

## BILHARZIASIS IN NATAL.

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### INTRODUCTION.

In the following pages I shall endeavour to describe the present state of our knowledge of Bilharziasis in Natal and the result of some personal observations extending over the last seven years. The investigations of the Bilharzia Mission to Egypt<sup>1</sup> have drawn attention to the various forms of cercariae which infest several species of fresh-water snail in African rivers. The cercariae which give rise to Bilharzia disease in Egypt are furcocercous cercariae, *i.e.* they possess a tail which is divided for a greater or shorter portion of its length. They are characterised, further, by the absence of a pharynx at the commencement of the oesophagus. Similar cercariae are present in one of the species of fresh-water snail which abounds in the infected areas of Natal.

In some localities, the custom of the South African natives in anointing the skin with grease and oil may be nature's own preventative against the skin-infection through cercariae. During the visit of the Bilharzia Mission to Egypt it was observed that cercariae would attack all parts of the bodies and limbs of young rats and mice that were immersed in water containing them.

As long ago as 1872, T. S. Cobbold was of opinion that the disease was caused as a result of drinking water containing snails or the little cercariae which are often present in snails from the rushes of slow-running streams or stagnant bathing-pools; but only recently has it been known that infected snails were so intimately associated with the disease.

<sup>1</sup> *Journ. Roy. Army Med. Corps* for 1915—July, August and September.

I have confined myself to the disease as met with in Natal, where it would seem that it is contracted in every instance from bathing in infected rivers and stagnant pools. Since the danger of bathing in these places has become known, and since the municipalities have filtered or strained their town water-supply, the disease has become very much less frequent. I have treated cases from Rhodesia which have presented the same symptoms as those with which we are familiar in Natal.

Treatment consists in the avoidance of re-infection from fresh-water bathing-places and in combating the complications that arise in the severer cases. The disease as generally met with is not a very serious one and its interest lies largely in its relation to Life Insurance.

#### HISTORY.

Bilharziasis has existed in Africa for many centuries. Specimens preserved from the XIIth dynasty, which is estimated from 2500 to 4000 years before Christ, are said to prove its existence at that period. The cause of the disease was not known until 1852, when T. Bilharz published the report of his discovery of a bisexual distome and successfully established a definite relationship between this trematode worm and the symptoms of dysentery and haematuria, which result from its presence in the human subject<sup>1</sup>. In 1864, J. Harley reported that the endemic haematuria, common to certain parts of Natal and the Cape of Good Hope, was also due to a species of *Bilharzia*<sup>2</sup>. In 1871 he published his attempts to obtain direct infection by the mouth in two young rabbits and two dogs; but no trace of *Bilharzia* could be found<sup>3</sup>. In 1872 T. S. Cobbold published an article on "The Development of *Bilharzia* haematobia," showing how he had endeavoured to trace the life-cycle through an invertebrate intermediary host<sup>4</sup>. He failed to induce the ciliated embryos to enter the bodies of different species of *Planorbis* and other fresh-water molluscs, but the experiments were undertaken in England. In 1888, Jas F. Allen, of Pietermaritzburg, reported "nearly all the youths bathing in the Umsindusi and Dorp spruit were infected; whilst the girls, who do not bathe, remained free from the disease<sup>5</sup>." This view of infection by bathing is commonly held by all general practitioners in Natal with any experience of the disease.

In a popular lecture to the Rhodesia Scientific Association in Jan. 1914, Sir Patrick Manson said, after briefly outlining the development of the liver-fluke, "In a similar way I believe the germ of *Bilharzia* disease, so common in this country, especially in young people, and probably contracted whilst bathing in pools and rivers, is acquired and spread."

<sup>1</sup> *Zeitschr. f. wiss. Zool.* iv. pp. 53, 72-76, 454.

<sup>2</sup> *Med.-Chir. Trans.* London, XLVII. pp. 55-72, 2 pls.

<sup>3</sup> *Ibid.* LIV. pp. 47-62, 1 pl.

