ON SCHISTOSOMIASIS:

A COMPARATIVE STUDY OF THE CONDITION AS FOUND IN BERBER PROVINCE, ANGLO EGYPTIAN SUDAN WITH REFERENCE TO RECENT LITERATURE.

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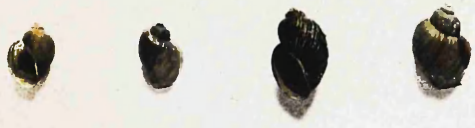
above,
SHELLS OBTAINED FROM WASHED
RIVER SAND.



BULINUS spp.

PHYSOPSIS spp.

LANISTES spp

MELANOIDES spp.

CLEOPATRA spp.

SNAILS LOCALLY INFESTED WITH *furcocercous* AND *non-furcocercous* CERCARIÆ.

R.M. BUCHANAN

CONTENTS.

ILLUSTRATIONS.

I N T R O D U C T O R Y.

The accepted clinical outlook on Bilharzial infestation is today too closely focussed on the advanced, or even terminal, stages of the disease. In effect, the whole is coloured by the literature of the Egyptian School, which deals almost exclusively with cases drawn from the hyper-infested population of the lower Nile and Delta.

The following paper is primarily concerned with infestations due to S. haematobium and S. mansoni as they occur in that part of the Sudan known as Berber Province.*

Both urinary and intestinal - or rather, visceral - schistosomiasis are endemic in the Sudan.[1] The former in particular is widespread, occurring in the three northern riverain provinces, in cultivated zones,[2] and seasonally about the water holes and pools of the western provinces. Further reference will be made to seasonal incidence. The infection due to S. mansoni, variously labelled intestinal, rectal, or visceral schistosomiasis, occurs in the White Nile Province south of Khartoum, and has lately been noted (1933-34) by the author in Berber Province, where there is a comparatively circumscribed zone of hyper endemic infection. It is virtually peculiar to this area, and/

* Now amalgamated with the province of Dongola, and Halfa, as the Northern Province.

and it is of great interest to note that no cases of vesical infection were found in the affected villages. This peculiarly localised distribution is noteworthy, and is subsequently discussed in detail.

An attempt has been made by various writers to establish and label a definite syndrome for the schistosomal infestations. A distinct, uncomplicated case of Bilharziasis is a rarity, at least in so far as this province is concerned. In the author's opinion the clinical aspect is here considered as dependent upon individual intensity of infestation, and it must not be forgotten - and this a point insufficiently stressed in the literature generally - that coincident disease, as for instance malaria, will greatly modify the clinical picture. According to local topography incidence in and affect on the population has been found to vary. It may confidently be said that a Bilharzial area will be malarious, although the converse does not hold: and the influence of chronic malarial infection and concurrent dysentery (a too common association in the population under review) may alter or even mask the results of the disease proper. It is certain that coincident malarial infection has not been sufficiently considered in relation to splenic enlargement as occurring in the Bilharzial infections, particularly in that due to infection/

infection with S. mansoni. The relation is well displayed in the series of 128 cases graphed subsequently.

It is only within recent years that note has been taken of the existence of "carriers", whom themselves unaffected to appreciable extent, are yet a constant source of dissemination. Detectable only on survey, such cases were found to constitute a considerable percentage of the total infected in my series, and are probably more common than generally supposed. A review of the series suggests the possibility of a variable degree of developed immunity to infection - though it is here considered that apparent "immunity" may in fact be due merely to low intensity of initial infection and escape from repeated infection, rather than to any development of resistance to infection by elaboration of specific "antibodies".

In this connection it may be remarked that no use has been made of the dermal sensitivity and complement deviation reactions of Fairley and others. This much regretted omission is primarily due to lack of laboratory facilities for preparation of suitable antigen. Further, this reaction, being of group type, does not differentiate between infections with S. mansoni and S. haematobium and is of limited use in relation to a population of which almost all/

all adult males have, or have recovered from, infestation with S. haematobium. Attention is subsequently drawn to the high incidence of the latter condition, and also to the very different age incidence of the two infections. The attitude of the patient had also to be considered - the bulk being unsophisticated, but bigoted and ignorant peasants, among whom the appearance of even one marked intradermal reaction would almost certainly result in concealment of infection wherever possible.

