SCHISTOSOMIASIS -

WITH PARTICULAR REFERENCE TO THE DISEASE IN SOUTHERN RHODESIA TOGETHER WITH SOME NEW OBSERVATIONS

Andrew Paton Martin.
M.B. Ch.B. (Cape) D.P.H. (Cantab)
D.T.M.&H. (Cantab).

CHAPTER I

DEFINITION:

Schistosomiasis is the name given to the disease which results from the morbid changes induced in the tissues of the human host by the presence and activities of male and female Trematode worms belonging to the Schistosomidae family.

In Southern Rhodesia, as in Africa generally, two forms of this disease, associated with two distinct species of Schistosome, are at present recognised; one, the urinary form, associated with the presence in the human body of Sch.haematobium; and the other, the intestinal form, associated with the presence of Sch.mansoni. Both forms of this disease can be present in the same individual at the one time. The urinary form of the disease is characterised by various bladder disturbances, by the exhibition of haematuria generally occurring at the end of micturition and by the presence of eggs in the urine; the intestinal form, by various dysenteric symptoms and by the presence of eggs in the stool.

The eggs laid by the female worm are responsible for by far the greater part of the morbid changes which occur in the tissues of the human host. These changes arise not merely from the presence of the eggs acting as foreign bodies, but are actively induced by the toxins elaborated by the eggs to ensure their own penetration of the tissues. In addition many pathological changes result from the movements of the growing and adult worms, and from the action of the various toxins which these worms produce.

CHAPTER II

HISTORICAL REVIEW:

There is ample evidence to support the belief that this disease originated in ancient Egypt. The classic symptoms of the condition, such as haematuria were known in Egypt from the earliest times, and the ancient papyri are strewn with references to the presence of blood in the urine as a condition common in incidence and necessitating medical attention. Eber's papyri contain prescriptions for

the treatment of haematuria, and there are various other references in the Kahun papyrus of the 12th Dynasty (3000 B.C.) and in the Hearst, the London and the Berlin papyri of the 18th Dynasty.

In addition, vesical calculi have been found in mummies of various dynasties; and prehistoric bodies, even those of young children, have been discovered exhibiting not only stones in the bladder, but other even more definite pathological changes which in the light of our present day knowledge, can only be regarded as attributable to schistosomial infection.

Sir Armund Ruffer (1910) found Schistosome ova in mummies dating back to 3000 - 4000 B.C.; he has also demonstrated calcified Schistosome ova in the nuclei of urinary calculi and in sections of the Cortex of Kidney taken from mummies belonging to the 20th Dynasty.

Further evidence of the frequency of incidence and of the views held by the Ancient Egyptians as to the method of contraction of the disease can be found in the two main measures of prophylaxis which they adopted for its prevention and which are described in their ancient writings and wall-paintings. From these it is evident that the Ancient Egyptians believed that infection took place through the urethra.

1. The Penis Shield:- Figures wearing this appliance can be seen on the walls of some of the temples. It is interesting to note the persistence of this prophylactic measure to recent times. Leiper has recorded the advice given in comparatively recent years to British soldiers proceeding to Egypt to use the modern equivalent of the Penis Shield, the French-letter, for protection against the contraction of Schistosomiasis, when bathing.
2. Circumcision:- Whatever its religious significance, the practice of this rite had, in Egypt, a very definite hygienic basis as a measure of prophylaxis against infection by Schistosomiasis.

Frequent references to haematuria occur both in ancient and mediaeval writings and various remedies are suggested for its

treatment. For instance, Ch.de la Roncière (1924) has recorded as the result of his historical investigations that the Caravan-Men who journeyed across the desert between Tafilalet and Timbuktu in the 14th Century were affected by haematuria which was probably of schistosomial origin. Again, Army Surgeons who accompanied Napoleon's troops when they invaded Egypt (1799 - 1801) have described how severely the men were attacked by haematuria and how extensive the incidence of this disease was amongst the Native population. Finally, in 1847 Pruner, a director of Kasr-el-Aini, published a description of Schistosomiasis of the bladder, but it was not until four years later that the significance of this description was appreciated and the cause of the condition discovered.

Theodore Bilharz, (1825 - 62), whilst working at Kasr-el-Aini as assistant Professor of Medicine, discovered, in 1851, a Trematode worm in the portal vein of a young man. In May of that year he wrote to his former teacher, von Siebold, and announced his discovery of a bisexual distome which he termed "Distoma haematobium". Further, he succeeded in establishing a definite relation between this trematode and the symptoms of haematuria and dysentery resulting from the lesions in the bladder and in the intestines which this worm produced. This discovery was announced by von Siebold to the Congress of Naturalists held at Gotha and was published in the Zeitschr. f. Wissenschaft Zoologie in 1852. In 1856, Bilharz published a lengthy paper on "Distoma haematobium and its relation to Pathological Changes in the Human Urinary Organs." Most of the observations made in that paper hold good today. Three of them deserve special mention:-

1. He estimated the incidence of infection amongst Egyptians as 50%.
2. He classified the pathological changes in the Urinary passages as -
   (a) Catarrhal Cystitis and Ureteritis.
   (b) Induration.
   (c) Polypoid Growth.
   (d) Ulceration.
3. He associated these morbid changes with the deposition of ova.

Bilharz does not appear to have made any observations on the life-history of the parasite or on the probable mode of spread of

the disease. He did, however, make one error in his writings which led to great confusion and controversy and undoubtedly delayed, for many years, the elucidation of the problem of the life-history. He recorded the presence of two types of ova in the uterus of the same female worm; one with a terminal spine, the other with a lateral spine, Owing to the authority of his position in the scientific world, established by his brilliant and careful researches, this statement was accepted as accurate and remained unchallenged for many years, even when new facts were coming to light which suggested that in this regard Bilharz had been mistaken. Controversy after controversy broke out in the scientific world whenever any new fact was announced which seemed to dispute this observation, until finally the question was conclusively settled by the Zoological investigations of Piraja da Silva and by the brilliant researches of Leiper.

In 1859, Cobbold suggested that this new genus of Trematode worm should be called "Bilharzia". Unfortunately Weinland some three months earlier in 1858 had already given to the new genus the term "Schistosome" and according to the Laws of Priority, this latter designation had to be accepted for use by the scientific world.

In 1854 Griesinger described and figured two forms of eggs, one with a terminal, and the other with a lateral spine. In 1864 Harley found, in certain localities in the Cape Colony and Natal, peculiar terminal-spined ova in the urine of patients affected by haematuria and demonstrated that they were the ova of a species of Schistosome which he termed "Bilharzia Capensis". In the same paper, following the observations of von Siebold, Harley suggested that between the ciliated embryo (the miracidium) and the adult sexual animal there were probably two other distinct forms which served to complete the chain of metamorphosis between the two extremes of development. "The ciliated miracidium", he wrote, "is adapted for an aquatic existence. Swimming freely about these minute organisms probably come into contact with certain mollusca and begin developing in them into what have been called "cercaria sacs"." This was an interesting and valuable suggestion

and served as a sign-post to further research.

In the meantime, Sonsino and others in Egypt were investigating the problem of the life-history of the parasite and the mode of infection of the human host. In 1871, Sonsino published a paper entitled "Schistosoma haematobium and its relation to Endemic Haematuria in Egypt." This was in many respects the first and most sustained effort in the elucidation of the problem, and correlated and set in proper perspective many of the known facts concerning the parasite.

In 1888, Allan in Natal first suggested the hypothesis that an unknown larval stage might enter the body through the skin. This hypothesis was strongly supported by Brock and others in 1894, but in so far as it suggested an infective / form of the parasite different from the miracidium was savagely criticised by Arther Looss who had constantly maintained on biological grounds that the miracidium was the infective agent. Looss was a brilliant controversalist and an able investigator. He had come to Egypt in 1894 to take over the work of research on Schistosomiasis from Leuckart, who had retired in that year. Looss worked at Kasr-El-Aini and conducted many valueable investigations which contributed to the elucidation of many problems in the parasite's life-history. But his dialectical ability was more of a hindrance than a help, since he argued with great aggressiveness and, like Socrates, was able by his skill in debate to make a poor side appear the better. On this occasion, as on others, he held strongly that the miracidium was the infective agent and that it developed into a sporocyst in the liver of the human host, and that it was from the Sporocyst that the adult forms were generated. By a series of skilful experiments he was able to demonstrate that human gastric juices killed the miracidia and so disposed of the theory that infection took place through the stomach as the result of drinking infected water. He gave his usual strongly worded support to the theory of skin infection but was implacable in his assertion that the miracidium was the infective agent.

Another important point upon which Looss' attitude delayed the earlier solution of the problem was in connection with the differentiation of the species. Basing his opinion on Bilharz's original

single observation of lateral-spined and terminal-spined ova in the same female, Looss again with an almost fanatical partiality maintained the theory of the unity of the species. As a result of his advocacy many investigators endeavoured to find explanations for the appearance of the lateral-spined egg and many ingenious solutions were put forward, none of which, however, could bear close scrutiny. Finally, the attempts were abandoned, and the subject remained neglected until Manson in 1903, as a result of an examination of a patient suffering from Intestinal Schistosomiasis who had never been to Africa, and in whose faeces he found only lateral-spined ova, postulated the theory that there were probably two species of Schistosome, one with lateral-spined ova depositing its eggs in the Rectum only, and the other with terminal-spined ova haunting the Rectum or the Bladder indifferently. Manson's patient had never had haematuria, and came from a country where the urinary form of Schistosomiasis was unknown. From this starting-point, proofs rapidly accumulated as to the truth of Manson's theory.

In 1907, Sambon in a series of papers elaborated Manson's suggestion and proposed that the species now be divided into two classes. He formally created the new species and named it Schistosoma mansoni, after Sir Patrick Manson. Sambon's action brought down upon his head all the vials of the wrath of Looss and his supporters, and a bitter controversy resulted. The truth, however, was finally established by accumulated proofs forthcoming from many workers and was put beyond all question of doubt or dispute by the brilliant and conclusive researches of Leiper.

Shortly after the outbreak of the Great War, Looss, a German subject, was removed from Egypt, and the work of research was entrusted to Leiper. In a series of magnificent researches, 1915 - 1918, this brilliant worker not only settled the question of the existence of two species but in addition discovered the whole life-history of the worm and solved most of the problems connected with it.

Since that time, a great deal of work has been carried out in the epidemiology and treatment of the disease and in the morbid anatomy of the human tissues. Ruffer, Symmers, Fergusson and Aly Pasha Ibrahim in Egypt have all contributed substantially to our

knowledge of the condition, and Girges in a particularly able publication in 1931, correlated and classified the facts established concerning the disease up to that time; the latter writer in addition investigated the distribution of the lesions and the pathogenic factors concerned in their production, and made a valuable contribution to the stock of knowledge.

The surgery of Schistosomiasis was first put on a scientific basis by Herbert Milton, and later workers including Frank Milton, Richard, Madden and Aly Pasha Ibrahim, elaborated his technique and laid down clear and sound rules for surgical procedures.

On the medical side, the introduction of the use of tartar emetic by Christopherson in 1917, completely revolutionised the treatment of the disease and in many cases rendered surgical procedure unnecessary. Later, Khalil, head of the Research Section of the Egyptian Public Health Department, brought to the notice of the medical profession, another antimonial preparation, since called Fouadin, which was less toxic in effect and extremely valuable in the treatment of children.

In Southern Rhodesia the existence of the disease was recognised from the earliest days of the Occupation in 1890, but until recent years little consideration was given to the extent of its incidence or to the malevolent effect it had on the health of the people. The more dramatic and the more easily recognised effects of malaria obscured the Schistosomial position and for many years practically every form of indefinite illness was considered to be some or other manifestation of the former infection. In 1927 a research worker from the London Tropical School of Medicine investigated the parasitological aspect of Schistosomiasis in Southern Rhodesia, but little use was made of the facts resulting from this enquiry and the position in the Colony remained unchanged. However, in 1937, it appeared to me probable that the Trustees of the newly constituted State Lotteries might be induced to use some of their funds for the prosecution of a campaign of research into the two main diseases which beset the people of this country, namely Malaria and Schistosomiasis. I accordingly formulated the plan described later under the heading "Control", presented it to Government for

acceptance, and was successful in persuading the Trustees to finance it.

In 1938, the Medical Research Unit of Southern Rhodesia was duly inaugurated and one of the main functions of this body is an investigation into the incidence of Schistosomiasis and its causal factors in this country with a view to their reduction or if possible, their elimination.

ILLUSTRATION.

THE DISTRIBUTION OF SCHISTOSOMIAL DISEASE

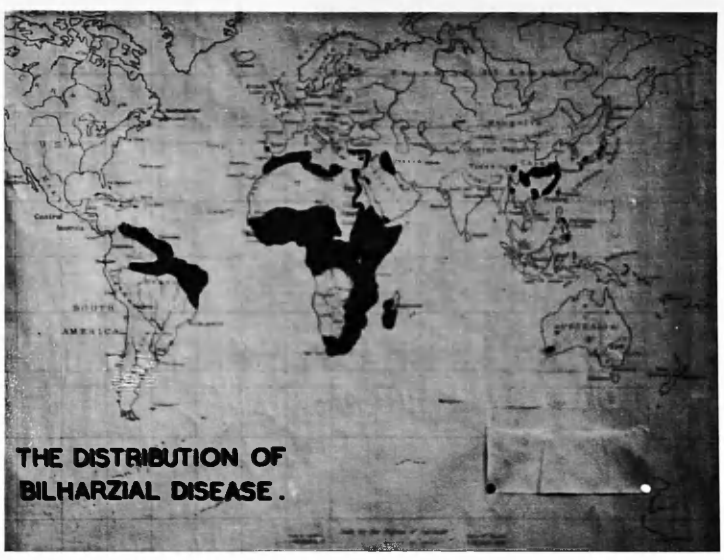

Fig.1.

CHAPTER III

GEOGRAPHICAL DISTRIBUTION:

The geographical distribution of the disease in Africa is shown in the accompanying map; and attention is specially drawn to the still peculiarly limited distribution of a disease which is known to have been in existence for at least four thousand years. This limitation is correlated with the life-history of the parasite and its dependance upon specialised intermediary hosts whose distribution is circumscribed by factors relating to food-supply and to a suitable climatic environment. Manson drew attention to this fact long before the life-history of the parasite was known, and deduced from it the probable existence of an intermediate stage, spent in an intermediate host of limited distribution.

Fig.1

From a study of the distribution of the disease in Southern Rhodesia, support is given to the opinion that the spread of the disease follows along the lines of a country's road communications. In Southern Rhodesia, the condition was at first confined almost entirely to the Eastern Districts of the Colony and to the areas developed from them. Today, however, practically all the rivers in some part or other of their course afford accommodation to the carrier-snails and are infected. I do not mean to suggest that the infection is universal throughout this Colony. On the contrary there are many streams which are entirely free from Schistosomiasis. But I do definitely mean that at some part or other of their main course or of their subsidiary tributaries, almost every river in Southern Rhodesia harbours carrier-snails, and that part of the river where these are present is almost always infected. In two different Rhodesian streams examined during the past year, the carrier-snail population exceeded one thousand individuals to the square yard of the stream bottom.

Whilst no definite evidence is obtainable, it is interesting, nevertheless, in view of the Colony's history, to speculate on the method of the disease's introduction to the country and its subsequent lines of extension. Very little is known of Southern Rhodesia's history prior to the invasion of Matabeleland by the Matabele under Umziligasi in 1842, and the occupation of the North-Eastern province of Mashonaland by the Pioneer Column sent by Rhodes in 1890. The

Pioneers established themselves principally in two areas; the district in and around the present capital city of Salisbury, and the district in and around the now abandoned site of the first township of Fort Victoria. This latter site was close to the Eastern boundaries of the province which adjoined Portuguese Territory; and near to the site of their township the pioneers found the massive ruins of an ancient fort and temple, obviously the remains of some previous civilization and culture. Later it was discovered that from this fort there extended, almost in a straight line, a chain of smaller forts or possibly trading-stations which led through Portuguese Territory due eastwards to the small but ancient harbour of Sofala, a little town on the coast of Portuguese East Africa. Now it was in the district round Fort Victoria that the first cases of Schistosomiasis were observed, and later on as the country became more settled and investigation became possible, it was in this area that by far the heaviest concentration of the disease was found. This, of course, may be correlated with two known facts; first that Fort Victoria was one of the earliest stations to have a Hospital and proper medical facilities, and secondly that even from the earliest days of the Occupation a comparatively large number of poor whites of Dutch origin settled in this area. However, these two facts do not altogether cover all aspects of the case.

A further interesting point in the history of those times is that communications between the warlike Matabele and the poverty-stricken terrified Mashona were confined almost entirely to one or two periodic annual raids by the former, in which they killed as many Mashonas as possible, stole all the cattle and grain they could find, and carried away as slaves and concubines as many of the young women as pleased them. There was thus no friendly communication between the two peoples, and the Mashonas departed with all haste to their mountain tops on the earliest rumours of a Matabele raid. Now it is a curious fact that although Schistosomiasis particularly of the urinary type was found to be rife amongst the Mashonas from the earliest days of the Occupation, very little evidence indeed of this disease was found among the Matabele. And correlated with that observation is the further fact that the

disease first manifested itself in the Europeans resident in the Eastern section and for many years was totally unknown amongst Europeans in the Matabeleland end of the territory.

