The Clinical Features of Intestinal Schistosomiasis in Rhodesia*

BY

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I was happy to accept your kind invitation to visit your medical school and to address the students. Already your graduates are finding their way into Rhodesia, where they are more than holding their own in the medical field, and I am equally aware of the fine name your school has earned for itself, not only in Africa, but far beyond. But before I begin this talk on the subject which is the main purpose of my visit, I would like to pay tribute to the memory of Dr. F. Gordon Cawston, a pioneer in the study of schistosomiasis and who did much research for many years on this disease in Natal. He died in your city in March, 1949, and was a good friend of the African.

It is interesting to remark that at one time it was held that *S. mansoni* was rarely encountered in South Africa. For instance, in 1908 Turner recorded that he had only seen lateral spiral eggs in Africans from the territories to the north of the Republic, and even as recently as 1934 Pijper too claimed that he personally had never seen a case of *mansoni* in S. Africa (quoted de Meillon, 1948); but in 1938 Porter had reported its existence in several places in the Transvaal; and since then, notably through the publications of Pitchford (1952), it has been shown to be a common disorder with a wide distribution in the Transvaal.

There are three reasons for my having chosen to speak to you on the intestinal parasite *Schistosoma mansoni*. First of all, in clinical research the emphasis has been on *Schistosoma haematobium* and so I believe we do not know enough about the natural development of the intestinal form of the disease in Africa. This may be due partly to the fact that *S. haematobium* is the more common of the two parasites and partly because *S. haematobium* is much easier to study, as it is far less difficult to collect a sample of urine than a specimen of stool.

Secondly, some clinicians regard *S. mansoni* as probably being asymptomatic. I remember a professor from Uganda who wondered about the wisdom of submitting a patient with intestinal schistosomiasis to a prolonged and dangerous course of treatment in view of the fact that, in his experience, *S. mansoni* did not appear to cause symptoms in children. I had previously believed the disease to be a serious ailment, not only because it interfered with the general well-being of the patient, but because of the possibility of its producing serious complications like cirrhosis of the liver. There has been much heated debate as to whether or not either of the common schistosomal parasites lead to cirrhosis of the liver and pulmonary hypertension. If it can be proved that *S. mansoni* is responsible for cirrhosis of the liver, or heart failure, as is believed by the Egyptian and South American workers, then any person harbouring these parasites should be treated whether or not he is showing symptoms. If it is proved that *S. mansoni* is as harmless as some believe *Entamoeba coli* to be, then we should only treat patients in whom the disease is actively causing symptoms.

The third reason for my interest in *S. mansoni* is that the general impression of doctors practising in Rhodesia is that it is more difficult to

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destroy the intestinal parasite with antimony than it is the urinary one, and that relapses tend to be more frequent, but eventually most European cases are cured. If the intestinal parasite is more difficult to eradicate than the urinary type it seems that one should expect more patients to remain harbouring it and therefore there should be more with complications of *S. mansoni* than of *S. haematobium*.

In 1960, therefore, I decided to study the clinical features of intestinal schistosomiasis with as fresh an approach as possible in order to obtain a better appraisal of this disease. This was difficult, as many of my African patients suffer at the same time from one or more concomitant diseases such as malaria, malnutrition, liver disease and anaemia, and it would be extremely risky to attribute any symptom of which the patient complained to *S. mansoni*. This is a very important point, as I believe that many of the clinical descriptions of *S. mansoni* in the African are vitiated by this fact. We should be careful before stating that any symptom in a subject, infested with other parasites or suffering from some other disorder, is due to *S. mansoni*. Fortunately in Rhodesia there is a European, Coloured and Indian population which rarely suffers from more than one disorder at a time. Thus in a European child harbouring *S. mansoni* it is reasonable to assume that any symptoms complained of are due to that disease. I think this applies also to Coloured and Indian children, who also belong to relatively well-off communities in Rhodesia, and so I decided to interrogate closely every European and Coloured child found to have *S. mansoni* ova in the stool in order to learn the symptoms complained of and obtain some idea of the clinical import of the disease in its early phases. At the same time I arranged with the Bilharzial Research Laboratory to study the distribution of *S. mansoni* in selected viscera of the body as well as their frequency as compared with *S. haematobium* in autopsy cases. This work was a follow-up of the study I made in 1947 and another with Fraser Ross in 1949. I consider these investigations important because we should know which organs are likely to contain ova and therefore show lesions in life.