<sup>4</sup> *Brit. Med. Journ.* II. pp. 89-92.

<sup>5</sup> *Practitioner*, XL. pp. 310-320.

In the *Tropical Diseases Bulletin*, March 30, 1914, Fleet Surgeon Kumagawa comments on the publication of K. Mirairi and M. Tuzuki<sup>1</sup>. "The authors noticed that, when ox faeces which contain the eggs of *Schistosomum japonicum* are kept for one or two hours in a suitable temperature, mixed with water, the majority of the miracidia come out, breaking the shell and swimming about very vivaciously. They noticed also that, in the infected locality, there are many snails in the waterways and ditches." The authors carefully picked up a number of young non-infected snails and tried whether the miracidia entered their bodies or not. They found that the miracidia entered the body of the snail. After twelve days the first rediae appeared and gradually concentrated to the hepatic ducts. The authors put mice into the vessels in which the full-grown snails were fed for three hours every day, and repeated this experiment for four days. After three weeks they found a great many *Schistosomum japonicum* in the livers of the mice. The authors concluded that this kind of snail was an intermediary host of *Schistosomum japonicum*. These observations were confirmed by the Wandsworth Expedition of the London School of Tropical Medicine, in the spring of 1914.

#### EXPERIMENTS IN NATAL.

In May 1915 I made repeated attempts to infect specimens of *Limnaea natalensis* with miracidia obtained from recently passed urine. There was practically no possibility of these fresh-water organisms being infected with *Bilharzia* when the experiments were commenced, nor could any evidence of infection be detected at the end of several weeks. These experiments were repeated in September, but again with negative results. From further study I am of opinion that *Limnaea natalensis* is not susceptible to infection with the miracidia.

Judging from the reproduction of digenetic trematodes, Dr E. Warren, the Director of the Natal Government Museum, was of opinion that the miracidia had the asexual phase of its development in the mollusca; that it would die if unable to harbour in a suitable mollusc in 24 to 48 hours and that, if it was able to migrate to the liver of a mollusc, the sporocyst—a smooth-walled elongated sac—would develop in the course of a week or two. He expected to find the sporocyst giving rise by budding of its wall to daughter-sporocysts and to contain a large number of cercariae with bifid tails.

At the end of May 1915, I supplied him with urine obtained from a patient suffering from Bilharziasis. He diluted this urine, which swarmed with ova, and added it to a vessel of water containing snails, *Physopsis africana*. The bathing season had not commenced and the snails were probably free from infection when the experiments began. Four weeks later, he wrote me a letter in which he said: "I have found some undoubted cercariae in infected snails, and the question now is whether they belong to *Bilharzia* or to some

<sup>1</sup> *Tokio Med. Journ.* Sept. 1913.

other trematode. Unfortunately, it is quite possible that the sporocysts and cercariae found are common in the snails and that the urine added to the water did not really infect them." In 1916, during the months of February to June, I collected and examined 500 specimens of this genus of fresh-water snail from various parts of Natal. Some of these were infested with cercariae; but I found cercariae, typical of *Bilharzia* infection, possessing a divided tail and showing no muscular pharynx, only in specimens from two bathing-places which are known to be associated with Bilharziasis amongst swimmers.

The sporocyst which contained the cercariae which Dr Warren found in the specimens of snail he had exposed to *Bilharzia* infection was an elongated body without oral sucker or alimentary canal. Through its transparent walls could be seen a large number of cercariae in various stages of development. When teased out and stained, the cercariae were seen to consist of a body and a tail. The body possessed a terminal oral and a ventral sucker. There were no pigment spots and no muscular pharynx. The tail was bifid. There was no cuticular keel along either side of the prongs of the tail.

In a stagnant pool near Durban which is known to give rise to Bilharziasis amongst bathers, I have found 15 sporocysts answering to this description and containing similar cercariae, in specimens of this same snail.