Although it may be objected that the present paper deals with a limited population in a circumscribed area, the conclusions drawn are in the main applicable to any zone of endemic Bilharzial infestation. The numbers examined form a considerable series, and the distribution and age and seasonal incidence of the contrasting infections are of some interest. A personal relation with those infected has been, and still is, maintained.

For the above reasons, and to allow of detailed study and effective comparison between the disease in its vesical and visceral forms, the following study on a known population under continued personal observation has been submitted.

II. 1

The population of the Province is divisible, broadly, into three groups:

a. Settled Agricultural,

b. Nomadic,

c. Industrial and Urban.

The material of this paper is drawn mainly from surveys carried out in the first, and largest, group; of which the people inhabit ribbon Cultivation on either side of the river. The highest density of population is in the neighbourhood of large pump schemes, (MAP No. II) of which there are 28 in the Province, four being Government owned. They are grouped also in relation to "Basins" - large tracts of land lying below the level of the maximum flood peak of the Nile, and affording seasonal cultivation. It may be mentioned in passing that the Nile shows two flood "peaks" - one occurring about the middle of August, and the other, less marked, occurring in the first week of September. It will be shown that this rise and fall of the river exerts a direct seasonal influence on the incidence of the disease, and that the methods of agriculture - particularly of irrigation - affect the local density of infection.

It was not found possible, unfortunately, to determine/

determine incidence of Schistosomal infestation among the nomadic group, who live in the "back-lands", and take advantage of seasonal pools, "rain crops" and grazing.[3] From the few encountered it would appear that intensity of infection is high: and it is probable such nomads form a reservoir of infection. Coming as they do in the latter part of the dry season to the fringe of the riverain cultivated areas, they may re-establish infestation in a cleared area. In the absence of reliable information this group is not further discussed. They are not however considered as a menace to the settled population, among whom unfortunately a sufficiently high proportion of carriers exists to ensure perpetuation of infection. Mention is later made to this in considering the problem of elimination of infection.

The industrial and urban group - principally the artizans and officials of the Sudan Railways in Atbara, with the associated traders, - present few cases of infection. Infection is not acquired in Atbara, and cases are found either (a) to arrive infected, or are (b) infected or re-infected when on leave, or on duty in out stations. Only one case of infection in a European (British) official has been found - detected only in the course of routine examination while he was in hospital with malaria. (He himself/

himself was unaware of the condition, which must have been contracted while duck-shooting in one of the Southern Provinces.) The case is an interesting example of minimal infection, which had continued over three years without causing the patient any obvious discomfort. The ova passed were found to be non-viable, and no specific treatment was considered necessary. Attention will later be drawn to other such cases cited in the literature in discussion of symptomatology.

II. 2

From a glance at GRAPH No. III it is evident that distribution of Bilharziasis throughout the Province is not uniform. It is considered that the factors principally affecting incidence are, first, the method of irrigation, and second, the rise and fall of the river. Irrigation is carried out by -

a. Waterwheels (Sagia) drawing either from a well, or direct from the river (see photographs).

b. By "shaddoof" - a counter-balanced bucket, swinging on a beam.

c. By mechanically driven pump.

d./

d. The seasonal rise of the river is also used to advantage by the aid of a series of canals and dykes: by which large areas of level low lying land - the Basins formerly mentioned - are temporarily flood covered. Such Basins, though presenting a serious sanitary problem (from the point of malaria) do not appear greatly to affect the Bilharzial incidence.