The 1947 and 1949 studies showed:

(1) That *S. haematobium* is the more common parasite.

(2) That the *S. mansoni* infestation is by no means uncommon.

(3) It is rare to meet a pure *S. mansoni* infection; the greater number of subjects had *S. haematobium* as well. In fact, this was so noticeable in all three studies that it can be assumed in Central Africa that if an African subject harbours *S. mansoni* he is likely to have *S. haematobium* as well. But the reverse is not true, as many schistosomal subjects harbour the *S. haematobium* parasite only. This is not apparent in ordinary clinical routine work, as often only one parasite is found, and as a rule little effort is made (such as by rectal snip) to search for the other as well. I am doubtful whether this applies to the European or Coloured child, since he is not usually so greatly exposed to the disease as is the African. I noted the symptoms complained of by African patients with *S. mansoni*, paying particular attention to those appertaining to the bowels and liver. A certain number of rectal biopsies were also performed, noting carefully the condition of the mucosa in order to detect any changes in its pattern, as occur so dramatically in the bladder with *S. haematobium*. I also took snips in a number of subjects in whose stools the laboratory had found positive ova to determine the correlation between the two findings. Snips were not taken in those in whose stools ova were not found, in order to study the frequency with which they could be detected in the snip, although the stool was negative. This is probably more common than we believe, as I have often met cases with no ova in the stool although they were present in the rectum.

A most important advance in the recognition of schistosomiasis came in 1943, when Ottolino and Attencio in South America showed that the chances of demonstrating the presence of *S. mansoni* were greatly enhanced by taking and examining a small snip of mucosa from the rectum. If three snips were taken from different sites, preferably near the first rectal valve, the chance of finding ova was increased. Later it was shown in Rhodesia that even in *S. haematobium* infections ova were found in rectal snips, although not as frequently as with *S. mansoni*. Ova discovered in the snip may be viable or dead. If viable, treatment is obviously needed. If dead, it is a little more difficult to decide whether to treat or not, particularly in a person who has not been treated before. In such a case I would advise a course of treatment in case the infection is still active, but in a patient with dead ova who has already been treated it is safe to assume that treatment has been effective.

I have found both the ordinary faecal smear and the rectal biopsy of great value in practice. Neither is absolutely reliable, although on the whole the two correspond closely. Usually, if ova are found in one they are likely to be found in the other, but in about 30 per cent. of cases the stool specimen may be negative and the snip positive or vice versa.

Table I shows that by taking only a single snip from any part of the rectal mucosa about 66 per cent. of the patients with positive stools had ova of *S. mansoni*. Thus this method is not fool-proof. On the other hand, about 33 per cent. of the snips also revealed the ova of *S. haematobium*, demonstrating a clear use for this method of investigation. There are, of course, a number of cases whose stools do not contain lateral spiral ova, although the snip does.

Table I

Comparison of Positive Stools (Mansoni Ova) with a Simple Rectal Snip Taken in Each Case (Africans Only)

Number with *S. mansoni* in stool	21
Number with *S. mansoni* in rectal snip	14 (66.6%)
Number negative in snip	4
Number with *S. haematobium in snip*	7 (three had *S. mansoni* as well)

Work in Salisbury has shown that a snip can be taken from any part of the rectal mucosa. The simplest technique is to use a proctoscope, but a small sigmoidoscope is best in a child. But whichever instrument is employed, it is essential to have a good light. It is not necessary for a preliminary washout to be given, as in most instances enough mucosa from which to take a snip can be seen. It is most essential that care should be taken not to snip a vein. Once this is remembered the procedure is very simple. I have found that one snip generally suffices and it should be about 2 to 4 mm. in size. This tissue is placed on a glass slide and another pressed firmly on top of it so that the whole circle of tissue is squeezed flat and can be examined immediately with the low-power lens. When the ova is alive this can be detected by movement of the miracidium inside the shell of the egg, whereas the dead ova shows no such movement and is generally blackened.