The appearance of both sporocyst and cercariae corresponded to those described by Leiper and Atkinson<sup>1</sup> as characteristic of Asiatic schistosomiasis.

#### RECENT RESEARCHES.

The *Bilharzia* Mission to Egypt, according to a full report by Lt-Col. Leiper<sup>2</sup>, demonstrated that the parasitic worms which gave rise to Bilharziasis in Egypt developed from the cercariae which infested certain species of fresh-water snails. The Mission found that infection might take place orally or cutaneously. The report is illustrated, and one microgram shows the cercariae in the act of passing through the unbroken skin of a newly-born mouse. Later, Lt-Col. Leiper<sup>3</sup> reported that those cercariae which infest specimens of *Bullinus* (sic) invariably develop into *Schistosomum haematobium*; whilst the cercariae which infest the *Planorbinae* of Egypt always produce *Schistosomum mansoni*.

#### DISTRIBUTION.

Practically all the rivers between Pietermaritzburg and the coast are said to be infected with *Bilharzia*, though it would seem that the disease is not to be caught at higher altitudes in Natal. During a somewhat extended tour of Natal in the spring of 1916, I visited the following places to ascertain the prevalence of susceptible fresh-water snails in infected areas. I found specimens of *Physopsis africana* difficult to obtain in any places except where the water was stagnant or slow-running.

<sup>1</sup> *Brit. Med. Journ.* Jan. 30, 1915.

<sup>2</sup> *Journ. Roy. Army Med. Corps*, 1915.

<sup>3</sup> *Brit. Med. Journ.* March 18, 1916.

Date	Place	River	Observations
Feb. 14	Verulam	Umhloti	Reeds clear after rains
" 16	Avoca	Little Umhlanga	<i>Physopsis</i> plentiful in infected pools
" 16	Toll Gate (Durban)	Brickfields	<i>Physopsis</i> , <i>Planorbis</i> , <i>Limnaea</i> and <i>Isidore</i> plentiful in infected pools
" 17	Hill Crest	Umhlatazana	Reeds clear in river, bathing not allowed
" 18	Umlaas Bridge	Umlaas	<i>Physopsis</i> plentiful in infected river
	Maritzburg	Quarry	Only <i>Limnaea</i> , doubtful infection
Mar. 10	"	Boshoff St. pool	Bathing forbidden. No <i>Bilharzia</i> cercariae. <i>Physopsis</i> , <i>Limnaea</i> , <i>Planorbinae</i> and <i>Ancylus</i> plentiful
" 31	"	Umsindusi	<i>Physopsis</i> and <i>Limnaea</i> plentiful. <i>Bilharzia</i> cercariae in <i>Physopsis</i> . Infected river
	Greytown	River	Reeds clear and no infection known
Feb. 16	Durban	Umgeni	Reeds clear
May 1	"	"	<i>Physopsis</i> and <i>Planorbis</i> . An infected place
" 13	Henley	Umsindusi (near source)	Reeds clear in river. No infection known
June 23	Maritzburg	Brickfields	Reeds clear. No bathing

THE FRESH-WATER SNAILS OF NATAL.

I have encountered the following species of snail in Natal rivers and fresh-water pools:

*Limnaea natalensis* is a common light brown snail with a semi-transparent dextral shell. Some specimens are infested with "tadpole" cercariae.

*Physopsis africana* is a common black snail with a blunt-pointed sinistral shell, the shell is characterised by a truncate columella. This snail harbours both the "tadpole" and furcocercous cercariae; but *I have only found the latter forms of cercariae in bathing-places which are known to give rise to Bilharziasis.*

*Planorbis pfeifferi* is similar to the intermediary host of *S. mansoni* in Egypt. It is a common brown snail with a round, flat shell.

*Planorbis leucocheilus* is a light brown, less common snail. The shell is small and flat. *Planorbis pfeifferi* harbours many "tadpole" cercariae.

*Isidora tropica* is a fairly common, dark brown snail with a blunt-pointed, sinistral shell. I have never found cercariae in this species.