In the course of this investigation it has been found that irrigation by water wheel, or by "shaddoof", does not tend to establish a focus of infection; but that the reverse is the case with pump fed cultivation. LEIPER[4] stresses the effect of change from basin to perennial irrigation, and consequent increase in human Bilharzial infection from 10 to 90% (in the Delta) - the direct result of the great increase in the snail population. Water drawn from the river by man-power, or by cattle driven wheels, is carefully husbanded, and strictly doled out in such manner that effective drying takes place between waterings, and is run through narrow channels giving a rapid flow: when supplied by pump, however, the native cultivator "over-waters" the crop, and if not strictly supervised, uses the secondary and tertiary canals, in which flow is sluggish, for all his domestic offices - thereby setting up a cycle of infection. Snails abound in the canals of pump schemes, but I have not yet found them in the sagia - ditches.[5]

Under optimal conditions breeding of snails may take/

take place throughout the year. HUMPHREYS[2] found that snails were established in irrigation canals 18 months after their opening and had spread throughout the scheme in 30 months.

There is no question but that snails are drawn into canals through the pumps; that is, canals even if treated, are promptly re-infested on occurrence of the first watering. I have found that in many areas, particularly in the cataracts north of Atbara, the silt may be composed of myriads of miniature snails. On no account should pumps draw other than from the free running stream.

In addition to the above noted primary factors, local topography may vary the incidence. The characters of river and river bank vary not only from district to district, but with the time of year. Where there are wide shallow stretches with numerous rocky pools, where there are grassy banks with weed covered pools, and above all, where long shallow creeks ("Kheiran") run some distance inland, there one will find a locally high incidence in Bilharzial - and malarial - infections. It has been suggested,[5] locally confirmed, that the incidence of Schistosomiasis mansoni is greatest about end-canals of pump-fed irrigation systems. (MAP NO. V.)

For convenience in reference, six "survey zones" north and six such south of Atbara have been marked out (MAP/

(MAP No. II). The principal Surveys referred to in this paper were carried out by the author in zones 1, 2, 3 north of Atbara, and in 1 and 2 south of Atbara.

II. 3

The seasonal incidence of the disease is of interest, marked in vesical (S. haematobium) infection, it is much less evident in visceral (S. mansoni) infection. This is related, in all probability, to seasonal exacerbation from fresh repeated infection with the more common S. haematobium; and possibly to drop in individual resistance following on continued labouring on the fields, to concurrent malarial infection, and in some instances, to malnutrition. During the fall of the river the number of cases coming up for treatment gradually increases (GRAPH NO. IV) and the greatest number present during the months of March, April and May. It may be, however, that records are appreciably affected by the demand for agricultural labour in preceding months, when the cultivator has no time to attend at his local dispensary. Others have drawn attention to this seasonal variation. ARCHIBALD[1] is of opinion that "infection is acquired during the months of the year when water is sufficiently shallow to/

to permit of a high concentration of cercaria. In the case of inland lakes and water courses, the seasonal incidence is from October to the end of January; in the Nile backwaters, the danger period is usually February to June." In the Province the rise is attributed to the pools and stagnant creeks left by the falling river. Figures show that it is in villages in the immediate neighbourhood of such that the most sudden and marked increase in infection occurs.

II.4. The snails most frequently found may be grouped under the following genera, Bulinus, Cleopatra, Melanoides, Planorbis, and Bythnia. Bulinus spp. and Melanoides spp. are widespread throughout pump schemes and Cleopatra spp. may be found almost anywhere in the Province, both north and south of Atbara. Planorbis is relatively uncommon, but has been noted in certain of the pump schemes. It is, however, noteworthy that Planorbis spp. have not been detected in any number in relation to the zones of visceral infection: and that in these areas Bulinus spp. and Melanoides spp. are the common varieties met with. Of the two, Melanoides spp. (Melanoides tuberculata) is considered the probable host. GOPSILL[6] in reporting on schistosomiasis in Nyassaland, gives Melanoides tuberculata as an intermediate host of S. haematobium, and of S. mansoni. ARCHIBALD,[1] on laboratory investigation, found that in the Sudan "Bulinus/

