"Haemosiderosis and Haemochromatosis in South African Natives with a Comment on the Etiology of Haemochromatosis"

Thesis submitted for the M. D. degree of the University of Glascow

by

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INTRODUCTION.

Haemosiderosis, the deposition of haemosiderin in various organs of the body e.g. liver, spleen, lymph glands and kidneys, may be the result of processes associated with anaemia or may be one feature of the condition known under the name of haemochromatosis.

Haemosiderin is a brownish or yellowish brown pigment of granular character containing "free iron", as can readily be demonstrated by its reaction with reagents like the combination of potassium ferrocyanide and hydrochloric acid.

As its name indicates, this pigment, at least under the majority of pathological conditions, is derived from the iron-containing moiety of the haemoglobin of the erythrocytes. Neumann, quoted by Wells (/.), maintains that haemosiderin is formed only under the influence of living cells and in the presence of oxygen: but Brown, also quoted by Wells (/.), has shown that haemosiderin may be formed in the liver during autolysis of that organ, especially if air be present.

A pigment, indistinguishable from haemosiderin, can also be formed during autolysis, from the iron-containing protein of cells, quite independent of haemoglobin. Milner, quoted by Wells (/.), states that during autolysis yet another iron-containing pigment may be formed, which differs from haemosiderin in having the iron so combined that it cannot react with the usual reagents, and that that pigment may later change into haemosiderin. This observation may be of importance in relationship to haemochromatosis and the pigment haemofuscin.

Haemochromatosis was the name given by von Recklinghausen to a condition, in which the organs and tissues throughout the body are infiltrated with two pigments, one iron-containing identical with haemosiderin, the other, known as haemofuscin which he described to be non-iron-containing. Subsequent work to which reference will be made later, would seem to indicate that haemofuscin also contains iron, though in masked form and that this pigment may ultimately be converted into haemosiderin, particularly in certain organs and more especially in the liver.

Shaw Dunn, in opening the discussion on haemochromatosis which took place at the eighty-ninth annual meeting of the British Medical Association, and which is reported in the British Medical Journal, 1921, Vol. ii., does not mention haemofuscin as an essential characteristic of the condition.

The description he gives is as follows.

> "A peculiar morbid condition, characterised by the accumulation of free iron-containing pigment in certain parenchymatous organs and in the skin, associated with interstitial fibrosis of the liver and pancreas; in a certain number of cases glycosuria develops.
>
> The skin especially of the exposed parts of the body exhibits a brownish or greyish brown pigmentation.
>
> The liver is more or less cirrhotic but is usually not much if at all diminished in size.
>
> The liver has a peculiar rusty colour which may be fairly homogeneous or may be paler in small rounded nodules, which represent hyperplastic liver tissue. When tested for the presence of free iron in a mixture of potassium ferrocyanide and hydrochloric acid, the liver gives an intense Prussian blue reaction.
>
> The pancreas shows, a rusty colour similar to that of the liver, though less marked, and also gives an intense reaction for free iron.

The/

The retro-peritoneal lymphatic glands in the upper part of the abdomen are more intensely rusty in colour than s/ ~~in~~ the liver and are laden with iron pigment.

Most of the other organs although they may not be obviously pigmented, give the iron reaction on testing.

Spleen reacts intensely, the heart, lungs, stomach and thyroid may show a fairly deep colouration; the kidneys usually react rather faintly as do the intestines and the skin.

In cases where the bone marrow is examined, it is usually observed that there is no special overgrowth of the red marrow, the condition being in marked contrast with that observed in pernicious anaemia and other haemolytic conditions in which free iron is deposited in the viscera."

In a series of autopsies, carried out at the General Hospital, Johannesburg, several cases, occurring in natives, presented a picture closely resembling that given above, but naturally the pigmentation of the skin was masked by its normal colour; in advanced cases however, pigmentation of the palate and of the mucous membrane of the mouth was observed, the pigment giving the iron reaction.

This has been reported in European cases also (2.).

In addition to these cases in which the picture was complete a considerable number of others showed gross rusty pigmentation of liver,

spleen and upper retro-peritoneal lymph glands, without the generalised deposit in the other organs such as pancreas, heart, thyroid gland, supra-renal glands, stomach and skin.

The incidence of this haemosiderosis was so striking and relatively so common that a routine examination for haemosiderin was made in almost every native autopsy. This was done even when there was no obvious sign macroscopically of iron pigmentation.

The method adopted for testing was that indicated by Shaw Dunn as mentioned above - a mixture of potassium ferrocyanide and hydrochloric acid (equal quantities of 4% solutions); this was used only in the cold, as heat gives usually a more intense reaction from the liberation of loosely combined iron.

As autolysis might have been considered to play a part in the production of the free iron, tests were carried out on Europeans as well as Natives and note taken as to the periods which elapsed between the time of death and the time of autopsy; from the results obtained it was obvious that post-mortem autolysis did not play a part in the production of the haemosiderosis. The reaction when obtained as the result of

autolysis/

autolysis, differs from that found in true haemosiderosis; the latter gives a sharply defined Prussian blue reaction while the former gives a discrete diffuse greenish colouration.

Bacteria of certain types are also said to play a part in the production of haemosiderosis, but an investigation into the bacterial flora of livers obtained post - mortem showed such inconstancy and variability in the results that this line of investigation was considered of little value in any attempt to elucidate the condition.

In appendix i the results of the investigation into the incidence of haemosiderosis in natives, are tabulated and also the cause of death in each case.

The results for Europeans are not tabulated as the number of positive cases is relatively scanty; a few cases of haemochromatosis among Europeans will be mentioned when the discussion of that condition is undertaken.

In the literature which is available in Johannesburg, no reference was found to the occurrence of haemochromatosis in natives, yet as is seen in appendix i, the tendency to the development of this condition may be

considered very marked.

Some explanation of this phenomenon has been sought in the hope that light might be thrown on the etiology of the condition.

Investigations have been made along several different lines. One of the first to suggest itself was that of diet. The diet of the native differs considerably from that of the European, both in type and in mode of preparation.

The details of the diet and their importance are discussed in Section III.

Another line of investigation concerned the fragility of the erythrocytes. Roque, Chalier and Nove' Josseraud (3.) in France and Howard and Stevens (4.) in America report excessive fragility of the red blood corpuscles in their cases of haemochromatosis. These reports indicated that an investigation into the resistance of the erythrocytes to hypotonic saline might prove of value.

A series of tests was carried out on two hundred natives, presumably normal, or at least not suffering from any serious disability which might invalidate the test. The method adopted in this investigation is described and the results, which are shown in appendix (ii) are discussed in Section IV.

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Though Shaw Dunn and others lay little emphasis

on the occurrence of haemofuscin as an essential feature of haemochromatosis, Mallory (5.) insists on its importance. He gives as his interpretation of the sequence of events, that the pigment haemofuscin, originally named and described by von Recklinghausen in 1889, is first deposited in the endothelium lining the sinusoids and in the parenchymatous cells of the liver and that subsequently it is changed to haemosiderin by a slow process of transformation involving months or years; the other features of the disease occur subsequent to this.

The interpretation implies pigmentation of the liver before cirrhosis.

The results obtained and recorded in appendix (i) showed that pigmentation without obvious cirrhosis was not uncommon amongst natives. In the light of this, it seemed advisable towards the end of the whole investigation to carry out a series of examinations on consecutive cases for the presence of haemofuscin in the liver.

The results of these examinations are tabulated in appendix iii and are discussed in Section ii.

Experimentally pigment cirrhosis and conditions resembling haemochromatosis have been produced in

animals/

animals by chronic copper poisoning (5) (8) and by injections of relatively enormous quantities of homologous citrated blood (10a). In the investigations outlined above it appeared that neither of these could adequately account for the incidence of haemosiderosis observed among natives.

During the investigation of the diet and the modes of preparation of the food, it appeared that metals other than copper might be factors particularly zinc and tin. A few experiments were carried out on rabbits with these metals; zinc, tin and copper salts were used; in some cases these were administered alone, in other cases associated with alcohol. The details of these experiments are shown in appendix IV and the results are discussed in Section V.

In Section VI the problem is considered and discussed as a whole in the light of the sum of these investigations.

SECTION II.

Incidence of the conditions to be discussed.

In Section I, reference was made to the fact that pigmentation of the organs of natives, coming to autopsy, was observed to occur relatively frequently and consequently a routine investigation of the incidence of haemosiderosis was set on foot.

In the year 1924, one case of haemochromatosis occurred, it was associated with diabetes and diabetic gangrene; the patient was a native woman, aged 50. In the same year there were in addition four cases of pigment cirrhosis, which did not show all the features of haemochromatosis. These five cases were the only five tested that year, in which two hundred and twenty-four native autopsies were performed.

These two hundred and twenty-four with the eight hundred and seventy-six recorded in appendix i make a total of eleven hundred. On the last page of the appendix are collected thirty-two cases of pigmentation of an extent and degree usually found in haemochromatosis. Thus in this series of eleven hundred natives, there were observed thirty-three cases of iron pigmentation of a degree comparable with that of haemochromatosis . Of these cases,

twelve/

twelve occurred in females and twenty-one in males.

Though accuracy in age determination among natives is not to be expected, the ages given closely approximate to the estimated age. This must be taken into account when the age incidence of this severe degree of pigmentation is being considered. With this qualification it may be stated that.

5	cases	occurred	between	the	ages	of	30-35 years
5	"	"	"	"	"	"	35-40 "
10	"	"	"	"	"	"	40-50 "
7	"	"	"	"	"	"	50-60 "
6	"	"	"	"	"	"	60-70 "

In 1924 there was 1 case

In 1925 there were 2 cases

In 1926 there were 12 cases

In 1927 there were 9 cases

In 1928 there were 9 cases

During the years 1925-1928 the test for haemosiderosis was applied systematically in seven hundred and forty-five cases. In addition to the cases of haemochromatosis mentioned above, haemosiderin was found in the liver, spleen and lymph glands in three hundred and sixty-eight cases. Of the seven hundred and forty-five cases, four

hundred and twenty-three were over thirty years of age. Of the three hundred and sixty-eight cases of haemosiderosis three hundred and ten were over thirty years of age. If to the cases of haemosiderosis are added those of haemochromatosis, a total of three hundred and forty two cases showing definite iron pigmentation is obtained out of the four hundred and twenty three that were over thirty years of age; i.e. in eighty per cent of these autopsies, obvious haemosiderin occurred.

Of the cases of haemosiderosis, one hundred and fifty-one were associated with tuberculosis; of these one hundred and twenty-one were over thirty years of age; of the cases of haemochromatosis eight were associated with tuberculosis.

The total number of cases of tuberculosis was three hundred and thirty-seven; sixteen of these occurred in natives under five years, an incidence of 17%, ninety-five occurred between the ages of five and thirty years - an incidence of 23.4%, and two hundred and twenty-six occurred over thirty years, an incidence of 35%.

Tuberculosis in the native has a distribution which closely resembles that found in the experimental

animal./

animal. Involvement of the spleen is exceedingly common and this organ may reach several pounds in weight and be the seat of multiple caseous foci of varying sizes. This involvement of the spleen is not infrequently associated with the occurrence of tubercles in the liver, and where haemosiderosis is well-marked the caseous nodules stand out sharply from the rusty pigmented substance of the liver. In addition where the upper retro-peritoneal lymphatic glands are involved the caseous process stands out against the rusty back ground of the pigmented gland.

In some cases of tuberculo-silicosis - a peculiar form of fibroid tuberculosis associated with silicosis which in the lung assumes initially an islet character similar to that of simple silicosis - the upper retro-peritoneal glands may be the seat of pigmentation and fibrosis due to the combined presence of tuberculosis and silica; in addition pigmentation from haemosiderin may also be present.

It would appear from sections of tissues showing the occurrence of tuberculosis and haemosiderosis or of tuberculosis, silicosis and haemosiderosis, that the iron pigmentation had been the primary condition and that tuberculosis had been superimposed. In other words that although tuberculosis

may be found associated with haemosiderosis in 50% of the cases of haemosiderosis under thirty years of age and in 37% of cases over thirty years of age, it does not necessarily follow that tuberculosis is an important etiological factor in the production of the haemosiderosis. It has been stated that erythrophagocytosis is increased in tuberculosis (12.), but such erythrophagocytosis could not produce the degree of haemosiderosis which has been observed. If tuberculosis were an important factor in the production of the iron pigmentation, then one would expect to find it in native children as marked as in adults, but iron pigmentation in children is conspicuous by its absence. The type of tuberculosis found im children and in adults in the majority of cases is closely similar - apart from the cases where silicosis and tuberculosis are found together. Only rarely in about five per cent of cases does one find evidence of old healed arrested tuberculosis, in the form of a calcareous gland or calcareous focus in the lung; the vast majority of cases of all ages show even on careful search no evidence of chronic tuberculosis:- cases of

of bone tuberculosis are in a class apart as some of these show chronic changes but in the material dealt with at the autopsies only seven of this type occurred in a total of three hundred and thirty seven.

A further fact which suggests that tuberculosis is not a factor in the haemosiderosis is that the organs in Europeans who died of acute tuberculosis did not give a positive reaction for haemosiderin in all three tissues - liver, spleen and lymph glands; the spleen might give a positive reaction and the liver show a trace but among Europeans, haemosiderosis in the sense used in this investigation, did not occur as the result of tuberculosis.

Another disease which commonly affects the South African native is schistosomiasis; infestations with Schistosoma haematobium and Schistosoma mansoni are not infrequent. Ninety-six cases of obvious bilharzia were noted in this series. It would seem, however, that the incidence of this condition is even greater than is borne out by the record of cases with obvious lesions; in the routine histological examination of the liver for haemosiderin and haemofuscin pigments, a number of specimens showed considerable deposits of bilharzia pigment even when no bilharzial lesion had been noted in the organs at autopsy. One of the manifestations of this disease

amongst the natives is an enlargement of the spleen; the enlargement may be of such a degree that the organ weight is two to three pounds. In the enlarged spleen ova of both S.haemotobium and S. mansoni have been found, thus bringing this type of splenomegaly into line with that of the Egyptian splenomegaly described by Ferguson (13.) and Day (14.).

Incidentally it may be mentioned that ova of the Schistosoma have been found in lung, liver, pancreas, spleen, omentum, appendix, fallopian tube, kidney, ureter as well as in the bladder and in the large intestine. Consequently it would appear that in certain cases of schistosomiasis there may be a general stimulation of the reticulo-endothelial system and this may play a part in the production of haemosiderosis.

Increase in erythrophagocytosis has been reported in certain, diseases, particularly tuberculosis, typhoid fever, pneumonia and in some cases of cirrhosis of the liver.(12) In this series of cases, seventy-eight died of typhoid fever or of one of its complications; in a proportion of these haemo-siderosis was present. There were one hundred and one cases of lobar pneumonia, five of which were associated with haemochromatosis.

In addition to the thirty-three cases of haemochromatosis in which cirrhotic changes of

varying/

varying degree occurred in the liver, forty-four cases of obvious cirrhosis were noted i.e. there were seventy-seven cases showing macroscopic cirrhotic changes. Of these seventy-seven, two were under twenty years of age and a further ten under thirty years of age.

The routine histological investigation of the liver in natives indicates however that early cirrhotic changes are much commoner than would appear from the naked eye examination. Cellular infiltrations in and around the portal tracts are an almost unvarying feature of the histological picture and this cellular infiltration is almost identical with that seen as the earliest manifestation of cirrhotic changes in the experimental animal.(31) (32)

A further indication that the liver is commonly the seat of change in the native is seen in the relative incidence of carcinoma. The number of primary carcinomata observed was forty-one in the series; of these, twenty-two were primary carcinomata of the liver, nineteen occurred in males and three in females. The sites of the other primary carcinomata were - oesophagus 3, thyroid 3, stomach 3, pancreas 2, breast 2, colon 2, bladder 2, skin 1, ovaries 1.

Of the carcinomata of the liver, three were associated with haemochromatosis and sixteen with cirrhosis of the liver.

De Vogel, quoted by Mallory (6.) refers to the high frequency of carcinoma of the liver in the Javanese and Chinese and states that ninety per cent of such carcinomas were found in cirrhotic livers. A similar relationship is found in the South African native.

Though only twenty-two cases of primary carcinoma of the liver occurred in the hospital autopsies during the years 1924-1928, forty-five other cases were reported in 1926 and 1927 from material sent to the South African Institute for Medical Research for examination. (Vide Reports for 1926 and 1927).

Metastases were noted in a few cases in the regional lymph glands and in the lungs.

Other diseases which may be of importance in relationship to haemosiderosis - being associated with activity of the reticulo-endothelial system - are malaria, typhus, amoebiasis, scurvy and the anaemias. No case of typhus came to post-mortem. Of malaria there were six cases of cerebral type. Twenty-four cases occurred of amoebiasis, the majority showing liver involvement.
Eight cases of scurvy were noted and in six this was the cause of death.

As regards the anaemias and leukaemias, though clinical records and clinical pathological examinations show that these do occur, only one case of aplastic anaemia and one case of myelogenous leukaemia reached autopsy.

Diseases numerically important among the causes of death were endocarditis (all varieties, fifty-five cases) and nephritis (all varieties, forty-two cases).

Aschoff,(15) in a lecture on atherosclerosis indicates that atheromatosis may be found in childhood, at puberty, and in senescence, and maintains that this condition is associated with an increase of cholesterin or cholesterin esters in the circulating plasma; in childhood and at puberty the condition is a reversible one and may disappear, but in the atheromatosis of senescence an irreversible condition is established. One of the manifestations in the childhood type, is a lesion of the septal curtain of the mitral valve. This type of lesion has been found in native children, e.g. in a child of three dying of acute infection. Similar lesions have been found however in the mitral valve at all ages; practically no native heart is free from it and a considerable number of Europeans of all ages show the same lesion.

The site of the lesion is commonly the seat of calcification in the later years of life.

Degenerative changes of atheromatous type are the rule in the arterial system of the natives, and the atheromatosis corresponds to the senescent form described by Aschoff. This lesion may be diffuse in individuals just over thirty years of age, and is usually gross in the sixth decade of life. In every case that reaches the age of sixty years calcification is always present, in the aorta or in the iliac and femoral vessels. Calcification may occur much earlier than this both in Europeans and in natives, for example a large calcified plaque was seen in the case of a European female, (South African born) aged twenty-one, who died from meningitis as the result of an accident; this plaque occurred in the ascending aorta just above the aortic cusps.

The relationship of diet and of climatic conditions, such as excessive sunlight, to this calcification has yet to be investigated; it is suggestive however that ergosterol in large doses may produce calcification in the experimental animal.(16)

If, as Aschoff maintains, atheromatosis, the infiltration of the intima with cholesterin esters, cannot occur without the presence in the blood plasma of

increased/

increased cholesterin substances and if the cholesterin metabolism is controlled by the liver, which is the accepted view, then it would follow from the incidence of atheromatosis in natives that some change, possibly functional but presumably organic was present almost constantly in the liver of the native.

As regards syphilitic lesions, these have not been noted to any large extent in spite of the fact that more than twenty five per cent of natives tested gave a positive Wassermann reaction.

In the discussion on haemochromatosis previously referred to, the importance of haemofuscin as an essential feature of the condition was discounted; emphasis was laid on the deposit of haemosiderin while haemofuscin was considered as an intermediate stage between haemoglobin and haemosiderin. Towards the end of this investigation it appeared that haemofuscin was not a disintegration product of haemoglobin but rather a combination of haemoglobin with other chemical substances.

This necessitated a reconsideration of the importance of haemofuscin and required an examination into its incidence in the livers of natives. Only a short series of examinations was made in consecutive cases coming to post-mortem. Between forty and fifty cases were examined. The chief test applied was that suggested

by/

by Mallory - the differential staining of the pigment with an alcoholic solution of basic fuchsin; haemosiderin does not stain with the basic fuchsin and haemofuscin does not give the reaction for free iron with hydrochloric acid and potassium ferrocyanide. (5) (7)

The tests were applied in all cases to sections of the liver and in a few cases to sections of spleen and of kidney.

Haemofuscin was found in appreciable amount in twenty-five cases, and in traces in the remainder with the exception of three which were negative. In practically all the positive cases the haemofuscin was present associated with haemosiderin in the same cells; in one case of cirrhosis, in the regenerating areas haemofuscin was present alone; in one case without cirrhosis haemofuscin was associated with only very small traces of haemosiderin.