Further, until the advent of Responsible Government in 1924, communications inside the Colony were confined to the Railway and to a very few roads which were only usable during the dry season. In 1925, however, the Government of the day embarked upon a large road-building programme and within a few years the country was covered with a net-work of public highways and subsidiary roads which opened up almost all parts of the territory. Since that date, it has been extremely interesting to note the rapid spread of Schistosomiasis throughout the Colony and to observe how it extended itself, first along the rivers and streams, lying adjacent to the highways, and latterly into many of the natural waters of the country in the vicinity of which its human host could be found. Today, the rivers and streams of Matabeleland are almost as heavily infected as those of Mashonaland. In the light of these facts it seems reasonable to believe that the disease was introduced into this country in early times by traders from Egypt and the North who used the town of Sofala as their port of entry and who built the line of forts between that station and the important centre of their activity in Eastern Mashonaland, Great Zimbabwe, the name which has been given to the mysterious mass of ruins adjacent to the town of Fort Victoria. Since natural conditions in and around Zimbabwe are very favourable to the growth and development of the carrier-snails, it is extremely likely that the disease became prevalent there and that most of the Native inhabitants in the surrounding areas became infected. Indeed, it is not unreasonable to surmise that the spread of both Schistosomiasis and Malaria played some part in the final abandonment of this former centre of civilisation. However, these are mere speculations, and interesting though they are, they cannot now be supported by proof.

A further point in connection with the difference in the distribution and prevalence of the two types of the disease in this Colony is to me of extreme interest. Whilst Sch.haematobium is the more dominant species throughout the Colony, Sch.Mansoni is also fairly widespread and has of recent years extended its presence more

widely and more rapidly. In some small areas of the Colony it is the sole species and appears to have established itself in strong predominance wherever it has found particularly suitable conditions.

This is peculiarly noticeable among rural European communities whose standards of life are low and especially so amongst the poor white Dutch class whose habits of life are in many instances even less modest than those of the African Native. The African Native defaecates alone and under the most secret conditions he can create for himself in the circumstances. This he does, not from any European ideas of modesty, but because he believed, or his fore-fathers believed and he inherits the habit, if not the belief, that any enemy having access to his faeces might, through charms and spells exercised over them, be able to do him a severe injury or perhaps cause his death. For this reason, he endeavours to hide all traces of his faeces and takes what measures he can to prevent anyone knowing even the site of their depositing. He does not mind urinating into a stream, because he reasonably considers that the volume of water so dilutes his urine that it would be impossible for any evil-wisher to recover any portion of it. Hence, whilst his ideas and precautions in regard to the disposal of his faeces acted as a deterrent to the spread of Sch.mansoni, his views on the subject of his urine had no similar effect in the case of Sch.haematobium. One further factor which, as far as the Native is concerned, undoubtedly plays a part in the curtailment of the spread of Sch.mansoni, is the common native custom of pig-breeding. Very many natives keep pigs and these animals, running loose as they do in the veldt, serve as scavengers for the areas round the kraals, thus keeping the place clear of faeces and refuse. This native practice is a further deterrent to the spread of intestinal schistosomiasis.

The poor white Dutch class and the lower type of European rural dweller had none of these practices or beliefs and in the primitive conditions under which they lived, they and their families used the veldt and the streams around their homesteads as their toilet-chambers, and thus ensured an efficient and powerful concentration of both types of the disease. For these reasons we find that in certain small parts of the rural areas, both types of the disease are common amongst the local European inhabitants, and Sch.mansoni much more prevalent than in other parts of the country.

CHAPTER IV

ETIOLOGY:

1. Residence: One of the foremost factors in the etiology of the disease is residence in an area where there is a sufficient concentration of infected persons and suitable snail hosts, and where, either due to lack of education or to the absence of adequate sanitary arrangements, the habits of some large section of the people are still primitive.

In Southern Rhodesia these conditions are to be found only in the rural areas and the smaller villages, so that with us the opportunities of infection are almost entirely confined to the country districts. Many European town-dwellers, however, owing to the Sunday picnic habit of the Colony, and the proximity of the countryside to the town, acquire the disease; generally through bathing in infected pools.

2. Occupation: Schistosomiasis being a disease associated with streams and natural waters, farmers and land-workers of all types and those whose occupations bring them into contact with water, e.g. small mine-workers, comprise the bulk of the infected population.

3. Sex: The Schistosome shows no regard for sex, and given the opportunity attack males and females with complete impartiality. The exigencies of occupation and the more noticeable penchant of the male for bathing in rivers and streams accounts for the greater number of cases occurring amongst the male population.

4. Age: No age-period is immune to the attacks of Schistosomiasis, but the greater activity of youth between the ages of ten and twenty - five years, accounts for the apparently greater susceptibility found in this age group.

In Southern Rhodesia, Schistosomiasis is a disease acquired in youth and paid for in early middle life. Save for the conditions mentioned later, the disease seldom appears to give rise to any serious systemic disturbance in the 'teens and twenties. This is due to the greater resilience of young tissues in effecting repair, and to the fact that the effects of the disease result from the number of eggs which failing to reach their objective, namely, ejection from the body in the contents of the excretory system,

wander around in the human body, and gradually accumulate there over a period of years.

In early middle life the effects of this accumulation of eggs in the tissues, no longer so active in defence and repair, become evident in the various manifestations of the disease which now occur. Obstructed or completely blocked ureters leading to hydro- or pyonephrosis; papillomata of the bladder with cystitis; papillomata of the bowel associated with chronic intestinal symptoms indicative of inflammation and ulceration; and many other similar signs of the disturbances taking place in the involved tissues now make their appearance, and the disease acquired in youth now claims its toll on the health of the individual concerned.

5. Season: The season of most frequent infections corresponds with the time of greatest snail activity, and in Southern Rhodesia, as in Egypt and elsewhere, snail activity is predominant in the warm wet months of the year; with us from November until April. Further, as that is the period during which river bathing is most frequently indulged in, the opportunities of infection are thereby greatly increased. In the winter-time when the river-waters are low and the temperature relatively cold, propagation ceases, and snails tend to disappear.

CHAPTER V

PARASITOLOGY:

The Schistosomes are members of the Trematode Group of the Worm Family. In the blood-vessels of man, three different species of Schistosomes have been found; these are -

Sch.haematobium.
Sch.mansoni.
Sch.japonicum.

General Characters: The Schistosomes differ from other Trematoda in having separate sexes. They present marked sexual dimorphism.

A. Male 1. Size: Size from about 6.9 to 1.5 c.m. in length depending on species.

2. Suckers: Has 2 suckers; one placed orally and the other ventrally.

3. Gynaecophoric Canal: Behind the ventral sucker the sides of the body become in-rolled forming a long ventral duct, the Gynaecophoric Canal, in which the female worm lies free.

4. Alimentary Canal: Commences at Oesophagus which leads into two Intestinal Caeca; these Caeca run separately for a short distance, then unite to form one Caecum.

5. Genital Glands: Four to eight Testes connected by vesiculoses seminalis opening into a Genital Pore behind the ventral sucker.

B. Female

1. Size: 1.2 cm. to 2 cm.

2. Suckers: Two, one of which is placed orally and the other ventrally as in male.

3. Alimentary Canal: As in male.

+Fig. 1a.

4. +Genital Glands: Occupy all the rest of the body and are arranged along one side in the following order from behind forwards.

(a) Vitelloria and Yolk Glands.
(b) Oviduct and Ovary.
(c) Shell Glands.
(d) Ootype.
(e) Uterus.

DIFFERENTIATING CHARACTERS

	Haematobium	Mansoni	Japonicum
Male	1.5 c.m. long	1 c.m.	0.9 - 0.6 c.m.
Cuticle	Finely tuberculated.	Grossly tuberculated.	Not tuberculated.
Testes	4 large Testes.	8 Testes - smaller.	8- Elliptical.
Female	2 c.m. long.	1.5 c.m.	1.2 c.m.
Intestine	Unite in Posterior half of body.	Unite in Anterior half of body.	Unite in Posterior half of body.
Egg	Spine Terminal. Urine, but may be in stools.	Lateral. Usually in Stools and rarely in urine.	Lateral rounded Knob. Only in stools.
Intermediate Host.	Bullinus. In Southern Rhodesia Physopsis globosa.	Planorbis. In Southern Rhodesia Planorbis pfeifferi.	Blanfordia or Hypsobia.

ILLUSTRATION.

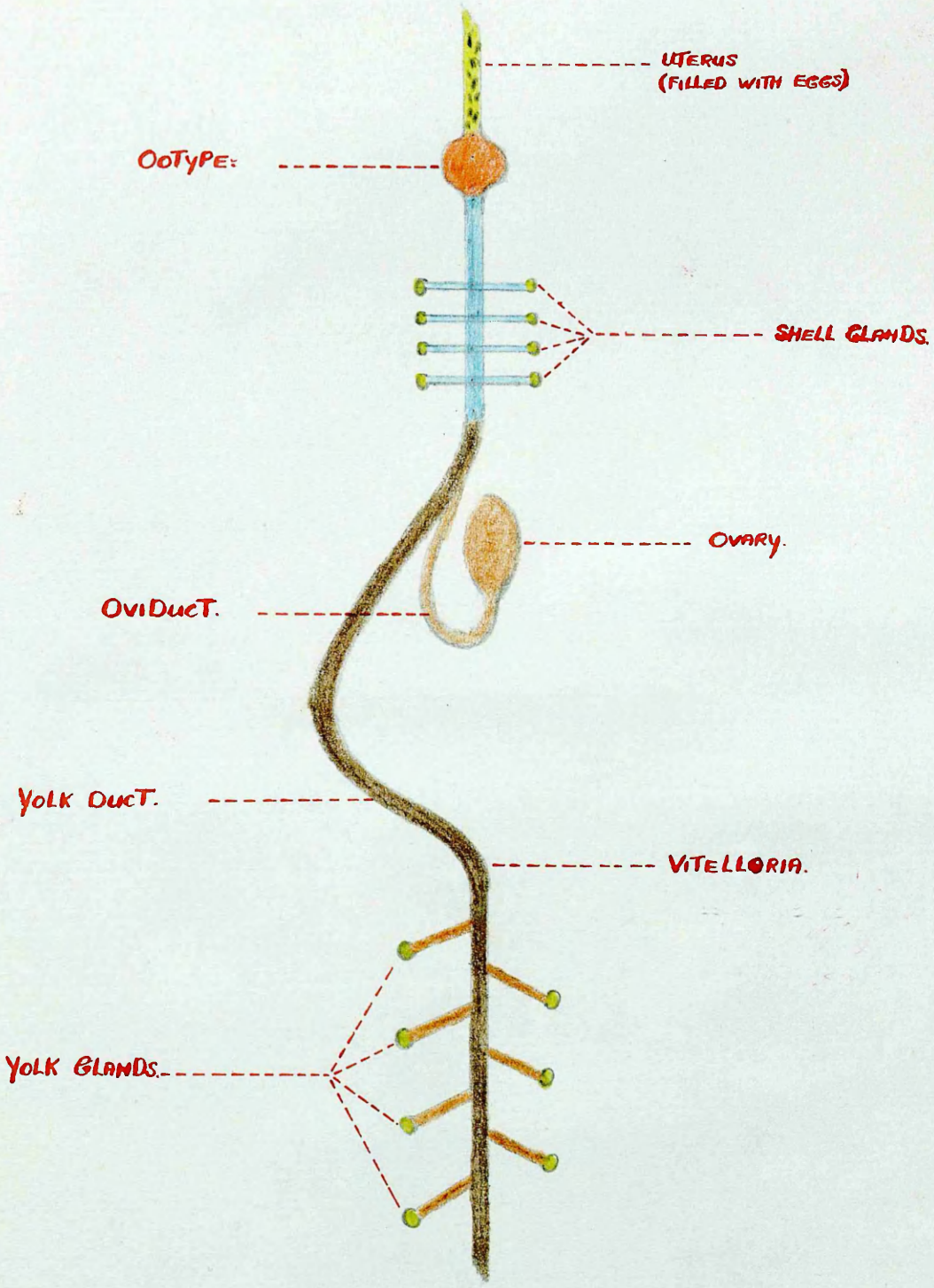

Fig.1a.

Life History: When an infected person voids a schistosome egg in his urine or faeces, that egg contains a minute ciliated embryo called a "miracidium", which can remain viable inside the shell for several days (up to five), and whose further existence will depend upon many factors. The miracidium hatches out of the egg by a transverse rupture of the shell and by this fact is at once differentiated from other types of trematoda whose "miracidia" come almost entirely from operculated eggs, that is from eggs with a specially designed lid which lifts at the moment of hatching to allow the miracidium egress.

The Schistosome miracidium is a free-swimming organism designed for an aquatic existence. It is a ciliated structure with a solid vestigial stomach sac which does not function, and has no alimentary canal. It has a primitive body space in which are contained the Cephalic Glands flanking the vestigial stomach on both sides and each opening by a short thin tube near to the anterior end of the organism. Behind the Cephalic Glands lie the two dense masses of Lateral Glands; these secrete a mucoid substance which passes through the Lateral Gland Ducts opening to the exterior at the junction of the anterior portion with the posterior three-quarters of the body.

In addition a central nervous system has been described, composed of two masses of cells lying behind the Lateral Glands. From these nerve cells emerge three pairs of nerve fibres, one pair passing to the anterior of the body-space, another pair passing to the sides, and a third pair to the posterior end. The primitive excretory system of Flame-Cells connected by excretory tubules which unite to form a single Excretory Duct has also been described. This Excretory Duct opens to the exterior through an Excretory Pore towards the Posterior end of the body. The miracidium possesses one solid organ, a Proboscis at its anterior end.

The miracidium of Sch.haematobium can live for a period of 5 days within its shell; the miracidium of Sch.mansoni up to a maximum period of 10 days; some investigators say the latter can survive within the shell for a period of 3 weeks, but this statement is not generally accepted.

The miracidium on emerging from the shell, immediately begins swimming about, but unless the urine or faeces have been mixed with fresh water, it will die within 24 hours. This is an important fact in the life-history and has been made use of in devising methods of prevention against Schistosomiasis.

If the urine or faeces have been voided in or mixed with fresh water, the miracidium not only hatches out the quicker from the shell, but also continues to live and rush around in search of its host, the snail. This distinctive type of snail it must reach within 36 hours or it will die. Further, it must reach a definite type of snail; not any snail will serve its purpose. For the Sch.haematobium, it must be a snail of the Bullinus species; for the Sch.mansoni, it must be a snail of the Planorbis type.

These two further facts in the life history of the Schistosome are of immense importance and render great assistance in the control and prevention of the disease.

If, however, the miracidium has been fortunate enough to find its distinctive snail, it then penetrates the antennae of the mollusc, casts its cilia, and becomes converted into a "Sporocyst", in the interior of which daughter Sporocysts bud and develop. These latter migrate to the liver of the snail where they rapidly multiply. Presently numerous "bifid cercariae" develop within the sporocysts and these on maturing make their way from the liver through the excretory organs of the snail and are voided into the water. The period of the parasite's life-history, from the time the miracidium enters the snail until the time the cercaria emerges, covers about two months.

The cercaria is the infective stage of the parasite and is a minute organism with a forked tail. It has no pharynx and no eye spots, but it has 2 suckers, one anterior, and one ventral, and it has in addition several pairs of salivary glands opening near the oral region.

The cercaria's life-time is, however, as difficult and as limited as that of the miracidium. The cercaria also must find its distinctive host within 24-36 hours, or death will overtake it.

This fact also is made use of in preventive measures, and as far as man is concerned it is an extremely fortunate provision that the

mere storage of infected water for 48 hours will ensure the death of all these infective organisms.

The Cercaria have little time at their disposal and immediately commence an active search for their distinctive host. If they are able to make contact with man, they immediately make their way through his skin or mucous membrane, depending upon the site of contact, and enter into the lymphatics or blood-vessels, and from their point of entrance commence their journey through the vessels of the body to the right heart, the lungs, the left heart, and eventually through the portal system to the veins of the liver where they come to rest and undergo their period of development into immature males and females.

Whilst developing in the veins of the liver, the sexes of the worms live apart, but before they reach full maturity the female joins up with the male and lies free in his gynaecophoric canal; then travelling against the blood-stream they, together, make their way downwards through the veins to their definitive positions; the Sch.haematobium to the pelvic and rectal plexuses, the Sch.mansoni to the inferior mesenteric and middle and inferior haemorrhoidal veins. The male worm is enabled to perform this journey by means of his suckers and the warty projections on his body surface. During this journey the worms feed on the blood corpuscles of the host and excrete into the blood-stream toxic products which may provoke some degree of fever. Having arrived at their selected positions the female worm leaves the male and proceeds against the blood-stream into the smaller venules of the plexuses where it begins to deposit its eggs in enormous numbers. It commences this task in the smallest venules and leaves its eggs behind it, as it gradually withdraws to meet and link up again with the male in the larger venule where he has been waiting for her.

The ova first adhere to the inside of the vessel wall and provoke an inflammatory reaction which results in the formation of a fibrin deposit around them. This enveloping projection is very noticeable in the lumen of the vessel. By means of digestive enzymes which they elaborate, the ova open their way through the venule wall and reach the surrounding perivascular tissue. From here by a further

ILLUSTRATIONS.