*Isidora forskali* is a rare, brown snail with a conical shell, I have only seen about three specimens of it.

In one brickfield, I found a large number of a small oval snail; which is a new species of *Ancylus* not yet identified.

The following table shows the percentage of snails harbouring *Bilharzia* cercariae between the months of April and July, 1916.

Month	Source	No. infected	Percentage	Form of cercaria
April	Toll Gate (Durban) (bathers infected)	1 out of 7	14	human
May	" " " "	13 " 85	15	12 human (1 eye-spotted)
June	" " " "	2 " 13	15	human
July	" " " "	8 " 131	6	"
April	Umsindusi (bathing not allowed)	30 " 197	15	1 or 2 human
May	" " " "	38 " 170	22	"
June	" " " "	7 " 30	23	almost all <i>C. secobii</i>
July	" " " "	0 " 6	0	
June	Boshoff St. (no bathing)	0 " 20	0	
July	" " " "	0 " 6	0	

## FURTHER EXPERIMENTS IN NATAL.

In the spring of 1916, I made an exhaustive study of the commoner fresh-water snails of Natal. I examined, microscopically, the livers of 1500 specimens. They included seven different species of snail. I could find no *Bilharzia* cercariae in any species of snail except in *Physopsis africana*. I examined 250 *Planorbinae*. It is interesting to note that *Schistosomum mansoni* which has this species of snail in Egypt as its intermediary host is unknown in Natal. I encountered three different forms of *Bilharzia* cercariae in the specimens of *Physopsis africana* which I examined, one was an eye-spotted form, for which I have suggested the name of *Cercaria bilharziella lunata*, in view of the crescentic form of its eye-spots. Another, for which I have suggested the name of *Cercaria secobii*, has very long prongs to its divided tail; the third form corresponds to that which causes Schistosomiasis in Egypt and the Far East. In one bathing-place which is known to be a source of Bilharziasis, I found 14 % of the specimens of *Physopsis africana* infected with this human form of cercaria. In the April number of the *Medical Journal of South Africa*, Dr J. G. Becker reported that he had found similar cercariae in three out of 13 specimens of this same snail which he had collected from an infected bathing-pool at Nijlstroom in the Transvaal.

I then undertook experiments to infect with miracidia, obtained from the urine of *Bilharzia* patients, a number of this species of snail.

On April 22, I found that 14 out of 31 specimens, obtained from the Umsindusi river, which I had exposed to infection by miracidia three weeks previously, contained *Bilharzia* cercariae—i.e. 45 %. Examination of 197 specimens direct from the river showed an infection of only 15 %.

During May, when 22 % of the specimens in the river were infested with *Bilharzia* forms, the percentage of infected snails amongst 19 which had been exposed to infection by miracidia 36 days previously was 37 %.

On June 9th, 60 specimens from the Umsindusi which had been exposed to infection on May 20 were examined for cercariae. Sixteen contained *Bilharzia* forms. Of 30 specimens from the same source, which had been kept for three weeks in clean water, seven were infected.

Similar experiments with *Planorbinae* and *Limnaea* proved entirely negative.

Whilst these experiments were in progress, I attempted to produce *Bilharzia* infection in 12 white rats, six guinea-pigs and six pigeons. During 1915, Dr E. Warren had undertaken similar experiments, submitting rabbits and mice to hypodermic infection with *Bilharzia* cercariae, but with negative results.

In my experiments, I immersed rats and guinea-pigs for an hour on several days in water swarming with *Bilharzia* cercariae; to others, I gave water containing cercariae by the mouth or rectum. No symptoms were produced at the end of three months and no parasitic worms were discovered post-mortem.

I am not aware that Bilharziasis has been demonstrated in any animal in South Africa, except in man. Dr Leipoldt, the Medical Inspector of Schools in the Transvaal, tells me that he has not succeeded in infecting guinea-pigs. However, he is certain that the house-fly carries the egg from the urine and that it can infect monkeys in this way, but, in view of the recent observations reported from Egypt on the life history of both forms of the parasite, this opinion would seem to be of little importance.

