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The Patterns of Disease in Africa*

by

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I suppose it is only correct and proper, when addressing medical students, to confine my remarks to a medical subject and preferably one which deals with diseases on this Continent. It is true there is much to say, indeed so much to think and speak about, that I find it a little difficult to know on what aspects I should concentrate. Again my audience is a large one with varied interests, which also makes it hard to decide, but perhaps you would like to hear something about my practice in Rhodesia; where possible I shall refer to the experience of others in Africa.

When I was about your age — quite a few years ago — I used to wander amongst the pine and oak trees on the slopes of this mountain, and on one of these jaunts met a young lady in her third year at the university. She told me she came from Bulawayo, just a name to me, except that I knew it was in Rhodesia. Nevertheless, on qualifying I said goodbye to my beloved Cape and left from Monument Station bound for the far North to marry my young lady. I had no idea what awaited me in the regions of the Zambesi.

It did not take long to see that there were striking differences in the pattern of disease and its frequency in the European and African races. This impressed and puzzled me. I asked myself why the African never seemed to develop a coronary infarct or angina of effort. I saw many acute abdomens but hardly ever gallstone colic, but met many cases of volvulus in otherwise very fit young adult males.

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Thus whilst it would be foolhardy to declare that certain diseases never attack the African and vice versa, it is true to say that there are a fairly large number of ailments that have a different prevalence in the two races, and now with the accumulated experience from many parts of Africa we can make this pronouncement with confidence.

Diseases Common or Well Known in the European, but Uncommon in the African

Several examples of these can be quoted.

1. Coronary thrombosis.
2. Thyrotoxicosis.
3. Cushing's syndrome (very rare in the African).
4. Addison's disease.
5. Leukaemia in infants.
6. Diabetes mellitus in infants and young children.
7. Ulcerative colitis.
8. Myopia.
9. Femoral hernia.
10. Carcinoma of the breast.
11. Calculi (in general).

Diseases Rare or Uncommon in the European, but Common in the African

1. Acute non-specific polyarteritis.
2. Kaposi's sarcoma (although this is common in certain Europeans).
3. Tropical ulcer.
4. Tropical myositis.
5. Primary carcinoma of liver.
6. Onyalai.

Let us discuss some of the factors which may account for the frequency or rarity of certain diseases.

The Influence of Environment

(a) *Geographical Pathology and Parasitism*

This factor struck me forcibly from my earliest contact with the African, who, most living close to nature, was liable to contract the more serious parasitic diseases of Africa. Thus we can expect

him to show more frequent or much heavier infestations of malaria, hookworm disease, trypanosomiasis and bilharziasis. Further, his contact with nature is clearly likely to take place from the earliest days of life, from when he leaves his mother's arms. Therefore three parasitic diseases (which perhaps I may refer to as the Big Three) — malaria, bilharziasis and ankylostomiasis — are probably the most frequently and intensively encountered during childhood. This does not mean that other important parasitic diseases are not common, but the big three are probably the most universal on the African Continent. In regions where we encounter diseases like leishmaniasis, relapsing fever and trypanosomiasis, we must also consider these diseases as part of the clinical picture. Thus we must know the geographical pathology of the region from which our patients come.

(b) *Nutrition*

Anyone deeply engaged in African medical practice soon comes to think almost automatically about food or rather its lack. Whatever factors are associated with malnutrition the clinician soon learns that there are a number of well-defined clinical disorders of nutritional origin. Again the prevalent nutritional disorders depend to some extent on the staple diet of the area, possibly also on the extent and severity of parasitic infestation and perhaps on the customs of the population. As I have indicated, the problem is most complicated. Thus, whilst we have good evidence to show that clinical pellagra can be expected in maize-eating regions, there is no doubt that persons consuming large amounts of alcohol are often attacked by it. Much of the malnutrition I meet comes from the tribal trust lands and perhaps European farms, but not from the African townships, contrary to what I first expected.

Whilst we meet so many African patients with nutritional disorders we must never fall into the trap of diagnosing a case of kwashiorkor as necessarily due primarily to lack of protein, because probably no less than 20 per cent. of children with this syndrome admitted under me are suffering from primary tuberculosis. No amount of protein will cure such patients, but once a course of anti-tuberculosis therapy is given the oedema clears, the rash disappears, the fever drops and the child makes an uninterrupted recovery. The patient with a cirrhotic liver, which is so common, or one with a chronic dysenteric disorder may assume some of the manifestations of malnutrition and so in the African we have to be specially careful not to label a nutritional disturbance as due solely to a poor diet, as a serious underlying visceral disease may be present and may have precipitated the patient, already on a border line diet, into a state of malnutrition.

