. Standard Soln. Containing 1.5 µg ^{59}Fe	
1	174 mg	434	21,923	0.017
2	96 mg	7842		0.560

REMARKS: This is the same case as No.6

Case No. 18 Sex F Age: 40 years

Nature of Operation Excision of mixed parotid tumour

Tissue Examined Normal parotid gland

S.I. 96 µg/100 ml.

U.I.B.C. 272 µg/100 ml.

T.I.B.C. 368 µg/100 ml.

% Saturation 26.3

Tube No.	Vol. of Serum	^{59}Fe Soln. 30µg ^{59}Fe/ml.	Fe Soln. 28µg Fe/ml.	^{59}Fe	Fe	Total Iron Added	% Saturation
1	8 ml	0.24 ml		7.2 µg		7.2 µg	50.1
2	8 ml	0.24 ml	0.50 ml	7.2 µg	14 µg	21.2 µg	96.3

Tube No.	Tissue Weight	Counts/100 Seconds		Tissue Uptake of ^{59}Fe µg/100mg Tissue
		4 ml. Tissue Solution	4 ml. Standard Soln. Containing 1.5 µg ^{59}Fe	
1	104 mg	374	22,790	0.024
2	93 mg	1155		0.076

Case No. 19 Sex F Age: 55 years

Nature of Operation: Removal of mixed parotid tumour

Tissue Examined: Normal parotid gland

S.I. 65 µg/100 ml.

U.I.B.C. 247 µg/100 ml.

T.I.B.C. 312 µg/100 ml

% Saturation 20.8

Tube No.	Vol. of Serum	^{59}Fe Soln. 30 µg ^{59}Fe/ml.	Fe Soln 28µg Fe/ml.	^{59}Fe	Fe	Total Iron Added	% Saturation
1	8 ml	0.25		7.5 µg		7.5 µg	51.0
2	8 ml	0.25	0.42	7.5 µg	11.8 µg	19.3 µg	98.1

Tube No.	Tissue Weight	Counts/100 Seconds		Tissue Uptake of ^{59}Fe µg/100mg Tissue
		4 ml. Tissue Solution	4 ml. Standard Soln. Containing 1.5 µg ^{59}Fe	
1	67 mg	155	32, 736	0.011
2	64 mg	2790		0.200

Case No. 20 Sex F Age: 32 years

Nature of Operation Mitral valvotomy - mitral stenosis.

Tissue Examined Myocardium of auticular appendage

S.I. 45 µg/100 ml

U.I.B.C. 233 µg/100 ml

T.I.B.C. 278 µg/100 ml.

% Saturation 16.2

Tube No.	Vol. of Serum.	^{59}Fe Soln. 30µg ^{59}Fe/ml.	Fe Soln. 28µg Fe/ml	^{59}Fe	Fe	Total Iron Added	% Saturation
1	8 ml	0.25 ml		7.5 µg		7.5 µg	50.0
2	8 ml	0.25 ml	0.35 ml	7.5 µg	9.8 µg	17.3 µg	93.9

Tube No.	Tissue Weight	Counts/100 Seconds		Tissue Uptake of ^{59}Fe µg/100 mg Tissue
		4 ml. Tissue Solution	4 ml. Standard Soln. Containing 1.5 µg ^{59}Fe	
1	125 mg	264	26,292	0.012
2	139 mg	1769		0.073

Case No. 21 Sex M Age: 50 years

Nature of Operation: Partial gastrectomy for peptic ulcer

Tissue Examined: Smooth muscle from duodenal wall

S.I.: 60 µg/100 ml

U.I.B.C.: 245 µg/100 ml

T.I.B.C.: 305 µg/100 ml.

% Saturation: 19.7

Tube No.	Vol. of Serum	^{59}Fe Soln. 30µg ^{59}Fe/ml.	Fe Soln. 28µg Fe/ml.	^{59}Fe	Fe	Total Iron Added	% Saturation
1	8 ml	0.25 ml		7.5 µg		7.5 µg	50.5
2	8 ml	0.25 ml	0.35 ml	7.5 µg	9.8 µg	17.3 µg	90.5

Tube No.	Tissue Weight	Counts/100 Seconds		Tissue Uptake of ^{59}Fe µg/100 mg Tissue
		4 ml. Tissue Solution	4 ml. Standard Soln. Containing 1.5 µg ^{59}Fe	
1	27 mg	194	20, 918	0.053
2	36 mg	300		0.060

Case No. 22 Sex M Age: 30 years

Nature of Operation Repair of traumatic rupture of bowel

Tissue Examined Rectus abdominus muscle

S.I. 60 µg/100 ml.

U.I.B.C. 245 µg/100 ml.

T.I.B.C. 305 µg/100 ml.

% Saturation 19.7

Tube No.	Vol. of Serum	^{59}Fe Soln. 30µg ^{59}Fe/ml.	Fe Soln. 28µg Fe/ml	^{59}Fe	Fe	Total Iron Added	% Saturation
1	8 ml	0.25 ml		7.5 µg		7.5 µg	50.5
2	8 ml	0.25 ml	0.35 ml	7.5 µg	9.8 µg	17.3 µg	90.5

Tube No.	Tissue Weight	Counts/100 Seconds		Tissue Uptake of ^{59}Feµg 100 mg. Tissue
		4 ml. Tissue Solution	4 ml. Standard Soln. Containing 1.5 µg ^{59}Fe	
1	69 mg	128	18.407	0.015
2	102 mg	837		0.068

Case No. 23 Sex M Age: 35 years

Nature of Operation: Thoracotomy for empyema

Tissue Examined: Latissimus dorsi muscle

S.I. 121 µg/100 ml.