Though this series is small the results suggest that the deposition of haemofuscin is relatively common in the liver of the South African Native.

As regards the age incidence, several of the cases showing appreciable quantities of the pigment were only twenty years of age. Appendix i shows that haemosiderin deposit in the liver is very common even at twenty years of age, though deposits in the other organs may not be appreciable. The age

incidence/

incidence suggests a factor which acts early in life and continues more or less constantly.

The occurrence of cases showing mainly haemofuscin deposit and of cases showing the association of haemofuscin with haemosiderin in the same cells (the transformation of haemofuscin to haemosiderin) supports the contention that the deposit of haemofuscin is antecedent to the haemosiderosis. The rate of transformation of haemofuscin to haemosiderin may vary in different cases and this may account for the different pictures obtained.

D I E T

S E C T I O N III

Most authorities consider that haemochromatosis is a condition which requires years for its development, so that if diet plays any part in its production, that diet must have been adopted for a similarly long period. In this respect the diet of the native in his kraal must assume some considerable importance. In type it is the diet which dominates his life, even when the native is in towns, the modifications there being mainly in the type of alcohol he consumes.

An investigation into the diet of the South African Native in his kraal was carried out some twenty years ago by Dr. G.A.Turner, then Medical Officer to the Witwatersrand Native Labour Association, Johannesburg. The report of this investigation is to be found in volume four of the Transvaal Medical Journal.

No apparent change has taken place in the natives' diet nor in his habits since that time so that the comments made by Dr. Turner are still worthy of consideration. Some extracts from the articles in the Medical Journal may be quoted, as they tend to throw some light on the incidence of disease or its

causation/

causation amongst the natives, and as they may also be relevant to the subject of haemosiderosis.

> "The native is proverbially improvident, he has to contend with erratic seasons and consequently it often happens that he has only a very indifferent diet upon which to exist."
>
> "Their improvidence combined with a constitutional inability to commence work except under compulsion causes the natives in many parts at certain seasons of the year to deteriorate physically."
>
> "Though the natives are able to endure hardships on a limited food supply, it has been found on several occasions that if after privation, they are given muscular labour, even when supplied with a good nutritious diet, they are liable within the first few months of commencing work to develope scurvy."
>
> "The natives of South Africa have become accustomed to a diet containing a higher percentage of carbohydrates than is found amongst European races."

Meat does not enter into the every-day diet, it is a luxury reserved for special occasions; eggs are not a popular form of food and fish is reckoned unclean by most tribes. The staple form of diet is of a 'vegetarian' type, although on rare occasions the natives at times will eat enormous quantities of meat. Even in the towns the native still adheres to this type of diet.

Though Dr. Turner mentions a great number of carbhydrate foods that may be used by the natives, e.g. mealies, potatoes, sweet potatoes, sugar cane, pumpkins,

avocadopears/

avocadopears, paw-paws, pineapples, prickly pears, etc., yet, so far as the natives are concerned upon whom this haemosiderosis investigation was carried out, almost the only carbohydrate food used is the mealie - a variety of the zea mays -. The mealie may be cookedwhole or may be ground for the preparation of mealie porridge, etc.

The fats of the diet were at one time derived mainly from milk, usually taken sour and often precipitated, but where milk is not available, vegetable fats may be employed, derived for example from nuts, or animal fats obtained at ceremonial times when meat is eaten. These fats are incorporated as a rule in the prepared mealies. Mealies contain a certain amount of protein (the meal contains 70% carbohydrate, 14% protein and 3.8% fat) and further proteins may be added to the diet from leguminous foods e.g. peas and beans and also from nuts.

As regards alcohol Dr. Turner states:-

> "The native races of South Africa generally, in common with other uncivilised people, are mentally incapable of resisting alcohol. Consequently in parts where spirits and wines are obtainable at all seasons, the native population is a drunken one from its childhood up."
>
> "On the other hand, in places where Kaffir Beer is the national drink, the native is by no means a drunkard in the true sense of the word; he doubtless at times consumes enormous quantities of beer ---- but he contents himself with these periodic bouts. Even this beer-drinking class,

however/

> however, gives way to drink when brought in contact with civilisation, unless kept more or less under close observation."

Dr. Peter Allan,(17) who is at present carrying out a tuberculosis survey in the Native Territories, informsme that there the diet consists almost entirely of mealies (3 pounds or more per day). These may be roasted whole or coarsely ground and made into porridge to which a little pumpkin or a few beans or peas may be added when available; animal fat may also be mixed into the porridge.

Meat is eaten at ceremonial times, once or twice a month. At these times the native indulges in kaffir beer orgies which may last for a week; the beer appears to have a highly intoxicating effect and the native eats nothing during the drinking bout, but when the bout is ended, he may eat enormous quantities of meat. Apart from ceremonial times the diet is essentially mealies.

Dr. Allan also confirms information that I have received that the native calabashes and cooking utensils are disappearing and being replaced by paraffin tins, petrol tins and tins of all sorts and sizes both for the storage of food and for its preparation. That this change had taken place in the towns has been a matter of common observation for years and it has also spread to the native territories.

Dr. Allan's communication to me indicates that there

has/

has been no change in the type of diet among the natives for the past twenty years and that Kaffir Beer is still as popular as ever with them.

Dr. Turner in his remarks on Kaffir Beer states that "it must be looked upon as the national drink of the Kaffirs and one universally appreciated by the Bantu race". It is made from various kinds of kaffir corn, a kind of millet (sorghum vulgaris). Mealies are also frequently employed when the other forms of grain are not available.

Properly made and used at the proper time, namely within a few hours of its preparation, the kaffir beer appears to be a valuable addition to the diet. One Missionary quoted by Dr. Turner says "It is in my view more than a luxurious and supererogatory beverage, it is rather a very admirable, very beneficial, even perhaps very necessary form of food". The suggested source of this "necessary form of food" is mentioned as being in the yeast which the liquor contains when drunk.

The mode of preparation of the kaffir beer resembles that of ordinary beer, but the various stages are not controlled as regards temperature, time, and materials. Consequently the type of fermentation produced differs from that of ordinary beer, and the end-product may show a well-marked acetous fermentation and contain a considerable quantity of acetic acid (18).

A malt is prepared by inducing the kaffir corn to germinate, and arresting the germination process by drying, preferably sun-drying. A certain amount of this malt is mashed with mealie meal in water, preferably hot water. The mass is allowed to stand in a covered vessel exposed to the rays of the sun for from one to three days. It then becomes a thick fermenting mass of sour-sweet taste with a distinctly putrid smell. This is regarded as a kind of mother wort.

Parts of this wort are added to boiling water and the whole boiled for the best part of a day, when it is put into vessels and allowed to cool for twenty-four hours. After this period of cooling a certain quantity of the kaffir corn/malt is now mashed into the wort to produce the main alcoholic fermentation. This is supposed to be at its height in twenty-four hours. Then the beer is strained into other vessels and is ready for consumption. If the beer is kept over for a day or so longer, the alcoholic content increases. The beer ought to be consumed as soon as prepared, for if stored at all it becomes more potent and more acid.

Dr. Klein (18) in an article on kaffir beer and its manufacture on the Rand, indicates the marked tendency for acetification to take place in the process just described. After quoting analyses of various specimens of the beer, which were made by the Government Analyst he shows that so far as the Rand is concerned the native " in order to feel satisfactorily seasoned will require/

require about 2,000 gms. of the beer which will give him the benefit of 50 gms. of acetic acid and 80 gms. of alcohol".

Even when properly prepared kaffir beer remains sweet only for about fifteen hours after its preparation, thereafter it becomes too acid and sour to be fit for human consumption. As prepared and drunk by the natives in and around Johannesburg, it is already too acid; even then its acidity is much increased by the addition of adulterants e.g. sulphuric acid.

Dr. Turner comments on this as follows:-

> "Unfortunately some of the native women are losing the art of making good beer or are too lazy to do so properly. To get over this failing and in some cases to increase the potency of the ordinary product they add adulterants. For the purpose some use cheap dop brandy or whisky and I have heard of paraffin, copper sulphate and even sulphuric acid being added."

Other types of alcohol and the methods of their preparation are noted in the appendices, where are copies of notes received from the Criminal Investigation Department, Johannesburg by Professor J.M.Watt of the Department of Pharmacology in the University of the Witwatersrand.

Formerly the preparation of food and drink was done in calabashes and native pots but nowadays these have been superseded to a large extent by the paraffin tin, the petrol tins and by galvanised receptacles.

Though/

Though the native is in many ways cleanly in his habits yet custom permits and sometimes demands that food and liquor be kept more or less continuously in the same receptacle, particularly in these processes where fermentation plays a part. Thus it happens that metal receptacles and utensils become corroded and the food contaminated by the corroded metals. Where tin receptacles are used, tin contamination will take place and it is obvious with an acid fluid like kaffir beer, this may be considerable. Similarly with zinc lined receptacles e.g. water tanks, which commonly are of galvanised metal. Thus, quite apart from any metallic content of the food as food, contaminations with zinc and tin are more or less constant.

That such contamination may be considerable can be readily appreciated in the light of the work of Sale and Badger (19). They investigated a series of cases of poisoning from bottled wort beer and found that the beer contained 229 milligramms of zinc per litre; this zinc content they presumed to be derived from galvanised pails in which the beer had been kept before bottling. They therefore investigated the rate at which various liquids dissolved the zinc from ordinary galvanised iron pails.

Some of their results may be quoted to show the

rapidity with which some fluids may dissolve this metal.

"Amount of zinc in mgms. per litre

	After 17 hours	After 41 hours
Tap Water	5	21
Distilled Water	9	27
Carbonated Water	193	181
Milk	438	1,054
Orangeade	530	854
Lemonade	1,411	2,700."

These results indicate that the native food and liquor contains appreciable metallic content from contamination.

Quite apart, however, from contamination, the native's diet does contain an appreciable quantity of zinc.

Bertrand and Beuger (20) as the results of their experiments show that zinc is an element of great physiological importance; it is found in appreciable quantities in substances rich in Vitamin B. They state that it is found in notable quantities in bran, yeast, butter, milk and cod liver oil, and that the organs richest in zinc are the liver, brain and heart.

Bertrand and Wakamara (21) maintain that from the physiological aspect zinc is more important to the organism than iron.

A certain minimal amount of zinc must therefore be ingested/

ingested in the food.

Bückner (22.) in an investigation into the zinc content of food reports the following quantities of zinc per thousand grammes.

"Tap water	0.25 mgm.
Yolk of egg	56.71 mgm.
Pea (dry)	34.53 mgm.
Yeast	414.86 mgm.
Corn(Zea Mays)	25.24 mgm.
Wheat Bran	139.2 mgm.
Wheat	84.0 mgm.
Bean	14.65 mgm.
Milk	4-5 mgm.
Precipitate of Cow's milk	674.3 mgm."

In a vegetarian type of diet, therefore, quite apart from contamination, appreciable amounts of zinc are ingested: if yeast made beers are associated with the diet, as in the case in some kaffir beers, the amount is much increased.

An investigation into the effect of long continued ingestion of zinc by cats and dogs, together with observations upon the excretion and storage of zinc has been carried out by Drinker, Thompson and Marsh (23.). From their work it would appear that absorbed zinc begins to be excreted promptly after its absorption. A small fraction leaves the body in the urine, the major amount however is excreted along the alimentary tract, some of it in the bile, probably some of it in the pancreatic juice. If the absorbed zinc is

greater/

greater in amount than the excretory mechanism can handle at once, zinc is temporarily stored, chiefly in the liver, and liberated by this organ as fast as it can conveniently be excreted. A small amount of zinc is excreted in sweat and probably some also in milk. (Both cow's milk and human milk contain appreciable quantities of zinc).

In their experiments three heavily zinced cats developed fibrosis of the pancreas with nodularity. Their observations have led these workers to conceive of the possibility that the pancreas may serve normally as one of the agents by which zinc or possibly other heavy metals, are excreted into the gastro-intestinal tract, and that the fibrosis observed may have resulted from an excessive demand upon the pancreas to exercise this function.

The organs involved in the storage and excretion of zinc viz. the liver, pancreas and skin, have some significance when considered in the light of the incidence of the pigmentation in haemochromatosis.

To sum up the salient features of the native diet, it is mainly vegetarian in type and is associated with an alcohol which degenerates rapidly to a crude form of vinegar; it is liable to contamination both with salts of tin and of zinc, and zinc is already present in the food in relatively large quantity.

It was presumed that these two metals might be factors in the incidence of haemosiderosis and of

haemochromatosis. The investigation into the relationship between zinc and tin feeding and the early changes associated with experimental pigment cirrhosis is dealt with in Section V.

FRAGILITY OF THE ERYTHROCYTES.

SECTION IV.

There are many tests whereby the resistence of the erythrocytes to varying strengths of hypotonic saline may be investigated. That employed in this series of examinations was originally elaborated by Simson (24), while working on certain types of anaemia and I am indebted to him for information as to the normal obtained over some five hundred examinations.

The ideal treatment of the blood to be examined is defibrination as soon as it is withdrawn from the patient, but as my specimens were collected several miles from the laboratory such treatment was impossible. Instead the blood was collected in a tube treated with potassium oxalate, only just sufficient of the anti-coagulant being present as was necessary to prevent clotting.

From 5 c.cs. to 10 c.cs. of blood were withdrawn from a vein, shaken up in the tube with the oxalate and sent to the laboratory where the specimens were received usually

within/

within one hour and never later than two hours after they had been taken.

The oxalated blood was then washed in six changes of saline (0.85%), the washing being done in the centrifuge. From the deposit of washed cells, a five per cent suspension was made in 0.85% saline and this suspension was used in the test against varying strengths of hypotonic saline.

The test tubes in the experiment were calibrated so that the diameters were approximately equal, and that equal volumes of fluid stood at the same level, thus giving equal volumes of fluid for colorimetric comparison. The standard chosen for colorimetric comparison was that of a sixty per cent haemolysis; this had been proved by experiment to be the most satisfactory.

The standard was obtained as follows:- 0.5 c.c. of the cell suspension was placed in one of the calibrated test tubes and to this were added 2 c.cs. of distilled water. The contents of the tubewere thoroughly mixed and the corpuscles underwent complete haemolysis. From this fluid, showing one hundred per cent haemolysis, 1.5 c.c. were taken and added to 1 c.c. of distilled water in another calibrated tube: the contents were mixed thoroughly and a standard of sixty per cent haemolysis thus obtained.

A separate standard was made for each specimen tested.

In carrying out the test eight calibrated tubes were used in addition to the standard; the test was duplicated for each blood examined so that an average might be obtained. In no case did the duplicate tests differ in results.

The eight tubes were placed in a convenient rack and numbered from 1 to 8. To each tube were added 2 c.cs. of saline of varying concentrations of salt; thereafter 0.5 c.cs. of the cell suspension in 0.85% saline; immediately, the tubes were rapidly inverted and the contents thoroughly mixed; the mixing was begun in the lowest concentration in tube 8 and continued in rapid succession up to tube 1. The finger or thumb used in the process of mixing was dried between two successive tubes.

The tubes were allowed to stand at room temperature overnight.

To minimise the percentage of experimental error, large quantities of the various concentrations of salt were prepared. No fresh solutions were required in the series of two hundred and fifty bloods examined. Before preparing the solutions the salt was heated and then cooled before weighing out the desired quantities; in this way an accurate saline concentration was

obtained/

obtained.

As a further precaution against error, all titrations were carried out with the same pipette - a standardised 2 c.c. pipette.- The pipette was washed out thoroughly by means of a suction pump, with the concentration of saline to be used in the titration.

The contents of the tube at the end of the experiment were as follows.

Tube 1 2 c.c. of 0.85% saline + 0.5 c.c. cell suspension giving saline concentration 0.85%

Tube 2 2 c.c. of 0.425% saline + 0.5 c.c. cell suspension giving saline concentration 0.51%

Tube 3 2 c.c. of 0.400% saline + 0.5 c.c. cell suspension giving saline concentration 0.49%

Tube 4 2 c.c. of 0.375% saline + 0.5 c.c. cell suspension giving saline concentration 0.47%

Tube 5 2 c.c. of 0.35% saline + 0.5 c.c. cell suspension giving saline concentration 0.45%

Tube 6 2 c.c. of 0.325% saline + 0.5 c.c. cell suspension giving saline concentration 0.43%

Tube 7 2 c.c. of 0.30% saline + 0.5 c.c. cell suspension giving saline concentration 0.41%

Tube 8 2 c.c. of 0.275% saline + 0.5 c.c. cell suspension giving saline concentration 0.39%

In the normal saline control i.e. tube 1, the cells sedimented and no haemolysis occurred. In a proportion of the remaining tubes haemolysis took place to a varying degree; this degree of haemolysis was compared colorimetrically with the

standard/

standard sixty per cent which had been prepared. That concentration of salt in the hypotonic saline which corresponded to the standard.could then be readily estimated.

As will be noted, the variation in saline concentration was arranged so that, when combined with the saline in the cell suspension, there should be a final variation of 0.02%.

In his work Simson found that by this method, using sixty per cent haemolysis as a standard, the normal limits for Europeans in England were from 0.41% cencentration to 0.43% concentration with an average of 0.42%, that is,to obtain a sixty per cent haemolysis of normal erythrocytes by means of hypotonic saline, an average concentration of 0.42% salt is required.

The term "normal fragility to hypotonic saline" will be used in this sense.

Accepting the figures 0.41% to 0.43% of salt concentration as a basis for the 'normal fragility to hypotonic saline', I assumed that if I found a similar normal variation among Europeans in Johannesburg that there was no need to carry out an extended survey among them. Fifty cases of varying ages were examined; they were unselected, the specimens being taken from patients sent to the

laboratory/

laboratory for the collection of blood for the Wassermann reaction.

The average result obtained was 0.42%

The maximum obtained was 0.445%

The minimum " " 0.40%

Two hundred native cases were also examined. The specimens were obtained by the courtesy of the Medical Officers of the Witwatersrand Native Labour Association and were taken from natives awaiting repatriation after service on the Mines, or from patients suffering from slight disabilities requiring their detention for a few days in a surgical ward. No case of obvious serious disease was examined.

In a proportion of the cases a red-cell count was done and an estimation of the haemoglobin, no serious departure from the normal was noted.

Though age is an uncertain quantity amongst natives approximations were given in each case.

The average age of the two hundred is 28.5 years.

The average fragility to hypotonic saline obtained was 0.437%

The maximum fragility to hypotonic saline obtained was 0.48%

The minimum fragility to hypotonic saline obtained was 0.40%

Among the two hundred cases there were several tribes represented - Shangaan 55, Xosa 37, Mchopi 27, Nyambaan 23, Pondo 21, Msutu 20, Swazi 7, Bechuana 6

and/

and four others.

The results abstracted from the tribal point of view are as follows -

	Average Age	Average Fragility
Shangaan	27.78 years	0.444%
Xosa	29.46 "	0.434%
Mchopi	27.55 "	0.439%
Nyambaan	29.78 "	0.427%
Pondo	27.9 "	0.438%
Msutu	28.65 "	0.434%
Swazi	30.1 "	0.443%
Bechuana	28.66 "	0.431%

The details of the results are seen in the tables in appendix ii.

From the results, it is seen that amongst the batch of natives examined, the fragility of the erythrocytes to hypotonic saline is greater than for Europeans.

Of the two hundred cases, one hundred and thirty were between the ages of twenty and thirty years and the average fragility for this group is 0.433%; seventy were over thirty years and the average fragility for this group is 0.44%.

This age grouping is also suggested in the tribal abstracts, particularly in the larger groups.

Between the ages of twenty years and thirty the fragility of the native corpuscles approximates to the upper limit of the European normal but above the age of thirty years the fragility is definitely increased.

Hughes and Shrivastara (25) investigated the fragility among Indians, with particular reference

to/

to cases of splenic enlargement and of cirrhosis of the liver. The method of test they adopted was that described by D. Orohovats (26.), in which the degree of haemolysis is determined by means of counts of the numbers of undamaged cells in the saline solution after a fixed definite period; the normal "fragility to hypotonic saline", i.e. for sixty per cent haemolysis, by this method is approximately 0.41%.: the results by the two methods of test are therefore comparable.