#### PROGNOSIS.

The duration of the disease is extremely variable. In the milder cases there is seldom any pain and the slight haematuria is not noticeable for more than a few weeks. In others, the disease runs a very protracted course, extending over many years. Many cases recover and show no after-effects from the disease, but sterility is common amongst patients who have suffered severely in their younger days. Even in its severest forms, Bilharziasis as met with in Natal seldom proves fatal; although, when complicated by an attack of cystitis or renal stone, fatal symptoms may and do occur. Favourable symptoms are—an absence of anaemia and renal colic, an absence of mucus in the urine and only slight and occasional haematuria. Renal colic is common and does not necessarily point to the presence of renal calculus; it is almost certainly due to the accumulation of ova in the kidney substance. These ova have become loose in the blood-stream and have not made their way up the ureters from the bladder, as popularly supposed.

The occasional presence in the urine of blood and mucus, or of shreds of mucous membrane, indicates the need for urinary antiseptics and diuretics, if permanent damage to the bladder-wall is to be avoided. Microscopic examination of the urine will often reveal the presence of ova, even though the absence of symptoms of the disease for several years had indicated that the patient was cured. In one Government School in the Transvaal, Rustenburg, between 30 and 40 % of the boys showed symptoms of Bilharziasis; microscopic examination of the urine revealed the fact that 76 % of the boys were infected. It is probable that the eggs will continue to escape from the body for some months after the worms themselves are dead. Occasional aggravation of symptoms in patients indicates that they continue to harbour parasites in the blood-stream, even though they have not exposed themselves to fresh infection for many years.

#### LIFE INSURANCE.

In course of conversation with the Resident Manager for Natal of the National Mutual Life Association of Australasia, he stated that the Company would not accept a case of Bilharziasis so long as the disease was sufficient to produce albumen in the urine. This attitude is a commendable one, for one could not say for certain whether the presence of albumen was entirely due to the disease of itself. But this statement was of especial interest in view of

the fact that I had recently advised a further examination of an applicant whose urine contained a slight trace of albumen. Later, I heard that the applicant had suffered from Bilharziasis, and a subsequent examination of the urine revealed the presence of ova containing miracidia. The Manager stated, further, that the Company would accept at the usual rates for Life Insurance persons with a past history of the disease whose urine was free from albumen.

The Manager for Natal of the Southern Life Association did not consider a history of Bilharziasis in the past very unfavourable, but he drew attention to the risk of gonorrhoeal infection in patients with a past history of the disease.

If an applicant for Life Insurance had noticed no symptoms of the disease for the last twelve months and occasional examination of the urine during the last six months had shown no presence of ova or other abnormality, I should recommend a Company to accept the case without a loading.

If examination of the urine revealed the presence of ova, I should advise a fresh examination in three months and not advise that the case be accepted until the urine had been clear for six months.

If renal colic had been severe and frequent, albuminuria present, a low specific gravity of the urine which is a not uncommon after-effect of the disease, or other sign of permanent kidney mischief, I would naturally advise refusal of the applicant.

The following opinions of Natal practitioners given in 1893 and 1906 in regard to the seriousness with which Bilharziasis should be considered in its relation to Life Insurance is instructive, and it is interesting to note how little they altered their views, after further experience of the effects of the disease. In one case, only the opinion expressed in 1893 can be quoted:

1. Have you had any death attributable to Bilharziasis?

Dr. A. 1893 (after eight years practice in Natal and Cape). No.

Dr B. 1893 (after ten years practice in Natal). No.

1906 (after twenty-three years practice). From secondary effects, but extremely rare; I know of two cases only.

Dr C. 1893 (after ten years practice in Natal). No.

1906 (after twenty-three years practice). No.

Dr D. 1893 (after ten years practice in Natal). No.

1906 (after twenty-three years practice). No.