"Bulinus truncatus (including the species B. contortus, B. dybowskii, B. innesi) is the intermediate host of S. haematobium, and Planorbis boissyi, P. pfeifferi, P. alexandrinus and P. herbeni the intermediate hosts of S. mansoni." VAN DEN BERGHE[7] also implicates Melanoides as harbouring schistosome cercariae. M. nodicunata has been shown[8] to act as the intermediate host of S. haematobium, and ZAVATTARI[9] is of opinion that M. tuberculata may act as an intermediate host. (BAYLIS[10] gives a list of corrected names of vectors of human schistosomiasis, Melanoides spp. not being included; it is not claimed that the list embraces all vectors.)

I have obtained both furcocercous and non-furcocercous cercariae from Melanoides spp. and double infection of the snail has been noted: Cleopatra spp., common throughout the province, also have been noted as infected with furcocercous cercariae, and with non-furcocercous of two types. The non-furcocercous are broadly divisible as pigmented and non-pigmented - the pigment being disposed anteriorly in the form of "eye spots". The furcocercous cercariae are invariably non-pigmented. Snails of Planorbis and Bythnia spp. have also been found to harbour furcocercous cercariae. It is not, of course, suggested that the furcocercous cercariae noted are necessarily those of S. haematobium or/

or of S. mansoni, although presumptive evidence is strongly suggestive: and it is greatly regretted that pressure of routine work did not allow of detailed investigation of the morphology of the cercariae noted. (In this connection I am greatly indebted to Sir Robert Archibald, late Director of the Welcome Tropical Research Laboratories, Khartoum both for his advice on technique and for the use of media and apparatus, and it is hoped that it may be possible to return to this study in the near future.) Reference must be made to the studies of ARCHIBALD and MARSHALL on the morphology of the cercariae of S. haematobium and S. mansoni, and on unclassified cercariae obtained from molluscs in the Sudan.[11,12,13] Emphasis is laid on LEIPER'S postulate that "the identification of any species of cercaria is only practically possible by recovering the adult worm from experimentally infected animals." Further observations on the anatomical structure and identification of cercariae may be found under reference numbers 14, 15, 16.

As noted above, many snails show evidence of infection not necessarily of Bilharzial type - being intermediate hosts to trematodes ultimately parasitic upon animals, birds or fish. Extensive investigation of the gut and other viscera of cattle, sheep and goats butchered in Atbara/

Atbara failed, however, to show evidence of local infection of animals. ARCHIBALD[1] states that bovine schistosomiasis (S. bovis) is fairly prevalent in the Sudan; and numerous schistosome infections of cattle, sheep and goats are described in the literature. VEGLIA and LE ROUX [17,18] have described a sheep schistosome with terminal spined eggs and have named it S. mattheei. Goats, cattle and water buffaloes in India are found naturally infected with S.spindale[19]. S. bovis is noted[20] as parasitic in horses, donkeys and mules in Irak, and infections are essentially portal and intestinal: and it is further stated that "S. mattheei as described by VEGLIA and LE ROUX[1929] is indistinguishable from S. bovis as the latter occurs in Irak". VAN DEN BERGE[7] has noted S. bovis and S. mattheei in sheep, oxen and man.

II. 5

The possibility of aberrant animal infection in man is of great interest, and was fully considered in the course of surveys detailed in this paper, but has never been noted, and ARCHIBALD (loc. cit.), in much wider enquiry, has not found the "characteristic ova of S. bovis ... in the faeces or urine of natives of the Sudan." BLACKIE[15] implicates S./

S. mattheei as a parasite of man, and claims to have found this parasite in ten men: in eight of these diagnosis was made on the egg form, in two, the adults were found post mortem. In the latter, S. haematobium was also present. He is of opinion that S. mattheei, in this locality,* is not a negligible parasite of man. But reliable diagnosis cannot be made on egg forms and measurements, as such may show - and in material gathered on Survey did show - marked variation even in the same individual (PLATE, No.XI.). In support of this, reference may again be made to the findings of MacHATTIE and CHADWICK[20] (loc. cit.) who have shown that a diseased S. bovis may produce eggs corresponding in shape and size to those of S. haematobium. The egg illustrated by BLACKIE as that of S. mattheei is indistinguishable from that of S. bovis. They illustrate consecutive eggs from a uterus, one S. bovis-shaped, the other S. haematobium-shaped, and immature eggs of S. haematobium from human urine showing elongation and spindle shape.