Multiplicity of Affections

Anyone settling in a rural African community in tropical or sub-tropical Africa will quickly notice that his African patients are often attacked by more than one disease at a time.. Thus a patient with pneumonia, tuberculosis, malaria, dysentery or carcinoma is more than likely as well to be infested by one or more of the parasites endemic in the region in which he lives. Usually the parasitic infestation is not the main reason for his admission to hospital as the non-parasitic ailment is more pressing. However, once he has been treated for the primary illness the time is opportune to deal with the parasitic infection. On the other hand, since the African has a well established tolerance to these infestations, many of them do not seem to be disturbed by them and so the patient is often discharged without treatment for it. But I do not favour this course. My attitude is that the parasite may affect the body adversely even if only slightly. For example *P. falciparum* in the blood must cause the destruction of some of the red cells which the patient can ill afford, especially if his diet is doubtful or merely adequate as is so often the case. The same can be said of *S. haematobium* as an African patient with the infestation cannot spare even the few c.c. of blood each day passed in his urine. This appertains as well to *S. mansoni* which may also attack the liver. The ankylostome too lives by imbibing blood, and even if the infestation is mild a person should not be losing blood, more especially a pregnant woman, who is so liable to develop a severe anaemia. Whilst we accept this law of the multiplicity of disease in the African, we must qualify it by pointing out that the additional disease is usually a parasitic one and the clinician should not expect to find two ordinary diseases in the same case.

Principle of Coincidence

The clinician in practice in tropical Africa is often faced with problems arising out of the multiplicity of disease so frequently found in a single patient, due to the occurrence of a parasitic infestation in addition to some other disorder. For instance, a patient may be suffering from an anaemia as well as passing ova of *S. haematobium* or hookworm. Thus it is not clear whether the parasite has caused sufficient blood loss to account for the anaemia or whether it is due to some other factor. Again a juvenile or young adult with a hard liver and splenomegaly may have ova of

S. mansoni in his stool. Is the liver disorder due to cirrhosis or to clay pipe stem fibrosis following *S.mansoni* infestation? One has to be very careful before attributing an anaemia or liver disease to one of these parasites, since the latter may be present coincidentally. I have suggested that this situation be referred to as the principle of coincidence.

Not uncommonly I have seen patients suffering from typhoid fever, lobar pneumonia, measles or severe anaemia, in whom the parasite was an incidental discovery and not responsible for the primary illness. Since parasitic infestation is less common in the European, if a parasite such as the plasmodium or schistosome is found the chances are that this is the cause of the primary condition.

VARIABLE PICTURE OF A DISEASE IN THE AFRICAN AND EUROPEAN

In Africa one finds that a disease may appear with a certain degree of severity in one race yet in the other it is the reverse. Thus in the white person measles is mostly mild and seldom complicated by one of its serious manifestations, but in the African child in tropical Africa not only do we meet a high death rate, but often severe complications like blindness, bronchopneumonia or tuberculosis. No doubt the difference may well be attributed to the poorer nutritional status of the African child; the better fed the infant, the better he can be expected to stand up to the infection.

The clinical picture of tuberculosis also shows some differences in the two races. In the European adult the acute miliary form of the disease is not very common, whereas in the African it is far from being regarded largely as an ailment of childhood. On the other hand, in the African certain organs are seldom attacked by the disease. Examples of this are laryngeal and renal tuberculosis and tuberculosis of the epididymis and suprarenal glands.

MALIGNANCY IN THE TWO RACES

It would be relevant at this point to refer to the important subject of new growths in European and African.

Table I compares the relative frequency of the various sites of carcinomatous growths in England and Wales with those noted over ten years in Rhodesia by Dr. Malcolm Ross, the Senior Government Pathologist in Salisbury.

Table I

ORDER OF RELATIVE INCIDENCE OF VARIOUS SITES OF CANCER IN ENGLAND AND WALES (1961-1963) (REGISTRAR GENERAL'S REPORT) AND THE FREQUENCY OF CANCER IN RHODESIAN AFRICANS BASED ON MALCOLM ROSS'S STUDY.

England and Wales (European)	Rhodesian (African)
1. Breast	Skin
2. Stomach	Bladder
3. Intestine	Retino-endothelial growths
4. Lung	Cervix
5. Ovary	Oesophagus
6. Cervix	Liver
7. Rectum	Lung

It will be seen that in the United Kingdom the stomach is shown to be the second most common site, followed by the intestine, whereas in Mashonaland (Rhodesia) the stomach and intestine are very much less often the site of malignant growths, whereas the oesophagus is a fairly important one although intestinal cancer is relatively uncommon. When we look at the breakdown of intestinal growths we find a fair number of Africans with cancer of the rectum. In my experience carcinoma of the colon is very uncommon, but carcinoma of the rectum is by no means rare.