U.I.B.C. 204 µg/100 ml.

T.I.B.C. 325 µg/100 ml.

% Saturation 37.2

Tube No.	Vol. of Serum	^{59}Fe Soln. 30µg ^{59}Fe/ml.	Fe Soln. 28µg Fe/ml	^{59}Fe	Fe	Total Iron Added	% Saturation
1	8 ml	0.15 ml		4.5 µg		4.5 µg	54.5
2	8 ml	0.15 ml	0.35 ml	4.5 µg	9.8 µg	14.3 µg	92.3

Tube No.	Tissue Weight	Counts/100 Seconds		Tissue Uptake of ^{59}Fe µg/100 mg. Tissue
		4 ml. Tissue Solution	4 ml. Standard Soln. Containing 1.5 µg ^{59}Fe	
1	119 mg	280	18,237	0.019
2	114 mg	420		0.030

Case No. 24 Sex F Age: 35 years

Nature of Operation Laparotomy - chronic gastric ulcer.

Tissue Examined Rectus abdominus muscle

S.I. 20 µg/100 ml.

U.I.B.C. 512 µg/100 ml

T.I.B.C. 532 µg/100 ml.

% Saturation 3.8

Tube No.	Vol. of Serum	^{59}Fe Soln. 30µg ^{59}Fe/ml.	Fe Soln. 28µg Fe/ml	^{59}Fe	Fe	Total Iron Added	% Saturation
1	8 ml	0.45 ml		19.5 µg		19.5 µg	49.6
2	8 ml	0.45 ml	0.65 ml.	19.5 µg	18.2 µg	37.7 µg	92.3

Tube No.	Tissue Weight	Counts/100 Seconds		Tissue Uptake ^{59}Fe µg/100 g Tissue
		4 ml. Tissue Solution	4 ml. Standard Soln. Containing 3.2 µg ^{59}Fe	
1	76	547	28,810	0.038
2	155	1491		0.050

REMARKS: Patient is same as Case No. 3

Case No. 25 Sex F Age: 20 years

Nature of Operation: Debridement of leg ulcer

Tissue Examined: Gastrocnemius muscle

S.I. 9 µg/100 ml.

U.I.B.C. 411 µg/100 ml.

T.I.B.C. 420 µg/100 ml.

% Saturation 2.1

Tube No.	Vol. of Serum	^{59}Fe Soln. 30µg ^{59}Fe/ml.	Fe Soln. 28µg Fe/ml	^{59}Fe	Fe	Total Iron Added	% Saturation
1	8 ml	0.55 ml		16.5 µg		16.5 µg	51.2
2	8 ml	0.55 ml	0.55 ml	16.5 µg	15.4 µg	31.9 µg	97.1

Tube No.	Tissue Weight	Counts/100 Seconds		Tissue Uptake of ^{59}Fe µg/100 mg Tissue
		4 ml. Tissue Solution	4 ml. Standard Soln. Containing 1.5 µg ^{59}Fe	
1	70	1181	29,847	0.085
2	44	984		0.112

REMARKS: Patient was suffering from severe malnutrition and iron deficiency anaemia.

Case No. 26 Sex F Age: 52 years

Nature of Operation: Laparotomy - carcinoma of stomach

Tissue Examined: Liver

S.I.: 10 µg/100 ml

U.I.B.C.: 274 µg/100 ml.

T.I.B.C.: 284 µg/100 ml.

% Saturation: 3.5

Tube No.	Vol. of Serum	^{59}Fe Soln. 30µg ^{59}Fe/ml.	Fe Soln. 28 µg Fe/ml.	^{59}Fe	Fe	Total Iron Added	% Saturation
1.	7 ml	0.30 ml		9 µg		9.0 µg	49.0
2	7 ml	0.30 ml	0.33 ml	9 µg	9.24 µg	18.24 µg	95.4

Tube No.	Tissue Weight	Counts/100 Seconds		Tissue Uptake of ^{59}Fe µg/100 mg Tissue
		4 ml. Tissue Solution	4 ml. Standard Soln. Containing 1.5 µg ^{59}Fe	
1	64 mg	503	30,964	0.038
2	36 mg	17,419		2.35

REMARKS: Patient was suffering from severe iron deficiency anaemia.

Case No. 27 Sex M Age: 26 years

Nature of Operation: Splenectomy - mobile spleen

Tissue Examined: Spleen

S.I.: 110 µg/100 ml

U.I.B.C.: 288 µg/100 ml

T.I.B.C.: 398 µg/100 ml

% Saturation: 27.6

Tube No.	Vol. of Serum	^{59}Fe Soln. 30µg ^{59}Fe/ml.	Fe Soln. 28 µg Fe/ml.	^{59}Fe	Fe	Total Iron Added	% Saturation
1	8 ml	0.25 ml		7.5 µg		7.5 µg	51.5
2	8 ml	0.25 ml	0.50 ml	7.5 µg	14 µg	21.5 µg	95.3

Tube No.	Tissue Weight	Counts/100 Seconds		Tissue Uptake ^{59}Fe µg/100 mg Tissue
		4 ml. Tissue Solution	4 ml. Standard Soln. Containing 1.5 µg ^{59}Fe	
1	130 mg	52	11,473	0.005
2	156 mg	201		0.015

REMARKS: Same patient as Case No.2