For some reason which I cannot understand the rectal mucosa is mostly normal in appearance to the naked eye, despite the fact that the mucosa of the bladder is so highly altered in appearance in urinary schistosomiasis. In fact, it is so changed that it is relatively easy to recognise the urinary form of the disease by cystoscopy. I think it is better to describe the rectal mucosa as normal, although to one who does a great many snips it seems that it is perhaps a little more swollen or oedematous than healthy mucosa, its surface perhaps coarser and more irregular, with often a few scattered pin-point haemorrhages. But I have only exceptionally encountered the more severe lesions such as the bilharzial papillomata or tumours described in Egypt and elsewhere.

Table II

Changes in Rectal Mucosa in S. mansoni Infestation

	Total No. with *S. mansoni*	No. with Normal Mucosa	Changes
A. Africans	20	17 (85%)	3 (minute haem.)
B. Europeans	8	7	1 (child with small papilloma)
C. Coloureds	10	8 (80%)	2 (minute haem.)

The main purpose of this lecture is to speak about the clinical features on what I shall refer to as the uncomplicated or simple case of intestinal schistosomiasis when, as yet, there are no other alleged complications such as cirrhosis of the liver or pulmonary hypertension.

As I mentioned earlier, from our autopsy studies in the African, at any rate, it is risky to speak of a pure *S. mansoni* infection, as the great majority have *S. haematobium* as well. In the Coloured and Indian children, too, a fair number have both infestations, and in the European we probably encounter a good proportion with only one parasitic infestation. Thus in my view it would be incorrect to speak of the symptoms of the disease in Africans who come from a district in which both infections occur.

Table III gives a summary of our autopsy findings on 100 consecutive adult Africans coming from both Rhodesias, Nyasaland and Mozambique. This clearly shows how rare it is to meet with a pure *S. mansoni* infestation in the African of Central Africa.

Table III

The frequency of *S. mansoni* and *S. haematobium* at autopsy (Gelfand, Clarke and Simpson) performed on 100 consecutive adult Africans dying in the Salisbury African hospital and drawn from Rhodesia, Nyasaland, Mozambique. Portions of different organs subjected to digestion in KOH.

Number of autopsies	100
Positive (bilharzial ova)	64
Negative	36

The 64 positive cases showed the following types of ova:

Pure *S. haematobium* infestation	40
Mixed *S. haematobium* and *S. mansoni*	21
Pure *S. mansoni* infestation	3

Thus in 24 autopsy cases showing *S. mansoni*, *S. haematobium* ova were demonstrated in 21 (87.5 per cent.). Elsewhere in Africa others have also observed the frequency with which the two forms occur in the same individual, although little comment is made on it. For instance, Schneider (1954) found that double infections occur frequently in Africans from the Northern and Eastern Transvaal.

In the living, however, such a high frequency for the two parasites is difficult to show. Nevertheless, in my Coloured and European series I was able to show that in the former 22 per cent. had *S. haematobium* as well, and in the latter 23 per cent.—a significantly high association which ought to be remembered in clinical work.

Table IV

NUMBER OF COLOURED AND EUROPEAN SUBJECTS SHOWN TO HAVE DOUBLE INFECTIONS

A. *Coloured and Indian Children from 9 to 17 years*—

Number of cases of *S. mansoni*	40
Number who also had *S. haematobium*	9 (22%)
Number who probably also had *S. haematobium*	3

B. *Europeans (Mostly Young Adults)*—

Number of cases of *S. mansoni*	27
Number who also had *S. haematobium*	6 (23%)
Number who possibly also had *S. haematobium*	2

It would therefore be better to study Nelson's description of the disease in the West Nile district of Uganda; but even there I feel we are on dangerous ground, because the cases originate in a malarial endemic zone where, too, the diet is not all it should be, and so it would be very easy to erroneously attribute symptoms such as tiredness to *S. mansoni*. Thus, in my view, the best groups in which to study symptoms are the European and Coloured children as seen in Salisbury, where malaria is rarely seen nowadays and the nutritional factor is very satisfactory on the whole. Now in them I found that about 30 per cent. of the children were unaware that anything was amiss and the disease was discovered through routine school medical examinations by the Health Department. I have questioned a number of them and there is no doubt that many of them believed that they enjoyed good health and were unaware of anything unusual and somewhat surprised to find that they had to undergo a course of treatment. According to some authorities, such as Loveridge *et al.* (1948) and Jordan and Randall (1962), schistosomal infection may in fact improve the scholarly attainment of the child in some instances, but I must say this has not been my experience.