Table II

CARCINOMATA OF THE ALIMENTARY TRACT (1955-1966) IN AFRICANS IN RHODESIA (M. ROSS).

Type of Tumour	Total number	% in alimentary tract	% of all tumours
Adeno-carcinoma of stomach	135	24.2	3.3
Carcinoma of small intestine	6	1.1	
Carcinoma of caecum	11	2.0	
Carcinoma of colon	25	4.5	
Carcinoma of rectum	65	11.7	
? site	6	1.1	
Argentaffinoma (carcinoid)	2	0.4	2.5

We must not forget that we are dealing with a much younger age group of population in the African than in the European and it is quite possible that as the African comes to live longer this presenting picture will change. We shall certainly expect a greater number with carcinoma of the stomach, colon and rectum.

Coming from a bilharzial infected country, my mind naturally turns to a possible relationship with *S. mansoni*, but I cannot be definite on this

point. In this connection an interesting hypothesis may be advanced. Bremner (1964) of Baragwanath Hospital has shown that the African in Johannesburg has a daily bowel action just short of a diarrhoea; 41 per cent. of his cases interrogated had a bowel action more than twice a day. This he attributed to the high residue diet and the frequent use of aperients by his African patients. As a result of this more frequent evacuation of bowel contents there is a greater chance of more effective removal of possible carcinogins from the bowel as compared with Europeans in whom the longer contact may result in the eventual development of malignancy.

Much has been written about the high frequency of carcinoma of the oesophagus in many parts of Africa. We are struck by the apparent rise of its frequency in Durban (Shonland and Bradshaw, 1969). Experience in Salisbury supports the Durban finding. Yet in other parts of the Continent this high frequency is not seen.

Table III

FREQUENCY OF CANCER OF OESOPHAGUS FROM 1958 TO 1967 IN HARARE HOSPITAL SURGICAL WARDS UNDER MR. HAMMAR.

Year	Males	Females	Total
1958	15	0	15
1959	24	2	26
1960	37	0	37
1961	33	1	34
1962	42	0	42
1963	54	2	56
1964	36	0	36
1965	54	0	54
1966	59	0	59
1967			74

RACIAL AND GENETIC DIFFERENCES

There would appear to be a few anatomical or structural differences, which though slight might account for the difference in occurrence of certain diseases in the two races. For instance, Cook's researches in Uganda in 1966 demonstrated that a specific lactose deficiency was not uncommon in certain tribes of Uganda, but that the Bahima people nearby did not show this defect. The latter are a cattle-owning people whilst the Baganda have little or no milk, so they may not have adapted themselves to this food.

There are two other conditions which may be included in the group of racial differences. It is more or less generally accepted that indirect inguinal hernia is relatively frequent in the African. An anatomic variation has been noted in a series of careful dissections in the muscular and fascial layers of the inguinal canal from that of the European. White (1962) suggests that the frequency of inguinal hernia in the African may possibly be related to the frequency of this unusual fixation of the mesentery to the small intestine of the African child. Elongation of the mesentery with increased length of the small and large intestine is a common observation in a race whose diet is largely of a residual kind. This elongation is acquired and has been shown to predispose to inguinal hernia. White also draws attention to the fact that the age and sex incidence of oblique inguinal hernia and of volvulus of the small bowel correspond very closely and that similar predisposing factors are operative in both conditions.

Evans (1961) has estimated that 9 per cent. of an unselected group of young adult Africans seeking employment in Rhodesia has external herniae. This contrasts with 2 per cent in the U.K. It has also been suggested that the almost universal occurrence of umbilical hernia in the African child is due to a genetic difference. But the subject of inguinal hernia in the African is difficult to follow. For instance, compared with Europe, direct inguinal hernia is much more common and strangulated hernia more often found in Mulago and Ibadan, but distinctly infrequent in comparison with the indirect type seen in Bulawayo and Nairobi. Again, femoral hernia is extremely uncommon in Africa except in the Baganda of Uganda living around Kampala and Jinja (Davey, 1968). Recent work suggests that it is liable to occur in people in whom the conjoined tendon has a narrow medial insertion into the ileopectineal line. West African women have smaller pelves than their European counterparts and this has been advanced as a cause for the difference in the frequency of femoral hernia in the two races.

I am able to state with some confidence that hiatus hernia is extremely uncommon in my hospital patients despite the many barium meals that have been performed. In 1969 there is mention of four cases of diaphragmatic hernia.