"About 1% of females show many typically shaped S. bovis eggs in utero and in the same uterus one or more typically shaped S. haematobium eggs having the same measurements as the egg of this parasite in the human subject..."

"Approximately 0.2% of females contain solely eggs of S./

* i.e., RHODESIA.

S. haematobium shape and measurement. Such females are only distinguishable from this parasite in man in that the vitellaria occupy approximately one half of the total length of the female."[20]

MacHATTIE and CHADWICK are of opinion "that BLACKIE has failed to produce any convincing evidence in support of his statement incriminating cattle and sheep as constituting a potential reservoir of human urinary schistosomiasis."

Spurious parasitism with S. mattheei is noted by BLACKIE (in the same Survey as that implicating man as a host for this schistosome), ova being noted in a case where a half raw ox-gut had been consumed on the previous day.

FISHER[21] in the study of intestinal schistosomiasis in the Stanleyville District of the Belgian Congo holds that the schistosome implicated forms a new species, schistosoma intercalatum. But "none of the morphological characteristics of this parasite, apart from the ovum, are such as to enable it to be differentiated clearly from either S. haematobium or S. bovis".

As pointed out above, however, neither size nor shape - in which great variation has been recorded in the present Survey - are reliable criteria, and of themselves do not justify the labelling of a new species. One might as well classify/

classify farmyard fowls in the shape, size and colour of their eggs! That the lesions produced were located between anus and pelvirectal junction is of interest, but terminal spine ova in and from such areas have not uncommonly been noted in S. haematobium infections. Moreover, SCHWETZ and BAUMAN[22] found that in the Stanleyville district S. haematobium is more often the cause of intestinal infection than is S. mansoni. REYNAL[20] has collected references as to aberrant localisations for schistosomes in man: ova of S. haematobium have been found in stools, alone and in conjunction with those of S. mansoni: and in the urine coincidentally ova of S. mansoni have been found, both singly and in conjunction with those of S. haematobium in the stool. (BRUMPT[24] has collected the literature of cases showing ova of S. mansoni in urine, and ova of S. haematobium in the faeces.)

In spite of co-existant S. haematobium and S. mansoni infections within one agricultural scheme in this province double infection has not been noted. Yet only a few Kilometres distance intervened between villages showing, in one instance vesical and in the other intestinal infection. Even among the children (commonly the principal group in vesical infection) in the known zone of rectal infection, no case has yet been found to show co-incident urinary and faecal infection.

II. 6.

It is suggested that this quite peculiar feature is due to variation in snail hosts, and this variation in turn to the chemical reaction of the water irrigating the affected zone. The chemical characteristics, it is considered, may render the water suitable only for snails of certain species, thereby limiting the spread and propagation of the optimum intermediate host; and may even affect the motility and penetrative powers of miracidia and cercariae alike. In point of fact, the water of adjacent irrigation zones has been found to vary in pH., and in the laboratory I have found that snails are very susceptible to the reaction and oxygen content of water. Alkaline waters may harbour Bulinus, but acid waters do not favour its development[25].

HASSAN[26] has found miracidia of S. haematobium and S. mansoni very susceptible to alteration in pH. The following percentages of chlorides killed in 4 hours: Na,0.9%; K, Mg., (NH_3), 0.7%; Ca., 0.5%. The relation between reaction of water and development of intermediate host and parasite has not sufficiently been studied; and it is much regretted that no facilities were here available for a more detailed study than that undertaken.