It would appear then, that the limits of the "normal fragility to hypotonic saline" are the same for Europeans and for Indians and presumably the limits ought to be the same for the South African Native, and therefore where variation occurs, it is related to some pathological process.

Hughes and Shrivastara (25.) are of the opinion that increased fragility is related to increased activity of the reticulo-endothelial system and they demonstrated that, in cases of splenomegaly associated with cirrhosis of the liver, there is increased fragility of the corpuscles in the early stages, and that as the cirrhosis advances this increased fragility becomes altered to increased resistance.

In Section ii attention was drawn to the incidence of gross cirrhosis amongst South African Natives and to the relatively enormous incidence of early cirrhotic changes, presenting a picture almost identical with that of the first manifestations of experimental cirrhosis. According to Hughes, one should expect amongst these natives an increased fragility of the erythrocytes to saline solutions, and that is borne out by the results recorded in appendix II. It may be emphasised again that so far as clinical examinations showed, these natives were not suffering from any condition that would be likely to invalidate the test, though schistosomiasis cannot be excluded entirely, as a fair percentage of mine natives suffer from this condition.

Stimulation of the reticulo-endothelial system occurs in a great number of diseases, tuberculosis, typhoid, schistosomiasis, amoebiasis, subacute bacterial endocarditis and a great many more and in Section ii it was shown that the diseases in which such stimulation occurs are exceedingly common amongst the natives.

Among Europeans, in the conditions where abnormality in relationship to the fragility of

the/

the corpuscles has been noted, there has also been demonstrated some disturbance of the relationship between the lecithin and cholesterin in the blood: as regards the natives no such data are available, but the great incidence of atheromatosis emphasised in Section ii certainly suggests that there is some departure from the normal in this respect.

E X P E R I M E N T A L.

SECTION V.

Only a few experiments were undertaken and the experimental animal used was the rabbit. In spite of the fact that several workers have taken exception to the use of the rabbit in experimental work, whose end result is cirrhosis, that animal presents certain advantages in any preliminary investigation of this type. The chief objection lies in the occurrence of coccidiosis in a considerable proportion of these laboratory animals, but even the presence of coccidiosis should not lead to misinterpretation of results, as the lesions associated with this condition are so characteristic and so localised. The great advantage that the rabbit presents over other laboratory animals is the relative ease with which cirrhotic changes may be induced in the rabbit's liver as compared with the others. For this reason only rabbits have been used in the investigation recorded, which was initiated as a preliminary investigation.

An abstract of the experiments is presented in Appendix IV.

Only a small series of animals was used and consequently, such subsidiary, but relevant examinations as the effect produced by the experi-

ments/

experiments on the fragility of the erythrocytes, have not been made; isolated examinations of this type would be of little value.

The animals received the ordinary laboratory food, greens and bran mash , with in addition the modifications mentioned in the appendix under each. No estimation was made of the metallic content of the food as control animals were fed on the diet without any modification. The modification of the normal diet consisted in the administration of a metallic salt in solution or suspension; in addition, in three cases, methylated spirits was administered.

The solutions or suspensions of the salts were originally made up in a ten per cent concentration and the salts used were stannous chloride, zinc chloride and copper sulphate.

The calculated amount of tin in the stannous chloride solution was 5.3 gms. in 100 c.c.

The calculated amount of zinc in the zinc chloride solution was 4.8 gms. in 100 c.c.

The calculated amount of copper in the copper sulphate solution was 2.5 gms. in 100 c.c.

In the case of the tin salt and of the zinc salt these concentrations were found to be borne easily by the animals in the doses administered, but as is seen in the case of rabbit No. 3, the dose and

concentration of the copper salt solution proved to be too toxic and subsequently the concentration was reduced to one per cent.

Of these solutions the daily dose was 0.1 c.c. which was accurately measured in a pipette and made up with water to 1 c.c. This was then administered by pipette into the rabbit's mouth and care taken to see that the rabbit swallowed the full dose. The solutions were administered daily at 9 a.m. for six days in the week; Sundays were omitted. At the same time the rabbits were weighed daily.

In the case of the rabbits receiving the tin and the zinc salts there never was any suggestion of toxic disturbance nor of loss of weight - females became pregnant and their offspring was healthy; in the case of the copper animals even with the one per cent concentration some toxic disturbance did take place as in seen in rabbits No. 6 and No. 7.

At the end of the experiments the animals were anaesthetised and killed by chbroform; a post-mortem examination was made immediately and the tissues fixed in formalin for subsequent histological examination. Paraffin sections were made and stained by haematoxylin and eosin, by van Gieson's stain, by Mallory's method for haemofuscin - basic fuschin - and also for haemosiderin.

No case of coccidiosis occurred amongst the rabbits. Rabbit No. 3 died of an acute haemorrhagic nephritis before the experiment had progressed sufficiently to show any changes in the liver, it received doses of the ten per cent copper sulphate colution and it was because of this result that the concentration of this solution was reduced.

Rabbit No. 4 suffered a fracture of one of its hind legs and had to be killed; its place was taken by rabbit No. 4A.

Two series of experiments were made; in the first the metallic salts were associated with alcohol in the form of methylated spirits; in the second no alcohol was administered; a control animal receiving only methylated spirits was also investigated.

The organs in which haemofuscin has been demonstrated in the experimental animal are the liver and the spleen and the bone marrow; reports only on the liver and the spleen will be made in this series.

The examination of the livers of control animals and these were fairly numerous, never revealed any abnormality.

In rabbit No. 5, to which only methylated spirits was administered, the liver showed well-marked fatty degeneration but there was no periportal

cellular/

cellular infiltration nor any increase in fibrous tissue. There was no evidence of haemosiderin but the sections stained for haemofuscin showed an occasional cell filled with pigment giving the differential staining reactions of haemofuscin. The cells were scanty but when affected showed large numbers of the granules; a possible explanation of the occurrence of these isolated cells lies in the fact that the bran of the food contains a certain quantity of zinc, possibly just enough to produce the minimal effects noted. It may be, too, that the fatty change associated with the spirit may render possible the deposition of the pigment in scanty quantity, under conditions in which the normal animal will not show any changes.

Hall and Butt(8) maintain that alcohol has an inhibitory effect on the deposition of haemofuscin pigment but in the short series of experiments mentioned here, such has not been my experience: the series however is too small to permit of a definite opinion.

In rabbit No. 2 to which zinc and methylated spirits were administered, definite changes appeared in the liver; there was a cellular infiltration of

mononuclear/

mononuclear type in the portal tracts, and fine fibrils of fibrous tissue were noted in it. Haemosiderin was absent but haemofuscin was present in abundance, liver cells and Kupffer cells both showing the pigment in great quantity. There was no gross cirrhosis but the periportal cellular infiltration with the fine fibrils of fibrous tissue suggests that if the experiments had been prolonged, a definite cirrhosis would have developed.

It is noteworthy that these changes were induced by such a small daily dose of zinc as 4.8 mgms.

Rabbits 1 and 4A received the tin salt, while 1 also received methylated spirits. Similar changes were found in these rabbits - periportal infiltration of mononuclear cells with fine fibrils of fibrous tissue and haemofuscin in liver cells and Kupffer cells; in rabbit 1 there was transformation in some of the liver cells to haemosiderin but this was patchy in character.

Rabbits 6 and 7 received the copper salt; the changes in the liver were similar to those of the other animals viz. periportal cellular infiltration with deposition of haemofuscin in the liver cells and in the Kupffer cells.

In all the metallic fed animals, the spleen showed deposition of both haemofuscin and haemosiderin and in some cells both pigments were present.

As regards the relative degree of deposition of the haemofuscin in the rabbits, the zinc rabbit

showed/

showed the greatest degree, while the copper and tin animals showed distinctly less; in this respect it is to be noted that the dose of copper was only 0.25 mgm. and also that, as Mallory has pointed out, even rabbits of the same litter react differently to the same doses of metals.

The only definite conclusion which may be made from this preliminary investigation is that haemofuscin may be deposited in the liver and spleen of the rabbit after the administration of relatively small doses of salts of tin, zinc and copper; it is also suggested from these experiments that long administration of small doses of the salts may ultimately lead to cirrhosis.

D I S C U S S I O N.

SECTION VI.

Haemochromatosis has not been confined to natives in its incidence in Johannesburg, for during the period in which thirty-three cases occurred amongst natives, there were seven amongst Europeans. The total number of European autopsies was eight hundred and seventy.

In addition to the seven cases of haemochromatosis all associated with glycosuria, there were five cases of pigment cirrhosis.

Unfortunately both in European and in native cases, full clinical histories have not been available, and where some attempt at history has been made, the details are usually so meagre as to be valueless. The only points of any relevance which have been obtained are the age, the occupation and history of alcoholism. These are given below.

Case (1) W.W.McL. - Aet. 44 - Draughtsman
No history - Cause of death - Malaria.

Case (2) F.G.G. - Aet. 64 - Gold miner
No history of alcohol - Carcinoma of liver - pulmonary tuberculosis.

Case (3)/

Case (3) W.T.O'B. - Aet. 66 - Compositor
Heavy brandy drinker - Cause of death - Prostatic abscess - Pyelonephritis.

Case (4) R.J.B. - Aet. 60 - Caretaker
No history available - Diabetes.

Case (5) S.R.B. - Aet. 50 - Musician
No history available - died in diabetic coma.

Case (6) L.S. - Aet. 50 - Barman
Alcoholic history - Diabetes

Case (7) --- - Aet. 46 - Gold miner
Alcoholic history.

These seven cases all occurred in males.

Pigment cirrhosis Cases.

(1) S.S. - female - aet 59.
No history available.

(2) J.W.I. - Aet 73 - Plumber.
Heavy whisky and beer drinker.

(3) P. O'K.- Aet 63 - Pensioner (presumably from mines)
No history available - had carcinoma of tongue.

(4) --- - Aet 40 + Gold miner
Alcoholic

Mallory (5) in a discussion of the relationship of copper poisoning to haemachromatosis gives the occupation of nine cases of pigment cirrhosis some of which showed the complete picture of haemochromatosis with diabetes; these are now quoted.

(1) Male Aet 63 - Two years milling and planing brass and then 36 years working steel in same room.

(2) Male Aet 55 - worked in railroad shop where copper was worked.

(3) Male Aet 55 - work in polishing brass pipe wrenches.

(4) Male Aet 46 - cable and telegraph/linesman for 23 years.

(5) Male Aet 41 - 14 years milling and turning brass.

(6) Male Aet 51 - Barkeeper

(7) Male Aet 50 - Importer of Italian liquors.

(8) Male Aet 84 - Longshoreman

(9) Male Aet 42 - Teamster

In the same article he also mentions three cases showing deposition of haemofuscin and haemosiderin pigments without the occurrence of cirrhosis.

These were in (1) male 49 - worked for years in brass foundry.

(2) male 60 - a copper worker since the age of 15

(3) male 52 - worked in a brass foundry for 6 years - and suffered from zinc colic.

In yet another article (6.), Mallory mentions that out of a total of 6,340 autopsies there occurred 329 case of cirrhosis of the liver and of these cases of cirrhosis 98 were associated with haemosiderosis. In my series of 870 autopsies there occurred 30 cases of cirrhosis of which 11 were associated with haemosiderosis; in the series of 1,100 natives there were 78 cases of cirrhosis and almost every one associated with haemosiderosis.

As regards the sequence of events which leads up to the terminal picture of cirrhosis of the

liver,/

liver, haemochromatosis and glycosuria, it is now generally accepted that the fibrosis of the pancreas with the associated glycosuria is a terminal phenomenon and that the diabetes plays no part in the etiology of the pigment deposition.

To quote Garrod (27.) "the question resolves itself into that of the precedence of the two remaining events, cirrhosis of the liver and haemochromatosis and we may either suppose that the haemochromatosis is the primary event, and that the deposition of the iron-containing pigment in the liver and pancreas gives rise to the fibrosis; or again that haemochromatosis results from a disturbance of iron metabolism brought about by the hepatic cirrhosis; or lastly that the cirrhosis and haemochromatosis are parallel effects of a common cause".

"if it can be shown that true haemochromatosis does actually occur in cases in which the liver exhibits post-mortem no cirrhotic changes, it will be necessary to abandon the theory that the cirrhosis is the primary event, and the necessary antecedent of haemochromatosis."

I have quoted three cases reported by Mallory in which pigment de-posit occurred without cirrhosis: also in the series of native livers tested for haemofuscin, the two pigments occurred without cirrhosis. Thus it would appear that haemochromatosis does occur without antecedent fibrosis. In the experimental animals too the pigments were deposited without cirrhotic changes.

Though Peyton Rous (11) interpreted the results of his experiments as indicating that cirrhosis preceded pigmentation, Mallory has since shown that a different interpretation is possible, and has also

recorded/

recorded the fact that the intravenous injection of haemoglobin is followed by a deposition of haemofuscin pigment in the liver.

The incidence of the haemosiderosis in natives and of haemochromatosis, is in complete accord with the view promulgated by Mallory as to the sequence of events in haemochromatosis, a view which necessitates the abandonment of the theory that the cirrhosis is the primary event and necessarily antecedent to the haemochromatosis.

The sequence of events is that "haemofuscin is deposited in the endothelium lining the sinusoids and in the parenchymatous cells of the liver. In the course of time it is changed to haemosiderin. The transformation is very slow and requires at least months and probably years; when the parenchymatous cells are filled with pigment beyond a certain degree they undergo necrosis and the pigment is taken up by endothelial leucocytes which often collect in numbers in the periportal connective tissue; following necrosis regeneration of liver cells occurs diffusely and in islands. The new cells in turn become pigmented with haemofuscin which later changes to haemosiderin.

Owing to the necrosis and disappearance of the liver cells, the stroma in places is relatively increased in amount by coalescence resulting in Sclerosis. In the foci where the liver cells regenerate new stroma is formed as in a tumour. In this way the connective tissue is gradually increased in amount

After the liver cells have taken up about all the pigment they can hold, it begins to be deposited in the other organs and tissues, especially in the pancreas, the cortex of the adrenal gland, the lymph nodes in the upper part of the abdomen, the heart, thyroid, skin of the extremities etc."

As regards the age incidence of haemochromatosis

most/

most of the cases recorded are over forty years of age, one or two are just over thirty; the age incidence in natives corresponds with that recorded for Europeans, five occurred between the ages of thirty and thirty five, five between thirty-five and forty, and the remainder were over forty; in the European series the ages are all over forty.

As regards sex incidence, both sexes are liable amongst natives, and from the percentage of cases in total post-mortems they appear to be equally liable. One would naturally expect this to be the case where the factors producing the condition are at work equally on the two sexes.

The factors which may be responsible for the development of the pigmentation may arise in the course of disease or may be derived from diet. As regards the possibility of a disease factor that might be suggested from the slight but definite increase in the fragility of the erythrocytes which has been noted among natives, particularly as that is thought to be associated with conditions in which there is stimulation of the reticulo-endothelial system, it has been noted too that an alteration of diet towards carbohydrate excess (9.) is associated with an increased formation of bile so that diet of this type may also produce an effect on the fragility of the corpuscles.

These two factors stimulation of the reticulo-endothelial system and excess of carbohydrate in the diet are common amongst the South African Native. The increased fragility of the corpuscles is not necessarily associated with anaemia, but it does render available more frequently the end products of erythrocyte destruction for combination with any substances having affinity for them. Peyton Rous (10) maintains that the normal method of destruction of the erythrocytes is by fragmentation, poikilocytes being simply cells in process of fragmentation and disintegration; the end product of the fragmentation being a fine haemoglobin-containing dust which undergoes phagocytosis.

Phagocytosis of erythrocytes also occurs normally and this may be increased under various pathological conditions, but normally it is not a dominant method of destruction of the erythrocytes.

As destruction of the red blood corpuscles is a process which is continuous, there must always be available a certain amount of haemoglobin material for combination with substances for which it has an affinity. Diseases which may alter the rate of destruction of the corpuscles may render available more of the haemoglobin dust stage for such combination and it seems to me that this is the method by which disease with stimulation of the reticulo-endothelial cells, may play a part in haemosiderosis, in conditions where there is no apparent abnormality

in the amount of blood destruction. At best it can only be an adjuvant and not a primary factor.

If one accepts the view mentioned above of the sequence of events which leads to haemochromatosis terminating in bronzed diabetes, there are two theories to be considered as to the etiology of the condition. Firstly, that advanced by Marie in 1895 (quoted by Mallory) that the lesions are due to some as yet unknown toxin which is responsible for the haemosiderosis; secondly that the source of the pigment is due to a perverted iron metabolism as a result of which the cells of the various organs and tissues retain most of the iron which reaches them through the circulation - as some put it briefly - an increased abnormal avidity of the tissues for iron.

Sheldon (2.) has shown that the blood iron in haemochromatosis tends to be lower than in the normal individual and consequently that the condition cannot be due merely to a failure of excretion of iron with accumulation in the body for in such a case the level of the blood iron would be raised.

Garrod (27.) in commenting on the case reported by him and his co-workers, mentions that the urine and bile were alike iron-free, thus suggesting that the failure to eliminate is due rather to the

existence of the iron in a form not suitable for excretion rather than to defect in any special excreting organ".

Mallory is inclined to support Marie's theory and puts forward the view that the unknown toxin suggested by Marie is chronic copper poisoning. In support of his view he adduces evidence derived from clinical histories e.g. occupation, from food contamination, from alcohol contamination and from experiments with copper poisoning. He has produced pigment cirrhosis by means of copper salts, but he also makes the observation that chronic poisoning with zinc will also produce lesions similar to those produced by copper. It is significant too, that of the cases quoted as being exposed to copper poisoning a number were also exposed to zinc poisoning. The importance of zinc in occupational disease has been emphasised mainly in regard to metal fume fever, an acute manifestation, due to conditions in which the zinc is oxidised and the fumes of zinc oxide occur in the atmosphere. This is a common feature in brass foundries and in oxya-cetylene welding, in galvanising iron.

In brass foundries in America Thompson (29.) found as a result of the processes, the workmen would inhale in the day 139 mgms of zinc and that

he would retain 69 mgms. In experimental animals, Drinker and Drinker,[28.] have shown "that inhaled zinc oxide, even in large amounts is rapidly removed from the lungs of the animals .... as zinc is removed from the lungs and its concentration there, diminishes, the zinc concentration of the liver, gall-bladder and the bile rise; simultaneously the zinc concentration of the pancreas also rises as it does with zinc fed animals".

Thus in the case of brass workers, the risk of chronic zinc poisoning is every bit as great as, if not greater than, the risk of copper poisoning.

Mallory mentions in support of his theory that copper can form a cuprohemol compound in the blood and that in some marine animals it takes the place of iron in haemoglobin; a zinc compound similar to cuprohemol has also been demonstrated in oysters [30.] so that zinc too can form a similar compound in combination with haemoglobin, and as the liver is the chief organ which deals with the excretion of zinc and with its storage such a compound would first be stored there. It would seem therefore that though copper poisoning may produce haemochromatosis, not every case of haemochromatosis is due to copper poisoning. Zinc may be the

dominant/

dominant factor in some cases and other metals in other cases.

Among the South African Natives enquiries did not reveal any possible source of chronic copper poisoning but there is evidence strongly suggesting the probability of both zinc and tin poisoning. The preliminary experiments recorded also support the view that zinc and tin may be factors in the production of haemochromatosis. If the source of the poisoning is in the diet then the incidence should be great and the record set out in the appendices shows that it is great.

As regards the alcoholic factor, that is always present in the native.

Pigment having the characters of haemofuscin has been found in the liver (1) as the result of intravenous injection of haemoglobin, (2) as the result of the administration of copper, of zinc and of tin, and a pigment resembling haemofuscin, but not identical with it, as the result of manganese administration.(31.) It would appear that these metals combine with the haemoglobin to form a chemical combination still retaining the iron of the haemoglobin and that this combination is stored in the endothelial cells and in the parenchymatous cells of the organ. After a considerable time this combination

may be broken down and haemosiderin set free in these cells. If there is a constant supply of any of these metals, a considerable proportion of the iron in the body comes to take this form e.g. cuprohemol, a form unsuitable for elimination and thus there would appear to be an increased avidity of the tissues for iron. With different combination of metals with the haemoglobin, there will necessarily be differences in the pigment deposited, which may show in minor microscopic details, but the general features are similar and the end result of the vital reaction of the cells containing the pigment is haemosiderin.