I enquired of each child attending school whether his work altered since becoming ill with schistosomiasis. It is difficult to draw any conclusions except to say that in some the standard drops. More often than not no alteration is observed and very occasionally is it improved.

Table V

EFFECT ON SCHOOL WORK AS DENOTED BY DIRECT QUESTIONING OF THE PUPIL

	Number of Scholars	Impaired	Unimpaired	Improved
1. Europeans	8	4	3	1
2. Coloured and Indian	15	4	11	—

Double Infections (*S. mansoni* and *S. haematobium*)

	Number of Scholars	Impaired	Unimpaired	Improved
Coloured and Indian	8	1	7	—

There can be no doubt that in the majority of the cases with *S. mansoni* infestation there is a lowering of the normal standards, and I think the feature that stands out more in this than in any other tropical infestation I know is the great tiredness, listlessness and lack of energy that so many children openly admit they feel. Often this is the only complaint, but at times it can be quite severe—almost like a myasthenia. This lethargy is noticed by the mother or father. The child is apathetic, falls behind in games and lacks enthusiasm for anything. This tiredness is most striking at times when it is even more marked than that found in children with diseases like tuberculosis.

There are many children who are not so tired, but on questioning confess that they tire easily during games or other activities.

Table VI

EFFECT ON ENERGY

1. *Europeans, Mostly Between 12 and 25 Years*—

Number of subjects	23
Energy impaired	18 (78%)
Number unimpaired	5

2. *Coloured and Indian Children, Mostly Between 9 and 17 Years*—

Number of subjects	25
Energy impaired	15 (60%)
Number unimpaired	10

When we consider the important subject of intestinal symptoms it is not clear in the literature how much actual disturbance there is and how frequent and severe it is. On the whole, workers in the Sudan and Egypt stress the severe diarrhoea with the passage of blood and mucus (Greany, 1952; Gheita, 1955). I think it is safe to say that a true dysentery is rarely encountered. In the European and Coloured patients I carefully investigated I found it absent. The same applies to the African, in whom a true dysentery seems to be uncommon. It is not easy in the African to be certain that we are not dealing with other complicating diseases. I would estimate that 40 per cent. of patients with *S. mansoni* did not admit having noticed any bowel disorder. In the remaining 60 per cent. of my patients diarrhoea, or more commonly termed a looseness of the bowels, was the most usual symptom, although as a rule not sufficiently marked as to worry the patient, as in most cases it was only mentioned after direct questioning on that point. Apart from diarrhoea the most significant symptoms that drew the patient's attention to the bowels was the passage of blood with or without mucus in the stools. This was not severe, but a fair number remarked that the stools contained blood with or without mucus. Seldom was much mucus passed without blood. Once blood and mucus were passed, the patient became concerned and reported to a doctor. Very few mentioned constipation as a feature of the disease.

Perhaps I should mention a little more about abdominal pain, which I believe to be an important feature of mansonal disease. Many patients complained of these pains, which were more often experienced in the upper half of the abdomen than in the lower part; and although I have not met cases resembling peptic ulcer or cholecystitis, the atypical distribution of abdominal pains should cause one to think of schistosomiasis. The pains were never severe; they were irregular in time and duration, but they were distinct enough for the patient to volunteer this information on his own.

Table VII

EFFECT ON BOWEL FUNCTIONS

A. *Africans (Mostly Young and Middle-Aged Adults)*—

Total number	73
Normal bowels	45
Abnormal	28 (33.3%)

Frequency of Symptoms Mentioned by the 28 Subjects on Questioning:

Pains	23
Diarrhoea	21
Blood	12
Mucus	6
Constipation	1
Pain and diarrhoea	9
Diarrhoea and blood	5
Blood and mucus	0
Diarrhoea, blood and mucus	2
Pain, blood and mucus	1
Pain, diarrhoea, blood and mucus	1

Pains mentioned on 20 occasions in the 45 cases with normal bowel function.