II. 7.

Snails are found most abundantly in the weeds and mud fringing secondary and tertiary canals. Temperature affects the rate of breeding, but breeding takes place throughout the year. The spread of snails is effected in the main by irrigation, though it is difficult to account for the infestation of scattered seasonal pools. Carriage of snails and eggs may be effected by birds, as suggested by HUMPHREYS[2]. The eggs are found on floating and submerged vegetation, which also affords harbourage for snails, as does the brickwork of bridge and "escapes" and posts or stakes in the canal. Palm fronds immersed in the canals have been used as traps. The canals may be cleared of snails by (a) drying and re-excavation, (b) treatment of the water in partially emptied canals with Sizoline or Ialine (Commercial Carbolic Acids). Both methods are here in use. (Cf. Section VI.)

Reinfestation of cleared irrigation zones takes place from the river, via pump and main canal. The "trapping" of canals has conclusively shown that snails are constantly thrown into the main canal during pumping and that they may be found settled in mud and on vegetation even at one Kilometre above intake. Snail concentration becomes progressively higher as one approaches the terminal stretches/

stretches of secondary and tertiary canals; and breeding is most marked in the sluggish water of these terminal ditches.

LEIPER, cited by KHALIL[30] has suggested that "further investigations are required to determine whether there is not an upward migration from the smaller to the larger canals at certain times of the year." In so far as this province is concerned it is positively affirmed that no such migration takes place; and that, though in certain circumstances snails will float up to and actively move on, the surface layer of the water, the probability is against migration in any area. As noted above the influx of snails is from the river and main canal, though conditions for multiplication are optimal in the terminal canals. A sufficient distinction is not always drawn between <u>infestation</u> of river and canals with snails and <u>infection</u> of these localities with bilharzia.

The main sources of infection of man are the small canals and drains, in which snails abound. They are found in great numbers in the swirl pits at the proximal part of the tertiary canals. Urination and defaecation into these pits and the supply canals is of frequent occurrence, in spite of prohibition. The pits ultimately contain stagnant water which has to be bailed out (by hand) as an antimalarial measure, and this completes the infective cycle.

The/

The free running water of the river is not a source of infection, but infection may occur (as previously noted) where the falling river leaves numerous stagnant pools among the grassy hollows of the bank. Snails swarm in the mud and on the vegetation of such pools. During low river, snails and their "eggs" may be collected from the main stream, especially where sluggish, by the use of traps of brush-wood, or palm fronds. For this reason, sanitary approval is given only to irrigation schemes in which the pump draws from the main stream.

Sagia (waterwheel) irrigation is comparatively free from danger, as snails do not swarm in the sagia pits or wells, and will not withstand the sudden and repeated drying of the narrow quick flowing sagia ditches.

The river though the source of the intermediate host is not a source of bilharzial infection.

JANE[27] has reported on the distribution in a major pump scheme and during the investigation was associated with the writer, who has repeated his observations and entirely agrees with his conclusions. It is considered that but few snails survive the drying out of canals, and that they play little or no part in subsequent re-infestation of new flooded canals. Snail "eggs" will not withstand drying, even for a brief period.

It has been suggested by GOPSILL[6], JANE (loc.cit.) and/

and others that snails may survive the dry period (of winter closure of canals) by burrowing into the soil while it is yet moist. GOPSILL has found Melanoides tuberculata at a depth of 5 feet in the mud of a dried river bed: but it is considered that such findings can be explained by silting, and by the cracking of drying canals and stream beds, the snails then following the gradual subsidence of water into these cracks. It has not proved possible to corroborate these findings locally, in spite of repeated and extensive examination of dried-out canal beds. Although a few snails are found buried in ooze at depths of two to three inches, it is obvious that they have been covered by silt and no evidence of "burrowing" has been substantiated. Excavation to a depth of two feet also failed to produce live snails.