The explanation of the pigmentation in Peyton Rous's experiments with whole blood in which a haemofuscin pigment is deposited, appears to me to be that haemoglobin as such or parahaemoglobin is deposited in the cells and subsequently broken down to haemosiderin; this would bring the pigment into line with the other haemofuscin pigments.

It seems to me therefore that the haemofuscin pigments related to haemochromatosis may be haemoglobin or an isomeric form of it, or haemoglobin in combination with other substances such as copper, zinc, tin or other metals and therefore are not disintegration products of haemoglobin. In this

connection/

connection a significant observation has been made by Hall and Butt (8.) that in their experiments with copper poisoning the degree of deposition of haemofuscin was in direct proportion to the amount of copper present.

SUMMARY.

Haemochromatosis is a not uncommon disease in the South African Native; the chief factor in its production appears to be in the diet, in which there is excess of carbohydrate and probable contamination with salts of zinc and of tin.

Haemofuscin is an essential feature of haemochromatosis; its deposition precedes the haemosiderosis. Haemofuscin would appear to be a pigment derived from the combination of haemoglobin with metals e.g. copper, zinc and tin; it thus contains iron in combined forms. After a period in the cells the pigment may be broken down and haemosiderin set free; this period apparently varies in different animals and with different metals.

In its formation, haemofuscin necessitates the abstraction from the blood of haemoglobin, and its conversion into a form unsuitable for the easy liberation of the contained iron. This would explain the lowering of the level of blood iron, and the diminished excretion of iron, a proportion being

in/

in the form of haemofuscin and unsuitable for elimination.

The development of the complete picture of bronzed diabetes depends on the degree of deposition of the pigment and the rate of its deposition. In mild cases pigmentation alone may occur, this was seen in the younger natives; in slightly more severe cases, some cirrhosis may be added and so on until the complete picture is produced. All stages have been noted in the South African Native.

SECTION VII

APPENDIX I.

This is a record of autopsies from January 1925 to September 1928; it includes the sex and age of the cases, the incidence of haemosiderin and the cause of death.

The incidence of haemosiderin is noted in the liver, spleen and lymphatic glands, and also under the heading "alia" where the pigmentation is co-extensive with that found in haemochromatosis.

The record is not complete in respect of the haemosiderosis as systematic tests were not carried out in the earlier cases.

Arbitrary signs used in the appendix are related to the presence of free iron in the organs.

+++ = very marked to marked.

++ = marked.

\+ = slight but definite.

± = trace.

\- = negative.

o = not examined.

No.	Race	Sex	Age	Liver	Spleen	Glands	Alia	Cause of death.
1	Msutu	F.	23	o	o	o	o	Acute Aortic Endocarditis Pericarditis
2	Zulu	F.	18	o	o	o	o	Influenza
3	"	M.	30	o	o	o	o	Tuberculo-silicosis
4	"	M.	23	o	o	o	o	Typhoid
5	"	M.	35	o	o	o	o	Typhoid
6	"	M.	60	o	o	o	o	Genito-urinary Tuberculosis etc.
7	"	M.	4/52	o	o	o	o	Gastro-enteritis
8	Msutu	M.	25	o	o	o	o	Typhoid
9	Xosa	F.	29	o	o	o	o	Perforation of ileum - peritonitis
10	Msutu	F.	30	o	o	o	o	Pulmonary Tuberculosis
11	Zulu	F.	20	o	o	o	o	Typhoid
12	Xosa	M.	3	o	o	o	o	Tuberculous Broncho-Pneumonia
13	"	M.	8/12	o	o	o	o	Enteritis
14	"	M.	9	o	o	o	o	Pulmonary Tuberculosis
15	Zulu	M.	65	+++	+++	++	-	Lymphosarcoma - Cirrhosis of Liver
16	"	M.	40	o	o	o	o	Cerebro-spinal Meningitis
17	"	M.	45	+++	+++	++	-	Generalised Tuberculosis
18	"	F.	30	o	o	o	o	Sapraemia
19	"	M.	1/12	o	o	o	o	Enteritis
20	"	M.	20	++	++	-	-	Cerebro-spinal Meningitis - fine cirrhosis
21	"	F.	23	o	o	o	o	Bacillary Dysentery
22	"	F.	3	o	o	o	o	Basal Meningitis
23	"	M.	40	o	o	o	o	Lobar Pneumonia
24	"	F.	10/12	o	o	o	o	Enteritis
25	Msutu	M.	25	o	o	o	o	Typhoid

No.	Race	Sex	Age	Liver	Spleen	Glands	Alia	Cause of Death.
26	Msutu	M.	32	o	o	o	o	Tuberculo-silicosis - Enteritis
27	"	M.	30	o	o	o	o	Generalised Tuberculosis
28	"	M.	45	++	++	+	-	Lobar Pneumonia
29	"	F.	25	o	o	o	o	Pulmonary Tuberculosis
30	"	M.	38	++	++	+	-	Perforation of amoebic ulcer - fine cirrhosis
31	"	M.	50	o	o	o	o	Tuberculous Pericarditis
32	"	M.	45	o	o	o	o	Appendicitis - General Peritonitis
33	"	M.	35	o	o	o	o	Ulcerative Endocarditis
34	"	M.	40	o	o	o	o	Bronchitis, Emphysema, Cardiac failure
35	"	M.	40	++	++	+	-	Tuberculous Meningitis
36	"	F.	34	o	o	o	o	Pulmonary Tuberculosis - Enteritis
37	"	M.	30	o	o	o	o	Arteriosclerosis - Syphilitic Mesaortitis
38	"	F.	19	o	o	o	o	Acute Endocarditis (Aortic and Mitral)
39	"	M.	60	o	o	o	o	Purulent bronchitis - Emphysema
40	"	M.	35	o	o	o	o	Tuberculoma brain - Generalised Tuberculosis
41	"	F.	24	o	o	o	o	Typhoid
42	Swazi	M.	78	++	++	+	-	Enlarged Prostate - Pyelo-nephritis
43	"	M.	36	o	o	o	o	Acute Yellow Atrophy of liver
44	"	M.	18	o	o	o	o	Tuberculous enteritis - Tabes Mesenterica
45	"	M.	4/12	o	o	o	o	Enteritis - Broncho-Pneumonia
46	"	F.	5	o	o	o	o	Generalised Tuberculosis
47	Msutu	M.	28	o	o	o	o	Psoas abscess - Tuberculous Meningitis
48	"	F.	25	o	o	o	o	Perforation of bowel (foreign body) Peritonitis
49	"	F.	27	o	o	o	o	Generalised Tuberculosis
50	Xosa	M.	50	o	o	o	o	Cardiospasm (?)

51/

No.	Race	Sex	Age	Liver	Spleen	Glands	Alia	Cause of Death.
51	Xosa	M.	36	o	o	o	o	Carcinoma of Liver
52	"	F.	60	o	o	o	o	Carcinoma of Breast - Cancer en Cuirasse
53	"	M.	17	++	++	-	o	Carcinoma of Liver with Coarse Cirrhosis
54	"	M.	30	o	o	o	o	Typhoid fever
55	"	M.	28	o	o	o	o	Haemorrhage in cerebral abscess - Cirrhosis
56	"	M.	28	+++	++	+	-	Septicaemia - fine cirrhosis
57	"	M.	30	o	o	o	o	Pyaemia
58	"	M.	22	o	o	o	o	Typhoid
59	Msutu	F.	21	o	o	o	o	Pulmonary Tuberculosis
60	"	M.	3	o	o	o	o	Broncho Pneumonia
61	"	M.	36	o	o	o	o	Myo-cardial Degeneration
62	"	M.	30	o	o	o	o	Generalised Tuberculosis
63	"	M.	60	o	o	o	o	Tuberculo-silicosis
64	"	F.	-	o	o	o	o	Pulmonary tuberculosis
65	"	F.	40	+++	+++	+++	++	Carcinoma of Liver - Haemochromatosis
66	Msutu	M.	35	o	o	o	o	Septicaemia - Ulceration Mandible
67	"	M.	35	++	+	-	-	Nephritis - fine cirrhosis
68	Xosa	M.	30	o	o	o	o	Lobar Pneumonia
69	"	M.	60	o	o	o	o	Hydatid disease of brain
70	"	M.	20	o	o	o	o	Typhoid fever
71	"	F.	13	o	o	o	o	Sarcoma of tonsil
72	"	M.	30	o	o	o	o	Typhoid
73	"	M.	38	o	o	o	o	Arteriosclerosis with Cardiac failure
74	"	M.	25	o	o	o	o	Influenza
75	"	M.	45	++	++	+	-	Tuberculous Peritonitis

76/

No.	Race	Sex	Age	Liver	Spleen	Glands	Alia	Cause of Death.
76	Xosa	M.	22	o	o	o	o	Lobar Pneumonia
77	Msutu	M.	10	o	o	o	o	Typhoid
78	"	M.	28	+	-	-	-	Septic bronchitis; Pericarditis; fine cirrhosis
79	"	M.	30	o	o	o	o	Nephritis
80	Xosa	F.	30	o	o	o	o	Typhoid
81	"	F.	20	o	o	o	o	Pulmonary Tuberculosis
82	"	F.	70	o	o	o	o	Pericarditis - Syphilitic Aortitis - cirrhosis
83	"	M.	60	o	o	o	o	Pott's disease - Generalised tuberculosis
84	"	M.	40	o	o	o	o	Cerebral tumour
85	"	F.	53	o	o	o	o	Hysterectomy - General Pericarditis
86	"	M.	36	o	o	o	o	Tuberculous Pleurisy and Pericarditis
87	"	M.	28	++	++	-	-	Malaria - Subacute Nephritis
88	Swazi	F.	? 25	o	o	o	o	Septic Sinusitis - Hypostatic Pneumonis
89	Zulu	M.	30	o	o	o	o	Pulmonary Tuberculosis
90	Msutu	F.	29	o	o	o	o	Septic endometritis
91	"	M.	60	o	o	o	o	Septic Wound - Pyaemia
92	"	M.	30	o	o	o	o	Typhoid
93	Zulu	M.	20	o	o	o	o	Miliary Tuberculosis - Cirrhosis
94	"	F.	30	o	o	o	o	Pulmonary Tuberculosis
95	"	F.	32	o	o	o	o	Intestinal Obstruction
96	"	M.	28	o	o	o	o	Amoebiasis
97	Shangaan	M.	16	o	o	o	o	Cerebro-spinal Meningitis
98	Msutu	F.	30	o	o	o	o	Ruptured Ectopic Pregnancy - Pulmonary Tuberculosis
99	"	M.	40	+++	+++	+++	+	Influenza - Nephritis
100	Fingo	M.	60	o	o	o	o	Nephritis

No.	Race	Sex	Age	Liver	Spleen	Glands	Alia	Cause of Death.
101	Xosa	M.	25	o	o	o	o	Pulmonary Tuberculosis - Cirrhosis
102	Swazi	M.	1.8/12	o	o	o	o	Cerebro-spinal Meningitis
103	"	F.	46	o	o	o	o	Cerebral Tumour
104	Shangaan	M.	40	++	++	+	-	Chronic Nephritis
105	Msutu	M.	45	++	++	++	-	Scurvy
106	Tongo	M.	35	++	++	-	-	Generalised Tuberculosis
107	"	M.	40	o	o	o	o	Acute Nephritis
108	"	M.	36	o	o	o	o	Tuberculous Pericarditis
109	"	M.	36	o	o	o	o	Tuberculous Meningitis
110	"	M.	34	o	o	o	o	Abdominal Tuberculosis
111	Msutu	M.	25	+	+	-	-	Duodenal ulcer - Pylephlebitis
112	"	F.		+	+	-	-	Suppurative Nephritis
113	Shangaan	M.	40	++	++	+	-	Cerebral softening
114	Zulu	M.		+	+	-	-	Tuberculous Meningitis
115	"	F.	1	o	o	o	o	Cerebro-spinal Meningitis
116	Xosa	M.	45	o	o	o	o	? Tetanus
117	"	M.	40	++	++	+	-	Tuberculous Peritonitis
118	Zulu	M.	40	++	++	-	-	Tuberculo-silicosis
119	Msutu	F.	16	-	-	-	-	Status Lymphaticus
120	Shangaan	M.	30	+	+	-	-	Influenza - Phlebitis
121	"	M.	1.7/12	-	-	-	-	Enteritis - Status Lymphaticus
122	Bechuana	M.	29	++	++	-	-	Acute Phthisis
123	"	M.	27	+	+	-	-	Tabes Mesenterica
124	Zulu	M.	65	++	++	++	-	Amoebic Dysentery
125	"	F.	20	-	-	-	-	Ovarian abscess with pyaemia

126/

No.	Race	Sex	Age	Liver	Spleen	Glands	Alia	Cause of Death.
126	Zulu	M.	46	++	++	-	-	Pulmonary Tuberculosis with Enteritis
127	"	F.	38	-	++	-	-	Pulmonary tuberculosis
128	Pondo	M.	30	+	+	-	-	Tuberculo-silicosis
129	"	M.	25	++	+	-	-	Typhoid
130	"	M.	46	++	++	-	-	Tuberculous Meningitis
131	"	F.	28	-	-	-	-	Pregnancy - Acute Yellow Atrophy
132	Msutu	M.	26	-	-	-	-	Broncho-pneumonia
133	Zulu	M.	50	++	++	+	-	Pulmonary Tuberculosis
134	"	M.	30	++	++	-	-	Miliary Tuberculosis
135	Shangaan	M.	30	++	++	-	-	Purulent bronchitis
136	"	M.	40	++	++	+	-	Lobar Pneumonia
137	"	M.	36	++	++	-	-	Tuberculo-silicosis
138	"	M.	30	++	++	+	-	Tuberculous broncho-pneumonia
139	Zulu	F.	40	++	++	+	-	Salpingitis - Peritonitis
140	"	M.	38	++	++	+	-	Influenza
141	"	M.		o	o	o	o	Miliary tuberculosis
142	Msutu	M.	25	++	++	-	-	Generalised tuberculosis
143	Zulu	M.	30	++	++	++	-	Pulmonary tuberculosis
144	"	M.	28	++	++	+	-	Generalised tuberculosis
145	Pondo	M.	48	++	++	+	-	Tuberculo-silicosis with Pericarditis
146	Xosa	M.	8/12	o	o	o	o	Enteritis
147	"	F.	50	o	o	o	o	Cerebro-spinal Meningitis
148	Zulu	F.	30	o	o	o	o	Operation - Intestinal obstruction
149	Shangaan	M.	25	++	++	+	-	Pneumococcal Meningitis
150	Msutu	M.	28	++	+	-	-	Bacillary Dysentery

151/

No.	Race	Sex	Age	Liver	Spleen	Glands	Alia	Cause of Death.
151	Msutu	M.	42	++	++	+	-	Broncho-pneumonia Toxic Jaundice
152	Zulu	M.	32	++	++	+	-	Pulmonary Tuberculosis
153	Msutu	M.	17	++	++	-	-	Generalised Tuberculosis
154	"	F.	19	++	++	-	-	Tonsillar abscess - Broncho-pneumonia
155	"	F.	50	++	+	-	-	Myocarditis - Broncho-pneumonia
156	"	F.	38	++	+++	-	-	Chronic Nephritis Hypostatic pneumonia
157	"	M.	28	++	++	-	-	Broncho-pneumonia
158	"	M.	24	++	++	-	-	Pulmonary Tuberculosis
159	Shangaan	M.	30	++	++	+	-	Carcinoma of Liver with Cirrhosis
160	Msutu	M.	50	++	++	+	-	Scurvy , Broncho-pneumonia
161	"	M.	3/12	-	-	-	-	Enteritis - Broncho-pneumonia
162	Zulu	M.	46	++	++	+	-	Malaria
163	"	M.	29	-	-	-	-	Cirrhosis of Liver with Ascites
164	Msutu	M.	65	++	++	+	-	Aortic Incompetence
165	Swazi	M.	57	+++	+++	+	-	Myocarditis with Cardiac failure
166	Zulu	F.	25	-	-	-	-	Typhoid
167	"	F.	46	++	++	+	-	Arteriosclerosis - Myocarditis
168	Xosa	F.	30	++	++	+	-	Pulmonary Tuberculosis
169	Zulu	M.	36	+++	++	+	-	Cerebral Haemorrhage
170	"	M.	9/12	-	-	-	-	Enteritis Broncho-pneumonia
171	Swazi	F.	19	+	+	-	-	Ulcerative Colitis
172	Shangaan	M.	? 40	+++	+++	++	-	Pulmonary Tuberculosis
173	Msutu	M.	32	+++	+++	±	±	Atrophic Cirrhosis - Aortic Incompetence
174	"	M.	32	+++	+	-	-	Osteomyelitis - Pyaemia
175	Zulu	M.	40	+++	+++	++	-	Tuberculous Pericarditis

176/

No.	Race	Sex	Age	Liver	Spleen	Glands	Alia	Cause of Death.
176	Ndebele	M.	34	++	+++	+	-	Pulmonary Tuberculosis
177	Zulu	F.	65	+++	+++	++	-	Amoebic Dysentery
178	"	M.	36	++	++	+	-	Malaria
179	"	M.	48	+++	+++	++	-	Scurvy
180	"	M.	28	+++	+++	+	-	Pulmonary Tuberculosis
181	Shangaan	M.	40	+++	+++	+	-	Influenza
182	"	M.	45	+++	+++	++	-	Cystitis with Broncho-pneumonia
183	"	M.	54	+++	+++	+	-	Pulmonary Tuberculosis
184	Zulu	M.	40	++	+++	+	-	Pulmonary Tuberculosis
185	"	M.	41	++	++	-	-	Aortic Incompetence - Cirrhosis
186	"	F.	19		+++	-	-	General Peritonitis
187	Fingo	M.	50	+++	+++	++	-	Pulmonary Tuberculosis
188	Xosa	M.	6/12	-	+	-	-	Broncho-pneumonia
189	"	M.	37	+++	+++	+++	++	Lobar Pneumonia
190	"	F.	34	++	++	-	-	Subacute Nephritis
191	Shangaan	M.	32	++	++	+	-	Miliary Tuberculosis
192	"	M.	60	+++	+++	++	-	Miliary Tuberculosis
193	Msutu	F.	33	++	++	±	-	Tabes Mesenterica
194	Xosa	M.	26	++	++	±	-	Generalised Tuberculosis
195	"	M.	36	++	++	±	-	Tuberculo-silicosis
196	"	M.	72	++	++	+	-	Arteriosclerosis with myocarditis
197	Zulu	F.	24	+	+	-	-	Typhoid
198	"	F.	40	++	+++	+	-	Pulmonary Tuberculosis
199	"	M.	30	+	++	-	-	Lobar Pneumonia
200	Xosa	M.	20	++	+++	-	-	Typhoid

201/

No.	Race	Sex	Age	Liver	Spleen	Glands	Alia	Cause of Death.
201	Xosa	M.	22	+	++	-	-	Pulmonary Tuberculosis
202	"	M.	27	++	++	-	-	Typhoid
203	"	M.	32	++	++	±	-	Tuberculo-silicosis
204	Zulu	M.	18	++	++	-	-	Pott's Disease - Tabes Mesenterica
205	"	M.	40	++	++	+	-	Tuberculo-silicosis
206	"	M.	8/12	-	-	-	-	Enteritis
207	"	M.	29	++	++	+	-	Tuberculo-silicosis with Tuberculosis
208	"	M.	38	++	++	+	-	Tuberculo-silicosis Hydatid disease of heart
209	Ndebele	M.	30	++	+++	+	-	Cerebro-spinal Meningitis - Cirrhosis
210	"	M.	38	-	-	-	-	Gastric ulcer
211	Fingo	M.	50	+++	+++	++	-	Pulmonary Tuberculosis
212	"	M.	36	++	+++	+	-	Pulmonary Tuberculosis
213	Msutu	F.	3½	-	-	-	-	Mastoiditis - Meningitis
214	Zulu	M.	40	++	++	±	-	Penumococcal Meningitis - Cirrhosis
215	"	M.	30	++	++	-	-	Cerebral softening
216	Msutu	F.	50	+++	+++	+++	++	Septicaemia - Haemochromatosis
217	Nyasa	M.	30	++	++	+	-	Tuberculo-silicosis
218	Msutu	F.	5/12	-	-	-	-	Enteritis with broncho-pneumonia
219	"	M.	30	++	++	+	-	Pulmonary Tuberculosis
220	"	F.	14	-	-	-	-	Acute Appendicitis
221	"	M.	30	+	+	-	-	Aortic Incompetence
222	Shangaan	M.	19	-	-	-	-	Otitis media - Bilharzia
223	"	F.	38	+++	+++	+++	++	Generalised tuberculosis
224	"	M.	35	+++	+++	++	-	Generalised tuberculosis
225	Zulu	M.	28	+	+	-	-	Cerebro-spinal Meningitis