B. *Coloured and Indian Children (Up to 20 Years)*—

Total number with *mansoni*	24
Normal bowels	21
Abnormal bowel action	3 (12.5%)

Frequency of Symptoms Mentioned by the Three Cases:

Constipation	1
Diarrhoea	1
Blood	2
Pain	1

Of the 24 cases, six mentioned pain. Site of the pain: epigastric, 1; left side of abdomen, 1; left hypochondrium, 2; periumbilical, 1; liver area, 1; pain general, 1.

C. *Europeans*—

Total number	23
Bowels normal	12
Abnormal bowel function	11 (47.8%)

Frequency of Symptoms Mentioned by the Eleven Cases:

Constipation	0
Diarrhoea	3
Blood	4
Mucus	2
Diarrhoea, blood and mucus	1
Mucus, blood and constipation	1

Site of pains complained of in 11 subjects out of the 23: epigastric, 3; general, 2; right lumbar, 2; right hypochondrium, 2; right renal, 1; R.I.F., 1.

Dyspepsia I think is unusual and pain after food not to be expected, but loss of appetite is a much more valuable symptom due to the toxic effect of the disease. But patients did not often complain of an alteration in appetite.

Table VIII

Effect on Appetite

A. *Europeans*—

Number of cases	9
Appetite good	8
Appetite poor	1 (with dyspepsia)

B. *Coloured*—

Number of cases	20
Appetite good	14 (70%)
Appetite poor	6 (one had nausea)

A varying amount of weight loss is sometimes found.

Table IX

Effect on Weight in S. mansoni Infestations

Europeans: Youths and Young Adults—

Number of cases	14
No change	4
Loss	9 (64%)
Increase	1

In six the weight lost was given as 26, 15, 15, 18, 32 and 10 lb.

Coloured and Indian Children: Not recorded.

There are two signs which should be mentioned when dealing with uncomplicated intestinal schistosomiasis. The first is a mild fever which is worth watching for, as it is a valuable one and is rather liable to be overlooked. The temperature seldom rises above 99° F. or 99.4° F., and although it may continue for many weeks for as long as the patient is untreated, the temperature may drop to normal for a day or a few days and then rise again for a variable period. But occasionally the fever is marked, reaching perhaps 100° F., and then the patient himself became aware of it.

Table X

Temperature Recorded in S. mansoni Cases at the Time of Examination Only

A. *Coloured and Indian*—

Number of cases	11
Temperature elevated	10 (between 99° F. and 99.8° F.)
Normal temperature	1

B. *European*—

Number of cases	7
Temperature elevated	5 (mostly between 99° F. and 99.8° F.)
Normal temperature	2

Despite this fever and the activity of the disease, the B.S.R. was generally well within normal limits, but in some of the cases it was raised.

Table XI

Erythrocyte Sedimentation Rate (Westegren)

A. *Coloured and Indian*—

Number tested	15
Normal E.S.R.	9 (75%)
Raised E.S.R.	6 (18 mm., 27 mm., 17 mm., 25 mm., 16 mm.)

(Note.—Of these six cases, four had double infections.)

B. *European*—

Number tested	7
Normal E.S.R.	5
Raised E.S.R.	2 (20 mm., 90 mm.)

Perhaps at this point I should refer to the early phase of the disease, generally known as the Katayama syndrome. This may occur with either the urinary or intestinal parasite and is usually typical and is generally seen a few weeks after exposure to infected water. The child runs a mild or moderate fever similar to that in typhoid or abortus fever, he feels poorly and later notices weals on different parts of the body. At this stage the spleen and liver may be palpable. A few patients cough; others complain of abdominal discomfort. A characteristic feature is the eosinophilia which is high during this stage, the eosinophils often reaching 50 per cent. and over in the white cell count. Ova begin to appear in the stool two or more weeks after this. It is not without interest to mention that Walt (1954) recorded in Durban the clinical features of Katayama syndrome.

In my experience the typical picture of the Katayama syndrome is rare, but I think there are mild or atypical forms in which the fully-fledged picture is not apparent. I have not as yet met it in the African, which is surprising, as I would expect children with the severer forms at any rate to be admitted to hospital or taken to outpatients with it.