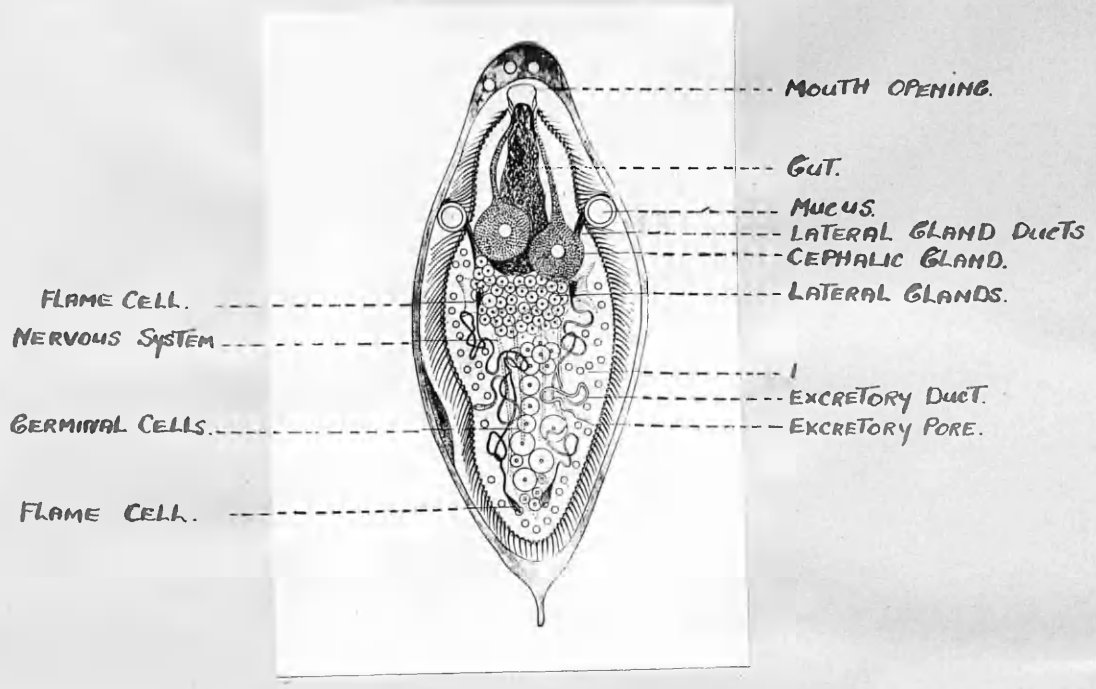

Fig.2. Fully developed ovum of S. HAEMATOBIUM. (After Girges).

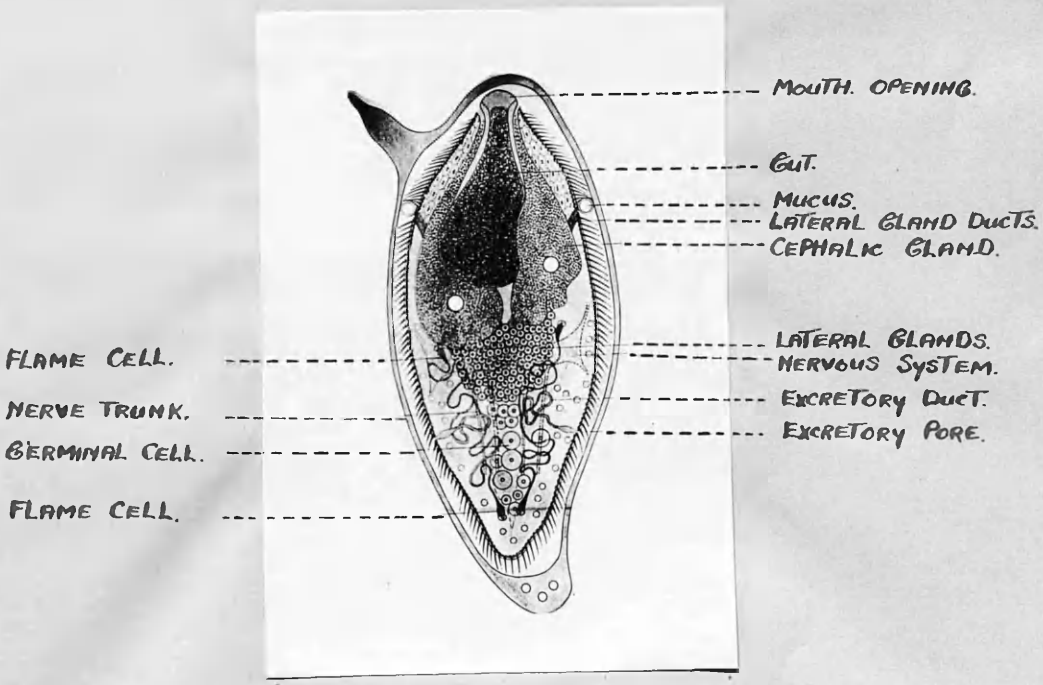

Fig.3. Fully developed ovum of S. MANSONI. (After Girges).

ILLUSTRATIONS.

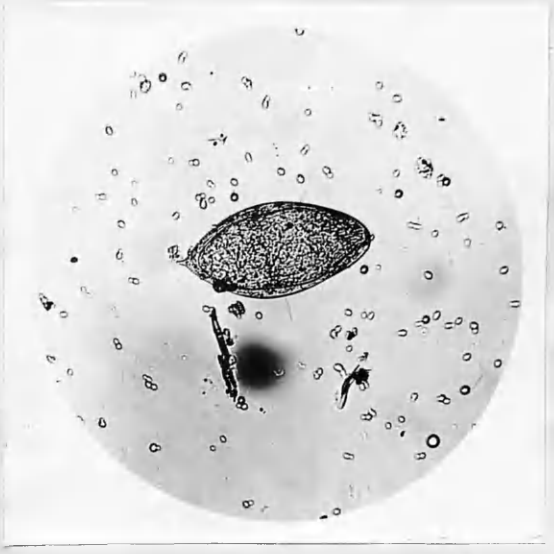

Fig.4. Microphotograph of ovum of S. HAEMATOBIUM in urinary deposit. x176

Fig.5. Microphotograph of ovum of S. MANSONI in faecal deposit. x115.

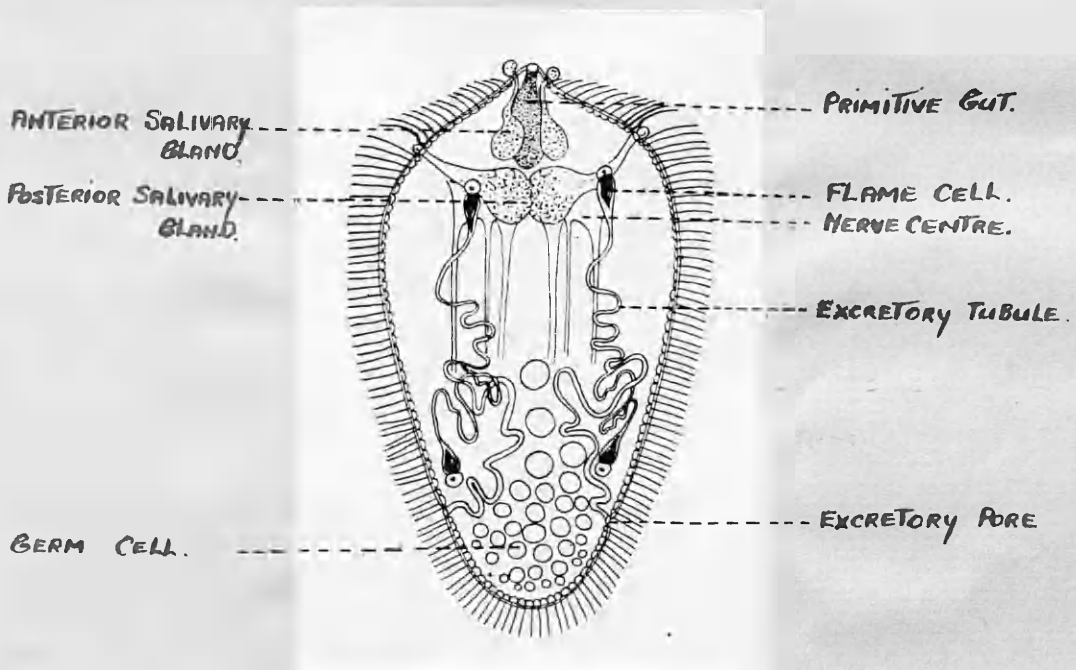

Fig.6. Miracidium of S. HAEMATOBIUM.

(After Faust)

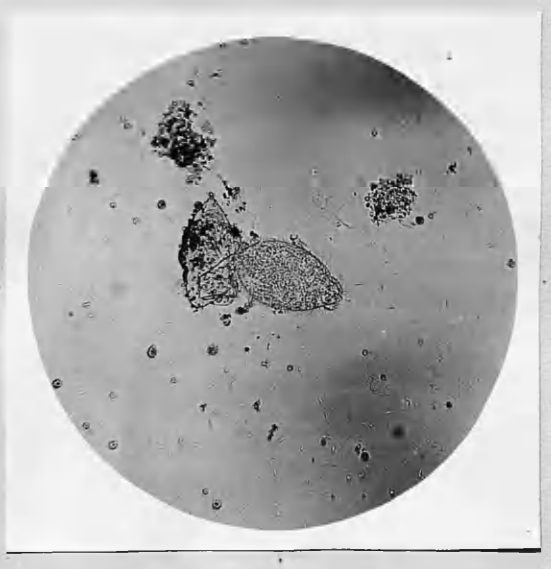

Fig.7. Microphotograph of MIRACIDIUM leaving ruptured shell. x115.

ILLUSTRATIONS.

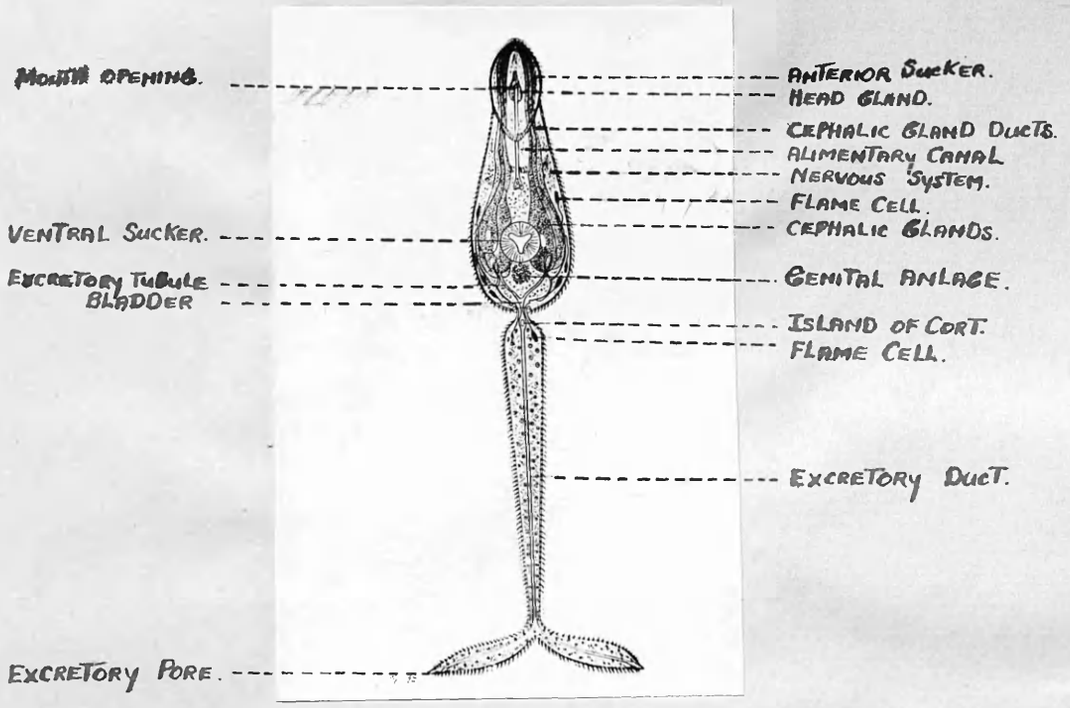

Fig.8. Fully developed CERCARIA of S. HAEMATOBIUM. (After Girges).

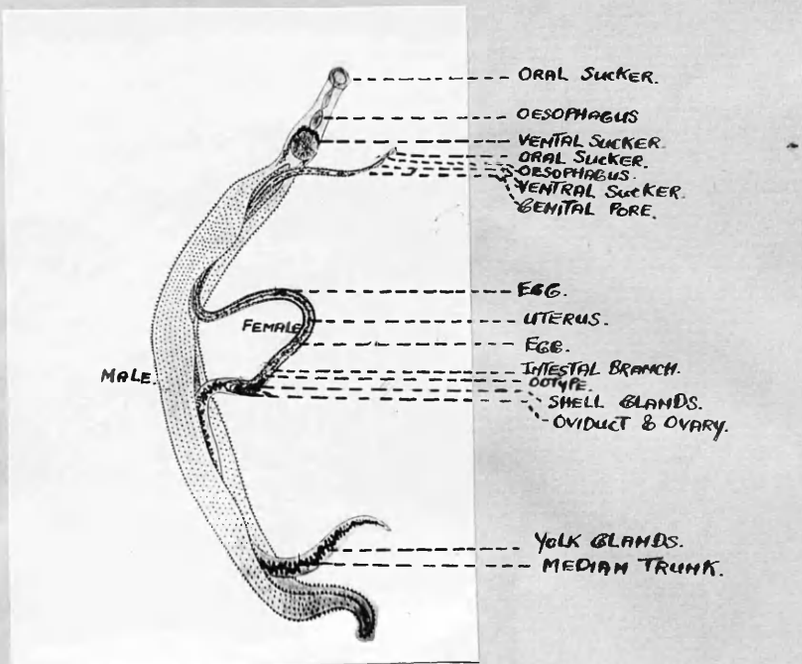

Fig.9. Male and female S. HAEMATOBIUM. (After Girges).

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digestive action they advance through the submucous and mucous layers until they eventually reach the lumen of the hollow organ (Bladder, Bowel, Ureter, Urethra, Seminal Vesicle, Vagina, etc.) where they are discharged from the body with the organ's contents, to begin again the life-cycle just described.

Severity of Infection: The severity of infection varies considerably, but in most cases it amounts to several hundred worms. In some cases, three hundred paired worms were found by Fergusson.

Duration of Infection: It has been estimated by various observers that the Schistosome worms can live in the veins of man from 15 to 25 years. A first attack confers a partial immunity in so far that as long as these worms are active within the body of their human host, no further infection takes place.

CHAPTER VI

PATHOGENESIS:

1. Stage of Invasion: When the cercariae pierce their way through the skin of man they provoke a reaction in the epidermis which appears in the form of small reddish-brown papules about 5m.m. in diameter and which are raised about 1 m.m. above the skin. If many cercariae attack the skin in one area, the resultant reaction may appear as a definite reddish-brown Rash, to which further reference will be made under Clinical Signs.

The skin reaction is partly the result of mechanical trauma and partly due to the action of the digestive enzymes secreted by the cercariae. Histologically this reaction is characterised by swelling, congestion, and by plasma-cell and eosinophil infiltration

Beyond the irritation caused by the Rash and in some instances the occurrence of some slight rise in temperature, the affected person at this stage rarely suffers any inconvenience.

2. Toxic Stage: In the next stage whilst the worms are growing in the veins of the liver, various toxaemic symptoms may appear and definite recurrent rises in temperature may accompany complaints of pain in the liver and spleen and in the upper part of the abdomen generally. Headache and vomiting are fairly frequent symptoms. The condition is often mistaken for a mild malarial attack, to which

it indeed bears a close resemblance. This reaction on the part of the host is undoubtedly due to the action of the various toxic products elaborated and excreted by the growing worms. This phase, however, soon passes and the host in most cases, appears to establish very rapidly a relative tolerance to the toxins produced.

3. Local Disease: In the next stage when the female worm is depositing her eggs in the smaller venules and during the period when the eggs are making their way to the lumen of the organ, very little systemic reaction is shown by the host. The local reaction produced by the cells in the vessel-walls and in the submucous and mucous layers of the organ has been already described. It is a purely local inflammatory reaction accompanied by a deposit of fibrin around the invading ovum. If the egg reaches the lumen of the organ and is discharged, little evidence of its passage remains, since the tissue reaction to the individual egg is very slight. But if masses of eggs are congregated together and are endeavouring to pass through the tissue at the same time, the patient may suffer local damage, and secondary infection by other organisms may occur. If every egg succeeded in reaching its objective, viz. the lumen of the hollow organ, very little permanent damage would be done to the host and the disease would not be of such serious import. Unfortunately great masses of eggs never reach the lumen of the organ but become lost in the surrounding tissues where their presence gives rise to serious consequences. The reaction of the tissues to these mass invaders is considered under the heading "Pathology".

CHAPTER VII

PATHOLOGY:

The essential pathological feature is the formation of a tubercle often referred to as a "pseudo-tubercle". It seems preferable to call this formation a tubercle since its structure is essentially a granuloma bearing some similarity to the granuloma characteristic of tuberculosis. The process starts around the ovum whether imbedded in the vessel-wall or free in the tissues. Presumably a toxin is elaborated by the miracidium inside the egg as the result of which an inflammatory reaction on the part of the tissues in the

ILLUSTRATIONS.

Fig.10. EARLY TYPE OF SCHISTOSOMIAL TUBERCLE.

Note, amorphous central core containing two ova. x160.

Fig.11. SCHISTOSOMIAL GIANT CELLS.

Tubercles containing ova are present, but note absence of ovum in tubercle on extreme right. x108.

Fig.12. PROLIFERATIVE TYPE OF SCHISTOSOMIAL TUBERCLE.