226/

No.	Race	Sex	Age	Liver	Spleen	Glands	Alia	Cause of Death.
226	Msutu	M.	36	+++	+++	++	-	Pyelonephritis
227	"	F.	22	++	++	-	-	Typhoid with Peritonitis
228	"	M.	7	-	-	-	-	Diphtheria - Broncho Pneumonia
229	"	M.	36	++	++	+	-	Carcinoma of Oesophagus
230	"	F.	4	-	-	-	-	Broncho Pneumonia
231	"	M.	8/12	-	-	-	-	Broncho Pneumonia
232	"	M.	34	+++	+++	+++	++	Suppurative Pyelonephritis - Cirrhosis
233	Shangaan	M.	58	++	++	+	-	Typhoid with perforation
234	"	M.	60	++	++	+	-	Pott's Disease Psoas abscess Generalised Tuberculosis
235	"	M.	25	++	+	-	-	Pneumonia, Bilharzia, Cirrhosis
236	"	M.	45	++	++	+	-	Tuberculo-silicosis
237	"	M.	22	++	+++	+	-	Pulmonary Tuberculosis
238	"	M.	42	o	o	o	o	Generalised Tuberculosis
239	Swazi	M.	18	±	±	-	-	Endocarditis with pericarditis
240	Xosa	M.	5	-	-	-	-	Tuberculomata Brain
241	"	M.	40	++	+++	+	-	Cerebral Aneurysm with rupture
242	Bechuana	M.	26	o	o	o	o	Pulmonary Tuberculosis
243	"	F.	20	-	-	-	-	Endometritis with peritonitis
244	"	M.	36	++	++	+	-	Generalised tuberculosis
245	"	M.	22	±	±	-	-	Aortic endocarditis
246	"	F.	39	o	o	o	o	Myocarditis
247	"	F.	26	+	++	-	-	Lobar Pneumonia
248	"	M.	23	++	+	-	-	Acute Enteritis with Nephritis
249	Zulu	M.	30	o	o	o	o	Blood cyst - brain - Epilepsy
250	"	F.	35	++	++	-	-	Cellulitis abdominal wall

251/

No.	Race	Sex	Age	Liver	Spleen	Glands	Alia	Cause of Death.
251	Zulu	M.	46	-	+	-	-	Tuberculo-silicosis - Bilharzia
252	"	M.	37	++	++	+	-	Intestinal obstruction
253	Xosa	M.	39	+++	+++	++	-	Tuberculo-silicosis - Generalised Tuberculosis
254	Zulu	M.	19	+++	+++	+	-	Generalised tuberculosis
255	"	M.	32	+	++	±	-	Pulmonary tuberculosis
256	Shangaan	M.	38	++	++	±	-	Arteriosclerosis, Tuberculous peritonitis
257	Bechuana	M.	30	+	±	-	-	Lobar Pneumonia
258	Shangaan	M.	18	++	+++	-	-	Pharyngeal abscess
259	"	M.	45	+++	+++	+++	±	Tuberculo-silicosis Chronic Nephritis
260	"	M.	12	+	+	-	-	Cerebral tumour
261	"	M.	18	-	+	-	-	Malaria
262	Msutu	M.	40	+++	+++	+++	-	Cirrhosis with Carcinoma of Liver
263	"	F.	1.6/12	-	+	-	-	Broncho-pneumonia
264	"	M.	40	+++	+++	++	-	Pulmonary Tuberculosis - Cirrhosis
265	"	F.	40	o	o	o	o	Arteriosclerosis
266	"	M.	40	o	o	o	o	Valvular disease of heart
267	"	M.	28	o	o	o	o	Broncho-pneumonia
268	"	M.	45	o	o	o	o	Tuberculo-silicosis
269	"	M.	60	o	o	o	o	Carcinoma of Pancreas
270	Zulu	M.	30	o	o	o	o	Lobar Pneumonia
271	"	M.	32	o	o	o	o	Arteriosclerosis - Cardiac failure
272	"	M.	48	o	o	o	o	Miliary tuberculosis
273	Msutu	M.	25	o	o	o	o	Lobar Pneumonia
274	Zulu	F.	30	o	o	o	o	Amoebic Dysentery
275	"	M.	45	+++	+++	+	-	Cirrhosis of Liver with Carcinoma

276/

No.	Race	Sex	Age	Liver	Spleen	Glands	Alia	Cause of Death.
276	Zulu	M.	21	+	+	-	-	Cerebro-spinal Meningitis
277	"	M.	45	o	o	o	o	Syphilitic Meningitis
278	Msutu	M.	30	+++	+++	+	-	Pulmonary Tuberculosis
279	Shangaan	M.	22	++	++	±	-	Tuberculous pericarditis
280	"	M.	65	+++	+++	++	-	Generalised Tuberculosis
281	"	M.	56	++	++	++	-	Lobar Pneumonia - Cysticerci Brain
282	Bechuana	F.	38	++	++	+	-	Generalised Tuberculosis
283	"	F.	65	+++	+++	+++	++	Arteriosclerosis - Cirrhosis of Liver
284	Msutu	M.	27	+++	+++	+	-	Cerebral Malaria
285	"	F.	24	+	+	-	-	Aneurysm of Aorta
286	Bechuana	M.	20	+++	+++	+	-	Tuberculous Broncho-pneumonia
287	"	M.	60	+++	+++	+++	±	Myocarditis (Cysticercus Cellulosae)
288	Xosa	M.	34	+++	+++	+++	±	Pulmonary Tuberculosis
289	"	F.	24	+	+	-	-	Pulmonary Tuberculosis - Salpingitis
290	"	M.	45	++	++	±	-	Cerebro-spinal Meningitis
291	Swazi	F.	25	+	+	±	-	Pulmonary Tuberculosis
292	Msutu	M.	40	++	++	±	-	Pneumococcal Meningitis - Cirrhosis
293	"	M.	48	+++	+++	++	-	Pulmonary Tuberculosis - Cirrhosis
294	Xosa	M.	72	+++	++	++	-	Scurvy
295	Msutu	M.	25	±	+	-	-	Ulcerative endocarditis
296	"	M.	22	±	++	-	-	Lobar Pneumonia
297	"	M.	22	+	+	-	-	Lobar Pneumonia
298	"	M.	36	+	+	-	-	Lobar Pneumonia
299	"	M.	40	++	++	++	-	Influenzal Broncho-pneumonia
300	"	F.	40	+	+	-	-	Lymphosarcoma

301/

No.	Race	Sex	Age	Liver	Spleen	Glands	Alia	Cause of Death.
301	Msutu	M.	30	++	++	-	-	Epilepsy
302	"	M.	21	+	+	-	-	Septic Arthritis - Septicaemia
303	Zulu	M.	31	+++	+++	+++	+++	Lobar Pneumonia - Haemochromatosis
304	"	F.	28	o	o	o	o	Generalised Tuberculosis
305	"	M.	10/12	-	-	-	-	Nephritis
306	Msutu	F.	19	±	±	-	-	Cerebro-spinal Meningitis
307	"	M.	40	++	++	+	-	Acute Endocarditis
308	"	F.	23	±	±	-	-	Suppurative Mediastinitis
309	Zulu	M.	49	o	o	o	o	Lobar Pneumonia
310	"	F.	28	o	o	o	o	Acute Bronchitis - Broncho-pneumonia
311	Xosa	M.	1/½	-	-	-	-	Tuberculous enteritis - Nephritis
312	Msutu	M.	35	++	++	+	-	Carcinoma of Liver
313	Zulu	M.	46	+++	+++	++	-	Aneurysm - Arteriosclerosis
314	"	F.	26	+	+	-	-	Septic Bronchitis - Broncho-pneumonia
315	Msutu	M.	46	+++	+++	+	±	Scurvy (Haemorrhage suprarenals)
316	"	F.	50	++	++	++	-	Carcinoma - Ovaries
317	Zulu	M.	30	++	++	+	-	Amoebiasis - Liver - Pericardium
318	"	M.	56	+++	+++	+++	++	Lobar Pneumonia - Haemochromatosis
319	Bacu	M.	29	-	-	-	-	Influenza
320	"	M.	20	+	+	-	-	Cerebro-spinal Meningitis
321	"	M.	32	++	++	±	-	Miliary Tuberculosis
322	"	F.	24	-	-	-	-	Carcinoma of Liver
323	Xosa	M.	48	+++	+++	+++	+	Pulmonary Tuberculosis
324	"	M.	45	+++	+++	+++	+	Pulmonary Tuberculosis
325	"	M.	22	-	-	-	-	Lobar Pneumonia

326/

NO.	Race	Sex	Age	Liver	Spleen	Glands	Alia	Cause of Death.
326	Xosa	M.	25	-	-	-	-	Pneumococcal Meningitis
327	"	M.	19	-	-	-	-	Acute Nephritis
328	"	F.	34	++	++	-	-	Subacute Nephritis
329	Zulu	M.	60	++	++	+	-	Aneurysm Aorta - Pulmonary tuberculosis
330	Swahili	M.	40	++	++	+	-	Lobar Pneumonia
331	Fingo	F.	38	++	+	-	-	Lobar Pneumonia
332	Zulu	M.	47	++	++	+	-	Septic Arthritis - Broncho-pneumonia
333	"	M.	45	+++	+++	++	-	Tuberculous Pleurisy
334	"	M.	45	+++	+++	++	-	Lobar Pneumonia
335	Shangaan	M.	25	++	++	-	-	Cerebro-spinal Meningitis
336	"	M.	49	+++	+++	++	-	Influenza with Pulmonary tuberculosis
337	"	F.	50	++	++	+	-	Influenza
338	"	M.	45	+++	++	++	-	Aortic Incompetence
339	Ndebele	F.	50	+++	+++	++	-	Carcinoma of stomach
340	"	M.	56	+++	+++	++	-	Emphyaema - Anaesthetic
341	"	M.	25	++	++	±	-	Pulmonary tuberculosis
342	Msutu	M.	41	+++	+++	++	-	Pulmonary tuberculosis
343	Xosa	M.	50	+++	++	±	-	Influenza
344	"	M.	48	+++	+++	++	-	Pulmonary tuberculosis
345	"	F.	48	++	++	+	-	Influenzal Broncho-pneumonia
346	Fingo	F.	27	-	-	-	-	Pneumococcal Meningitis
347	Zulu	M.	30	++	++	-	-	Carcinoma of Colon
348	Nyasa	M.	30	++	++	±	-	Influenza
349	"	M.	23	o	o	o	o	Amoebiasis Liver
350	Nchopi	F.	50	o	o	o	o	Gumma of Brain - Broncho-pneumonia

351/

NO.	Race	Sex	Age	Liver	Spleen	Glands	Alia	Cause of Death.
351	Shangaan	M.	50	+++	+++	++	-	Pulmonary tuberculosis
352	Pondo	F.	60	++	++	+	-	Interstitial fibrosis Lungs
353	Shangaan	M.	36	+++	+++	++	-	Nephritis - Arteriosclerosis
354	"	M.	30	o	o	o	o	Tuberculo-silicosis
355	"	M.	36	o	o	o	o	Septic Broncho-pneumonia
356	"	M.	35	o	o	o	o	Pylephlebitis
357	Zulu	F.	30	++	++	-	-	Mitral Stenosis
358	Msutu	M.	35	++	++	-	-	Tuberculo-silicosis
359	"	M.	27	++	++	-	-	Aortic Incompetence
360	"	M.	30	++	++	±	-	Aneurysm with Aortic Incompetence
361	"	M.	60	++	++	++	-	Haemorrhagic Colitis - Arteriosclerosis
362	"	F.	28	-	++	-	-	Gangrenous Endometritis - Bilharzia
363	Xosa	M.	50	++	++	+	-	Pulmonary tuberculosis
364	"	M.	50	++	++	+	-	Myocarditis
365	"	F.	30	++	++	-	-	Pulmonary Embolism (Operation)
366	"	M.	1.5/12	-	+	-	-	Tuberculous Broncho-pneumonia
367	Pondo	M.	50	++	++	+	-	Lobar Pneumonia with abscess
368	Msutu	M.	30	+++	++	++	-	Carcinoma of Liver with cirrhosis
369	"	M.	29	o	o	o	o	Aortic Incompetence
370	Pondo	M.	46	++	++	+	-	Pulmonary tuberculosis
371	Fingo	M.	41	++	++	+	-	Tuberculo-silicosis
372	"	F.	3.11/12	-	+	-	-	Tuberculous Broncho-pneumonia
373	Msutu	M.	1.4/12	-	-	-	-	Broncho-pneumonia
374	"	M.	60	-	-	-	-	Endocarditis - Cerebral Embolism
375	"	M.	40	++	++	+	-	Strangulated hernia

376/

No.	Race	Sex	Age	Liver	Spleen	Glands	Alia	Cause of Death.
376	Swazi	M.	22	-	-	-	-	Endocarditis - Meningitis
377	"	M.	38	++	++	+	-	Pulmonary tuberculosis
378	Msutu	F.	12	+	+	-	-	Mitral Endocarditis
379	"	M.	32	++	++	-	-	Lobar pneumonia
380	Fingo	M.	33	±	++	-	-	Lobar pneumonia
381	Msutu	F.	40	-	+	-	-	Suppurative Nephritis
382	"	M.	33	++	++	±	-	Lobar pneumonia
383	"	M.	28	++	++	±	-	Tuberculous peritonitis
384	"	M.	26	+++	+++	+	-	Lobar pneumonia
385	Msutu	M.	33	++	++	+	-	Meningitis
386	"	F.	48	++	++	+	-	Myocarditis; cerebral embolism
387	"	M.	40	++	++	+	-	Myocarditis
388	"	M.	3½	++	++	-	-	Amoebic Dysentery
389	"	M.	2¾	-	-	-	-	Enteritis Broncho-pneumonia
390	"	F.	23	+	++	-	-	Endometritis - Pulmonary tuberculosis
391	Shangaan	M.	48	+++	+++	+++	±	Scurvy, Pulmonary tuberculosis, Bilharzia
392	Xosa	M.	1.7/12	-	-	-	-	Broncho-pneumonia
393	"	F.	3/12	-	-	-	-	Enteritis - Broncho-pneumonia
394	Fingo	M.	28	+	+	-	-	Aortic Disease
395	"	M.	45	o	o	o	o	Lobar pneumonia
396	Zulu	M.	34	o	o	o	o	Broncho-pneumonia
397	"	M.	6	-	-	-	-	Tuberculous Enteritis
398	"	M.	45	+++	+++	++	-	Influenza - Myocarditis
399	"	F.	30	+++	+++	++	+	Influenza
400	"	M.	22	+	+	±	-	Typhoid

401/

No.	Race	Sex	Age	Liver	Spleen	Glands	Alia	Cause of Death.
401	Zulu	F.	61	+++	+++	++	-	Aortic Incompetence
402	"	F.	34	+	++	-	-	Pulmonary tuberculosis
403	"	F.	28	+	+	-	-	Typhoid
404	"	F.	34	+++	+++	++	±	Pulmonary tuberculosis
405	"	M.	50	+++	+++	+++	±	Influenzal Broncho-pneumonia
406	"	M.	40	+++	++	+++	-	Uraemia
407	"	M.	27	++	++	++	-	Pulmonary tuberculosis
408	"	M.	38	-	+	-	-	Bronchiectasis Bilharzia
409	"	M.	54	++	++	++	-	Pulmonary tuberculosis
410	"	M.	20	++	++	++	-	Nephritis
411	"	M.	32	o	o	o	o	Septic Broncho-pneumonia
412	Msutu	M.	40	o	o	o	o	Arteriosclerosis
413	"	M.	9/12	-	-	-	-	Pulmonary abscess
414	"	M.	25	++	++	-	-	Cerebro-spinal meningitis
415	"	M.	25	++	++	-	-	Lobar pneumonia
416	"	M.	50	+++	+++	++	-	Amoebiasis
417	"	F.	4/12	-	-	-	-	Enteritis
418	"	M.	25	+++	+++	±	-	Nephritis. Cirrhosis
419	"	M.	45	+++	+++	++	-	Pulmonary tuberculosis; cirrhosis
420	"	M.	45	++	++	++	-	Arteriosclerosis - Cardiac failure
421	"	F.	1	-	-	-	-	Broncho-pneumonia
422	"	M.	36	+++	+++	++	-	Tuberculo-silicosis - cirrhosis
423	Barotse	M.	30	o	o	o	o	Tuberculous pericarditis
424	Zulu	M.	30	++	++	+	-	Arteriosclerosis
425	"	F.	15	++	++	+	-	Typhoid

426/

No.	Race	Sex	Age	Liver	Spleen	Glands	Alia	Cause of death.
426	Zulu	M.	35	++	++	+	-	Generalised tuberculosis
427	"	F.	25	++	++	-	-	Typhoid
428	"	M.	28	++	++	+	-	Tabes Mesenterica
429	"	F.	17	++	++	-	-	Septic Endometritis - Peritonitis
430	"	M.	6/52	+++	++	+	-	Acute Oesophagitis (Scald)
431	Xosa	M.	32	+	+	-	-	Lobar pneumonia
432	"	M.	45	o	o	o	o	Emphysema - Cardiac failure
433	"	M.	39	+++	+++	++	-	Carcinoma of Liver
434	"	M.	18	++	++	-	-	Typhoid
435	"	M.	45	+++	+++	++	-	Generalised tuberculosis
436	"	M.	18	+	+	-	-	Emphysema
437	"	F.	10/12	-	-	-	-	Enteritis
438	Zulu	M.	35	+++	+++	++	-	Cerebral abscess - subcortical
439	Fingo	M.	40	+++	+++	++	-	Pulmonary tuberculosis
440	"	M.	57	+++	+++	+++	+	Carcinoma of liver - Cirrhosis
441	"	M.	29	+	±	-	-	Influenza
442	"	M.	15	++	++	+	-	Typhoid
443	"	M.	22	++	++	±	-	Typhoid
444	"	F.	45	++	++	++	-	Lobar pneumonia
445	"	M.	35	++	++	++	-	Influenza
446	"	F.	6	++	++	-	-	Typhoid
447	Xosa	M.	45	++	++	++	-	Pulmonary tuberculosis
448	Msutu	M.	35	++	++	+	-	Tuberculo-silicosis
449	"	M.	30	+	++	+	-	Tuberculous Meningitis
450	"	M.	25	++	++	++	-	Generalised tuberculosis

451/

No.	Race	Sex	Age	Liver	Spleen	Glands	Alia	Cause of Death.
451	Msutu	M.	11/12	-	-	-	-	Tuberculous peritonitis
452	"	M.	1½	-	-	-	-	Tuberculous Meningitis
453	"	M.	21	++	++	-	-	Typhoid
454	"	M.	40	++	++	++	-	Lobar pneumonia
455	"	M.	1.5/12	++	+	+	-	Broncho-pneumonia
456	"	M.	26	++	++	++	-	Subacute Nephritis
457	"	M.	45	++	++	++	-	Lobar pneumonia
458	"	M.	36	++	++	++	-	Tuberculous Broncho-pneumonia
459	"	M.	17	+	+	-	-	Typhoid
460	"	M.	40	++	++	+	-	Influenza
461	"	M.	39	++	++	++	-	Pulmonary tuberculosis
462	"	M.	30	++	++	++	-	Lobar pneumonia
463	"	M.	32	++	++	-	-	Septicaemia
464	"	M.	40	++	++	++	-	Aneurysm
465	"	M.	28	++	++	++	-	Lobar pneumonia
466	Zulu	M.	36	++	++	-	-	Typhoid
467	"	F.	6/12	+	+	-	-	Capillary bronchitis - enteritis
468	"	M.	6/12	+	+	-	-	Enteritis
469	"	F.	5/12	+	+	-	-	Enteritis
470	"	M.	9	++	++	-	-	Typhoid
471	"	M.	?	++	++	-	-	Cerebral haemorrhage
472	"	M.	7	-	-	-	-	Amoebic Dysentery
473	"	F.	4	-	-	-	-	Pulmonary tuberculosis
474	"	M.	55	++	++	+	-	Lobar pneumonia
475	"	M.	35	++	++	+	-	Typhoid