BARLOW[28] found that in Egypt Planorbis boissyi and Bulinus contortus survive the dry period by production of an epiphragm of dried slime. He obtained live, but non-infected, snails from a canal bed which had been dry for 30 days. (The production of this epiphragm in non-operculated snails has been the subject of recent interesting articles[29] in the Illustrated London News.)

NOTE: A LIST OF ARTICLES BEARING ON THE SECTION FOLLOWS OVERLEAF.
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III. 1.

SURVEYS.

The surveys on which this paper is based were carried out in February and March of 1933 and 1934, in areas Nos. 1, 2, 3, North, and 1, 2 South, (as indicated on May No. II).

In the northern zones 1083 persons were examined and 69 (6.4%) were found infected with S. haematobium. No infection with S. mansoni was at that time noted. (Two cases of intestinal Bilharzia have since been found, but in each case infection was found to have occurred during work in the Abu Selim area). In the Southern zones 1 & 2 1507 persons (all males) were examined, and 19 (1.3%) were found to have urinary (S. haematobium) infection.

In Zones 1 & 2 endemic intestinal infection was found. Attention had first been attracted to certain villages in these areas (Abu Selim, El Ogar, Umerab and Timerab, in Zones S1, and Rau, Fereikh and Aliab in Zone S2). In Zone S1. 310 persons were examined and 109 (35.1%) were found to show the ova of S. mansoni in their stools. In Zone S2, 167 were examined, and in 93 (55%) the ova of S. mansoni were demonstrable. Distribution of infection throughout/

throughout the villages is by no means uniform and is most marked in such as are related to subsidiary and end-canals. (Attention has already been drawn to the Snail population of such canals). It is of interest to note that the village of Hassobalab, which is not in intimate relation to any end-canal showed no infections among 70 persons (males) examined: and a further point of great interest is that no cases of S. haematobium infection were detected among children in Zone S1.

III. 2.

Analyses of the cases of urinary bilharzia showed by far the highest incidence of infection to occur in villages adjacent to narrow creeks, and collections of stagnant water. Incidence varied from 1.6% to 50% of persons examined.* Sagia (water wheel) cultivation had no relation to incidence. According to area from 85.7% to 92% of infected persons were under 21 years of age and the highest incidence of urinary bilharzia occurred in the "5 to 15 yrs." age group. In brief, urinary bilharziasis is primarily a disease of children (GRAPH No. VI.) Few cases were found over 25 yrs. of age. Similar findings are later recorded by DIXON[31] (in Katanga) who found the highest incidence between/

* According to Village.

between the ages of 10 and 15 years, and no infections among persons over 45. He ascribes this to the comparatively rapid production of immunity. In this, however, I am not disposed entirely to concur, holding that persistance of infection - and the grave complications found in higher endemic areas, such as the Nile Delta, - are dependent on the initial intensity of infection, and on repeated infections. CHRISTOPHERSON[32] has drawn attention to the danger of neglecting initial infection in the child, and to the results of added infection in adult life.

No gross lesions have here been noted as following on infection with S. haematobium. BLACKIE[15] noted in the course of Survey in Southern Rhodesia that gross clinical manifestations were rarely associated with (the local) urinary bilharziasis; but gives a warning, in that 12 post-mortem examinations showed a greater degree of tissue injury than clinical examination had suggested. He was, however, classing infections according to the egg count in the urine: and the numbers noted cannot be accepted as a guide to the actual worm load. PIJPER[33] is of opinion that in most adults the damage amounts to little more than periodic loss of blood, but that in children infection may have serious results. Incidentally, he notes that most local cases occur in the young.

A/

A sharp contrast, both in age incidence and clinical finding, is given by infection with S. mansoni.

This may affect all members of the population, incidence rises with age, and the resultant lesions are both extensive and severe, and especially involve liver and spleen. Here again, intensity of infection is considered the decisive factor in the production of the ultimate picture. Some 15% of persons examined present no obvious feature, and can only be detected on survey. These cases may complain only of slight and infrequent attacks of diarrhoea - for blood may never be obvious in the stools. GOPSILL[34] has stressed the value of the history of obvious dysentery of 7 days duration, even some years back, as indicative of the onset of bilharzial infection. Such history is here of no value, dysentery being of common occurrence among cultivators.