Note, granulation tissue arranged in concentric whorls around the remnants of ova. x130.

Fig.13. SCHISTOSOMIAL TUBERCLE, showing numerous giant-cells. Note, no ovum is present. x70.

ILLUSTRATIONS.

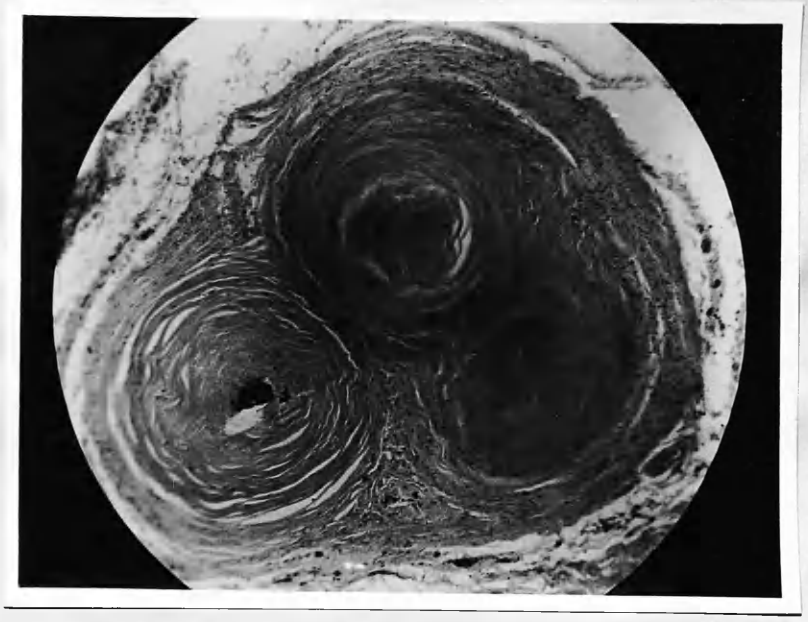

Fig.14. HEALED SCHISTOSOMIAL TUBERCLE. The microphotograph illustrates the end result of Schistosomial inflammation. Note, dense fibrous tissue formation which has occurred in the tubercle. x96.

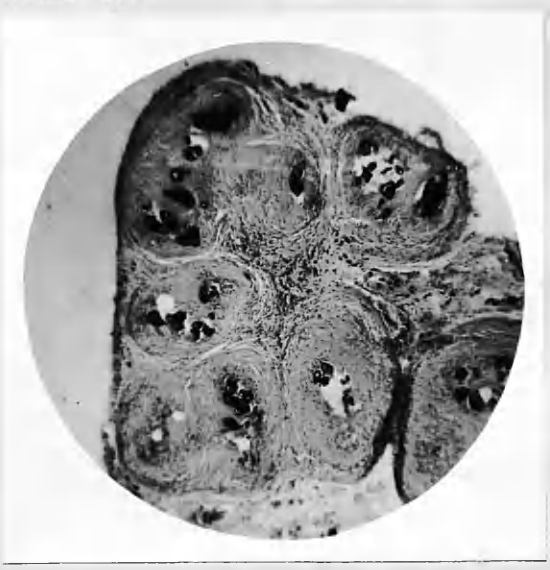

Fig.15. CONGLOMERATE TUBERCLE, showing microscopical features. x40.

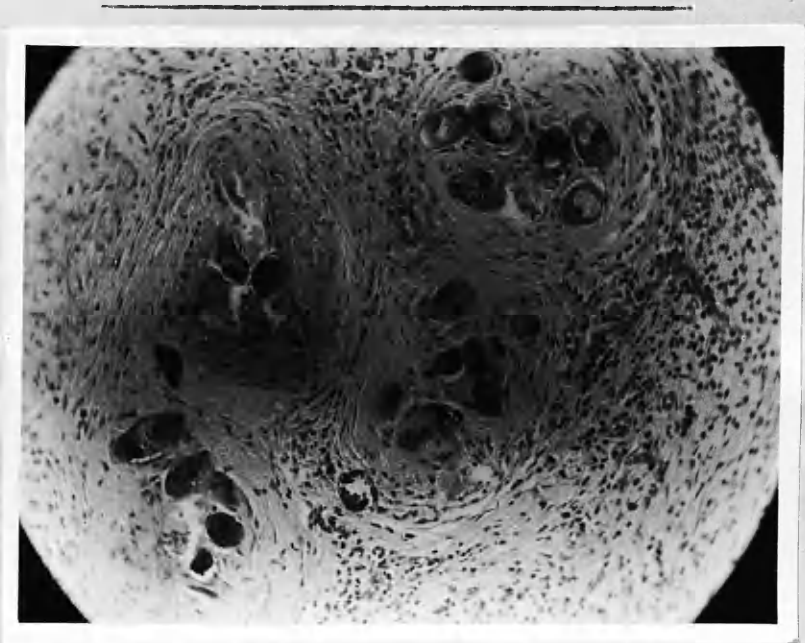

Fig.16. SCHISTOSOMIAL INFLAMMATION. Numerous ova are present together with fibrosis and chronic inflammatory cell infiltration of the intervening tissue. x150.

immediate neighbourhood of the ovum is produced. The first cell to appear in concentration around the ovum is the eosinophil; this appearance is characteristic and is not seen in any other granulomatous formation. Soon after their appearance, the eosinophils commence to disintegrate and to form an amorphous central core, whilst, simultaneously, wandering cells, mononuclear cells and young fibroblasts, begin to range themselves in a concentric fashion round
+Fig.10 this central degenerating mass+; multinucleated giant cells, indistinguishable from those found in tuberculous granulomata also make
+Fig.11 their appearance in this surrounding zone.+

Sooner or later, a wall or ring of fibrous tissue is laid down at the periphery of this tubercle, and this fibrous tissue formation ~~proceeds~~ extends from without inwards to replace the granulation
+Fig.12 tissue, which is the forerunner of fibrous tissue+. The ova are devitalised or killed and eventually become calcified. The Giant Cells obviously are scavenger cells and not infrequently are found to contain engulfed portions of the dead ova; this latter occurrence has been found frequently in tissues examined in the Public Health Laboratory, Salisbury. In addition, tubercles containing giant cells have been found in which no ova or remnants of ova are
+Fig.13 demonstrable+, whereas tubercles in the near neighbourhood of a more recent inflammatory process contain ova and remnants of ova. It seems reasonable to argue from this repeated observation that the giant cell has a marked phagocytic function and plays a large part in the digestion and ultimate disposal of ovular remnants. So far as I am aware, this observation has not hitherto been published by any previous writer. The end result is the eventual formation
+Fig.14 of a concentric fibrous scar.+

The tubercle is, therefore, essentially a chronic inflammatory process of a non-suppurative nature. Not infrequently these
+Fig.15 tubercles coalesce+, and this conglomerated mass is visible to the naked eye as the greyish nodules to be described later and which are characteristic of the macroscopic appearances of the disease. Between the individual tubercles themselves and between the masses of coalesced tubercles, fibrous granulation tissue is pres-
+Fig.16 ent, and eosinophil cells, plasma cells, and even ova infiltrate it+.

ILLUSTRATIONS.

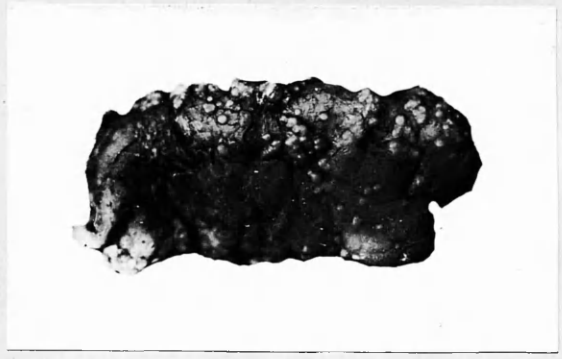

Fig.17. Appendix showing CONGLOMERATE TUBERCLES; sago-grain appearance. Nat. size.

A further feature is the presence in this granulation tissue of numerous capillary vessels. Thus the affected tissue of the part is gradually replaced first by granulation and subsequently by fibrous tissue formation.

It is not out of place here to compare the structure of the Schistosomal tubercle with that of the tubercle produced by Koch's Bacillus. They differ essentially in three important respects; first in the constancy of the eosinophil concentration in the Schistosomial Tubercle and the complete absence of this type of formation in the latter; and secondly, and above all, in the diagnostic presence of the characteristic Schistosomial ovum. Thirdly in tuberculosis, the tubercle tends to caseation, whilst in Schistosomiasis the tubercle is essentially hyperplastic with no tendency to softening.

"Morbid Anatomy". The affected part is generally thickened, hard and of a reddish hue, and contains abundant fibrous tissue. When the tissues are cut, a grating noise often accompanies the passage of the knife; this is due to the presence of calcified eggs. Usually, though not always, the presence of greyish-white nodules can be detected in the tissues by the naked eye; these are due to the presence of conglomerations of coalesced tubercles referred to above, and have a greyish lustre which makes them resemble "sago grains".+ In all organs this is the fundamental pathological tissue-change, except perhaps in the Spleen where it is difficult to find tubercle-formation and where the condition which presents itself is generally a purely cellular infiltration; nor can tubercles usually be demonstrated in the ureters, where, owing to the late stage at which inspection becomes possible, usually only the more advanced fibrous tissue formation stage is found.

+Fig.17

In addition, in the lungs, not infrequently, an endarteritis obliterans of the smaller and larger branches of the pulmonary vessels is present, with the production of emphysema or even occasionally of Ayerza's disease (recently described by Shaw and Ghareeb as occurring in Egypt - Vol.35 page 665 - T.B.+). In Southern Rhodesia, the morbid changes are usually those of fibrous tissue formation of the lung tissues, a pulmonary fibrosis, which has as its most frequent sequela the contraction of an acute

+Tropical Bulletin.

pneumonic infection from which death often results. In my view the high incidence of pneumonia amongst natives in Southern Rhodesia and its relatively higher death rate is due to the frequent occurrence of Schistosomial changes in the lung tissues.

Complications and Sequelae:

1. Fibrous-tissue formation, and contraction of scar-tissue with its well-known resultant complications and sequelae.

This is best seen in the hollow viscera, though it is also demonstrable in other organs:-

(a) in the Urinary Tract - Bladder, Ureters and Urethra. Here the contracting scar-tissue frequently gives rise to strictures which may result in hydronephrosis or, if pyogenic infection has been superimposed, to pyonephrosis and to uraemia. The ureters are often involved in the fibrous-tissue formation which takes place in the Bladder-wall; they are quite frequently involved independently of the Bladder.

(b) in the Bowel:- Where replacement of the mucous lining by fibrous tissue may lead to chronic catarrhal conditions and disturbances of function, and where the contracting scar-tissue may form strictures with resultant catarrhal and ulcerative processes.

(c) in the Appendix - Where as the result of fibrous-tissue formation and contraction, an acute inflammatory condition may be produced.

(d) in the Gall-Bladder - Where stricture may result and lead to a chronic inflammatory process and to the production of Gall-stones.

(e) in the Fallopian Tubes - Where fibrous-tissue formation and contraction may lead to the setting-up of a chronic inflammatory process or even to an acute Salpingitis indistinguishable from that due to the ordinary pyogenic organisms.

(f) in the Liver - Where severe impairment of liver function may result from the destruction of liver lobules by fibrous-tissue formation and which may result in ascites and complete liver failure.

(g) in the Lung - Where the destruction and replacement of lung-tissue by fibrous-tissue formation may lead to a condition of pulmonary fibrosis with its usual sequelae of Chronic Bronchitis, Emphysema and Pneumonia.
The effect of this pulmonary fibrosis in relation to Pulmonary Tuberculosis is discussed later.

One further observation which would seem to find its most appropriate place for record under this general heading is the occasional discovery of a patient who is suffering from Schistosomiasis and who is passing large quantities of sugar in the urine. These cases do not present the characteristic symptoms of Diabetes Mellitus nor is the condition progressive. Improvement rapidly follows the administration of antimony and the sugar disappears from the urine. During the past few years I have found a few cases of this type amongst native patients. (Erfan Vol.31 p.114)

2. Superimposed Secondary Infection.

This generally occurs behind a stricture or it may be consequent upon a schistosomial lesion where the tissue has been devitalised.

Examples of this are:-

(a) An acute Cystitis following upon Schistosomial lesions of the Bladder.

(b) Peri-Urethral Abcesses occurring behind urethral strictures.

(c) Pyelonephrosis arising from blocked ureters.

(d) Pneumonia superimposed upon a devitalised schistosomial lung.

A point in this connection not hitherto sufficiently appreciated is the tendency of the African Native, whose resistance has been lowered by the ravages of Schistosomiasis, to contract Tuberculosis, particularly of the pulmonary type, even on minimum exposures to infection. This statement is in direct contradiction of an observation recorded from Egypt some years ago where the writer stated that these two diseases were rarely found in the same patient. Our experience in Southern Rhodesia is so much the reverse that I hold that the presence of Schistosomiasis is often a contributory factor in the contraction of tuberculosis.

On the other hand, whilst Hookworm infestation is frequently associated with Schistosomial infection in the native of Southern Rhodesia - and this association has been remarked upon elsewhere, particularly in Egypt and Nyasaland - there is no reason to believe that the one predisposes to the other. The reason for the double infection would appear to be that the climatic and geographical features favouring the development of the one parasite are beneficial to the maintenance and spread of the other.

3. Calculus Formation is not infrequently found as the result of stricture formation in the Urogenital Tract and in the Biliary Tract.

4. Perhaps the most important complication of Schistosomial disease, next to those resulting from stricture formation, is the tendency to malignant disease in the affected organ. Despite many statements to the contrary, I am convinced that the high incidence of malignant disease in natives in Southern Rhodesia is due to Schistosomiasis.

The sites where this malignant condition is most usually found in order of frequency are:-

(1) The Bladder - transitional-celled carcinoma.
(2) The Liver - hepatoma superimposed upon a multilobular cirrhosis.
(3) The Rectum and Colon - adeno-carcinoma.
(4) The Testicle - seminoma.

Blood Picture Pathology:

4(a). Red Cells: The more or less continued loss of blood by the involvement of the urinary or intestinal tract often results in the

production of a hypochromic anaemia. This is generally mild, the typical count being between 3½ and 4 million, although severer forms where the count was as low as 2 millions have been encountered. Nevertheless in some cases where the loss of blood is slight, the red-cell count and haemoglobin estimation present no serious abnormality. The occurrence of other helminthic infestations such as hookworm may obscure the blood-picture and produce deficiencies not attributable to the Schistosomial infection. However, in Schistosomial Cirrhosis of the Liver there is practically always a hypochromic anaemia of moderate or even greater severity with a haemoglobin estimation of 40 to 60%. Although it is well known that in Cirrhosis of the Liver due to disturbance of the storage of the pernicious-anaemia preventing factor, a macrocytic anaemia may develop, I have not over a large series of cases been able to demonstrate an Addisonian type of anaemia in a Cirrhosis of the Liver due to Schistosomiasis.

4.(b) White Cells: The majority of patients show no increase in the leucocyte count. When an individual is infected, there is usually a marked alteration in certain elements of the leucocytes. This is best seen in the Eosinophil Count which may in the initial stages of the disease reach as high as 50%. As the disease progresses, however, the Eosinophil count drops considerably and not infrequently returns to normal. Over a series of 50 cases where Schistosomial infection was known to be present for many years, the following Eosinophil percentages were found:-

List of Blood Counts

Case No.	Eosinophiles	
1. (Native)	13%	(B.mansoni)
2. (Native)	13%	(B.haematobium)
3. (Native)	6%	(B.mansoni & B.haematobium)
4. (European)	10%	(B.mansoni)
5. (Native)	12%	(B.haematobium)
6. (Native)	19%	(B.haematobium)
7. (Native)	9%	(B.haematobium)
8. (Native)	5%	(B.mansoni & B.haematobium)
9. (Native)	15%	(B.haematobium)
10. (Native)	19%	(B.haematobium)
11. (Native)	7%	(S.mansoni)
12. (European)	7%	(B.mansoni)
13. (Native)	5%	(S.mansoni)
14. (Native)	5%	(B.haematobium)
15. (Native)	16%	(B.mansoni)
16. (European)	23%	(B.mansoni)
17. (Native)	14%	(B.mansoni)
18. (Native)	12%	(B.mansoni)
19. (European)	5%	(B.mansoni)
20. (European)	48%	(B.mansoni)

The remaining 30 cases showed no eosinophilia.

These cases were routine investigations and were late cases of the disease.

Roughly in 25% of cases, there may be a leucocytosis with a relative increase in the neutrophils. This leucocytosis is generally mild and in the neighbourhood of 12000 to 14000, but occasionally one finds it as high as 20000. In my opinion the leucocytosis is generally related to some superimposed infection.

In Schistosomial Cirrhosis of the Liver I have found, as other workers have found elsewhere, a leucopenia with a relative lymphocytosis.

CHAPTER VIII

CLINICAL MANIFESTATIONS:

It appears to me that as far as the clinician is concerned, too much stress has been laid upon dividing Schistosomial Disease into two types. If the Clinician knows the pathology of Schistosomiasis, it makes little difference to his treatment as to whether the infective organism is of the urinary or of the intestinal variety. A further reason for abolishing this division is that very many Natives suffer from both diseases 30% of those examined in the Salisbury Native Hospital during a six-monthly period showed both infections.