Another early sign that should always be sought is a slightly or moderately enlarged smooth liver. Although it is said to be a common finding in the West Nile districts, I have encountered it only in the minority of cases and not sufficiently often to be of much help in the diagnosis. I think the state of the liver in the African is often affected by malnutrition and, further, it commonly enlarges in malarial districts, therefore this sign is of very doubtful value. Splenomegaly is even more doubtful in regions where malaria is known to occur.

Personally I do not consider splenomegaly a feature of uncomplicated bilharziasis except perhaps in the early phase of the Katayama syndrome. Nor is icterus a characteristic of schistosomal hepatitis, although I once thought it possible. However, whilst a hepatomegaly is not often found, it is a sign that should not be overlooked, for though it is not specific of schistosomiasis, its presence should remind us of the possibility.

Table XII

Enlargement of Liver and Spleen

A. *Coloured and Indian Children (8 to 16 Years)*

	Total Number Examined	No Enlargement	Number Enlarged
Liver	35	30	5 (16.7%)
Spleen	35	35	0

B. *European (12 to 30 Years)*

	Total Number Examined	No Enlargement	Number Enlarged
Liver	20	17	3
Spleen	20	20	0

In the uncomplicated stages of intestinal bilharziasis the liver function tests are almost invariably normal.

Table XIII

Liver Function Tests in Uncomplicated S. mansoni Disease

A. *Coloured*—

(1) Flocculation Tests:

Number of cases 15
Normal flocculation tests 12
Abnormal flocculation tests 3

(2) S. Proteins:

Number of cases 13
Normal S. proteins 13
Abnormal S. proteins 0

(There were also five Coloured children with both *S. mansoni* and *S. haematobium* infestations, but liver function tests were normal.)

B. *Europeans*—

Number of cases 13
Normal flocculation tests 13
Normal S. protein 9
Abnormal flocculation tests 0
Abnormal S. proteins 2

(A.G. ratio just below one due to increased globulins)

Walker *et al.* (1954), on investigating the blood loss in stools of adult S. African Bantu with *S. mansoni* infestation, found that the blood loss of small moment. On the other hand, Nelson (1958), working in the West Nile district of Uganda, considered that *S. mansoni* may be considered one of the factors responsible for the low haemoglobin levels there.

In Table XIV is shown the main relatively simple haematological procedures carried out on this series of patients. It will be observed that more often than not the haemoglobin level of the blood is not changed, and if anaemia was found it was usually only mild. The total leucocyte is generally normal, but occasionally the total count may be increased. But of special diagnostic value is the presence of a blood eosinophile which is observed in over two-thirds of the subjects. Still, it is worth noting that in about a third no eosinophilia was recorded at the time of examination.

Table XIV

Haematological Findings (Including Haemoglobin Levels, Total White and Differential Counts)

A. Haemoglobin Levels

1. *Coloured and Indian*—

(a) For *S. mansoni* Infestation Only:

Number tested 8
Normal 3 (37.5%)
Under 85 per cent. 5

(b) For Both *S. Mansoni* and *S. haematobium* Infestations:

Number tested 10
Normal 7 (70%)
Below 85 per cent. 3

2. *European*—

(a) For *S. mansoni* Infestation Only—

Number tested 8
Normal 6 (75%)
Below 85 per cent. 2

B. Total Leucocyte Count

1. *Coloured and Indian*—

Number determined 16
Normal 13 (81%)
Increased ..... 3 (14,000, 17,000 and 18,000)
Decreased ..... —

(Of the 16 cases, five had a double infestation, but no special effect noted on count.)

2. *European*—

Number determined 15
Normal 13 (86%)

Increased	2 (12,300 and 18,000)
Decreased	—

C. Eosinophilia

1. *Coloured and Indian*—

Number Determined	Number Increased	Normal 0-4%
19	13 (68.4%)	6

Up to 4 per cent.	6 cases
Between 5 and 10 per cent.	5 „
Between 11 and 20 per cent.	6 „
Plus 21 per cent.	2 „

(Five had double infestations, but no special effect noticed on the degree of eosinophilia.)