ALLERGY AND TOLERANCE TO A PARASITIC DISEASE

The general picture of disease in Africa depends to a certain extent on vegetation and rainfall, but there is also another important factor already mentioned — that of race. Most of us in practice in Rhodesia find ourselves in a land where there are at least two main racial groups and our medical interest soon becomes aware that there are clear differences in the reaction to disease of the two races. It does not mean that particular diseases in the European and African have entirely different effects on each; similar ones can be

demonstrated. Yet it is true to say that the pattern tends to be different in one group to that in the other. We have long pondered on this. The reason that comes to mind immediately is the possibility of a racially distinct immunity or tolerance. The African has lived in tropical Africa for hundreds of years and so, with a few possible exceptions, such as the Besotho, must have been exposed to a similar kind of environment.

We have come to regard those parasites which live in the blood as contributing to these differences in the two races. This applies particularly to the malarial, filarial (*A. perstans*), sleeping sickness, schistosomal and amoebic parasites. On the whole, the European, infected for the first time with one of these parasites which invade the blood stream, is apt to show a much more severe and violent reaction than the African in whom an immunity has been established. On the other hand, Africans who have been brought up in non-endemic regions react similarly to the European. We know that the Evangelists, who came up from the Cape to Malawi, died of blackwater fever and cerebral malaria in much the same way as did Europeans. Schistosomiasis perhaps best describes this point. Here we have a disease in which, for practical purposes, the European in Central Africa has been exposed at the most for three or four generations and in the vast majority hardly for more than one. The African has survived this environment for centuries and he has built up a tolerance to the disease. We know that some of the babies born of African mothers, who were themselves born in Mashonaland, have a positive fluorescent antibody test. This baby is therefore different to the European infant who has not had this advantage, although it may be so temporary that it lasts only a few months.

We know that the katayama syndrome is seen in the European, yet despite a careful watch for it and for the slightly earlier manifestation, Kabure itch, also well known in the white man, neither has as yet been described in the African. (In Kabure itch we meet the itchy papules at the time of exposure, and in the katayama phase urticarial swellings and pronounced eosinophilia.)

We might have erred in defining the katayama phase as occurring only in the early stage before ova appear in the excreta, for ova may be found in the stool and urine, and yet this phase continues for some months. Thus we not only meet the classical katayama phase in Europeans, but often in the first months or year or two from the time of infestation; in our experience some very serious complications of the disease may be seen in him. A complication which may appear during this period of schistosomiasis is a paraplegia in which the individual develops a progressive weakness and sensory loss in both legs as well as urinary incompetence. Fortunately this severe effect may respond well to specific treatment, although the prognosis is not always so good. We are not sure whether it is due to interference with the blood supply to the segment of the cord because of the occlusion of a vessel by a worm or worms. This form of paraplegia is not normally seen in the African. There is fairly good evidence produced in publications, mostly from South Africa and Rhodesia, that early paraplegia in schistosomiasis has so far been seen only in the white man.

Clinicians have been impressed by the more striking constitutional symptoms complained of by the European compared to the African. Rarely is tiredness the presenting complaint of the African, yet as a rule he passes a far greater number of ova in his excreta than the European, and it is therefore presumed that there are much fewer worms in the European than in most African cases. We are therefore led to believe that the European must be more sensitive or allergic to the presence of the worms and their products.

Again, my experience has shown that it is not as simple as might be expected to cure the European with a schistosomal infection. He may be very resistant to cure even in an infestation that is mild as judged by the small numbers of ova passed. Many European children and adults too require three or more courses of different therapeutic agents before a cure can be pronounced. In contrast to this, we have frequently observed that an African, passing a large number of ova in his excreta, may respond readily to a single course of treatment. The difference perhaps lies in the degree of sensitivity which makes it more difficult for the European to be cured than the African in whom this reaction appears to be absent.

In malaria we often, although not always, see a vast clinical difference in the two races. The European who comes to Rhodesia for the first time frequently suffers very severely when infected, and some even develop cerebral malaria or one of the other more dangerous forms.

The story of the African brought up in an endemic malarial zone is well known. There is first a temporary passive immunity conferred on the new-born for about three months. It should be pointed out in all fairness that the mother's milk in itself possesses antimalarial virtues. Some workers have suggested that the body still contains some foetal haemoglobin which affords protection, and others that there is little attraction for the female mosquito in the skin of so fine a texture in a child of such tender age.

From the age of three months until about two to three years the effect of malaria on the African infant may be severe, and many deaths occur because of the lack of a properly established immunity. After that period the child is fairly well protected against the malarial parasite and by the age of about 20 years can withstand most attacks. Only occasionally do we meet with cerebral malaria, pernicious malaria or the other more serious forms of the disease. The same applies to blackwater fever. This condition was hardly seen in the indigenous African, although some three or more decades ago it was especially common in the European population. Today the treatment received is possibly more effective and people are more careful to complete a course of therapy. According to many workers, when quinine was used with less frequency, blackwater fever tended to disappear.