Fairley (Price's Textbook on Medicine) recognises three stages in the disease - (1) Invasive, (2) Toxic or Anaphylactoid, (3) Localised Disease, in which there is an Early and a Late Type. Whilst this division would fit in with the stages described in the Pathogenesis of the Disease, in my experience I have not found this of great clinical help, since the first two stages of the classification are very rarely observed as distinct separate phases, nor are they often complained of by the Patient. I would therefore prefer to classify the Disease into divisions which can be recognised clinically and which my own observations would appear to justify. The classification I have devised, therefore, is a division into 3 stages one a Primary or Invasion Stage, (2) Secondary or Stage of localised Disease, and (3) a Tertiary Stage of Complications.

1. The First Stage comprises the period when the cercariae enter the skin to the time when the adult females deposit their eggs in the venules of the venous plexuses. The complaints during this stage are general in character and mostly abdominal in type.

The patient may complain of Debility, Malaise, Abdominal Pain, Cough, Diarrhoea, Loss of Appetite. There may be some loss in weight. If the patient is examined, very few abnormal physical signs may be found to explain his condition, though the temperature may be raised, the spleen palpable, and in some more serious cases the typhoid state may be simulated. A marked Eosinophilia in the blood is generally present, and the presence of this altered blood picture in a patient resident in an affected area and known to have exposed himself, provided other causes of Eosinophilia are excluded, should lead the physician to suspect the possibility of Schistoso-

miasis infection. It is obvious that ova will not be present in the urine or stool, but the complement-fixation reaction, first described by Fairley, will often be found to be positive. There is only one distinctive symptom which may or may not be present, which can be regarded as at all diagnostic and that is the presence of Urticarial Rashes. These are, however, generally transient in character and unless one has the good fortune to find a patient exhibiting one or other of these rashes at the time of examination, there is little else in the general condition which would lead the physician to suspect Schistosomiasis.

This Primary Stage may last from several weeks up to a few months or even to one or two years. In my experience, however, it is seldom that we find the patient presenting himself for examinatia during this stage, and it is still more unusual to find the condition diagnosed in this stage. If the condition does bring the patient for examination, the diagnosis is generally that of malaria, since a great number of cases have malarial parasites in the blood; or perhaps one or other of the nematode worms infections, by which many of the Natives are affected, is held responsible for his symptoms.

2. <u>The next or Secondary Stage</u> should in most cases be easy of recognition and diagnosis, since the ova have now been deposited, in the case of Sch.haematobium in the Bladder, or in the case of Sch.mansoni in the bowel, and are already escaping through the lumina of these organs.

A. When the Bladder is involved the most important symptom complained of is the passage of blood in the urine; this haematuria is mostly a terminal one though not always; there may be frequency and pain which is generally felt at the end of micturition. The haematuria may persist for months or even years, and in the kraals of this Colony this haematuria is often regarded as normal micturition since a very large percentage of the Native population suffer from Schistosomial Disease. In the ordinary case, the haematuria progressively diminishes and tends to disappear, although the disease has not been cured. With reference to this symptom, one important feature must be stressed, namely that one frequently

encounters many people with definite Bladder involvement, but who have no vesical complaints and no visible blood present in the urine. The blood in these cases can only be found on microscopic examination and is then generally accompanied by ova. On examination of the patient no physical abnormalities may be detected beyond the characteristic cystoscopic appearances of the Bladder wall. In the early stages of Bladder involvement, one can frequently demonstrate the pale patches due to tubercles surrounded by a zone of congestion, or areas in which ulceration has already taken place. Later when fibrosis and calcification have occurred the "Sandy Grain" appearance is produced, viz. a dirty brown speckling generally best seen in the Fundus or Trigone region. An eosinophilia of mild or moderate degree is generally present in the blood, though not always. The complement-fixation may be found to be positive. Examination of the urine at this stage practically always shows the presence of albumen and blood provided ova are present in the urine. In a series of over 2000 urines tested, in no case were ova found without the accompanying presence of albumen and Red Blood Corpuscles. This statement is important and seems well supported by the large number of tests. In certain areas of Egypt the presence of albumen in the urine is regarded as a certain sign of Schistosomiasis and almost as long as twenty years ago, certain large Insurance Corporations in Durban refused to accept candidates for life insurance if, after treatment, Red Blood Corpuscles or Albumen were still present in the urine.

B. Should the disease be localized in the Bowel, and in this position it is almost always of Sch.mansoni origin, the history given by the patient will generally be one of diarrhoea accompanied by the passage of blood and mucus, occasionally sufficiently severe as to simulate a dysentery and often mis-diagnosed as Amoebiasis or Ulcerative Colitis. Tenesmus may be present but is generally of the mild type. As a rule there are periodic attacks of diarrhoea and the patient may have had the condition for many years before he presents himself for examination. The microscopic examination of the stool, especially if present of the muco-sanguineous discharge will reveal the presence of the ovum. It is essential that several specimens be examined over a

period of days in order to exclude the diagnosis of Schistosomiasis, and monthly or even three-monthly examinations should be performed if the symptoms remain and no ova are found. Should one fail to find the ova in the stool in a suspected case, a sigmoidoscopic examination should be proceeded with, and if small inflamed areas of mucosa are found, scrapings of the suspected sites with a curette should be obtained and examined for ova. An Eosinophilia may be present in the blood, and the complement-fixation test may be of assistance in establishing or confirming the diagnosis.

Whilst bowel symptoms may be present, it is important to realise that as in the case of the Bladder, there may be cases, which are not infrequent, where no bowel symptoms may be complained of. This has been frequently demonstrated in the Native Hospital where routine examination of the Stool and Urine frequently reveal the presence of the parasite in the excreta, when the patient may be suffering from some other ailment.

C. In the above paragraph, I have described the two common sites where the disease may localise itself, but there are, in addition, many other sites where the disease may be found and where its presence gives rise to greater disturbances and to which the complaints of the patient are referred. In such cases it is surprising to find how mild the Bladder or Bowel involvement may be.

Such a type of case is well illustrated in tumours of the Testicle due to Schistosomiasis. References in the literature to Schistosomial Testicular Tumours are extremely scanty. Perhaps the first mention was made by Gelfand & Davis in their paper entitled "Bilharzia Lesions of the Testicle in Southern Rhodesia", and published in the S.A. Medical Journal in 1939. In such cases the chief complaint is one of slowly progressive testicular enlargement; it is often painless, but the patient may complain of a heavy feeling in that region. There may be an accompanying hydrocele; the Wassermann Reaction is negative. Carcinoma of the Testicle is generally excluded by the long antecedent history. When the mass of growth is of a rapid nature, however, the differentation from Primary Carcinoma of the Testicle is practically impossible. A point which was not stressed by Gelfand & Davis in the differentation from Primary Carcinoma, but which I believe to be of value, is that in Schistoso-

ILLUSTRATIONS.

Fig.18. Case of SCHISTOSOMIAL ORCHITIS described in text. Note "golf-ball" appearance of testicle.

Fig.19. Another case of SCHISTOSOMIAL ORCHITIS in which the right testicle was mainly involved.

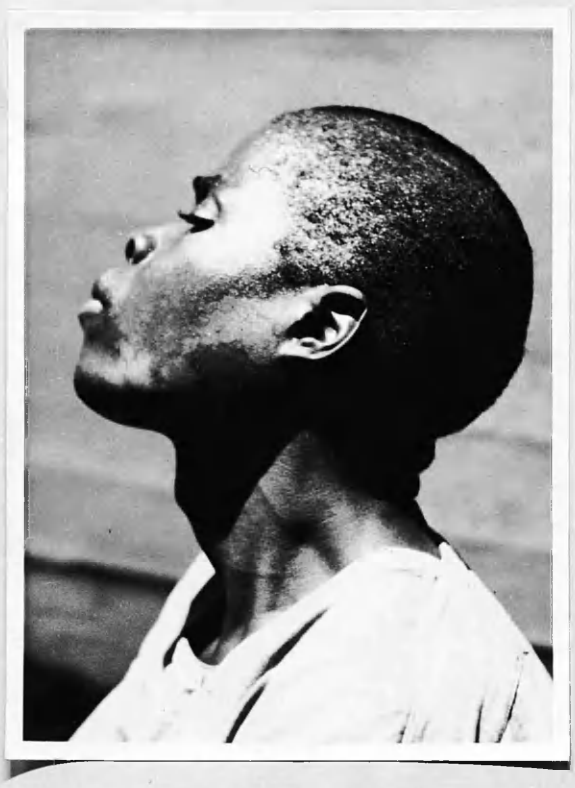

Fig.20. Photograph illustrating DEPIGMENTED SKIN ANOMALY.

Note distribution, merely in region of angle of jaw.

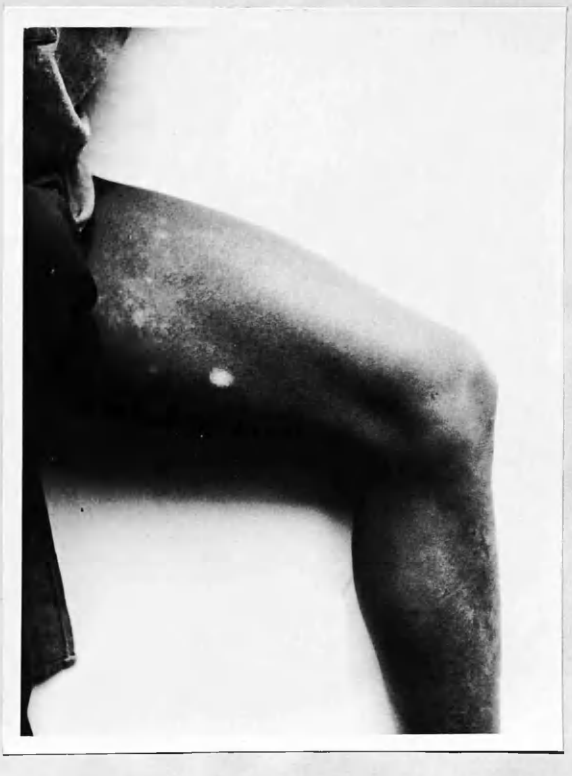

Fig.21. Photograph of DEPIGMENTED PATCH on inner side of thigh.

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mial Orchitis, the Epididymis and Testicle are both simultaneously involved in the process and cannot be distinguished, whereas in Primary Carcinoma of the Testicle the mass of growth can generally be felt distinct from and lying in front of the Testicle.

Case illustrating this point in the Differential Diagnosis under discussion:-

Adult Native Male aged 26 years. Admitted to Native Hospital complaining of swelling of left Testicle of "two weeks (?)" duration. Figs. 18 & 19. The left Testicle was the size of a golf-ball, rounded and smooth.+ Testicular sensation was absent. The Testis was indistinguishable from the Epididymis. The left cord was normal in thickness. There was no impulse on coughing. The right Testicle and its appendages were normal. Urine and Stool did not reveal the presence of ova, but on cystoscopic examination the typical "sandy grain" appearances were seen. The Wassermann Reaction was negative.

A course of antimony was administered but no appreciable improvement resulted. The infected testicle was removed surgically and the diagnosis of Schistosomial Orchitis was confirmed histologically. There was no evidence of malignancy.

D. In other cases the infection may localise itself in the Seminal Vesicles or Prostate. The main complaint here will be one of pain in the perineal region accompanied by attacks of Spermatorrhea or Haemospermia. In Southern Rhodesia the presence of Haemospermia in any patient must lead the physician to consider seriously the diagnosis of Schistosomiasis. Examination of the urine for ova and cystoscopic examination of the Bladder, or urethroscopic examination will clear up the diagnosis. In passing it may be mentioned here that sterility in the male as a result of these lesions is perhaps a not infrequent occurrence.

E. Schistosomial Urethritis may, and frequently does produce a urethral discharge, of a milky white appearance, and is often mistaken for gonorrhea. In my experience it is well in any Native case where there is a complaint of urethral discharge not only to stain a slide and examine for gonococci, but also to examine both the slide and the urine for ova.

F. Localized Disease of the Female Genital Tract is also not uncommon and is presenting a problem of growing importance in this

Colony. As a rule the uterus, cervix, fallopian tubes and ovaries are matted together and are involved simultaneously in the process. If the uterus is involved together with the cervix there will be menorrhagia and leucorrhea, and schistosome ova, generally Sch. haematobium, may be found in the curettings.

When, however, the Tubes alone are affected and the Cervix and Uterus escape, the diagnosis of Schistosomial Salpingitis is extremely difficult. One or both tubes may be involved. The symptoms complained of are pains in the back especially after exertion, and dysmenorrhea. If both tubes are involved, sterility will result. The pains may be acute and sudden, and simulate an acute abdominal emergency. In contrast to the usual case of Salpingitis, namely, that following Gonorrhea, or subsequent to tearing of the Cervix during Labour, there is no leucorrhea or menstrual disturbance since in these Schistosomial cases the cervix and the endometrium escape. On physical examination there will be tenderness in one or other iliac fossa and on vaginal examination the tender swollen appendage can be felt in the affected fornix. Ova may be found in the excreta or there may be an eosinophilia in the blood and the complement-fixation test will often be found to be positive. If ova are not found in the excreta, cystoscopic or sigmoidoscopic examination may give characteristic evidence of Schistosomiasis. The condition should therefore be suspected in any female patient complaining of persistent lower abdominal pain and exhibiting the presence of a tender mass in the Fornix. These cases generally pass into the hands of the surgeon for operation, but it is the pathologist who usually determines the actual causal agent of the condition.

Case: The case was that of a woman aged 30 years, married. The chief complaints were those of sterility, lower abdominal pain and dysmenorrhea. There was no history of leucorrhea or any serious menstrual disturbance. Just before admission to hospital, she developed very acute lower abdominal pain, accompanied by high temperature 103°F and vomiting. The surgeon, on examination, diagnosed a Tubal Abortion since there was a slight loss of blood per vaginam. At operation both tubes were grossly thickened and

fibrotic; there was no evidence of a tubal abortion. The tubes were removed and histological examination revealed a Salpingitis due to Sch.haematobium.

G. Rash: 1. Whether Schistosomiasis results in a definite disorder of the skin beyond that of the rash mentioned in connection with the invasion stage, is difficult of proof. Certainly, definite disorders of the skin do occur in patients suffering from this disease, and the occurrence of these rashes should lead the physician to suspect its presence. As far as I have been able to discover the first reference to such a skin condition was made in 1930 by Professor Sinderson of Baghdad who claimed that 20% of Schistosomiasis cases admitted to the Royal Hospital showed a butterfly pigmentation on the face; that is, a darkish-brown pigmentation on both cheeks and along the bridge and lateral aspects of the nose. He noticed that the butterfly sign was much rarer in the female than in the male. On studying the literature, I have been unable to trace any further reference to this condition. In Southern Rhodesia the presence of this anomaly of pigmentation has been noticed in probably 2% of the cases. The pigmentation may have the Butterfly distribution but in my experience it may be situated mainly on the forehead or on the upper part of the sides of the neck and on the exposed parts of the hands, arms and legs. The parts affected are generally a few small circumscribed areas. Again, the Butterfly Rash may be only partially developed, the rash appearing only on one side of the cheek and on the bridge of the nose. I have seen this pigmentation both in the male and female patient, but more commonly in the former and this may be due to the fact that the male is more exposed to the sun. The pigmentation does not disappear with treatment and seems to be a permanent disfigurement. It occurs in both the urinary and intestinal forms of the disease.

2. A second type of skin disorder is occasionally found in patients suffering from Schistosomiasis. In contrast to the first condition it is a depigmented anomaly of the skin and may aptly be described as leucodermia.[+] It may be confused with Leprosy, but is distinguished from this condition by the fact that no M.leprae can be found microscopically in scrapings, nor can any sensory

[+] Figs. 20 & 21

disturbance in the depigmented patch or in any other part of the body be demonstrated. On the other hand, in cases due to Schistosomiasis, ova will practically always be found in the stool or urine. It is slightly more frequent than the former skin anomaly and seems to be present in about 4% of cases. It may have a Butterfly distribution but is frequently met with in the upper region of the front of the neck, and may extend backwards over the occipital region. The affected skin is white, its margin slightly depressed below the level of the normal skin; it does not itch; it is resistant to treatment with antimony; and is apparently a permanent disfigurement.

The explanation of the two skin anomalies is difficult. Sinderson suggests that the pigmentation he found was probably due to the effects of solar light on a skin which had already been affected by Schistosomial toxins, and in the absence of any other feasible explanation, one must regard this as at all events the only explanation which is in harmony with the known facts.

H. Mental and Neurological Disorders due to Schistosomiasis have been referred to previously in the literature.

1. F.G. Cawston of Durban drew attention to the backwardness of pupils in South Africa who were suffering from Urinary Schistosomiasis. Our experience in Southern Rhodesia corroborates this observation though it should be remembered that the majority of these cases come from homes where are operative the same social factors of poverty, malnutrition, and disease which in other countries, where Schistosomiasis can be excluded, give rise to similar conditions of mental retardation. Nevertheless, it is quite certain that when all due allowance has been made for these factors there still remains a certain amount of mental backwardness which is beyond that accounted for by such social conditions.

2. Epileptiform convulsions due to Schistosomiasis have been described. For instance Nieva of Manila, in 1935, describes a case suffering from Urinary Schistosomiasis who for a year had convulsive attacks about twice a week. Following the administration of tartar emetic intravenously these attacks apparently ceased. This case was not proved at post mortem to have been due to Schistosomiasis although clinically the apparent cure following treatment was suggestive. It is admitted

that syphilis, ascariasis, and taeniasis probably account for a large number of these convulsive cases, but again there is a definite residue in which the only infective organism present is that of Schistosomiasis. It is reasonable therefore to assume that Schistosomiasis also is one of the conditions capable of causing these epileptiform seizures.