476/

No.	Race	Sex	Age	Liver	Spleen	Glands	Alia	Cause of Death.
476	Zulu	F.	26	+	+	-	-	Nephritis
477	"	F.	24	++	++	+	-	Pulmonary tuberculosis
478	"	F.	21	+	+	-	-	Aortic and Mitral disease
479	"	M.	2	-	-	-	-	Typhoid
480	"	F.	7/12	-	-	-	-	Enteritis
481	"	M.	1	-	-	-	-	Oesophagitis - Broncho-pneumonia
482	"	M.	35	++	++	++	-	Lobar pneumonia
483	"	M.	45	++	++	++	-	Generalised tuberculosis
484	"	M.	60	++	++	++	-	Pulmonary tuberculosis
485	"	F.	3	+	+	-	-	Pneumococcal meningitis
486	"	F.	34	++	++	+	-	Pulmonary tuberculosis with enteritis
487	"	F.	30	+	+	-	-	Pulmonary tuberculosis
488	"	F.	25	++	++	+	-	Pulmonary tuberculosis
489	"	F.	38	++	++	-	-	Typhoid with perforation
490	"	M.	30	++	++	-	-	Typhoid with pneumonia
491	Bechuana	M.	21	±	±	-	-	Typhoid with peritonitis
492	Zulu	M.	60	+	++	-	-	Lobar pneumonia
493	"	M.	2	-	-	-	-	Broncho-pneumonia
494	"	M.	36	+	+	-	-	Aortic endocarditis
495	"	F.	23	+	+	-	-	Carcinoma thyroid
496	"	M.	45	++	++	+	-	Nephritis - uraemia
497	"	M.	37	++	++	++	-	Pulmonary tuberculosis
498	"	M.	32	+	+	-	-	Typhoid with splenic abscess
499	"	M.	28	+	+	-	-	Amoebic dysentery
500	"	F.	31	+++	+++	+++	-	Cerebral softening with tuberculosis

501/

No.	Race	Sex	Age	Liver	Spleen	Glands	Alia	Cause of Death.
501	Zulu	M.	42	+++	+++	+++	+	Pulmonary tuberculosis
502	"	M.	32	++	++	-	-	Ulcerative endocarditis (aortic)
503	"	F.	19	++	++	±	-	Tuberculous peritonitis
504	"	M.	28	++	+	++	-	Generalised tuberculosis
505	Basuto	M.	38	+++	+++	+++	±	Appendix abscess with pylephlebitis
506	"	M.	40	+	++	-	-	Lobar pneumonia
507	"	F.	28	+	+	-	-	Carcinoma of groin
508	"	F.	6/12	-	-	-	-	Gastro-enteritis - Broncho-pneumonia
509	"	F.	36	±	±	-	-	Tuberculous enteritis
510	Msutu	M.	33	+++	+++	+++	-	Tabes mesenterica
511	"	M.	32	++	++	++	-	Tuberculous meningitis
512	Fingo	M.	5	-	+	-	-	Bacillary dysentery
513	Msutu	M.	1	-	-	-	-	Gastro-enteritis - Broncho-pneumonia
514	"	M.	16	-	-	-	-	Aortic endocarditis
515	"	M.	28	+	+	±	-	Typhoid
516	Zulu	M.	11/12	-	+	-	-	Broncho-pneumonia with ricketts
517	"	F.	60	-	+	-	-	Carcinoma thyroid
518	"	M.	32	+	+	±	-	Typhoid
519	Fingo	M.	54	+++	+++	+++	+	Lobar pneumonia with abscess
520	Msutu	F.	26	-	-	-	-	Cerebro-spinal meningitis
521	Zulu	M.	22	++	++	-	-	Pneumococcal meningitis
522	"	M.	48	+++	+++	+++	±	Pyonephrosis
523	Msutu	M.	7/12	-	-	-	-	Gastro-enteritis
524	Xosa	F.	25	+++	+++	+++	±	Pulmonary tuberculosis
525	Msutu	F.	20	-	-	-	-	Ulcerative endocarditis

526/

No.	Race	Sex	Age	Liver	Spleen	Glands	Alia	Cause of Death.
526	Msutu	M.	45	++	++	++	-	Tuberculo-silicosis with tuberculous enteritis
527	"	M.	6	++	++	+	-	Subacute Nephritis
528	"	M.	28	+	++	-	-	Cellulitis scrotum
529	"	M.	46	++	++	±	-	Tuberculo-silicosis
530	Zulu	F.	3½	±	±	-	-	Typhoid
531	Swazi	M.	42	++	++	++	+	Pulmonary tuberculosis with Scurvy
532	Msutu	M.	4/12	-	-	-	-	Broncho-pneumonia
533	"	M.	25	++	++	+	-	Typhoid
534	Xosa	F.	46	++	++	++	-	Pulmonary tuberculosis
535	"	M.	30	++	++	+	-	Typhoid
536	Msutu	M.	34	++	++	++	-	Lobar pneumonia
537	"	M.	30	++	++	±	-	Lobar pneumonia
538	"	F.	25	+	+	±	-	Pulmonary tuberculosis
539	"	F.	11/12	-	+	-	-	Amoebic dysentery
540	"	F.	27	+	+	-	-	Pulmonary tuberculosis
541	Zulu	F.	17	+	+	-	-	Generalised tuberculosis
542	Fingo	M.	2/12	-	-	-	-	Gastro-enteritis
543	Msutu	M.	35	++	++	+	-	Generalised tuberculosis
544	"	F.	3/52	+	+	-	-	Broncho-pneumonia
545	Swazi	M.	48	+++	+++	++	-	Generalised tuberculosis
546	Ndebele	F.	17	-	-	-	-	Mitral disease
547	Zulu	M.	25	+	+	-	-	Lympho sarcoma
548	"	M.	38	++	++	++	-	Gangrene of lung
549	Msutu	M.	28	+	+	-	-	Lobar pneumonia
550	Katanga	M.	27	++	++	-	-	Generalised tuberculosis
551	"	M.	20	+	+	-	-	Typhoid

552/

No.	Race	Sex	Age	Liver	Spleen	Glands	Alia	Cause of Death.
552	Katang	M.	30	++	++	++	-	Tuberculosis, lungs prostate etc.
553	Xosa	F.	68	+++	+++	++	+	Generalised tuberculosis
554	"	M.	28	+	+	-	-	Cerebral haemorrhage
555	"	M.	47	++	++	++	-	Lobar pneumonia
556	Zulu	M.	45	+++	+++	+++	-	Lobar pneumonia
557	"	M.	17	+	+	+	-	Influenzal broncho-pneumonia
558	"	F.	24	+	+	+	-	Typhoid
559	"	F.	50	++	++	++	-	Cerebral haemorrhage
560	"	M.	34	++	++	+	-	Pulmonary tuberculosis with miliary
561	Ndebele	M.	25	+	+	-	-	Lobar pneumonia
562	Shangaan	M.	10	-	-	-	-	Aortic and Mitral disease
563	Zulu	F.	24	-	-	-	-	Broncho-pneumonia
564	Msutu	F.	21	±	±	-	-	Nephritis; pontine haemorrhage
565	"	F.	7	-	-	-	-	Sarcoma of skull
566	Fingo	M.	34	++	++	+	-	Amoebiasis - liver, lung, brain
567	"	M.	40	++	++	+	-	Pott's disease with generalised tuberculosis
568	"	M.	45	+++	++	++	-	Tuberculous pleurisy
569	"	M.	40	++	++	±	-	Influenza
570	Mozambique	M.	25	++	++	+	-	Generalised tuberculosis
571	Zulu	F.	33	+++	+++	++	+	Pelvic abscess
572	"	F.	20	-	+	-	-	Typhoid
573	"	M.	36	++	++	+	-	Generalised tuberculosis
574	Fingo	M.	29	++	++	+	-	Pulmonary tuberculosis
575	Zulu	M.	58	+++	+++	+++	++	Splenic abscess
576	Swazi	M.	50	++	++	++	-	Pulmonary tuberculosis

577/

No.	Race	Sex	Age	Liver	Spleen	Glands	Alia	Cause of Death.
577	Swazi	F.	6	-	-	-	-	Pulmonary tuberculosis
578	"	M.	25	++	++	-	-	Lobar pneumonia
579	Katanga	M.	30	++	++	-	-	Cerebro-spinal meningitis
580	Msutu	F.	28	++	+	-	-	Tuberculous peritonitis
581	Zulu	M.	34	++	++	-	-	Mediastinitis - tuberculosis
582	Msutu	F.	6	+	+	-	-	Tuberculoma - brain
583	"	F.	38	+	+	-	-	Post-operative urinary obstruction
584	Morolong	F.	28	-	-	-	-	Lobar pneumonia
585	Msutu	M.	50	++	++	-	-	Dislocation of atlas - meningitis
586	Shangaan	M.	44	++	++	-	-	Lobar pneumonia
587	Zulu	M.	27	+++	++	+	-	Cirrhosis of liver with carcinoma
588	"	F.	38	++	++	-	-	Carcinoma of breast
589	"	M.	26	+++	+++	+++	-	Cirrhosis of liver - carcinoma
590	"	M.	36	++	++	-	-	Mitral disease
591	Msutu	M.	24	++	++	-	-	Typhoid with perforation
592	"	F.	3.2/12	-	+	-	-	Broncho-pneumonia
593	Zulu	M.	16	+	+	-	-	Broncho-pneumonia
594	Fingo	M.	7/12	-	-	-	-	Enteritis
595	Msutu	M.	20	±	±	-	-	Typhoid
596	"	M.	13	-	-	-	-	Mitral disease
597	Xosa	M.	45	+++	+++	+++	-	Arteriosclerosis
598	Mchopi	M.	50	++	++	++	-	Pachymeningitis haemorrhagica.
599	Zulu	M.	49	+++	+++	+++	±	Malaria
600	Msutu	M.	36	±	±	-	-	Miliary tuberculosis with meningitis

601/

No.	Race	Sex	Age	Liver	Spleen	Glands	Alia	Dause of Death.
601	Msutu	M.	30	++	++	+	-	Pulmonary tuberculosis
602	"	M.	40	++	++	+	-	Cerebro-spinal meningitis
603	"	M.	47	++	++	+	-	Lobar pneumonia
604	"	M.	56	++	++	+	-	Arteriosclerosis
605	"	M.	26	++	++	-	-	Lobar pneumonia - bilharzia
606	"	M.	43	++	++	++	-	Lobar pneumonia
607	"	F.	19/365	-	-	-	-	Gastro-enteritis
608	"	M.	72	-	-	-	-	Emphysema - bronchitis
609	"	M.	35	±	±	-	-	Tuberculo-silicosis
610	"	M.	25	++	++	-	-	Lobar pneumonia
611	"	M.	5/12	-	-	-	-	Broncho-pneumonia and pericarditis
612	"	F.	4/12	-	-	-	-	Pulmonary tuberculosis
613	"	M.	38	++	++	+	-	Arteriosclerosis - pericarditis
614	"	M.	29	+	++	+	-	Lobar pneumonia
615	"	M.	38	++	++	++	-	Tuberculous peritonitis
616	"	M.	18	++	++	-	-	Pulmonary tuberculosis
617	Zulu	F.	25	-	++	-	-	Nephritis
618	Xosa	F.	36	++	++	-	-	Nephritis
619	"	M.	37	++	++	++	-	Arteriosclerosis
620	"	M.	52	++	++	+	-	Arteriosclerosis
621	"	M.	23	++	++	-	-	Lobar pneumonia
622	"	M.	28	++	++	+	-	Cholangitis with biliary abscess
623	"	M.	40	++	++	+	-	Pulmonary tuberculosis
624	"	M.	12	-	-	-	-	Pott's disease
625	"	M.	30	±	±	-	-	Aneurysm Aortic Incompetence

626/

No.	Race	Sex	Age	Liver	Spleen	Glands	Alia	Cause of Death.
626	Xosa	M.	30	++	++	+	-	Miliary tuberculosis
627	Msutu	M.	47	++	++	+	-	Haemorrhage into pancreas
628	"	F.	35	++	++	+	-	Cerebro-spinal meningitis
629	"	M.	30	++	++	+	-	Tuberculous meningitis
630	"	M.	30	++	++	+	-	Lobar pneumonia
631	"	F.	50	±	±	-	-	Thyroid carcinoma
632	Tembu	F.	48	+++	+++	++	-	Cerebral abscess
633	"	M.	24	++	++	++	-	Lobar pneumonia
634	Xosa	M.	45	++	++	++	-	Pulmonary tuberculosis
635	"	M.	35	++	++	-	-	Suppurative pyelonephritis
636	"	M.	42	++	++	+	-	Endocarditis
637	"	M.	50	++	+++	++	+	Tuberculous pleurisy
638	"	M.	25	-	+	-	-	Amoebic dysentery
639	"	M.	49	++	++	+	-	Generalised tuberculosis
640	"	M.	22	++	++	++	-	Bilharzial dysentery
641	"	M.	50	+++	+++	+++	+++	Carcinoma of Oesophagus
642	"	M.	33	++	++	+	-	Thrombosis - pulmonary artery
643	Zulu	M.	35	++	++	+	-	Tabes Mesenterica with peritonitis
644	"	M.	75	-	-	-	-	Pulmonary embolism
645	Shangaan	M.	60	++	++	++	-	Lobar pneumonia - Bilharzia
646	"	F.	25	±	+	-	-	Typhoid
647	Zulu	M.	54	++	++	++	-	Generalised tuberculosis
648	"	M.	2/12	-	-	-	-	Enteritis
649	"	M.	35	++	++	+	-	Nephritis
650	"	M.	6	+	-	-	-	Acute Nephritis

651/

No.	Race	Sex	Age	Liver	Spleen	Glands	Alia	Cause of Death.
651	Mchopi	M.	38	++	++	+	-	Pulmonary-tuberculosis - Generalised
652	Zulu	M.	40	++	+	+	-	Lobar pneumonia
653	"	M.	45	++	++	+	-	Bronchitis - broncho-pneumonia
654	Morolong	F.	3	-	-	-	-	Pulmonary tuberculosis
655	"	M.	32	++	++	+	-	Pulmonary tuberculosis
656	Swazi	M.	15	+	+	-	-	Abdominal tuberculosis
657	Morolong	M.	23	++	++	++	-	Generalised tuberculosis
658	Zulu	M.	32	++	++	++	-	Pulmonary tuberculosis with enteritis
659	Mozambique	M.	55	+++	+++	++	-	Tuberculous pericarditis
660	Msutu	M.	18	±	+	-	-	Pulmonary tuberculosis
661	"	F.	9/12	-	-	-	-	Capillary bronchitis
662	"	F.	9	-	-	-	-	Tuberculous broncho-pneumonia
663	"	M.	30	+	+	+	-	Tuberculous peritonitis
664	Zulu	M.	30	±	±	-	-	Arteriosclerosis - Cardiac failure
665	Shangaan	M.	36	++	++	++	-	Pulmonary tuberculosis
666	Xosa	F.	24	+	+	-	-	Mitral disease - Amoebic dysentery
667	Msutu	F.	20	+	+	-	-	Pulmonary tuberculosis
668	"	F.	1.5/12	-	-	-	-	Pulmonary tuberculosis
669	Zulu	M.	48	++	++	+	-	Pulmonary tuberculosis
670	Swazi	M.	29	++	++	+	-	Pulmonary tuberculosis
671	Ndebele	M.	27	+	+	-	-	Lobar pneumonia
672	Msutu	M.	24	+	+	-	-	Cerebro-spinal meningitis
673	Zulu	M.	71	++	++	++	-	Myocarditis
674	Msutu	M.	35	±	±	-	-	Influenza
675	Zulu	M.	36	++	++	++	-	Nephritis - Scurvy

676/

No.	Race	Sex	Age	Liver	Spleen	Glands	Alia	Cause of Death.
676	Zulu	F.	18	++	++	++	-	Meningoencephalitis
677	"	F.	26	++	++	++	-	Pulmonary tuberculosis
678	"	M.	42	+++	+++	+++	++	Lobar pneumonia
679	"	M.	80	++	++	++	±	Arteriosclerosis
680	"	M.	50	+++	+++	+++	+	Arteriosclerosis
681	"	M.	35	++	++	++	-	Pulmonary tuberculosis with pericarditis
682	"	M.	34	±	±	-	-	Lobar pneumonia
683	"	F.	15	-	-	-	-	Generalised tuberculosis
684	"	M.	43	++	++	++	-	Pulmonary tuberculosis
685	"	M.	1.6/12	-	-	-	-	Pulmonary tuberculosis
686	"	M.	1.8/12	-	-	-	-	Enteritis.
687	Msutu	F.	30	+	+	-	-	Generalised tuberculosis
688	Zulu	M.	23	+	-	-	-	Chronic Endocarditis
689	Msutu	M.	32	+	+	-	-	Nephritis
690	"	M.	4/12	-	-	-	-	Enteritis
691	Xosa	M.	68	++	++	+	-	Typhoid
692	"	F.	25	+	+	-	-	Operation - Ovarian Cyst
693	Zulu	M.	12	-	-	-	-	Influenza
694	Xosa	M.	46	++	++	+	+	Empyaema
695	Msutu	M	2	++	+	-	-	Typhoid
696	Xosa	M.	6	-	-	-	-	Meningitis
697	Tonga	F.	30	+++	++	++	-	Carcinoma of liver
698	"	M.	70	++	++	++	-	Empyaema
699	"	M.	32	+	+	-	-	Miliary tuberculosis
700	Msutu	M.	1½	-	-	-	-	Hydrocephalus

701/

No.	Race	Sex	Age	Liver	Spleen	Glands	Alia	Cause of Death.
701	Fingo	F.	15	±	±	-	-	Influenza
702	Swazi	M.	55	++	++	+	-	Subacute Nephritis
703	"	F.	1.9/12	-	-	-	-	Broncho-pneumonia.
704	"	M.	28	+	+	-	-	Influenzal-Broncho-pneumonia
705	Zulu	M.	56	++	++	+	±	Lobar pneumonia
706	"	M.	25	-	-	-	-	Lobar pneumonia
707	"	M.	56	++	+	+	-	Plague
708	"	M.	35	±	+	±	-	Pulmonary tuberculosis
709	"	M.	24	+	+	-	-	Typhoid
710	"	M.	20	-	-	-	-	Lobar pneumonia
711	"	M.	22	-	-	-	-	Typhoid
712	"	M.	38	++	+	-	-	Amoebic dysentery
713	"	M.	30	+	+	-	-	Lobar pneumonia
714	"	M.	22	+	+	-	-	Influenza
715	"	M.	40	++	++	+	-	Typhoid
716	Katanga	M.	40	++	++	++	-	Pulmonary tuberculosis
717	"	M.	55	++	++	++	-	Abdominal tuberculosis
718	"	M.	19	-	-	-	-	Operation
719	"	M.	23	±	±	-	-	Lobar pneumonia
720	"	F.	70	+++	+++	+++	+	Broncho-pneumonia
721	"	M.	30	+++	+++	+++	++	Carcinoma of liver
722	"	M.	45	++	+++	++	+	Dysentery
723	"	M.	25	±	±	-	-	Typhoid
724	"	M.	30	++	++	++	-	Malaria
725	"	M.	29	++	++	++	-	Dysentery