Today it is interesting to note that cases of blackwater fever are becoming more common in Africans in Rhodesia. It is thought that such patients have lost their acquired resistance to malaria. However, it is possible that these cases were in fact not blackwater, but that the haemoglobinuria was due to some other cause such as the use of drugs like chloroquine or as a result of an acute infective state like typhoid fever or lobar pneumonia, or at times that it was related to a deficiency in the glucose 6-phosphate dehydrogenase enzyme.

Over the years a fair number of Africans with trypanosomiasis have been examined, and during the same period a good representative sample of Europeans suffering from the same disease. In both races when the nervous system is attacked the clinical picture and end result tend to be the same. In both the disease may attack the nervous system with dire results. What has been so striking, however, is the frequency with which an acute reddened chancre or sore develops in the European as the first sign of the disease. This does not mean that the African does not develop a sore, but from descriptions of it the reaction appears much milder than in the European. In the latter a sore, usually about half an inch or at times an inch in diameter, appears in a matter of hours. It is quite itchy and the patient thinks he has been bitten by an insect or has an urticarial bump. Almost immediately he develops a rising temperature, does not feel well, and within 24 hours is confined to bed. By now his spleen may have become palpable and the sore starts to vesiculate with a dirty greyish fluid.

We believe we can say the same about *Acanthocheilonema perstans* infection, which must now be accepted as pathogenic to man. A large number of Africans who harbour this parasite show little or no ill effect. Indeed, the general opinion that it did not appear in man was based on the experience of the disease in the African, in whom it follows a different course to that in the European. In the latter, not unusually, there appears a mild arthralgia, perhaps a variable degree of long-lasting fever and very exceptionally a hepatitis, pericarditis and even an encephalitic state. This should not be taken to mean that the African does not suffer severely, for indeed serious effects from the parasite have been described in him. However, it would seem that the white man suffers from the parasite more frequently than the African.

The malarial parasite in Africa possesses many dangerous propensities and its part in causing death, especially in the young, is well known. However, in endemic regions, which cover a vast extent of terrain in tropical Africa, many of the inhabitants carry the parasite in their blood without showing outward signs of ill effect. If such a person develops an acute disorder like typhoid fever, the finding of the malarial parasite may cloud the issue and the patient may be treated for acute malaria whilst the true nature of his illness is overlooked. However, in a non-immune subject with an acute febrile illness the presence of the malarial parasite denotes that it is the cause of the complaint.

Does parasitic disease suppress the development of auto-immune disease? Greenwood, Herrick and Voller (1970) remark on the rarity of the so-called auto-immune diseases such as Hashimoto's disease, myxoedema, rheumatoid arthritis, Addison's disease and the systemic collagen diseases. This they attribute to the high immunoglobulins (IgG and IgM) which are formed in Africans exposed to malaria and other parasitic diseases. If this can be shown to be true, then it can be argued that, whilst a disease may be detrimental to the body, it may, at the same time, confer certain protective functions.

The Stability of the African Endocrine System

Anyone practising in Africa over a number of years must be struck by the apparent rarity with which most endocrine disorders are encountered. It would be foolhardy to state that the African never develops Addison's disease, Cushing's disease, myxoedema or cretinism. There was a time when I never saw thyrotoxicosis, which, however, is more commonly encountered nowadays. Indeed, in Johannesburg Dr. Seftel informs me that it is very common amongst townsmen as compared with the farming community. Never-

theless, in practice the African appears to have an endocrine system that is extremely stable and in his own environment probably fully capable of coping with the various stresses and strains of life. Acromegaly is not altogether rare in my experience. The one exception in these disorders is the frequency with which diabetes mellitus is encountered in hospital practice today. One should perhaps include diabetes as an endocrine disorder, since the pancreas is so likely to be the cause of this disturbance.

Table IV
FREQUENCY OF ENDOCRINE DISORDERS AT HARARE HOSPITAL

	1967	1968	1969
Thyrotoxicosis	5	18	4
Diabetes mellitus	105	98	162
Cushing's syndrome	1	4	4
Acromegaly	—	2	3
Addison's disease	—	—	5
Myxoedema	4	7	2
Hyperparathyroidism	—	1	4
Pheochromocytoma	—	—	1
Cretinism	—	—	1