Dr E. 1893 (after ten years practice in Natal). Yes, indirectly. One case at P.M.: examination showed kidneys congested and deposits of gravel. Patient had suffered from gravel, albuminuria and renal colic. Ova were numerous and I attributed death to Bilharzia.

1906 (after twenty-three years practice). Not directly.

2. Have you had life shortened by this affection?

Dr A. 1893. In one case only, which I attributed to chronic cystitis, due to Bilharzia. Cystitis began at age of 10 years. Death occurred with convulsions at age of 16 years. P.M. examination showed much inflammation of the bladder, but no examination for Bilharzia was made at time of death, nor for two or three years before then.



- Dr B. 1893. No.  
 1906. Not apart from two cases mentioned.
- Dr C. 1893. No.  
 1906. No. The most serious case I have seen, with intense anaemia, got better in time with no treatment.
- Dr D. 1893. Cannot say.  
 1906. I should say no.
- Dr E. 1893. Yes, but very rarely.  
 1906. I think so, but only very occasionally.

3. Have you observed any serious symptoms attributable to Bilharzia?

- Dr A. 1893. With exception noted above, no.
- Dr B. 1893. Yes, I have formed the opinion that occasionally I have had symptoms in themselves serious, which I think may be attributed to Bilharzia if the theory of the ova forming nuclei for gravel be correct.  
 1906. I believe renal colic is not infrequent in lads who have had Bilharzia.
- Dr C. 1893. Yes. *Anaemia after constant drain from the bladder. I have only had one such case out of, say, 100 that have been under my treatment. If the theory of gravel arising from ova of Bilharzia be correct, and I think it is, from the fact of so many gravel cases having a history of Bilharzia, then I have had severe symptoms indirectly.*  
 1906. Anaemia is common but not serious, and only in aggravated cases and the young have I had slight albuminuria which passes off.  
 Calculi. It is common to find those suffering from above to have at one time had Bilharzia.
- Dr D. 1893. Can recollect two cases in which general health suffered consequent on Bilharzia.  
 1906. I have had cases where for a time the general health has been impaired but those have been patients grown up when they had the disease.
- Dr C. 1893. Yes. Not infrequently anaemia and renal symptoms. I had two cases aged 10 years and 15 years with chronic dysentery and griping pains suggesting an ulcerated condition of the bowel. These, when examined, showed Bilharzia and I attributed the condition of both to Bilharzia.  
 1906. Albuminuria rare and only in extreme cases. I have seen very severe haemorrhage in two or three cases, but in each it passed off quickly. Slight haemorrhage is common and occasionally anaemia. Sometimes, but rarely, renal colic from gravel.

4. Have you found any cure for the disease?

- Dr A. 1893. None, certain; the disease usually cures itself.
- Dr B. 1893. No. I sometimes give Iron and Turpentine and find at least that the patients are none the worse for it.  
 1906. No. In lads it will probably cure itself about the age of puberty.
- Dr C. 1893. No. The disease will probably cure itself in time.  
 1906. No. Salol, Santonin or tonics may do good. They often lessen bleeding.
- Dr D. 1893. No. Nature seems to cure in most cases.  
 1906. No.
- Dr E. 1893. Iron may assist, but they usually grow out of the affection.  
 1906. No. The disease tends to die out of its own accord.

5. How would the fact of a patient being the subject of Bilharzia affect him in medical examination for Insurance?

(a) When the symptoms are slightly marked:

- Dr A. 1893. No loading.

Dr B. 1893. Not at all.

1906. If no albuminuria, if symptoms slight, general look healthy, patient, say, 20 years of age, I should pass the life with a light loading of, say, three years to be taken off when the patient is quite better.

Dr C. 1893. Not at all.

1906. If symptoms slight, if patient 23 or 24 years old and if applicant has had the affection two or three years, I take no notice of it.

Dr D. 1893. If on examination of urine, ova are detected, add a few years for the protection of the Company, though you yourself may not think anything of it.