726/

No.	Race	Sex	Age	Liver	Spleen	Glands	Alia	Cause of Death.
726	Katanga	M.	36	++	++	++	-	Pulmonary tuberculosis
727	Xosa	F.	35	+++	+++	+++	++	Broncho-pneumonia
728	Msutu	F.	43	++	++	++	-	Tuberculous peritonitis
729	Xosa	F.	27	++	++	+	-	Cerebral embolism
730	"	M.	19	-	-	-	-	Pulmonary tuberculosis
731	"	F.	19	-	-	-	-	Influenza
732	Zulu	M.	25	+	+	-	-	Appemdicitis
733	Swazi	M.	21	++	++	++	-	Tuberculous peritonitis
734	Xosa	F.	11/12	-	-	-	-	Pulmonary tuberculosis
735	"	M.	37	+	++	+	-	Lobar pneumonia
736	"	M.	17	-	-	-	-	Endocarditis with cardiac failure
737	"	M.	25	++	++	+	-	Enteric - Pulmonary embolism
738	Zulu	F.	10/12	-	-	-	-	Broncho-pneumonia
739	Msutu	M.	50	+++	+++	++	±	Tuberculous pericarditis
740	"	M.	56	+++	+++	+++	++	Pontine haemorrhage
741	Zulu	M.	60	+++	+++	+++	++	Carcinoma pancreas
742	Msutu	M.	31	++	++	+	-	Aortic Incompetence
743	"	M.	25	++	++	-	-	Typhoid with perforation
744	Zulu	M.	35	++	++	±	-	Tuberculous meningitis
745	Msutu	M.	17	++	++	±	-	Tonsillitis - Septic pneumonia
746	"	M.	65	++	++	++	-	Chronic Nephritis - Carcinoma
747	"	M.	28	++	++	-	-	Haemorrhage from pancreas
748	"	M.	40	++	++	+	-	Pneumothorax
749	"	M.	38	++	++	++	-	Cerebellar abscess
750	"	M.	45	++	++	++	-	Emphysema - Cardiac failure

751/

No.	Race	Sex	Age	Liver	Spleen	Glands	Alia	Cause of Death.
751	Msutu	F.	2	-	-	-	-	Enteritis - Status Lymphaticus
752	Zulu	M.	32	++	++	-	-	Typhoid
753	"	F.	28	++	++	+	-	Pulmonary tuberculosis
754	Swazi	F.	21	-	+	-	-	Pulmonary abscess
755	Msutu	M.	17	++	++	-	-	Tuberculous meningitis
756	Zulu	M.	26	++	++	-	-	Typhoid
757	"	M.	2	-	-	-	-	Intussusception
758	"	M.	50	+++	+++	+++	-	Aortic Incompetence
759	Fingo	M.	16	+	±	-	-	Typhoid
760	Zulu	M.	22	±	+	-	-	Bronchiectasis
761	"	M.	27	+	+	-	-	Typhoid
762	Xosa	M.	38	++	++	+	-	Influenza
763	"	M.	36	++	++	+	-	Typhoid
764	Shangaan	M.	25	-	+	-	-	Lobar pneumonia
765	Zulu	M.	50	+++	+++	++	-	Influenza
766	"	F.	70	+++	+++	++	+	Mitral Incompetence
767	"	M.	19	-	-	-	-	Cerebro-spinal Meningitis
768	Msutu	F.	30	+	+	-	-	Nephritis
769	"	M.	18	++	+	-	-	Lobar pneumonia
770	"	M.	22	+	+	-	-	Bilharzia
771	"	M.	40	+++	+++	++	+	Malaria
772	Shangaan	M.	62	++	++	+	-	Arteriosclerosis
773	Zulu	M.	38	++	++	+	-	Myocarditis
774	Msutu	M.	26	+	+	-	-	Typhoid with haemorrhage
775	"	F.	2 4	-	-	-	-	Endocarditis (Heart Puncture)

776

No.	Race	Sex	Age	Liver	Spleen	Glands	Alia	Cause of Death.
776	Fingo	M.	52	+++	+++	++	-	Rupture of aneurysm of femoral artery
777	"	M.	45	+++	+++	+++	-	Pulmonary tuberculosis
778	Zulu	M.	23	++	++	+	-	Aplastic anaemia
779	"	M.	26	++	++	-	-	Typhoid with perforation
780	"	M.	40	++	++	++	-	Purulent bronchitis with broncho-pneumonia
781	"	M.	3	-	-	-	-	Glioma retina - operation
782	Morolong	M.	39	++	++	++	-	Cirrhosis of liver with carcinoma
783	Zulu	M.	19	-	-	-	-	Cirrhosis of liver with carcinoma
784	"	M.	30	++	++	++	-	Pulmonary tuberculosis etc.
785	"	M.	65	+++	+++	+++	±	Pulmonary tuberculosis etc.
786	Shangaan	M.	26	++	++	+	-	Tuberculosis Mediastinum with pericarditis
787	Msutu	M.	30	+	+	-	-	Typhoid - Cirrhosis
788	Fingo	F.	38	-	-	-	-	Puerperal septicaemia
789	Msutu	M.	44	++	++	±	-	Nephritis - bronchitis
790	Zulu	M.	33	+	+	-	-	Typhoid
791	"	M.	38	++	++	++	-	Rectoperitoneal sarcoma
792	"	M.	38	+++	+++	+++	-	Cirrhosis of liver, ascites etc.
793	"	F.	25	++	++	-	-	Pulmonary tuberculosis
794	"	M.	70	++	++	+	-	Aortic disease with pneumonia
795	"	M.	50	++	++	+	-	Influenza
796	Fingo	M.	3/12	-	-	-	-	Enteritis - Broncho-pneumonia
797	Msutu	M.	40	++	++	++	-	Pott's disease etc.
798	Shangaan	M.	40	++	++	++	-	Generalised tuberculosis
799	"	F.	24	-	-	-	-	Influenza
800	"	M.	60	o	o	o	o	Carcinoma of stomach, Metastasis liver

801/

No.	Race	Sex	Age	Liver	Spleen	Glands	Alia	Cause of Death.
801	Shangaan	M.	22	+	+	±	-	Lobar pneumonia
802	"	M.	29	+	+	±	-	Tuberculous bronhco-pneumonia
803	"	F.	30	+	+	+	-	Cystitis pyelonephritis
804	"	M.	41	++	++	++	-	Carcinoma of liver
805	Msutu	M.	20	+	+	-	-	Mitral Stenosis
806	Xosa	F.	2/52	-	-	-	-	Capillary bronchitis
807	Shangaan	M.	15	-	-	-	-	Suppurative meningitis
808	"	M.	30	+	+	-	-	Septic Meningitis - Bilharzia
809	Ndebele	M.	39	++	++	+	-	Confluent Broncho-pneumonia
810	Baca	M.	40	++	++	++	-	Lobar pneumonia - Meningitis
811	Msutu	M.	20	-	-	-	-	Typhoid - Broncho-pneumonia
812	Zulu	M.	65	++	++	++	-	Purulent bronchitis with tuberculosis
813	Swazi	M.	15/365	-	-	-	-	Broncho-pneumonia
814	Msutu	M.	22	++	++	-	-	Typhoid
815	Xosa	F.	27	+	+	-	-	Cirrhosis - Ascites - Haemorrhage
816	Pondo	M.	45	++	++	++	-	Pulmonary tuberculosis with enteritis
817	Msutu	M.	45	++	++	++	-	Pneumococcal Endocarditis, Arthritis and meningitis
818	Xosa	M.	49	++	++	++	-	Aneurysm of Aorta
819	Msutu	M.	34	++	++	+	-	Mastoiditis - Meningitis
820	Zulu	F.	24	+	+	-	-	Gangrenous endometritis
821	"	M.	26	+	+	+	-	Aortic and Mitral disease
822	"	M.	52	++	++	+	-	Carcinoma of Oesophagus
823	Msutu	F.	27	±	±	-	-	Transverse myelitis - Meningitis
824	Fingo	F.	40	+	+	+	-	Myelitis
825	Msutu	M.	30	±	±	-	-	Ulcerative endocarditis

826/

No.	Race	Sex	Age	Liver	Spleen	Glands	Alia	Cause of Death.
826	Zulu	F.	3/52	-	-	-	-	Toxic Necrosis of liver
827	"	M.	38	++	++	+	-	Carcinoma of bladder, pyonephrosis
828	Msutu	F.	20	-	-	-	-	Ectopic pregnancy; peritonitis
829	"	M.	55	++	++	+	-	Bronchitis, emphysema
830	"	M.	38	+	+	+	-	Generalised peritonitis
831	Swazi	F.	45	o	o	o	o	Influenza
832	Xosa	F.	30	o	o	o	o	Aortic Incompetence
833	Fingo	M.	39	+++	+++	+++	-	Tuberculo-silicosis with tuberculosis
834	Msutu	M.	37	++	++	++	-	Tuberculosis with broncho-pneumonia
835	"	M.	51	++	++	++	-	Pulmonary tuberculosis with enteritis
836	Misenda	M.	19	-	-	-	-	Pulmonary tuberculosis with status lymphaticus
837	Msutu	F.	22	-	-	-	-	Influenza with nephritis
838	Mozambique	M.	35	++	++	++	-	Pulmonary tuberculosis
839	"	F.	21	+	+	-	-	Pulmonary tuberculosis
840	Msutu	F.	55	++	++	++	-	Osteomyelitis - jaw - Pneumonia - Bilharzia
841	Xosa	M.	48	++	++	++	+	Myelogenous leukaemia
842	Shangaan	M.	40	++	++	+	-	Pulmonary tuberculosis
843	Xosa	F.	11	-	-	-	-	Aortic Mitral Endocarditis
844	Msutu	M.	44	++	++	+	-	Myocarditis
845	Morolong	F.	21	-	+	-	-	Puerperal septicaemia
846	Msutu	M.	50	++	++	++	-	Genito-urinary with generalised tuberculosis
847	Xosa	M.	49	++	++	++	-	Chronic Nephritis Tuberculous Mediastinitis
848	Msutu	M.	32	++	++	++	-	Broncho-pneumonia
849	Pondo	M.	65	++	++	++	-	Abdominal with Miliary tuberculosis
850	Msutu	M.	1	-	-	-	-	Olitis Media - Meningitis

851/

No.	Race	Sex	Age	Liver	Spleen	Glands	Alia	Cause of Death.
851	Pondo	M.	45	++	++	++	-	Pulmonary tuberculosis with abdominal
852	Morolong	M.	35	++	++	++	-	Lobar pneumonia
853	Zulu	F.	43	++	++	++	-	Cellulutis arm with septicaemia
854	"	M.	60	++	+++	++	-	Lobar pneumonia
855	"	F.	28	+	+	-	-	Lobar pneumonia
856	Bechuana	M.	23	+	+	-	-	Tuberculous pleurisy Abdominal tuberculosis
857	Msutu	M.	25	±	+	-	-	Pulmonary tuberculosis
858	"	M.	40	++	++	++	-	Tuberculous pleurisy
859	Shangaan	M.	36	++	++	++	-	Abdominal tuberculosis
860	Xosa	F.	40	++	++	+	-	Tumour of jaw - sarcoma ? Metastasis
861	Msutu	F.	9	-	-	-	-	Pulmonary embolism
862	Zulu	M.	38	++	++	++	-	Ulcerative endocarditis - Cerebral embolism
863	Shangaan	M.	46	++	++	++	-	Septic arthritis - Tuberculo-silicosis
864	Msutu	M.	32	+	+	-	-	Influenza
865	"	F.	2/12	-	-	-	-	Broncho-pneumonia
866	"	M.	45	++	++	++	-	Pott's disease with myelitis
867	Xosa	F.	38	++	++	+	-	Bacillary dysentery
868	Blantyre	M.	29	-	-	-	-	"Miliary" fever - Septicaemia
869	Msutu	F.	31	-	-	-	-	Puerperal septicaemia
870	Zulu	M.	22	±	+	±	-	Lobar pneumonia
871	"	F.	32	+	+	-	-	Influenza
872	Msutu	M.	36	++	++	+	-	Pulmonary tuberculosis - Influenza
873	Xosa	F.	33	-	+	-	-	Puerperal septicaemia - Endocarditis
874	Msutu	M.	35	++	-	+	-	Oedema glottidis - Ulceration larynx
875	"	M.	40	++	++	++	-	Aneurysm Aorta - Broncho-pneumonia
876	"	F.	?(104)	++	++	++	-	Carcinoma of ovaries - Peritonitis

Number of Cases of Haemochromatosis or of Extensive Haemosiderosis

No. 65	Female	40	Carcinoma of liver
" 99	Male	40	Influenzal nephritis
" 189	Male	37	Lobar pneumonia
" 216	Female	50	Puerperal septicaemia
" 223	Female	38	Generalised tuberculosis
" 233	Male	34	Suppurative pyelonephritis - Cirrhosis of liver
" 259	Male	45	Tuberculo-silicosis - Chronic Nephritis
" 283	Female	65	Arteriosclerosis - Cirrhosis of liver
" 303	Male	31	Lobar pneumonia
" 318	Male	56	Lobar pneumonia
" 323	Male	68	Pulmonary tuberculosis
" 324	Male	45	Pulmonary tuberculosis
" 399	Female	30	Influenza
" 440	Male	57	Carcinoma of liver - Cirrhosis
" 501	Male	42	Pulmonary tuberculosis
" 519	Male	54	Lobar pneumonia
" 553	Female	68	Generalised tuberculosis
" 571	Female	33	Pelvic abscess
" 575	Male	58	Splenic abscess
" 637	Male	50	Tuberculous pleurisy
" 641	Male	50	Carcinoma of oesophagus
" 678	Male	42	Lobar pneumonia
" 680	Male	50	Arteriosclerosis
" 720	Female	70	Broncho-pneumonia
" 721	Male	30	Carcinoma of liver
" 722	Male	45	Dysentery
" 727	Female	55	Haemochromatosis - Broncho-pneumonia
" 740	Male	56	Pontine haemorrhage
" 741	Female	60	Carcinoma of pancreas
" 766	Female	70	Mitral Incompetence
" 771	Male	40	Malaria
" 785	Male	65	Pulmonary tuberculosis

APPENDIX II.

The results of "fragility tests" on

50 European Cases

and

200 Native Cases

EUROPEANS.

No. of Case	Result.	No.of Case	Result.
1	0.44%	26	0.42%
2	0.43%	27	0.41%
3	0.41%	28	0.445%
4	0.415%	29	0.420%
5	0.44%	30	0.435%
6	0.42%	31	0.425%
7	0.435%	32	0.42%
8	0.435%	33	0.43%
9	0.41%	34	0.43%
10	0.415%	35	0.41%
11	0.420%	36	0.425%
12	0.415%	37	0.405%
13	0.42%	38	0.41%
14	0.43%	39	0.43%
15	0.43%	40	0.415%
16	0.415%	41	0.42%
17	0.41%	42	0.43%
18	0.425%	43	0.42%
19	0.41%	44	0.43%
20	0.40%	45	0.43%
21	0.40%	46	0.425%
22	0.42%	47	0.43%
23	0.41%	48	0.42%
24	0.43%	49	0.44%
25	0.425%	50	0.405%

Average fragility = 0.42%

Maximum fragility = 0.445%

Minimum fragility = 0.40%

SHANGAANS.

No.	Age	Result	No.	Age	Result
1	36	0.45%	29	23	0.445%
2	27	0.46%	30	30	0.465%
3	27	0.46%	31	38	0.455%
4	40	0.465%	32	38	0.45%
5	30	0.46%	33	40	0.43%
6	23	0.45%	34	20	0.45%
7	25	0.48%	35	30	0.455%
8	22	0.445%	36	23	0.41%
9	24	0.455%	37	23	0.435%
10	30	0.465%	38	24	0.415%
11	24	0.43%	39	25	0.45%
12	38	0.435%	40	30	0.45%
13	35	0.46%	41	25	0.42%
14	23	0.43%	42	32	0.42%
15	28	0.45%	43	21	0.42%
16	26	0.45%	44	27	0.437%
17	23	0.45%	45	22	0.45%
18	28	0.435%	46	20	0.40%
19	25	0.43%	47	37	0.455%
20	30	0.435%	48	25	0.428%
21	28	0.455%	49	22	0.445%
22	25	0.46%	50	26	0.46%
23	20	0.43%	51	27	0.437%
24	40	0.45%	52	22	0.44%
25	25	0.432%	53	30	0.435%
26	25	0.455%	54	28	0.45%
27	20	0.445%	55	38	0.45%
28	35	0.455%			

Average Age 27.78

Average fragility = 0.446%
Maximum fragility = 0.468%
Minimum fragility = 0.40%

Ages 20 - 30

Average fragility = 0.441%

Ages 30 - 40

Average fragility = 0.449%

XOSAS

NO.	Age	Result	No.	Age	Result
1	30	0.427%	20	45	0.435%
2	28	0.43%	21	25	0.435%
3	34	0.455%	22	22	0.415%
4	25	0.428%	23	22	0.44%
5	25	0.43%	24	27	0.45%
6	40	0.46%	25	32	0.44%
7	45	0.432%	26	22	0.43%
8	38	0.415%	27	30	0.435%
9	34	0.43%	28	28	0.43%
10	20	0.42%	29	27	0.46%
11	25	0.425%	30	24	0.43%
12	22	0.45%	31	48	0.45%
13	24	0.41%	32	22	0.43%
14	44	0.435%	33	40	0.455%
15	24	0.43%	34	23	0.435%
16	28	0.415%	35	24	0.432%
17	28	0.415%	36	30	0.40%
18	35	0.46%	37	22	0.425%
19	28	0.44%			

Average Age 29.46 years

Average fragility = 0.434%
Maximum fragility = 0.46%
Minimum fragility = 0.40%

Ages 20 - 30

Average fragility = 0.422%

Ages 30 - 40

Average fragility = 0.438%

Ages 40 - 50

Average fragility = 0.438%

MCHOPI

No.	Age	Result	No.	Age	Result
1	33	0.448%	15	23	0.455%
2	25	0.45%	16	23	0.432%
3	26	0.45%	17	20	0.40%
4	25	0.44%	18	30	0.42%
5	50	0.455%	19	38	0.435%
6	25	0.445%	20	30	0.44%
7	26	0.435%	21	23	0.44%
8	26	0.435%	22	25	0.445%
9	20	0.43%	23	46	0.41%
10	22	0.46%	24	22	0.43%
11	39	0.445%	25	40	0.445%
12	25	0.44%	26	28	0.45%
13	24	0.415%	27	24	0.438%
14	25	0.455%			

Average Age 27.55 years.

Average fragility = 0.439%
Maximum fragility = 0.465%
Minimum fragility = 0.40%

Ages 20 - 30

Average fragility = 0.439%

Over 30 years.

Average fragility = 0.439%

NYAMBAAN

No.	Age	Result	No.	Age	Result
1	22	0.43%	13	26	0.405%
2	25	0.435%	14	24	0.41%
3	22	0.43%	15	27	0.425%
4	23	0.43%	16	28	0.40%
5	28	0.43%	17	40	0.435%
6	23	0.426%	18	40	0.43%
7	25	0.41%	19	34	0.455%
8	25	0.44%	20	20	0.415%
9	24	0.445%	21	36	0.44%
10	28	0.41%	22	55	0.46%
11	22	0.425%	23	40	0.41%
12	28	0.43%			

Average Age 29.78 years

Average fragility = 0.427%
Maximum fragility = 0.46%
Minimum fragility 0.40%

Ages 20 - 30

Average fragility = 0.423%

Ages 30 - 40

Average fragility = 0.434%

PONDO

No.	Age	Result	No.	Age	Result
1	33	0.41%	12	24	0.432%
2	32	0.46%	13	24	0.445%
3	19	0.43%	14	25	0.45%
4	22	0.45%	15	32	0.42%
5	35	0.45%	16	25	0.415%
6	22	0.44%	17	27	0.43%
7	25	0.455%	18	39	0.43%
8	30	0.43%	19	36	0.44%
9	30	0.44%	20	24	0.44%
10	32	0.45%	21	28	0.425%
11	22	0.455%			

Average Age 27.9 years

Average fragility = 0.438%
Maximum fragility = 0.46%
Minimum fragility = 0.41%

Ages 20 - 30

Average fragility= 0.439%

Ages 30 - 40

Average fragility = 0.436%

MSUTU

No.	Age	Result	No.	Age	Result
1	30	0.43%	11	45	0.445%
2	23	0.44%	12	38	0.455%
3	27	0.435%	13	30	0.445%
4	28	0.45%	14	26	0.45%
5	32	0.43%	15	30	0.43%
6	20	0.455%	16	25	0.43%
7	27	0.415%	17	26	0.42%
8	26	0.46%	18	37	0.40%
9	26	0.43%	19	25	0.42%
10	26	0.445%	20	26	0.415%

Average Age 28.65 years

Average fragility = 0.434%
Maximum fragility = 0.46%
Minimum fragility = 0.40%

Ages 20 - 30

Average fragility = 0.435%

Above 30 years

Average fragility = 0.433%

SWAZI

No.	Age	Result
1	26	0.45%
2	28	0.443%
3	28	0.405%
4	30	0.43%
5	30	0.43%
6	43	0.48%
7	26	0.46%

BECHUANA

No.	Age	Result
1	38	0.415%
2	28	0.46%
3	25	0.46%
4	24	0.40%
5	35	0.435%
6	22	0.42%

Fingo	1	32	0.43%
Tonga	1	24	0.43%
Molotse	1	34	0.42%
Unknown	1	28	0.42%

APPENDIX III

The incidence of haemofuscin in a short series of Native Cases.