PARADOXICAL (CONTRADICTORY) SITUATIONS

There are a number of diseases in which a paradoxical or unexpected situation arises for no clear reason, as far as we know, which I have thought appropriate to refer to as paradoxical situations. We know that in many parts of Central and South Central Africa acute volvulus is a common acute abdominal emergency in the African, yet in Rwendi Urandi we are told that acute intussusception takes the place of volvulus, whilst in Uganda, which borders on this territory, volvulus is to be expected. Again, rheumatic fever is specially well known in Rhodesia, but in Nigeria it is seemingly uncommon. Again, endomyocardial fibrosis is frequent in Nigeria, whereas in Rhodesia this form of cardiomyopathy with extensive fibrosis is particularly infrequent. In Rhodesia we meet the South African type of cardiomyopathy. Gallstone colic is rare in almost any part of Africa, yet in the African in the Sudan it is quite common. We meet a very common form of pancreatitis and diabetes in Uganda and Nigeria characterised by calcification of the organ associated with steatorrhea and diabetes mellitus; this picture is seldom seen in the Cape, and then mostly in coloured people. In the north this condition is unrelated to alcoholism. The majority of the experts maintain that a protein lack in early life has led to this peculiar disorder. Yet the condition is hardly ever met with in the Bantu, although kwashiorkor is at least as common in Southern Africa as in the northern territories just mentioned. I have seen only two such cases of calcification of the pancreas, despite careful search. And yet the extraordinary situation exists that in Southern Africa we not unusually meet diabetes mellitus in an African with siderosis and porphyria. (The diabetic-siderotic-porphyric syndrome.)

When we look at cardiomyopathy in Africa we also find differences in its pattern. In South Africa cardiomyopathy accounts for apparently 20 per cent. of all cases seen with heart failure and there is roughly the same frequency in Uganda, Nigeria and other more northerly regions, but the type of cardiomyopathy seems to be quite different in the two divisions of Africa. In the northern part it is characterised by a dense fibrotic reaction obvious to the naked eye and is seen mostly in the left ventricle. In the Southern African type of cardiomyopathy the main changes are seen microscopically and little or no fibrosis is evident to the naked eye. Of course, I am not trying to infer that the two forms are the same in nature. All we can say at present is that we do not know what causes cardiomyopathy in Africa.

Workers seem to agree that whilst femoral hernia is very rarely encountered in the African, yet around Jinja in Uganda it is said to be not uncommon (Davey).

CHANGING PATTERN OF DISEASE

We notice now that the pattern of some diseases seems to be changing in Africa. Some are becoming less frequent in certain places and others more common. What factors cause this we can only guess. My experience teaches me that this is related in some way to a changing environment. For instance, I used to encounter many cases of tropical ulcer, tropical myositis and scurvy, and over the years these have become less frequent. Here I might argue that perhaps better standards of nutrition and improved labour conditions are responsible for their diminishing incidence. But what can be the explanation for the virtual disappearance of onyalai from Mashonaland, where there are still many areas in which the environment has changed very little?

Chigger disease about 70 years ago was a serious crippling disease due to a parasitic infection of the toes that caused severe pain and suffering to hundreds of labourers from Zambia and Malawi. Today this has entirely disappeared from large areas of Central Africa, and where it does occur is not nearly as bad as it used to be. Again one might attribute this to the better roads and footpaths.

Then on the other hand there seems to be a sudden upsurge in the prevalence of diabetes mellitus. It is difficult to give exact figures, but I personally am struck by the many more cases

I am seeing in my ward, arising not only in urban areas, but in rural ones as well. One tends to blame urban life for this, but the disease is by no means uncommon amongst the rural population. However, in the country areas there is a change in food habits. Perhaps it is due to eating too much fat, obtained in the form of dripping, now procurable from the local butcheries that are springing up throughout the country; or it may even result from the consumption of too much refined sugar, both of which were generally absent from the traditional diet.

I have already mentioned that we are just beginning to meet cases of coronary thrombosis and thyrotoxicosis. The same reasons, as for diabetes mellitus, can be advanced for their appearance in the African population.

As the African changes his environment and moves away from his closeness to nature, as for instance in the urban townships, we can expect less parasitic infestations. We can expect less bilharzial effects on the viscera and less malaria, but as the townsman is beginning to lose his acquired immunity to the latter, when he does expose himself to infection with *P. falciparum* he will be liable to develop blackwater fever, which was such a feature amongst Europeans until recently.

It is tempting to ascribe the difference in the food habits of the two races as the reason for the patterns of intestinal disease in them. Increasing numbers of Africans are adopting the Western way of eating and modifying their own. As certain diseases like peptic ulcer and appendicitis seem to be becoming more frequent, one is apt to attribute these changes to these dietetic factors. There may be some truth in this hypothesis. For instance, the rarity of ulcerative colitis in the African might be put down to particular differences between the African and European diets.