3. Involvement of the spinal cord with the production of myelitis has been seen by me on several occasions. It is most commonly mistaken for tuberculous disease of the spine. The lesions are generally found in the dorsal or upper lumbar segments of the cord with a resultant paraplegia producing both motor weakness and sensory loss. Unfortunately every case within my knowledge was diagnosed only at operation. In other countries Schistosome ova have been found in the brain at post mortem, but in Southern Rhodesia this finding has not yet been demonstrated. However, it is only within the last eighteen months that recognition has been given to the fact that Schistosomiasis may be a factor in the production of cerebral and spinal lesions, and though we have carefully sought for evidence at post-mortem, no case has yet occurred in which this factor has been demonstrable.

Case:- Miss. M: an unmarried European female, aged 43 years was admitted to Hospital on 6th December 1935. Her history was that of a slowly progressive Paresis involving the arms, legs, and sphincters. Her speech was stated to be slow, difficult, and inclined towards slurring. The patient was a senior matron in the employment of the Government Nursing Service. The symptoms described above had been noted by the Surgeon-in-charge of the Hospital where the patient was employed, and were described in a letter which she brought with her on admission to the Central General Hospital. Unfortunately at the hour of her arrival at Hospital the senior sister of the ward was temporarily absent and the patient was admitted by a sister much junior in rank to herself. The patient, contrary to Hospital Regulations insisted on proceeding to the bathroom to bath herself and refused to allow anyone to accompany her. The junior sister though protesting, allowed herself to be overruled on the point. As was to be expected, the patient fell on

trying to get out of the bath, and crashing heavily on the edge of the bath with the point of her chin, rendered herself unconscious. This unfortunate mishap complicated the whole issue and for some weeks it was impossible to tell how much of her cerebral symptoms were due to this known severe accident and how much to the vague undefined condition which had brought her to Hospital.

As the days went past and the effects of her accident wore off, it was possible to see that whatever the cause of the condition, there was definite cerebral and spinal involvement. The speech was slow, difficult, and inclined towards slurring; cerebration was sluggish and there was evidence of mental confusion and irritability. Restlessness and insomnia were marked. There was paresis of both arms and legs; the patient was incontinent, and complained of pains in the abdomen. The Wassermann Reaction was negative. The cerebro-spinal fluid showed a leucocyte count of 4 cells per cu.mm. The Nonne-Apelt Globulin test was negative.

The temperature was intermittent, going up at night to 99°- 100°F. and coming down to 97°F in the morning. Urine showed a very faint trace of Albumen, a few Red Blood Corpuscles, but no other abnormalities. Stool examination showed a few pus cells, Red Blood Corpuscles, and Charcot-Leyden crystals. No ova were found either in stool or urine. The blood count showed a leucocytosis of 14000 of which 56% were neutrophils and 25% were Eosinophils. In the absence of any schistosomial ova in stools or urine, this eosinophilia was believed to be due to Ascaris, though there was no other evidence to justify this view. The patient's condition continued to get worse and towards the latter part of January, the vague abdominal pains became a noticeable feature, and were considered to be due to Gall Stones. An X-Ray picture of the Gall Bladder taken at this time revealed no abnormalities and the Radiologist reported that the Gall Bladder was normal. On the 3rd and 4th February the abdominal pains became acute, and the abdominal wall was rigid over the Gall Bladder, and great tenderness was present. Operation was decided upon and the abdomen opened on 5th February. Although the case was not mine I was present at the operation and saw a Liver studded over with small round biscuit-coloured umbilicated nodules; which varied in size from mere dots to half an inch in diameter. The peritoneal lining of the bowel was also studded with these nodules, though here

they were of smaller size; the mesentery in addition showed a similar condition. The Gall Bladder was normal, and no stones were present. One of the nodules was excised and sent for histological examination. The Laboratory reported that "the material submitted consists of hepatic tissue enclosing a small nodule. This nodule presents the histological characters of a tuberculoma". On this report it was considered that the case was one of miliary tuberculosis.

The patient did not improve although the abdominal pains became milder in character and less frequent in attack. Eventually it was decided to ask the Laboratory for a complete sectioning of the nodule. This was done and the Laboratory reported the presence of a calcified Schistosomial adult worm in the centre of the nodule. The patient was at once put on a full course of antimony and general improvement was immediately evident. She was discharged from Hospital on 14th June 1936, six months after admission. Progress was in some respects slow, and 18 months subsequent to dishharge the patient was still walking with the aid of crutches and with great difficulty. The mental and cerebral symptoms cleared up fairly rapidly, and at the time of discharge, the patient was speaking normally and was mentally clear. Sphincter control also returned before the patient left Hospital.

Three years later the patient was walking without crutches and with no apparent difficulty though easily fatigued.

The case was one of Generalized Schistosomiasis involving both the viscera, the cerebrum and the spinal cord.

I. <u>Continuous Pyrexia</u>:-

A condition undoubtedly due to Schistosomiasis but not sufficient -ly recognised by the medical profession is that of continuous pyrexia. A patient is admitted to Hospital with a long previous history of pyrexia but with no other definite causal factor save the presence of Schistosomiasis. It is a remarkable fact that the temperature drops very soon after a few injections of antimony. The following case is very typical of many:-

Patient, a Native male, 18 years old. Admitted to Salisbury Native Hospital complaining of weakness and cough of several weeks duration On examination he was found to be wasted in appearance with pale conjunctivae, and with a temperature of 102°F, which remained in this vicinity continuously for a period of 3 weeks after his admission. The heart was normal in size and position, but a blowing systolic murmur was detectable at apex and base. The Wassermann Reaction was negative as was also the Widal Reaction. Blood culture was sterile; no malarial parasites were found in repeated smears and the agglutination tests for undulant fever due either to the bacillus melitensis or to abortus bacillus were negative. The spleen was very much enlarged and the lower edge was palpable two fingers breadth beneath the umbilicus. No abnormalities were found in the urine but Sch.haematobium was found in the stools. There were no other abnormalities in the stools. X-Ray of the chest gave negative results. Quinine was first administered for a period of one week, but without any beneficial effect on the temperature or on the general condition of the patient. It was after the third week that Antimony was given. The improvement was dramatic. The temperature dropped to normal after the third injection, the appetite began to improve, the weight increased, and the blood count which on admission showed 3½ millions Red Blood Corpuscles returned to a normal level shortly before the end o of the course of antimony treatment. The patient remained in hospital for a month after the last injection and during that time there was no recurrence of temperature. The patient felt so fit that he demanded to be discharged, and since that date he has remained completely well.

III. Tertiary Stage or the Stage of Complications:-

This stage generally sets in a few years or even as many as twenty years after the first evidences of infection. Although the lesions are present in the affected region and are slowly progressive, yet it is not generally until definite signs and symptoms have developed that the patient presents himself for treatment. In native cases, very often the patient is first seen in an almost terminal condition. Many of the more serious

ILLUSTRATIONS.

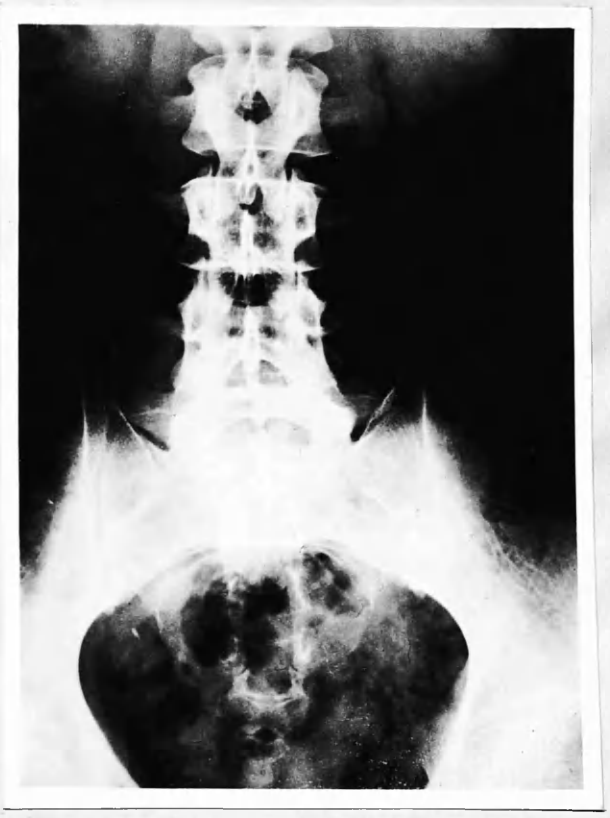

Fig.22. A straight radiogram revealing no abnormality in a patient known to have Urinary Schistosomiasis.

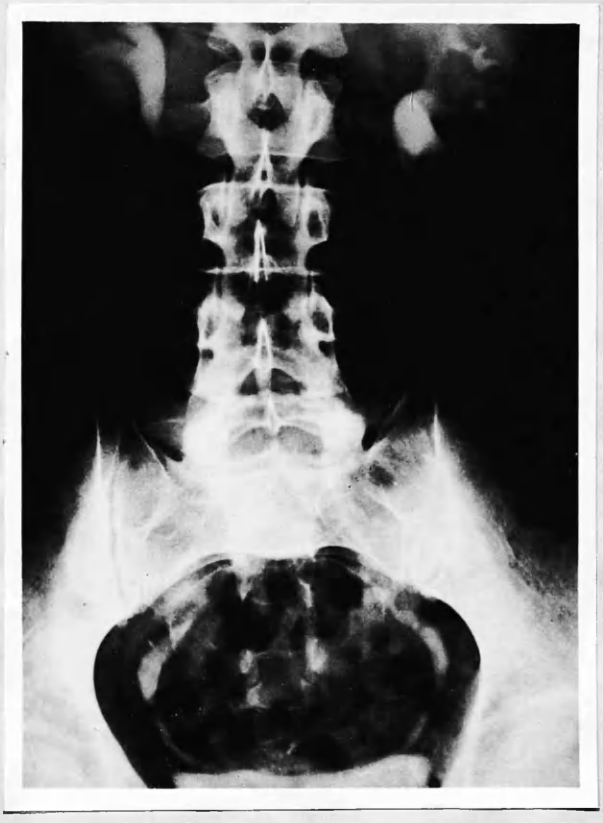

Fig.23. Same case after uroselectan showing strictures in lower third of ureters. This was proved by retrograde pyelography.

ravages of Schistosomiasis could be avoided if, when the disease was recognised in the Secondary or Stage of Localised Disease, the attendant physician were to warn the patient of the persistent nature of the condition, and, after a full course of antimony had been administered, to insist upon the patient's return for further periodic examinations. In my view it is indefensible for a medical man to send a schistosomial patient away after a single full course of antimony with the happy assurance that he is now cured. No physician should undertake the responsibility of pronouncing a cure until he has satisfied himself by the evidence of repeated examinations extending over a period of months that the condition is indeed no longer active.

The most important disorders in this Stage are:-

1) Lesions of the Urinary Tract.
2) Lesions of the Bowel including Appendicitis.
3) Cirrhosis of the Liver and Splenomegaly (Egyptian).
4) The liability of Schistosomatics to Tuberculosis and Pneumonia.

I. Lesions of the Urinary Tract:-

The most important complications here are those due to fibrosis with resultant stricture and secondary infection, and the liability to neoplastic formation. Stricture in one or other ureter is very frequent. It may occur along any part of the length of the ureter but is more frequently found in its lower third. The ureter above the stricture and the pelvis of the kidney begin to dilate, and sooner or later interfere markedly with the function of that Kidney. The patient not infrequently complains of colic on the affected side, frequency of micturition, and the hydronephrotic kidney can be frequently palpated. Infection of the hydronephrosis is liable to occur and calculi are not uncommon. If there is bilateral hydro- or pyonephrosis, death from uraemia probably ensues.

Figs. 22 & 23. The diagnosis of a stricture of the ureter is easily established by pyelography+ especially of the intravenous method. Any patient known to have suffered from vesical Schistosomiasis who develops symptoms of colic should be investigated from this point of view. It is insufficient merely to take straight radiographs of the abdomen and to look for renal calculi or evidence of enlargement of

the Kidney. Intravenous pyelography is the method of choice. It is simple and safe and the dilated and tortuous portion of the ureter above the stricture is always clearly outlined.

Stricture of the urethra is occasionally seen and this complication generally results in the retention of urine or the formation of periurethral abscesses.

Transitional Cell Carcinoma of Bladder

Tumours of the Bladder, due to Schistosomiasis, are commonly seen. Their progress and development may be exceedingly slow. At first the tumour assumes the structure of a simple villous or sessile papilloma, but its appearance is deceptive for it is very liable to become malignant at any time. When malignancy occurs the spread into the Bladder wall, into the Rectum and neighbouring lymph glands is generally very rapid. The diagnosis again is easily established since a patient who gives a history of long standing vesical Schistosomiasis, and who now comes complaining of haematuria or pain in the hypogastrium must be cystoscoped. The growth on the bladder wall will be readily recognised though in most instances only the pathologist can determine with certainty its innocent or malignant character.

2. Lesions of Bowel;

The most important complication here is that of neoplastic formation. The patient may present himself with a papilloma or a carcinoma in the rectum or colon.

The history will be that of the passage of blood and mucus in the stools. If the growth is annular and constricting there will be colic and alternating attacks of constipation and diarrhoea. Loss of weight is frequent. The growth is easy of recognition by the sigmoidoscope if it be in the rectum or sigmoid colon, but if above the level of these regions, a Barium Enema or a Barium Meal will usually demonstrate its presence. The attempt to demonstrate Colonic Papillomata by a simple Barium Enema is not of much value because of the/small frequent size of the growths; for this purpose a modification in the technique is advisable. The colon should first be filled with Barium, then evacuated, and thereupon insufflated with Air. If a radiogram is now taken, the papillomata will be demonstrable in relief.

ILLUSTRATIONS.

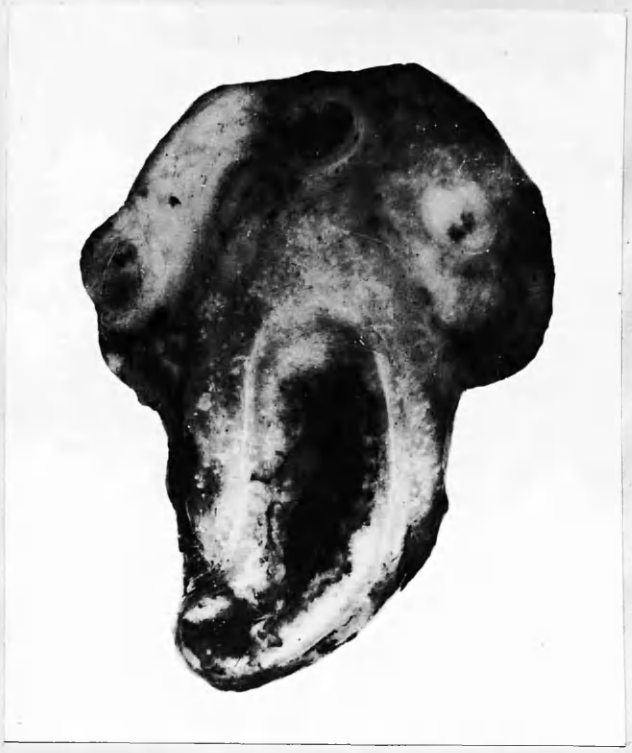

Fig.24. ACUTE SUPPURATIVE EXACERBATION IN CHRONIC SCHISTOSOMIAL APPENDIX.

Conglomerate tubercles are present in the musculature, in the mucous and submucous layers and in the greatly thickened subserous coat. Note distortion of lumen due to fibrous contration. Slightly enlarged.

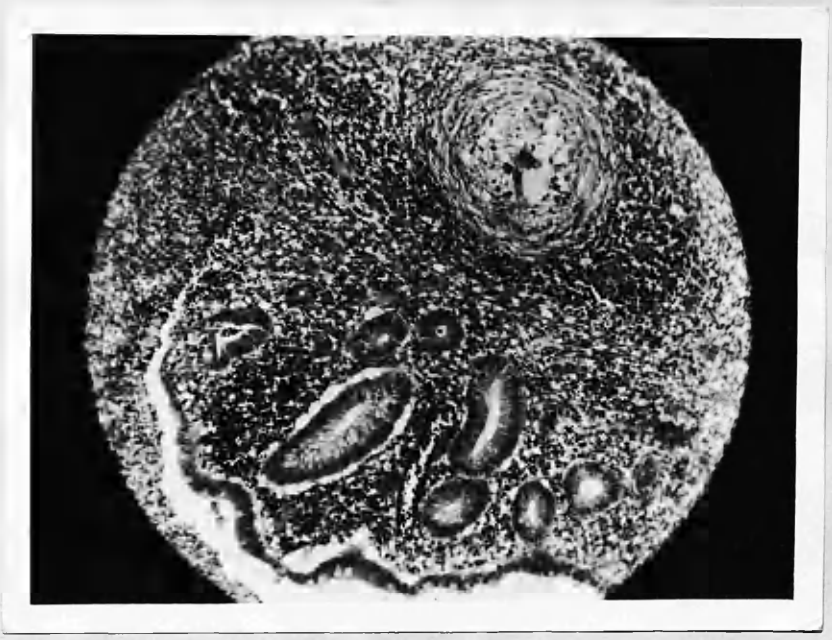

Fig.25. SCHISTOSOMIAL TUBERCLE behind an intact portion of mucosa.