2. *European*—

Number Determined	Number Increased	Normal 0-4%
17	12 (70%)	5

Between 0 and 4 per cent.	5 cases
Between 5 and 10 per cent.	3 „
Between 11 and 20 per cent.	6 „
Plus 21 per cent.	3 „

The Complicated Form of Intestinal Schistosomiasis

This is a necessary category in order to distinguish between the more clearly defined uncomplicated form of *S. mansoni* disease from its much more difficult and debatable later stages. For instance, many workers do not agree that hepatic cirrhosis with or without splenomegaly or pulmonary hypertension, consequent upon ovideposition, in the pulmonary circulation are late manifestations of the disease, yet the Egyptian and South American schools of thought seem to indicate that there is no doubt whatsoever about these relationships. Even if ova are found in sections of liver and lung tissue it does not mean they are responsible for the cirrhosis or pulmonary hypertension in that particular patient, as both may be present coincidentally, because schistosomiasis is so common. As a result, it becomes difficult to prove an association under such circumstances.

It would seem that all agree that schistosomal disease is not responsible for Laennec's cirrhosis (septal or multilobular cirrhosis) or that form of cirrhosis known as postnecrotic scarring. But authorities have always claimed that schistosomiasis, especially the intestinal form, is responsible for the clay pipestem cirrhosis in which there is a diffuse granulomatous schistosomal process in the portal tracts. The parenchyma is not affected except by secondary pressure caused by contracting fibrous tissue in the portal system. Although most workers consider that granulation tissue is produced by the ova themselves, others like Professor Gillman (1957), formerly of this university, believe that the lesions are primarily in the portal radicles and arise from an endophlebitis due to the presence of dead adult worms in the lumina of their vessels.

In contrast to Egypt, few workers in centres in other parts of Africa have been struck by the relationship of clay pipestem cirrhosis to schistosomiasis. In both Johannesburg and Durban it is either not encountered or seen only very rarely. Should such a relationship be established, schistosomiasis must be viewed with the utmost concern, as this form of hepatic disorder is accompanied by all the well-known complications of portal hypertension, viz., splenomegaly, oesophageal varices and the inevitable haematemesis which may follow this pressure. Many cases, too, are complicated by an ascites, anaemia and encephalopathy.

For a number of years I have been doubtful as to whether there is a definite relationship, although in 1950 I was prepared to accept that, in such patients with a general diffuse deposition of ova in the portal tracts, so much granulomatous and fibrous tissue may ensue that the liver is greatly disturbed by all the concomitant sequelae. This point of view was supported by the results of our liver studies at the Harare hospital. In 1962 Forbes and I published our biopsy findings in adult Africans (mostly young) affected by cirrhosis. Schistosomal lesions were exceptional. If clay pipestem cirrhosis were common, we expected to find at least a few cases in this series characterised by diffuse schistosomal granulomatous tissue in the portal tracts. We did not find this, although at least 60 per cent. of our population is affected by schistosomiasis. However, I noticed in my juvenile series of hepatic cirrhosis that *S. mansoni* was found much more frequently than one would expect in the general population and I suggested (Gelfand, 1961) that schistosomal cirrhosis of the liver might be a more important possibility than has been accepted in Southern Africa. I continued studying both my juvenile and adult cirrhotic patients by performing a liver biopsy on each, and to my surprise I have found that about half of my juveniles have schistosomal lesions in the liver, but only very few of the adults have them. It has therefore occurred to me that in this part of Africa, at any rate, schistosomal clay pipestem cirrhosis occurs mainly in the young, who are likely to be carried off by it before they reach adulthood. We do not know the natural course of hepatic cirrhosis in the young African, but being familiar with the severity of this disease I should

not be at all surprised to find that it is generally followed by early death, whereas the cirrhosis in the adult is of a different type. I consider I have enough evidence to show that bilharzial clay pipestem cirrhosis is found in the young, and therefore I should like to follow this up by stating that, as a result, when the infestation is heavy, schistosomiasis is a serious disease. Further, in the African, at any rate, hepatic cirrhosis due to schistosomiasis is not uncommon in the young, but in the adult cirrhosis of the liver usually has a different cause. In the juvenile, however, not all with cirrhosis have schistosomiasis. There are clearly, as in the adult, an appreciable number who do not suffer from schistosomiasis, but develop cirrhosis for other reasons.