We should remember some of the deleterious effects the Western form of diet has on those who follow it. One that comes to mind is the casual relationship between gluten and idiopathic steatorrhoea. We might also ask whether the rarity of coeliac disease and idiopathic diarrhoea in the traditional African may not be due to the low content of gluten in his diet? Some authorities have attributed the frequency of peptic ulcer and cholecystitis in the European to his diet so rich in wheat, animal fats and sugar.

Diverticular disease is apparently very rarely encountered in our Shona patients, and this is confirmed by the experience of surgeons with long practice in Harare hospital. It is said that in Europeans diverticula may be found in the colon barium enema in about 7 per cent. of cases submitted for this examination. Why is there this difference? Again it is tempting to attribute it to the dietetic habits of the two races. Because of the high residual diet of the African the sigmoid colon is probably distended, and this in turn induces a co-ordinated contracture and segmentation of the distal colon, making possible a good evacuation. In the European, on the other hand, with a low residual diet the sigmoid is not adequately distended, and as a result the colon below shows incordinated contraction and segmentation with an increase of tension, leading in turn to pouch formations (Reilly, 1969).

Town and Country

I feel certain that if we were to ask any physician practising at Harare hospital whether he sees much diabetes mellitus or any surgeon there whether acute appendicitis is common, the answer might well be in the affirmative. Indeed, this is what I find myself. Yet whenever I go to the rural areas and speak to the local clinicians, the answer is that both disorders are rare. In other words, in a strictly tribal trust area the doctor sees both those disorders very infrequently, and I am satisfied that he is able to recognise them, and in the case of appendicitis, if unable to operate himself, would refer the patient to a hospital from which he can obtain a reliable opinion.

There are two explanations for the apparently conflicting experience in these two diseases. With regard to diabetes mellitus, when the rural doctor meets such a patient in his area he refers him for advice and treatment to one of the big centres where special tests, like blood sugar, can be carried out and so these centres tend to gather large numbers of cases of diabetes. I am not altogether satisfied that there is a true rise in its incidence in the urban areas of Rhodesia. For instance, when Carr and I carried out a survey in Highfield township in Salisbury, we found the frequency of the disease very low (ref.: Gelfand and Carr). In the case of acute appendicitis my evidence is that here we have a true growth in its incidence in the urban African, but what the reason is for this I am not clear; I have already discussed this matter in another section of this paper.

Acute Intestinal Disorders

In African trauma figures, rupture of the stomach and intestine (especially the small bowel) are not uncommon injuries. The African is more exposed to injury than the European because of the nature of his work. Also, after a drinking bout or a heavy meal, if he becomes involved in a fight, a blow delivered to a full abdomen may result in rupture of the viscera.

In the European an acute abdomen is mostly

caused by an inflammatory disorder — especially in the appendix. The next most frequent condition is an obstructive lesion (mostly external hernia). In the African, on the other hand, the most important disorder producing an acute abdomen is obstruction due to external hernia, volvulus and intussusception, followed by the inflammatory group.

Table V

Europeans—
1. Inflamatory (especially appendicitis)
2. Obstructive (external hernia)

Africans—
1. Obstructive
2. Inflamatory group.

In 1963 I decided to study the place of origin and diet, at the time of the illness, of every patient admitted to the surgical wards of Harare hospital. I personally saw each patient after admission from 4th August, 1963, until 19th December, 1965. The nature of the acute abdominal disorder, the place at which the patient first became aware of his illness and the kind of diet he ate were recorded.

Table VII

TYPE OF DIET TAKEN BY PATIENTS WITH ACUTE ABDOMEN — GRADE OF AFRICAN WITH COMMON TYPES OF ACUTE ABDOMEN SEEN AT HARARE HOSPITAL (NONE REFERRED FROM A RURAL VILLAGE)

Condition	Grade I (traditional)	Grade II	Grade III (Europeanised)	Total
Perforated duodenal ulcer	4	3		7
Volvulus	21	1		22
Strangulated hernia	28			28
Appendicitis	24			24

It will be observed that both strangulated inguinal hernia and volvulus may occur in both the traditional village and in urban areas. Perforation of duodenal ulcers seemed to develop only in towns; acute appendicitis mostly in the towns. There was no case referred from a village. Why is this? I have frequently visited rural hospitals, many of which are cared for by efficient medical officers, who deny the existence of acute appendicitis in the strictly traditional village. Is this due to dietetic differences between the strictly traditional villager and the urban African who, although he may still be living largely on a traditional diet in town, has adopted some European foods such as bread, sugar, tea and jam for his breakfast? I must admit that the Shona in the rural areas are also beginning to have these items for breakfast. It is possible that urban Africans who develop acute appendicitis may be in domestic employment and not only as cooks, as some doctors have implied. Mr. John Gordon, a surgeon at Harare, has suggested that in towns the African cannot attend to his bowel habits with the same ease as in his village. He might have to control himself, which may lead to constipation, and perhaps this might lead to acute appendicitis. Again, doctors inform me that perforation only occurs in town and not in the traditional setting. Is it in some way linked with the stress of town life?