1906. If patient of strong physique, temperate and otherwise in good health, pass at ordinary rates.

Dr E. 1893. In case of three or four years' standing, without serious symptoms and no albuminuria. If ova have disappeared, ordinary rates.

1906. If applicant has been affected a good while and is getting better, I would (other circumstances being favourable) pass him as a first-class life.

(b) When the symptoms are well marked:

Dr B. 1893. Add three years or more, according to symptoms.

1906. Would defer the case, but not refuse it.

Dr C. 1893. Load the life three or four years. If anaemia profound or renal colic, refuse the life.

1906. Might load, because of after trouble from calculi.

Dr D. 1893. Would be regulated entirely by circumstances.

1906. Wait for a few months and then re-examine.

Dr E. 1893. Defer till better or slightly marked.

1906. In extreme cases, refuse the life. Moderately severe, without anaemia and albuminuria slight, I would load the life three to five years.

#### TREATMENT.

The treatment of Bilharziasis in Natal consists in:

- (a) avoiding fresh infection from the bathing-pools,
- (b) destroying the parasites which exist in the portal and mesenteric veins. So far we do not know of any effective remedy for this,
- (c) hastening the exit of the eggs from the bladder,
- (d) correcting the bacilluria and haematuria,
- (e) correcting the anaemia,
- (f) treating the renal colic, gravel and cystitis,
- (g) surgical remedies to remove calculus or to relieve retention of urine.

The consensus of opinion would seem to be that it is best to resort to the almost exclusive use of diuretics and urinary antiseptics in uncomplicated cases of the disease. The use of hexamine is of undoubted service in many cases and may be combined with small doses of Buchu. Where there is continuous renal colic, Sodium salicylate and Tr. Hyoscyamus are indicated.

Amongst the various remedies which are employed in Natal may be mentioned:

(a) Methylene blue, Male fern, Thymol, Santonin and Salol, to destroy the parasites in the blood-stream. Injections of Salvarsan have been tried without effect, and the mercuric-bichloride treatment of Egypt seems to be quite ineffectual in Natal.

(b) Buchu, Turpentine, Hexamine and Cystopurin, to accelerate the escape of eggs and to counteract the bacilluria.

(c) Adrenalin in severe cases of haematuria.

(d) Tr. Hyoscyamus and Sodium salicylate for the renal colic and injections of morphia where required.

(e) Iron and tonics for the anaemia.

It should be borne in mind that the disease is intractable and in many cases resists all forms of treatment; whilst it is the opinion of many with a large experience of the disease that where complications do not arise, the disease may be left to die out of itself without treatment of any kind. My own feeling is that, so long as there is haematuria and flakes of mucous membrane in a cloudy urine, the patient is well advised to persist in taking some form of urinary antiseptic; but, until we are in possession of a drug which is known to destroy the parasites, it is unwise to persist in the use of uncertain remedies.

#### CONCLUSIONS.

1. That *Bilharzia cercariae*, similar to those which have been demonstrated to cause Bilharziasis in Egypt, occur in Natal in specimens of *Physopsis africana*.

2. That specimens of *Physopsis africana* abound in infected localities, and that a large proportion of these are infected with *Bilharzia cercariae*.

3. That specimens of *Physopsis africana*, when exposed to infection from the urine of *Bilharzia* patients, increase in the number of infected forms.

4. That experimental infection of animals has been unsuccessful and, as far as is known, animals in Natal are not susceptible to the disease.

5. That it is rare to come across a person whose life has been shortened or whose death has been caused from Bilharziasis in Natal.

6. That, until some specific is known, palliative measures are the only ones that can be recommended in the treatment of the disease.

7. That municipalities would be well advised to follow the example of Durban in filtering their water-supply, or, where this is impracticable, the water should be strained through such copper ribbons as are in use at Pietermaritzburg which preclude the possibility of infected snails entering the reservoir from which the town supply of water is derived.

8. That, in infected areas, river bathing should be forbidden and suitable swimming-baths provided.