Table XV

Number with Bilharzial Lesions in Cirrhosis of Liver, Determined by Liver Biopsy

Adult Africans, From 20 Years Onwards—	
Total cases	31
Number showing bilharzial lesions	3 (10%)
Number without bilharzial lesions	28
Juvenile Africans up to 20 Years—	
Total cases	12
Number showing bilharzial lesions	6 (50%)
Number without bilharzial lesions	6

Similarly, the serious effects of ovideposition in the lungs have been claimed in numerous publications from Egypt and South America. Much of the evidence is convincing. Not only are the pulmonary arterioles occluded by ova, granulation tissue and ultimately by fibrous tissue, but a very typical lesion, the angiomata, develops near the site of occlusion. In other parts of Africa, outside Egypt, pulmonary hypertension with right heart failure is most unusual, and as far as I am aware has not been reported from South Africa. In Southern Rhodesia and also in Northern Rhodesia I have come to believe it occurs, as I have seen the occasional case, but I must confess such cases are few and far between. As with the liver, it can be argued that pulmonary hypertension and ova are coincidental. Although the Egyptians and South Americans find heart failure (cor pulmonale) a frequent and serious complication of schistosomal infestation, I believe that it is far less common in Central Africa.

I wish to refer briefly to the claim made by a doctor practising on the shores of Lake Victoria, in Uganda, that *S. mansoni* disease is sometimes accompanied by tumour masses, which may produce an obstruction in the bowel. Whilst I personally have not encountered such a case, nor have my surgical colleagues practising in the Harare hospital, I must admit this possibility, as his article is very impressive and convincing. Still, it must be very rare, and why it should be confined to the environs of Lake Victoria I cannot say.

It should be remembered that not always are the ova of *S. mansoni* present in the rectum, for in three out of 24 *mansoni* cases found at autopsy no ova were detected in the specimens studied, although they were to be found in the bladder. Thus it is quite possible that from time to time bladder symptoms such as hypogastric pain dysuria and haematuria may result from an intestinal infestation.

Conclusion

Intestinal schistosomiasis often produces marked tiredness, and this is possibly its most outstanding feature. Many, too, have a mild fever and the majority of subjects show a blood eosinophilia. Vague abdominal pains and a bowel upset not infrequently form part of the clinical feature, but it is rare in my experience to meet a true dysentery. Of the complications, probably its most serious effect is on the liver, but it would seem that clay pipestem cirrhosis is more often encountered in Central Africa in the relatively young person and that the adult is rarely affected by it. Pulmonary schistosomiasis leading to right heart failure would also seem to be rare.

These, then, are my findings, but I want to repeat that far more interest by the clinician should be shown in intestinal schistosomiasis, as much more useful information will be revealed.

REFERENCES

De Neukkir, G. (1948). *S. Afr. med. J.*, 22, 253.

Forbes, J. I. & Gelfand, M. (1962). *Trans. Roy. Soc. trop. Med. & Hyg.*, 56, 305.

Gelfand, M. & Ross, W. F. (1949). *Trans. Roy. Soc. trop. Med. & Hyg.*, 42, 559.

Gelfand, M. (1950). *Schistosomiasis in South Central Africa.* Juta & Co. Ltd.

Gelfand, M. (1961). Chapter 3, page 34, in *Cirrhosis and Primary Cancer of the Liver in Trans-Sahara Africa.* Union Internationalis contra Cancrum, edited by J. N. P. Davies.

Gelfand, M., V. de V. Clarke & Simpson, B. (1963). In preparation.

Gheita, A. (1955). *J. Egypt. med. Assoc.*, 38, 102.

Greany, W. H. (1952). *Ann. trop. Med. & Parasit.*, 46, 298.

Gillman, T. (1957). *Ann. trop. Med. & Parasit.*, 51, 409.

Jordan, R. & Randall, K. (1962). *J. trop. Med. and Hyg.*, 65, 1.

Loveridge, F. G., Ross, W. F. & Blair, D. M. (1948). *S. Afr. med. J.*, 22, 1.

Pitchford, R. J. (1952). *S. Afr. med. J.*, 26, 524.

Walt, F. (1954). *S. Afr. med. J.*, 28, 89.