No.	Sex	Age	Haemofuscin	Haemosiderin
1	F.	38	+	+
2	F.	3/52	-	-
3	M.	44	+	+
4	M.	35	+	+
5	M.	50	+	+
6	M.	36	+	+
7	M.	36	-	±
8	M.	46	+	+
9	F.	27	+	±
10	F.	40	+	±
11	F.	?	±	-
12	M.	30	±	-
13	M.	-	+	+
14	M.	30	+	+
15	M.	-	±	-
16	F.	33	+	+
17	F.	32	±	±
18	M.	60	+	+
19	F.	21	+	+
20	M.	55	+	+
21	M.	30+	-	-
22	M.	19	±	±
23	M.	38	+	+
24	M.	31	+	+
25	F.	30	+	+
26	M.	40	+	+
27	M.	39	+	+
28	M.	37	+	+
29	M.	21	+	+
30	M.	20	+	±

Fifteen other cases, mine boys were tested; their ages are not available but the majority were under thirty, all of them showed traces of haemofuscin, four showed appreciable quantities.

APPENDIX NO. IV.

Abstract of Experiments on

Rabbits with metallic salts (Tin, Zinc, Copper)

Rabbit No. 1:- (Stannous Chloride with Methylated Spirit).

Animal kept under observation and weighed daily for a fortnight before experiment begun.

Experiment begun on 9/11/26 when animal weighed 3,310 gms.

Rabbit fed on ordinary diet for laboratory animals and in addition received solution of tin chloride six days of the week, and methylated spirits on two days of the week.

Daily dose of tin chloride solution was 0.1 c.c. of a ten per cent solution of stannous chloride; this was made up to 1 c.c. in a pipette with distilled water. The dose was administered by pipette into the rabbit's mouth and care taken to see that the whole dose was swallowed.

The amount of tin in the daily dose was 5.3 mgms. The dose of methylated spirits administered was 1 c.c. The total quantity of tin administered throughout the experiment was 1.054 gms. The total amount of methylated spirit administered was .32 c.cs.

The experiment ended on 11/7/27 when animal weighed 3,220 gms.

The/

The animal was anaesthetised and killed with chloroform.

Post-mortem examination was carried out at once.

No obvious abnormality was detected. Tissues were fixed in formalin and sections taken for histological examination.

Rabbit No. 2:- (Zinc chloride with Methylated Spirit)

Animal kept under observation and weighed daily for a fortnight before experiment begun.

Experiment begun on 9/11/26 when animal weighed 2.400 gms.

Rabbit fed on ordinary diet and in addition received solution of zinc chloride six days of the week and methylated spirits on two days of the week.

Daily dose of zinc chloride solution was 0.1 c.c. of a ten per cent solution of zinc chloride; this was made up to 1 c.c. in a pipette with distilled water. The dose was administered by pipette as in the case of Rabbit 1.

The amount of zinc in the daily dose was 4.8 mgms. The dose of methylated spirits was 1 c.c.

The total quantity of zinc administered was 0.835 gm. The amount of methylated spirit administered was 24 c.cs.

During the experiment the rabbit became pregnant

and/

and gave birth to four young that survived and thrived.

The zinc administration was stopped from 4/12/26 to 6/1/27. when it was recommenced.

The experiment ended on 11/7/27 when the animal weighed 2560 gms.

The animal was anaesthetised and killed with chloroform.

Post-mortem examination was carried out at once. No obvious abnormality was detected.

Tissues were fixed in formalin and sections taken for histological examination.

Rabbit No. 3:- (Copper Sulphate with Methylated Spirit)

Kept under observation for a period before experiment begun.

Experiment begun on 9/11/26 when animal weighed 2,180 gms.

Fed on ordinary diet and in addition given daily 0.1 c.c. of 10% solution of copper sulphate on six days of the week; also 1 c.c. of methylated spirit on two days of the week.

Amount of copper in the dose of copper sulphate solution was 2.5 mgms.

12 doses given making total of 30 mgms in 12 days.

Animal died on 25/11/26 when it passed blood in the urine.

Post-mortem/

Post-mortem examination within half an hour of death revealed a haemorrhagic nephritis.

From this it was assumed that the dose of copper sulphate was too toxic for the animal concerned and consequently in experiments 6 and 7 the dose was diminished.

Rabbit No. 4 :- (Stannous Chloride).

This animal was one of the four young from rabbit 2, and it was kept under observation until experiment begun on 13/7/27. Unfortunately ten days after the commencement, its hind leg was broken and the animal was anaesthetised and killed

It was receiving a daily dose of 5.3 mgms of tin in the form of stannous chloride. No alcohol was given. Tissues were taken for examination as in the other rabbits.

Rabbit No. 4A.:- (Stannous Chloride).

This animal was substituted for No. 4.

The experiment began after a preliminary period of observation on 10/8/27 when the animal weighed 1950 gms.

The animal was fed on ordinary diet and in addition received 5.3 mgms of tin in the form of stannous chloride daily on six days of the week.

The experiment lasted till the 7/9/28 when the animal was anaesthetised and killed by chloroform. It then weighed 2,250 gms and was in good condition.

Post-mortem/

Post-mortem examination was made at once and the tissues fixed in formalin. Sections were prepared for histological examination.

The total amount of tin administered to the rabbit over the period of thirteen months was 1.765 gms.

Rabbit No. 5:- (Methylated Spirit)

Experiment was begun on 13/7/27 when the animal weighed 2,100 gms.

In addition to the ordinary diet this rabbit received twice weekly a dose of 1 c.c. of methylated spirit. administerd by pipette as in the other rabbits.

The total amount of spirit given was 123 c.cs. The experiment was ended on 6/9/28 when the animal weighed 2,100 gms and was in good condition.

Post-mortem was performed immediately and tissues preserved in formalin for histological examination.

No abnormality was detected macroscopically.

Rabbit No. 6:- (Copper sulphate with Methylated Spirit)

This animal was kept under observation from birth and experiment was begun when it was about seven months old.

It then weighed 1,400 gms.

In addition to ordinary diet it received daily

for/

for six days of the week 0.1 c.c. of a one per cent solution of copper sulphate and twice a week 1 c.c. of methylated spirit. The experiment lasted till the 21/9/28 when the animal was anaesthetised and killed by chloroform; it then weighed 1,690 gms.

After 49 doses of the copper sulphate the animal became sickly and the experiment was stopped for two weeks and then recommenced.

The daily dose of cooper was 0.25 mgm.

The total amount of copper administered was 86.5 mgms.

The total amount of spirit administered was 94 c.c.

No obvious abmormality was detected post-mortem and tissues were preserved in formalin for histological examination.

Rabbit No. 7:- (Copper Sulphate)

This animal was also kept under observation from birth and the experiment commenced when it was about seven months old on the 13/7/27. when it weighed 1,420 gms.

Apart from the methylated spirit which was not administered, this experiment was the same as in No. 6 with the period of intermission after 49 doses of the Copper sulphate.

The/

The daily dose of copper was 0.25 mgm.

The total amount administered was 86.5 mgm.

The animal was anaesthetised and killed with chloroform on 21/9/28 when it weighed 2,120 gms. At the post-mortem no obvious abnormality was detected and the tissues were preserved in formalin for histological examination.

APPENDIX V.

Copy of Information received from Criminal Investigation Department, Johannesburg, by Professor J.M. Watt, Pharmacology Department, University of the Witwatersrand.

KAFFIR BEER: "Umgomboti".

The beer in question is made of Kaffir corn and mealie meal.

The Kaffir corn is soaked for two or three days and when the grain commences to sprout, it is taken out of the water and allowed to dry - sun dried for preference.

This is then known as "Umtombo" or malt; the malt is thence ground into coarse meal, the grain being merely broken, this is then cooked into a thick porridge, when cooked it is placed into drums or barrels and allowed to cool off. When cool a very soft porridge of raw mealie and Kaffir corn meal is added, thence the necessary proportion of luke warm water according to the quantity of the Malt or Umtombo.

It is then allowed to stand for two or three days fermenting; after the fermentation it is strained and fit for consumption.

The liquor in question is a favourite beverage for the Cape Colony natives, Zulus and Swazis and even Basutos.

SKOKIAN.

Skokian is prepared by means of various yeasts such as the yeast obtained at the Breweries, Hattingh's yeast and compressed yeast.

A cake or two of yeast placed into a 4 gallon tin of hot water, sugar and syrup added and allowed to stand about four hours until it commences to ferment - an airtight utensil is preferable, as this causes a quicker fermentation. A quantity of Kaffir corn malt tied in a cloth and placed into Skokian gives it a better taste or kick.

Methylated spirits, pineapples, grapes and stale bread are also added.

QALI/

QALI.

Qali is prepared by means of a Hottentot fig known as "Insema"; the root is very bitter and is boiled several times to reduce its strength and bitterness, when the strength is sufficiently reduced, either black or white sugar and syrup are added.

About 4 gallons of luke warm water are added and allowed to cool off, the process taking about 24 hours, when it is ready for consumption.

The taste of Qali is bitter and it is a highly intoxicating beverage.

SIGOMFANA.

Two blocks of yeast and about 5 to 6 pounds of white sugar or black sugar to 4 gallons of luke warm water.

A rag containing Kaffir corn malt is placed into the utensil when fermenting, the object being to give it a Kaffir corn flavour, pineapple and even boiled potatoes are added, this is done merely for flavouring purposes.

Sigomfana is a highly intoxicating drink.

White Sigomfana is also prepared as Sigomfana - white sugar only being used.

The principal ingredients are yeast and white sugar; this liquor is also intoxicating, the strength depends on the amount of the yeast added.

The time allowed is 5 to 6 hours for fermentation; this is known as "Igwebu" and White Segumfana.

Copy of Notes from Carl Faye,

Department of Native Affairs, Maritzburg.

is'ko'keyana (isi'ko'keyana, ejective k).	yeast inpregnated froth, obtained from the Breweries of white people, from which a liquor is made by Natives by mixing with water and sugar or syrup. The water is first warmed, and then thoroughly sweetened with the sugar or syrup, before the yeastly froth is added. The name is used for both the froth and the liquor.
igwebu (iligwebu implosive b).	the same.
insema	bulbous root of a ground euphorbia (E.pugniformis and E.bupleurifolia, according to Bryant). The bulb is skinned, pounded and dried and then ground into a vile potent drink by mixing with warm water sweetened with sugar or syrup. Insema is the universal Zulu name for the bulb (each plant has one bulb only; it is largely used for a popular game among Zulu boys); the name has now also come into use for designating the liquor made from it.
isqat'avit'i (isiqat'avit'i aspirated t).	the liquor made from the insema. Name first used as euphemism for insema-liquor.
isk'welek'anda (isik'welek'anda aspirated k).	the liquor made from the insema. First used as euphemism.
umqombot'i (umqombot'i aspirated t).	X'oza and Sut'u for Zulu u'cwala, beer.
isgomfane (isgomfane)	Euphemism for is imeyana.
umzexa (umuzexe)	the same.
iq'ali (iliq'ali)	the same - vowels as in imali, money.
igcobozi (iligcobozi)	Swazi amabele beer.

The/

The meaning of these names are self-evident, but to save the trouble of writing them out, I give them here as briefly as possible

is'ko'keyana from 'ko'ka = 'ka'ka,= surround, hence that which surrounds or isolates the senses; the ending eyana indicates a subtle agent here.

igwebu = froth.

insema is descriptive of the bulb.

isqat'avit'i = that which is blinding and over-powering, from qat'a, arrive at, reach, alight upon (the eyes, understood), and vit'i (vit'iza) that which renders totally helpless.

isk'welek'anda = that which climbs upon the head, "goes to the head".

umqombot'i = that which makes the belly protude.

isgomfane = that which bows down (a person, understood, inducing prostration).

umzexe = that which cuts; refers to the "bite" and "kick" of the liquor.

iq'ali = that which induces stiffness, a cataleptic condition.

igcobozi from the shape and size of the vessel in which it is served: fromgcoboza, dip.

is'imeyana ="Intoxicating drink made with treacle (the name corrupted from machine)" -- according to Colense.

isi imeyana The name in the Zulu mind is also associated with ukut'i shime, to thrust oneself into the grass, to plunge into a maze, bog.

APPENDIX VI.

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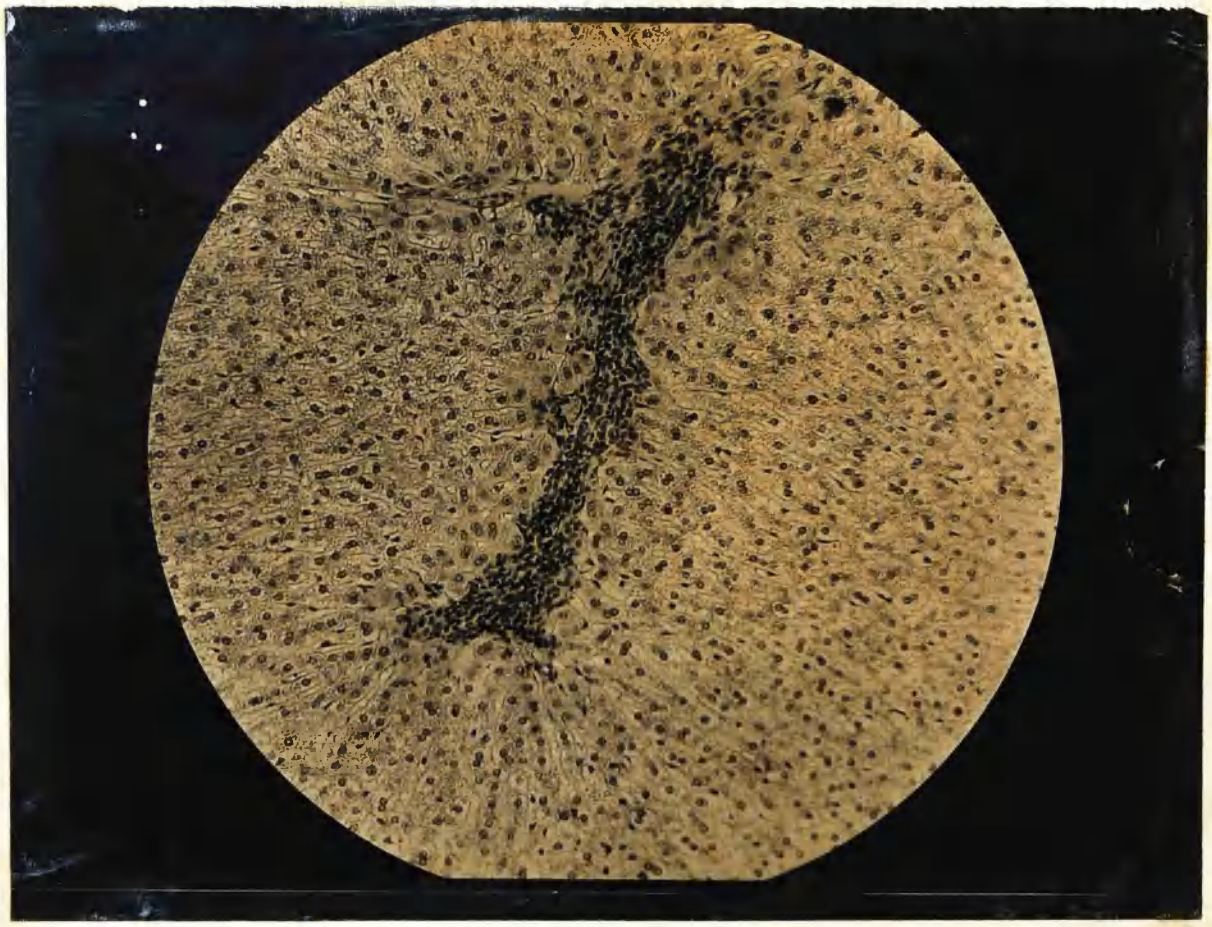
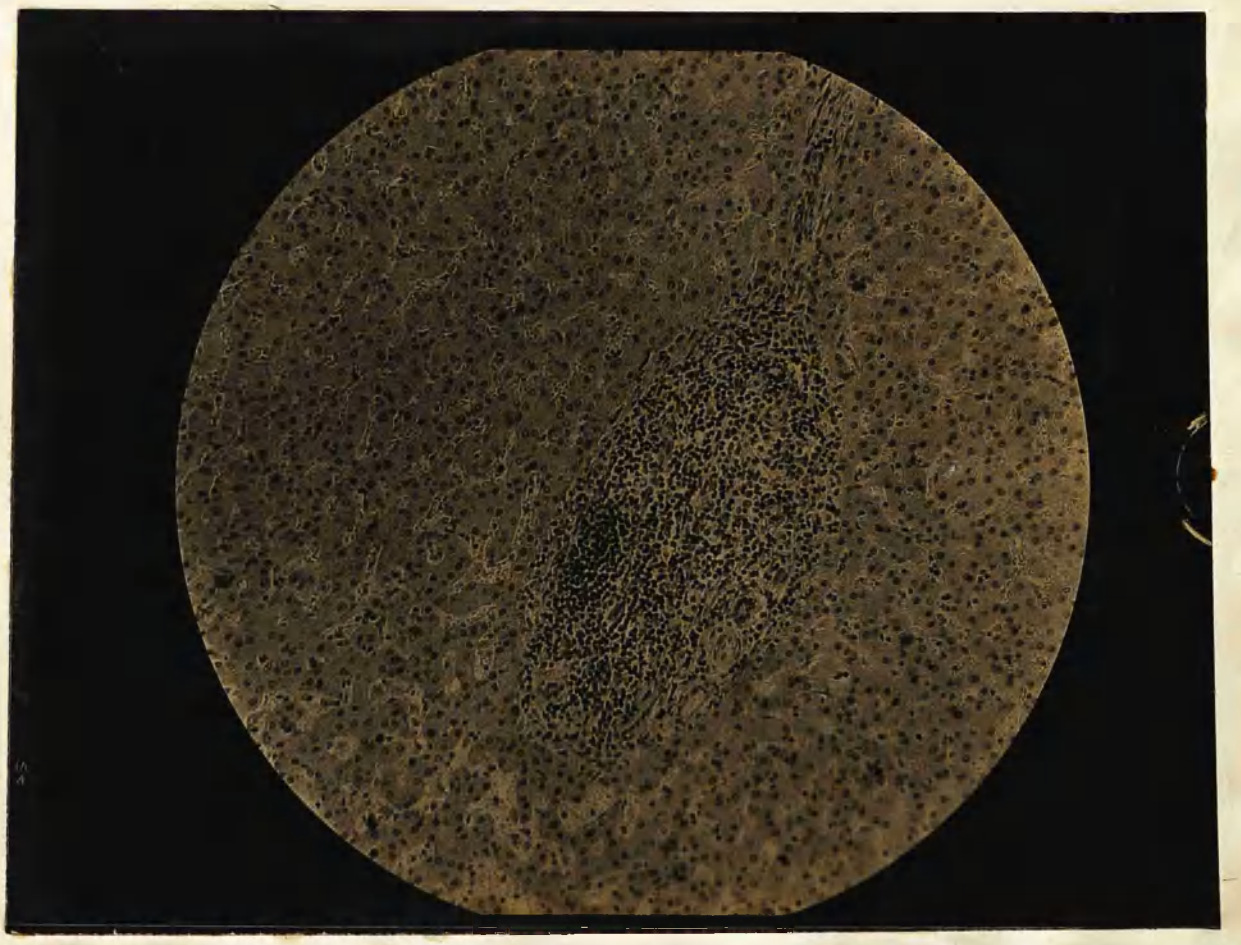


Section. Rabbit Liver. Zinc.



Section. Native Liver.