THE MIND (PSYCHE)

I am not in a position to state categorically or with any confidence or accuracy that the African has a different anatomical structure or function in any organ of the body. Although one might be tempted to think this is so, I am not convinced that a truly scientific observation can be made. I am aware that it has been claimed that the African might have an additional branch of the coronary artery. There is also a postulation that his brain has less Betz cells than the European. These statements have never convinced me largely because Vint's autopsies, carried out mainly on African patients, suffered from bias. It is very possible that a group of Africans, more than likely suffering from poor nutrition and diseases like malaria, might have had brains which had undergone some atrophy. We have good evidence that in kwashiorkor some damage occurs in the cerebrum with resultant loss of intelligence, and there are many Africans who have suffered from this disease in childhood.

My own contact with Africans shows me that they are a capable race and that the great bulk of them fall within the normal range of intelligence. I believe there is a great concentration of normals in their society with probably less mentally defectives and, on the other extreme, geniuses. The genius or awkward individual who wanted to change his society was regarded as a witch and driven out of the village. There was only room for those who conformed.

People ask why, if the African has a normal mind, did he appear so underdeveloped when the first white men came to the Continent? It is argued that he had no wheel or any of the mechanical devices that characterise the Western world. At first sight this appears to be the case, but in my study of the Shona I have come to the conclusion that this was almost a deliberate move to ensure that everyone was equal in material benefits and that the family lineages or segments of the clan shared their possessions equally. If

man is allowed to become acquisitive, jealousies and fighting result. Progress was sacrified for the good of the group as opposed to the interests of the individual. At all costs peace was maintained within the group and tensions reduced to a minimum. Therefore the Shona have developed a most involved, elaborate, disciplined code of behaviour in which every individual, according to his status in the family, is ensured of proper respect — the father, the mother, the father's sister, the mother's sister or brother, the grandparents, the sister and brother, and most of all the in-laws are accorded the right respect. Sharing was the rule. A brother gave help in times of need without any expectation of repayment. No one ever ate a meal alone. A man who needed bridewealth to procure a wife was helped by the other male members of the family who could afford some cattle. Every man was able to live on a piece of land sufficient for his needs, and so each was limited in the wealth he could acquire. He could not accumulate and become wealthy in the Western sense. As a result, a great brotherhood developed in which all was geared to a happy medium to produce a good and contented man. Therefore it is my contention that what appeared backward or "primitive" to the outside world was in reality an intelligent attempt to maintain equality and sameness in material wealth and thus peace within the clan. The Shona frowned upon change and what the modern world knows as progress. All this was achieved through their belief in ancestral worship and in witchcraft. According to this cult, the ancestors insist that their children and grandchildren live the lives they did on this earth and do not change or leave their home environment. Any change is bound to lead to weakening of the clan and the brotherhood so necessary to survive in this harsh and cruel world.

Does this concept of brotherhood have an effect on the wellbeing of the individual? A person who grows up in this environment has a feeling of being surrounded by love and a sense of support with the knowledge that one of his kinsmen will be ready to give him a helping hand whenever necessary. He knows that when he dies his wife and children will be taken over by his younger brother and so they will never want. We know from a number of reliable sources that suicide or attempted suicide is very much less in African than European society, despite the stresses and strains that most human beings undergo. It is a rare event for an African to be brought to the casualty department of Harare hospital after an attempt to take his own life. The comfort the individual derives from this philosophy of brotherhood is given as a reason for this difference. The sense of security under kinship protection that the traditional African has is not available to the Western world. Thus we can expect to find perhaps less coronary artery disease, less coronary thrombosis and kindred disorders of stress in the African than in the European.

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Table VI

The Place in Which the Acute Abdominal Crisis Occurred in Ninety-nine Cases

Type of Lesion	No. of Patients	Town	Rural	Farm	Mine
Strangulated inguinal hernia	28	11	11	5	1
Acute volvulus (s. intestinal type 13 sigmoid type or complicated, 7, ? type 2)	22	9	6	7	0
Intussusception	4				
Acute intestinal obstruction (3 with adhesions)	8				
Perforated duodenal ulcer	7	7	0	0	0
Perforation (other than duodenal)	3				
Acute appendicitis	24	22	0	1	1
Acute peritonitis	2				
Cholecystitis with stenosis of crystic duct	1				
	99				