Note exudate on left. x110.

ILLUSTRATIONS.

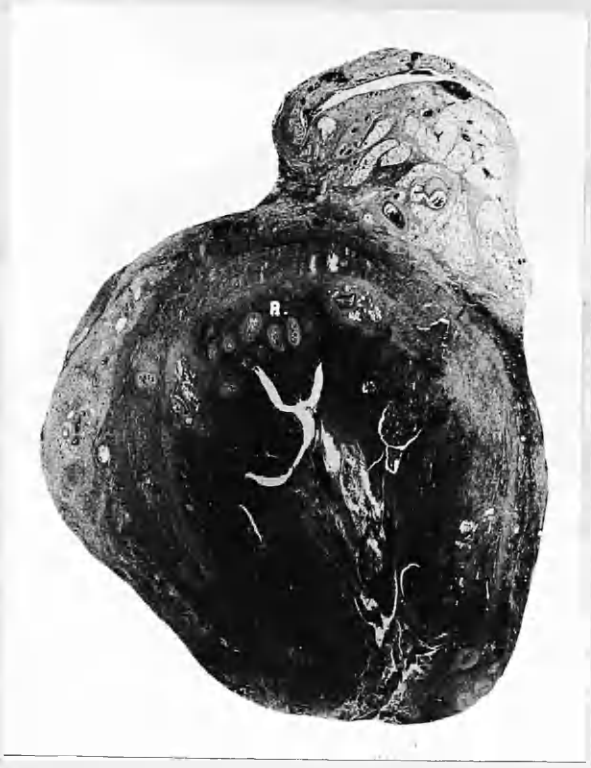

Fig.26. ACUTE GANGRENOUS APPENDICITIS WITH PERFORATION IN CHRONIC SCHISTOSOMIAL APPENDIX. Note presence of tubercles especially at point marked A. x7.

Fig.27. OBLITERATIVE APPENDICITIS DUE TO SCHISTOMIASIS. Note gross fibrosis with conglomerate tubercles. x13.

Appendicitis:

Schistosomial involvement of the appendix is not uncommonly seen in Southern Rhodesia and this would appear to be in keeping with the findings in other territories where the disease is prevalent. Barsoum in an article in the Journal of Tropical Medicine & Hygiene entitled "The Bilharzial Appendix" in 1934, stated that "Bilharzia does not cause or predispose to Appendicitis". This observation is certainly at variance with the experiences of other workers. As long ago as 1912 Temple Mursell recorded a case of perforated appendicitis in an appendix which was riddled with numerous Schistosomial eggs. Since then many references in the literature have appeared on the subject, in which the occurrence of an acute superimposed appendicitis has been described. Loret-Campbell and Rose working in West Africa published a report in 1936, in which they stated that acute symptoms clinically indistinguishable from pyogenic appendicitis are occasionally met with. 20% in a series of 35 individuals had gross macroscopic lesions which were held accountable for the severe appendicular symptoms described.

I have been able to collect a series of 50 cases of Schistosomial appendicitis and in 12% of these there was an acute suppurative process in addition to the helminthic infection.+ Two of the cases had gangrenous appendicitis.+ The lumen in a Schistosomial appendicitis not infrequently becomes stenosed and this stenosis predisposes to secondary infection and stercolith formation.+ It is therefore easy to appreciate the risks that may develop in such an appendix. The symptoms of an acutely inflamed appendix superimposed on the Schistosomial lesion are clinically indistinguishable from an ordinary pyogenic appendicitis. There is no definite means whereby the diagnosis of Schistosomial appendicitis in such cases can be established with certainty, but the knowledge that a patient is suffering from Schistosomiasis should lead one to suspect this possibility. A fact worth mentioning here is that in the 50 appendices examined 95% were due to Sch.haematobium; an interesting observation since it is well known that Sch.mansoni involves the intestines whereas Sch.haematobium generally involves the urinary tract. The explanation of this phenomenon is, as far as I know, unknown. It is frequently stated that acute appendicitis is extremely rare in the African

Figs. 24 & 25

Fig. 26

Fig. 27

Native. In Southern Rhodesia, however, it is not infrequent as a result of Schistosomiasis.

Case:- The patient was an adult Native male about 35 years of age. He came to the Out Patients' Department complaining of constipation and abdominal pain of five days duration. Unfortunately in the temporary absence of the medical officer he was not examined but was advised by the nurse in charge to take Castor Oil. Forty-eight hours later he was admitted to Hospital in a very serious condition. The temperature was moderately raised 102°- 103°F; the pulse was very rapid and of poor quality. The whole abdomen was rigid and tenderness was most marked in the Right Iliac Fossa. He died very soon after admission. At post-mortem, the terminal end of the Appendix had perforated and the appendix itself was markedly inflamed, swollen, and adherent beneath the caecum. There was an extensive and diffuse peritonitis. The appendix on histological examination presented the typical appearances of an acute suppurative process superimposed on an organ already grossly diseased by Schistosomiasis of the Urinary type.

III. Hepatic Disorders:-

The hepato-splenic syndrome due to Schistosomiasis has not previously been described in Southern Rhodesia. Its prevalence in Egypt, Japan, Northern Nyasaland and the Belgian Congo has been made the subject of many articles in the literature. In other words wherever Sch.japonicum or Sch.mansoni is found it is not unreasonable to expect to find this syndrome. In Japan it is known as Katayama Disease, and in Egypt and elsewhere, as Egyptian Splenomegaly. It would perhaps be far better to refer to it as Schistosomial Cirrhosis or as Schistosomial Hepato-lienal fibrosis.

The lesions are first set up in the liver by the ova; tubercle formation occurs and eventually fibrosis is induced. With a few eggs only, the damage is balanced by repair and thus function is not impaired. With more eggs, however, a multilobular cirrhosis ensues and in most cases so closely resembles Laennec's Cirrhosis or Gin-drinkers Liver as to be indistinguishable from the latter.

The splenomegaly is probably dependant upon venous stagnation. Some have explained this by the occurrence of a hyperplasia of the

splenic tissue, probably the result of the deposition of ova in the spleen. But whilst, in these cases I have not as yet failed to find Sch.mansoni in the liver, I have never recovered ova from the spleen.

The liver is as a rule palpable in most cases, the edge is sharp and hard, and generally the irregularity due to the hyperplastic nodules on the liver surface can be made out. Sometimes, however, the liver is shrunken (atrophic cirrhosis) and cannot be felt.

The spleen, however, is in the majority of cases markedly enlarged and the sharp notched anterior border not infrequently extends to below the umbilicus. The abdomen is distended by the enlargement of the spleen and liver or by the development of an ascites from which about 50% of these subjects suffer.

The Clinical Picture:-

This may be sub-divided into three classes.

1. Latent Cirrhosis of the Liver: Here the patient's condition is unsuspected during life and may only be found at post-mortem although the person may have died from a totally different disease. This accounts for 25% of the total cases.

2. Schistosomial Cirrhosis with Predominant Portal Obstruction:

The symptoms here develop insidiously and three stages may be recognised.

(a) The Pre-Ascitic Stage: The appetite is poor, and not infrequently there is discomfort and flatulence after food, and occasionally vomiting. The patient has periodic attacks of fever, may be short of breath, and complain of general weakness. Occasionally a severe Haematemesis due to rupture of oesophageal varices may set in and prove intractable and the patient dies. On examination the patient appears somewhat wasted; anaemia usually of the hypochromic type is present, and there is a leucopenia with a relative lymphocytosis. The liver is often, but not always, enlarged and palpable and the spleen generally easily palpated. There may be transient attacks of jaundice.

Figs.28 and 29

(b) The Ascitic Stage:+ The pre-ascitic stage may last many years, but once the ascitic stage develops the life of the individual may be counted in months, though some may survive for 2 or 3 years. The weakness and debility increase. He may lose more weight or he may now wish to be admitted to hospital because of the discomfort or disfigurement due to the distended abdomen. In many cases however, it is surprising, particularly in native cases, to find how few complaints are made despite this gross disturbance in the liver. Dilated veins due to collateral circulation may be seen in some cases on the abdomen, generally coursing upwards from the umbilicus and across the chest. If there is only a moderate degree of Ascites, the spleen and liver will be palpable, but often the enormous distension of the abdomen due to the fluid renders

this impossible. After tapping the abdomen, the spleen and liver can then easily be felt. The fluid recovered on tapping is a serous effusion, straw-coloured and of a specific gravity in the neighbourhood of 1020. There may be oedema of the ankles and legs due to pressure by the abdominal fluid on the inferior vena cava.

(c) The Terminal Stage: Death rapidly ensues either from Cholaemia in which case jaundice is marked or from pneumonia or peritonitis. Tuberculosis generally of the miliary type may set in and the patient's end thus hastened.

3. Schistosomial Cirrhosis with Predominant Jaundice: This accounts for about 25% of cases. The patient, as a rule, is admitted to Hospital complaining of jaundice of several weeks duration. He may give a history of recurrent attacks of icterus. The jaundice is generally well marked in the conjunctivae and produces a peculiar yellowish discolouration of the skin. The spleen is grossly enlarged and the liver is as a rule palpable. In this group of cases, Ascites is often absent. The Icteric Index is high, the van den Berg Reaction yields a direct positive or bi-phasic reaction. Gastro-intestinal disturbances and general ill-health are also present. Death sets in generally with the development of coma or a terminal Pneumonia.

Differential Diagnosis:-

1. For years Schistosomial Cirrhosis of the Liver and Splenomegaly has been confused with malaria. This fact was probably due to the reluctance of the practitioner to perform post-mortems or to the scarcity of competent pathologists in the country. The Spleen, however, is more grossly enlarged than the Splenomegaly found in Malaria. The presence, moreover, of the multilobular cirrhosis with a demonstration microscopically of the tubercle formation and of the ova in the liver has proved beyond question or doubt that these cases cannot be due to the malarial plasmodia. Quinine has not the slightest effect on the condition and in many of these patients no malarial parasites are to be found.

2. Kala-Azar, another disease in which splenic enlargement occurs, is not known in Southern Rhodesia.

3. Syphilis is frequently found in the native of Southern Rhodesia, but in this disease the Spleen is not usually enlarged. Further, the positive Wassermann Reaction in Syphilis distinguishes the condition from that of Schistosomiasis. In addition, the arsenicals

ILLUSTRATIONS.

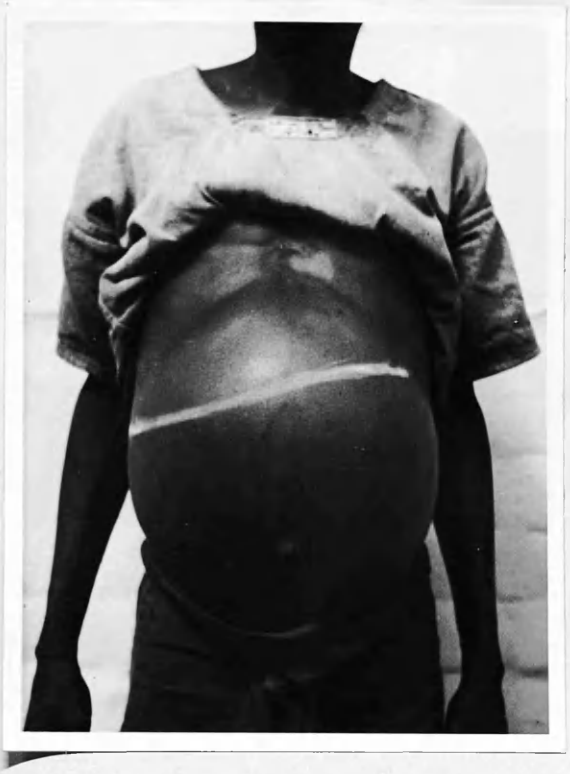

Fig.28. Case showing ASCITIC STAGE of SCHISTOSOMIAL CIRRHOSIS of LIVER with PREDOMINANT PORTAL OBSTRUCTION. Note enlarged superficial abdominal veins.

Fig.29. Side view of same case.

Fig.30. Case of LATENT CIRRHOSIS described in text.

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which are so useful in the treatment of Syphilis have no effect in Schistosomiasis.

4. Leukaemia is easily distinguished from Schistosomiasis by the characteristic blood-changes which occur in both types of the former disease, and by the enlargement of the lymphatic glands. ~~in the lymphocytic form of the disease~~.

Cases illustrative of condition:-

1. Case of Latent Cirrhosis: The patient was a male native aged 25 years. He had injured his left shoulder in an accident and had been admitted to Hospital. Except for this complaint, his clinical history was entirely negative. The liver was palpable 1½ inches below Right Costal Margin, its edge was hard and the surface irregular. The Spleen was enormous and could even be seen on
Fig.30 inspection of the abdomen.+ There was a rash on his face including the lower jaw; it was depigmented; there was some pallor of the conjunctivae; tongue was moist and clean. Examination of stool revealed Sch.mansoni. Urine was normal. No malarial parasites were ever demonstrated though microscopic examination was frequent. Wassermann Reaction was negative. As soon as his shoulder was better the patient left hospital.

2. Case illustrating Schistosomial Cirrhosis with Predominant Portal Obstruction:

The patient was an adult male native aged 35 years. He was admitted to Hospital complaining of a distended abdomen of about 12 months duration. A fluid thrill was present; the spleen was enlarged but the liver markedly so. There was some oedema of the legs. He was tapped twice but the distension soon reappeared. No ova were found in stool or urine. He died from a terminal Pneumonia, and at Autopsy, Schistosomial Cirrhosis due to Sch.mansoni, was found to be present in the liver.

3. Case of Schistosomial Cirrhosis with Predominant Jaundice: A young adult native male aged 24 years was admitted to hospital deeply jaundiced; this condition he had had for four weeks. The liver was palpable about 3 inches beneath the Right Costal Margin. Its edge was hard and sharp and its surface irregular. The Spleen was grossly enlarged, and its lower border reached to beyond the umbilicus. The patient became comatose and died. At necropsy, a

multilobular cirrhosis of the liver was found and the spleen weighed 700 grammes. Histological section of the liver revealed the presence of tubercle-formation, and the ova of Sch.mansoni were found on digestion of the liver tissue. No ova were found in the spleen, either microscopically or even on digestion.

Schistosomial Cholecystitis:-

The first case of Schistosomial Cholecystitis believed to have been reported in a living case was published in 1937 by Makar. The condition, however, had been recognised previously as the result of autopsy findings. Hashing in 1932, found at autopsy six cases of Schistosomial Cholecystitis in patients suffering from the intestinal form of the infection. Steyn (1938) encountered in Johannesburg a case at autopsy and recorded his pathological findings.

In spite of the very frequent involvement of the liver by Schistosomiasis, it is noteworthy that the Gall Bladder is seldom affected. It has been suggested that Schistosomial Cholecystitis is the result of direct spread from the neighbouring liver tissue. This would appear to be unlikely as I have frequently met with, at autopsy, Schistosomial Cirrhosis of Liver, where that portion of the liver surrounding the Gall Bladder was involved, and yet the Gall Bladder itself revealed no evidence whatsoever of any inflammatory process. It would appear further, that the infection lodges in the Gall Bladder as the result of a haematogenous spread in which embolic ova enter the arterial supply to the Gall Bladder and are held up in its distant radicles. In the same way hepatic syphilis is frequent whereas syphilis of Gall Bladder is practically unknown since Syphilis is at first essentially a disease commencing in the radicles or branches of the Hepatic Artery, the Cystic Artery tending to escape. The diagnosis of Schistosomial Cholecystitis is practically impossible for the clinical features are indistinguishable from those due to the ordinary case of Cholecystitis. Even should the patient give a history of Schistosomiasis, it is not justifiable, owing to the extreme rarity of this condition, should the patient develop symptoms of Gall Bladder disease, to assume that the latter organ is affected by Schistoso-

-miasis. The true nature of such an affected Gall Bladder after its removal at operation can be easily determined by the pathologist.

The following case is, I believe, the second case of Schistosomial Cholecystitis reported in the living.

Case History:- The patient was a European male aged 55 years. Except for previous attacks of "malaria" he had been perfectly well until one year before admission to Hospital when he began to develop pain in the right hypochondrium associated with a marked degree of flatulence and gastric upsets, especially after partaking of fatty foods. The reason for his admission to Hospital was a severe attack of Biliary Colic without Jaundice. A cholecystogram was taken and the Gall Bladder failed to concentrate any dye; no calculi were visible. His urine and stool were examined since this is routine Hospital procedure, but no abnormality was found in either of these dejecta. The diagnosis of Chronic Cholecystitis probably with stone, was made on the clinical history and the radiological findings. Operation was advised to which the patient consented. At no time was Schistosomial Disease, still less the presence of Schistosomial Cholecystitis suspected. The Gall Bladder was removed and presented the following features:-

On Gross Examination, it was of a deep red colour. The wall was much thickened, and the cavity, which was filled with numerous small infective calculi, was contracted. The inner lining was perfectly smooth. No Schistosomial tubercles were evident microscopically.

On Histological Section, fibrosis was very prominent with resultant replacement of the muscular tissue and mucous folds. Chronic inflammatory cells were found in abundance. Schistosomial tubercles of the pre-fibrotic type, indicating that the condition was of long-standing, were to be seen. A small portion of the Gall-Bladder was digested in 3% Caustic Potash, and numerous ova of Sch.haematobium were recovered.

The patient made an uneventful recovery, and has since undergone a course of antimony treatment.

Note Pages 61 and 62 have been transposed by Book-binder.

CHAPTER IX

TREATMENT:

In 1917, G.P. Christopherson, Principal Medical Officer of the Civil Hospital, Khartoum, introduced the antimonial preparations as specific remedies in the treatment of Schistosomiasis. Afterwards it was found that MacDonagh had been the first to use Tartar Emetic in Schistosomiasis and that he had published his results in 1915 in a book dealing with the Biology and Treatment of Venereal Diseases. Little attention had been paid to this portion of the work at the time, and no use was made of the discovery until in May 1917, Christopherson, working without knowledge of MacDonagh's findings, published the results which he had obtained by using Tartar Emetic intravenously in cases of Schistosomiasis. Christopherson had been led to experiment with Tartar Emetic by observing its very beneficial results in cases of Leishmaniasis. It is undoubtedly due to the work of Christopherson that Antimony therapy was established on a safe and sound basis. Prior to the introduction of Antimony in 1917, Emetine was the drug most commonly used in the treatment of this condition. It made its first appearance in the therapy in Cairo where it was used as a secret remedy. Later, many workers used it in the treatment of all forms of the condition.

Emetine is not as effective in its parasiticidal action as Antimony; in addition, it is more toxic to the host and possesses a myocardial toxin which acts adversely on the heart, and may actually produce organic disease; it may also cause peripheral neuritis of prolonged duration. Lastly it is very much more costly a drug than antimony.

In Southern Rhodesia, until a few years ago, we retained the use of this drug for the treatment of Schistosomiasis in children and obese patients whose veins were difficult to find, but the results were attended so often with disastrous side-effects that ultimately few practitioners cared to risk its use. Eventually the introduction of Fouadin provided a safer and more efficacious remedy for children and for other types of cases where Tartar Emetic intravenously was not well tolerated. Today, in Southern Rhodesia the use of Emetine in Schistosomiasis has been completely abandoned.

Diagnosis: The diagnosis of Schistosomiasis is in the vast majority of cases, simple, provided that the physician remembers the protean manifestations of this disease and the relatively simple investigations required to prove its presence.

1. The first essential is a history of exposure and residence in an infected area.

2. The Urine and Stool must be examined for the presence of ova. It is common knowledge that one single specimen of either stool or urine may fail to reveal the presence of ova. Single specimen investigation cannot be too strongly condemned, and many specimens should be examined if the disease be suspected, especially if albumen and Red Blood Corpuscles are found microscopically.

The most valuable method of urine examination is to obtain the last few drops of the excretion and place them on a watch-glass. This should then be placed under the microscope and examined carefully for ova. If this is not done, all the urine should be collected and centrifuged, and the deposit examined for the presence of eggs. Failure to discover the ova in the urine or stool should not cause the physician to cease investigating the case from this point of view.

3. Cystoscopy or Sigmoidoscopy may in many instances reveal the presence of the infection.

4. An eosinophilia in the blood picture is helpful, but in itself it is not sufficient evidence on which to base a diagnosis of Schistosomiasis. In the majority of cases the urine and stool investigations confirmed by Cystoscopy or Sigmoidoscopy are sufficient to establish the diagnosis.

5. In a few cases, however, where thorough examination on the above lines has not been helpful, a complement-fixation test or the Intra-Dermal Skin Tests may be tried.

In my view, much work still remains to be done in the perfecting of these two latter tests before they can be considered satisfactory

Differential Diagnosis:-

This has been fully dealt with in the discussion of the Clinical Manifestations of the disease.

Antimony therefore has been established as the drug of choice and the trivalent compounds are the most favoured of these. Potassium Antimony Tartrate and Sodium Antimony Tartrate are the two most commonly used.

Potassium Antimony Tartrate is a more toxic and rougher type of drug than the Sodium Salt. It is readily soluble in water and is easily sterilized by boiling. It produces its effects in lesser dosage than the Sodium Tartrate but its side-effects are stronger and may cause greater discomfort to the recipient.

Sodium Antimony Tartrate is probably the safer and less toxic of the two drugs, though it requires a slightly larger total dosage for full efficiency. It is more soluble and less nauseating than the Potassium Salt. In actual practice there is little to choose between the two drugs and each has its own supporters.

Dosage:- Our practice in Southern Rhodesia is to begin with a dose of ½gr. of the Potassium or Sodium Tartrate and to continue the injections on alternate days, giving increasing doses of the drug up to a maximum of 2 to 2½ grs; the rate of increase and the maximum amount of the individual dose depending on the drug used and the reactions of the patient. We limit the maximum individual dose of the Potassium Tartrate to 2 grs., but continue up to 2½ grs. with the Sodium Tartrate if the patient's condition permits. A total maximum dosage of 26 grs. is the amount aimed at.

Other trivalent compounds are in common use and of these Antimosan and its more recent preparation Fouadin are best known. The value of these preparations is that they can be used intramuscularly without causing pain or giving rise to necrosis. These preparations are more expensive than the Tartrates and have the additional disadvantage that they require to be introduced into the tissues in large volumes of solution (5-10 c.c.), thereby setting up a certain amount of local irritation. Fouadin, however, is extremely useful in the treatment of children.

Many pentavalent compounds of Antimony have been introduced in the therapy of Schistosomiasis, but none of them has any special value which would make its use preferable to the better known of the trivalent compounds. The commonest pentavalent compounds are

Stibenyl, Stibosan, and Stibamine, but the use of most of them has been discarded.

Antimony is a powerful and dangerous drug and requires both care and skill in its preparation and use. Only freshly made solutions should be used; if the tartrates are the drugs of choice, they must be injected intravenously and every possible care must be taken to ensure that the needle enters and is retained in the vein. Unless this is done, the drug may be injected or leak into the surrounding tissues where it will give rise to a nasty reaction with local necrosis.

Antimony exercises a specific lethal reaction on the Schistosomes and kills both adults and ova.

The first effect of the drug is to stop egg-production; the next is to cause the death of the ova in the shells, and the final result when the drug is in sufficient accumulation, is to destroy the adult worms. To obtain this end, sufficient concentration of the drug must be maintained in the circulation for at least 3 to 4 weeks.

The amounts of the drug required for this purpose do no damage to the host though the margin of safety between the therapeutic and the poisonous dose is not great. Symptoms of a metallic taste in the mouth accompanied by salivation, vertigo, nausea, weakness and headache indicate to the physician that the limit of tolerance has been exceeded. Cough is of no importance since almost every patient is seized with an attack of coughing of varying intensity, either during the injection or immediately after it; this is merely an indication that the drug has reached the pulmonary circulation. The symptoms of acute and chronic antimony poisoning can be found in the text-books.

The only criterion of complete cure is the final and permanent disappearance of living ova, and this can only be ascertained by repeated examination of urine or stool over a period of months after the course of treatment has been completed.

B. <u>General Treatment</u> - follows the usual lines of maintaining the strength of the patient and alleviating distressing symptoms.

In this disease with its many and varied aspects, the whole armamentarium of the physician and nurse may be called into action, and the special methods adopted will depend upon the manifestations

of the condition.

It is not until the Secondary Stage of Local Disease has been established that the patient will seek medical aid. If the condition is recognised and the specific treatment administered, the disease will be quickly brought under control and the symptoms alleviated. The only exception to that happy state of affairs is when cerebral or neurological symptoms show themselves suggesting the involvement of the central nervous system. In these circumstances, the maintenance of the general condition of the patient is important, and adequate nursing services must be combined with intelligent treatment.

The Third Stage or Stage of Complications is the phase where general treatment will be most frequently required, and this will follow the type of complication present and be adapted to its needs. There is no need to indicate here the many various lines which general treatment may take.

C. Surgical Treatment: The surgical treatment of Schistosomiasis has been admirably worked out by the American and Egyptian Schools of Surgeons, and ranges from the correct methods of passing a catheter to extripation of the Kidney and the Spleen. All this elaborate technique can be found in the various surgical text-books on the subject. Here I only wish to mention that the operation of Splenectomy in the treatment of the Hepatic-splenomegaly type of the disease does not seem to have resulted in the benefits expected and is gradually being discarded by most surgical experts. Further, it is well to note that all surgical procedures should be preceded by a full course of Antimony. It is extraordinary how often the need for surgical intervention disappears after a full course of specific treatment.

CHAPTER X.

CONTROL AND PREVENTION OF SCHISTOSOMIASIS:

In this section of the paper I intend to confine myself to a description of the plans and proposals I prepared in 1937 for the Southern Rhodesia Government, and which have since been adopted as the plan of campaign against Schistosomiasis in this Country. First however, it would be advisable to explain briefly the type of

population in the Colony and the bearing this feature has on our problem. The white population is relatively small, comprising in all only some 60,000 persons. While much of it is scattered all over the 150,354 square miles of the Colony's territory, there are relatively large concentrations in the towns of Salisbury and Bulawayo, and lesser concentrations in the smaller towns of Umtali, Gwelo, Fort Victoria, Gatooma, Que Que, Shabani and Gwanda. Outside of these townships, mining and agriculture form the two chief industries and most of the rural population are employed on one or other of these occupations. Within the white community there are two main social classes; the better educated type almost entirely of European extraction, and the poorly educated type of South African Dutch origin. The latter class live mostly in the rural areas and are close to the standards of the native population in their ideas of sanitation and personal hygiene. The introduction of compulsory education is doing much to raise the intellectual level of this class through the medium of their children.

Both these classes of white persons live in the midst of a large native population which, whilst it has its own protected areas of occupation, the Native Reserves, nevertheless provides the European population with its servants and workers. The native population in Southern Rhodesia corresponds to the working-class population of European countries, and whilst engaged on these functions, they either live on the employers' premises or land, or are accommodated in locations or compounds around the townships or within the vicinity of their places of employment. Further, the Native Reserves, large tracts of land specifically allocated for the habitation and use of the native, are not confined to any one part of the country, but are scattered throughout its length and breadth. The European population, therefore, lives in the midst of the Native population. This native population is of Bantu stock and until the occupation of the Colony in 1890 was completely barbarous. From 1890 until the advent of Responsible Government in 1924, there was no organised plan nor indeed any official attempt to educate and develop the native people. This work was left to the voluntary efforts of a few Missionary bodies unsupported by any Government contribution. Since 1924, however, successive Governments have

devoted more and more attention to the care of the native, and today the Native Affairs Department is a powerful State organisation which devotes the whole of its energies to the care and development of the native in his home, his schools, his lands, and in his various forms of employment. The Government Medical Department, working in conjunction with the Native Affairs Department has, since 1935, created 52 hospitals for his accommodation and treatment in times of sickness, and has so organised these medical stations as to make them the centres of teaching in methods of village hygiene and the maintenance of personal health. Whilst the native race cannot yet be said to be educated, and whilst its modes of life are still primitive and mostly unhygienic, nevertheless the leaven is working and the general tone of the people gradually rising. It seemed to me therefore, in 1937, that the time had come when some organised effort should be made to stamp out the two chief diseases which condemn not only the native, but also many members of the European community to permanent ill-health, namely malaria and schistosomiasis, and in that year I submitted to Government proposals which I will later relate as far as they concern the subject of this paper, the Disease of Schistosomiasis.

In the formulation of an anti-schistosomiasis campaign consideration must be given to the different stages of the parasite's life-history, and control measures based on the facts relative to these varying phases.

The Schistosome has two Stages in its' life history.

1. a Corporeal Stage:- during which adult worms, and ova with their contained miracidia, live in the vessels and tissues of man.

2. an Extracorporeal Stage:- which includes the following phases:-
   (a) Eggs and miracidia in the dejecta of man.
   (b) Sporocysts in Snail.
   (c) Cercariae in Water.

Every plan of campaign must take cognisance of these two Life-Stages and the condition and activities of the parasite during the phases which comprise them.

Two further facts are required before any plan with a reasonable chance of success can be adopted, namely:-

(a) A knowledge of the percentage of infected cases in the population.

(b) A knowledge of the general type of snail population and of the presence, distribution, habitats and habits of the particular carrier-snails.

In order to obtain information on the first point, namely the number of infected cases relative to the total population I directed that a blood, stool, and urine examination should be conducted on every school-child in the Colony and similar examinations performed as a matter of routine on every patient admitted to any Hospital in the Colony. As all Hospitals in this country are under Government administration, no difficulties were experienced in having these instructions carried out. Unfortunately the threat and later the outbreak of war with its resultant dislocation of Hospital procedure and shortage of staff interfered with the carrying out of these measures and finally caused their temporary abandonment. However, before that took place, sufficient evidence had accumulated to give me fairly accurate ideas of the position, and on the basis of these examinations it can be stated that the proportion of infected cases amongst the white population is 12% and amongst the Native race is 35%. Of this 12% amongst the white population 10% is due to infection by Sch.haematobium and 2% to infection by Sch.mansoni, whilst in the 35% amongst the Native population 29% is due to Sch. haematobium, 4% to Sch.mansoni and 2% to infections of both parasites.

To obtain evidence on the second point, namely, the general type of snail population in the Colony, and the presence, distribution, habitats and habits of the particular carrier-snails, I asked for and obtained the services of an eminent helminthologist Dr. Alan Mozley, who arrived in the Colony in June 1938 and has since been engaged on this work.

In the meantime I was now able to formulate a plan of campaign, and this I did on the following lines:-

1. Division of the country into areas.
2. Examination in Areas to determine percentage of Infected cases.

As each area had one or more Government Medical Officers stationed within it, and had one or more Government Hospitals and Government Schools within its boundaries, the examination of European and Native school-children and the routine examination of all patients admitted either to European or Native Hospitals enabled us to deal with fairly large samples of the population in each area.

3. Free treatment and where possible mass treatment of infected

cases in each area with particular reference to children and adolescents.

One great difficulty in carrying out this part of the scheme is occasioned by the length of the course. As soon as symptoms disappear, many people believe themselves cured and do not return for further injections. That difficulty we endeavoured to cope with by our next step.

4. Education of the whole population in regard to the seriousness of the disease and the principles of prevention by Lectures, Cinema Demonstrations, Pamphlets.

This can only be regarded as a necessary adjuvant. Its results are neither great nor stable, and the lessons learned are soon forgotten.

5. Education of the population particularly in rural areas in the use of latrines, and other methods of disposal of urine and faeces. Provision of Latrines of the bored-hole type in Native Villages.

6. Sanitary Location of Native Villages - away from water courses.

7. Provision of Public Baths for Europeans; and of snail-free bathing places for Europeans and Natives.

The first we have in great measure been able to do through the generosity of the State Lottery Trustees - an Institution of Southern Rhodesia - who provided the funds for the erection of public baths for the use of small communities unable to afford such expenditure.

In other instances we were able to render and maintain snail-free various bathing places much frequented by the public. From time to time and particularly before public holidays, we issue in the Press of the Colony lists of places which have been treated and which we consider safe for bathing.

8. Treatment and elimination of the carrier-snails and their habitats.

The usual lines adopted under this heading are as follows:-

(a) Drainage of Pools where carrier-snails exist.

(b) Clearing of banks of Rivers, Lakes and Pools, of vegetation, and the removal of all food debris.

(c) Use of Chemical Substances destructive to snails but not in sufficient concentration to be harmful to man or domestic animals.

(d) Planting of trees and shrubs whose leaves or fruit are inimical to Snails.

(e) Encouragement of natural enemies, e.g. ducks.

All these methods and many others are in use in Southern Rhodesia but I am somewhat handicapped in dealing with this part of the subject by my knowledge of the many new methods experimentally introduced by Dr. Mozley, some of which already show great promise of success. These methods, however, are Dr. Mozley's and not mine, and as he has not yet had the opportunity of publishing them, since some of them have not yet had sufficient time in which to reveal their ultimate value, I think it would be better if I confined myself to the statement that many new methods of control and elimination of snails and their habitats are at present under trial and some of these have already given great promise of a successful issue.

9. One further method which we strongly urge in rural areas is the provision of water-tanks, so that water required for domestic purposes can be stored for 48 hours prior to use. This makes use of the fact that the cercarie dies in 36 - 48 hours and so even if the water has been taken from an infected river or stream, the danger of infection can be eliminated.

The above is an outline of the plan designed by me for the control and elimination of schistosomiasis, and now operative in Southern Rhodesia, although to some extent modified owing to the exigencies resulting from the war.

CONCLUSION:-

In this paper it has been my endeavour to present a clear and orderly review of the disease of Schistosomiasis particularly as it affects the people of Southern Rhodesia.

With this object in mind I have divided the subject into the following parts:

1. Definition.
2. Historical Review.
3. Geographical Distribution.
4. Etiology.
5. Parasitology.
6. Pathogenesis.
7. Pathology.
8. Clinical Manifestations, Diagnosis, and Differential Diagnosis.
9. Treatment.
10. Methods of Control.

Though I am indebted to many authors for information in regard to some parts of the paper, particularly in regard to the Historical Review, the bulk of the work is my own and has been compiled from notes made during a ten years' period when I was Medical Superintendent in charge of the Salisbury European and Native Hospitals. Much of the paper, particularly where it refers to conditions in Southern Rhodesia, is entirely new, since, as far as I am aware, this aspect of the work has not previously been dealt with by any other writer.

I am indebted to Mr. Vance Carlisle, late of the Pathology Department of the Glasgow University and now a member of the Public Health Laboratory staff, Salisbury, for much valuable help in preparing the various photographs.

DIRECTOR OF MEDICAL SERVICES.
SALISBURY,
SOUTHERN RHODESIA.

10th February 1941.

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