All cases cannot be diagnosed on purely clinical grounds. The number of eggs noted in a faecal smear bears no direct relation to the severity of symptoms, and in about 15% of cases were present without clinical manifestation.

Splenic enlargement (in Zones S1 and S2) is of common occurrence. Nor can this splenomegaly be ascribed solely to coincident malarial infection. It is not the invariable rule; but the average history suggests that all cases/

cases ultimately show it, though infection may persist for months or years before enlargement occurs. (Further reference to this splenomegaly is made under the head of Clinical Study.) The incidence and degree of splenomegaly varies greatly according to locality. In Zone S2, 35% of infected persons show splenomegaly in contrast to 75% in the Abu Selim Area, and individually the increase is less extreme.

III. 3.

In investigation of S. mansoni infections the method of examination was by thick faecal smear. The portion of stool was emulsified with water, and such quantity placed on the slide as to give a "ground glass" appearance when the coverslip was added. No stool was considered as "presumably negative" until three separate portions had been emulsified and examined. Owing to lack of staff and time, concentrative methods could not be applied: and it is, of course, obvious that the method adopted cannot have displayed all infections - it is sufficiently amazing that the ova in 60% of cases were detected on initial examination, and that in one village (Abu Selim) 67.3% of persons examined were found infected!

A sedimentation-concentration method has been elaborated by HOFFMAN, PONS and JANER[35], and this will be/

be used when necessary in hospital practice.

In the investigation of S. haematobium infections, the urine of all cases was tested for Albumen. If Albumen, or obvious blood, was present the urine was then rapidly centrifuged and the deposit examined microscopically. In all cases in which blood was present in the urine ova were found, though not necessarily on the first examination. 25 adults and 7 children (all males) showed albuminuria only, without blood or ova. VAN DEN BERGHE[36] draws attention to the occurrence of ova without coincident albuminuria (in 22 out of 73 cases); and to the occurrence of albumen, pus and blood in 11 cases, in the absence of ova. While the latter is not improbable - and I have noted its occurrence - I have yet to find a case in which the passage of ova was not associated with albuminuria. RAMSAY[37] has elaborated an equation to evaluate the significance of abnormal constituents in the urine in areas of endemic S. haematobium infection, and to show that the "Coefficient of Association" is high (0.93), and that haematuria is an index of infection:

$$C = \frac{(AB)(ab) - (Ab)(aB)}{(AB)(ab) + (Ab)(aB)}$$

A = ova present. a = ova absent
B = Blood present. b = Blood absent C = coefficient.

It has not yet proved necessary to resort to this mathematical exposition for demonstration of infection.

The/

The figures given for urinary bilharziasis relate exclusively to males. It is alleged that the incidence among women and girls is low. (The figure of 0.85% infection among females as against 11.5% among males is quoted in the Annual Report (1932) of the Director, Sudan Medical Service). I have, however, no comparable figures for this Province: but admissions to hospital suggest that the ratio is rather higher.

It must not be forgotten that a very large proportion of S. haematobium infections are latent, or relatively without symptoms. It has been suggested[38] that such latent infection may be made evident by injection of Emetine (0.059gm - 0.19gm) with subsequent re-examination of the urine some five hours later. I am doubtful of the value of this procedure, and no occasion has yet occurred on which it could usefully be employed.

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IV. 1.

CLINICAL STUDY.

In its essential pathology schistosomiasis is considered by the writer to belong to the granulomata: the primary tissue reaction being that of fibrous proliferation with the development of multiple scattered granulomata, endarteritis, endophlebitis, and calcification. The diverse clinical appearances are dependent upon intensity (and repetition) of infection, and upon vascular communications: and are further complicated by septic infection and intercurrent disease. As in the case of tuberculosis distribution of lesion may be localised or widespread; as in syphilis there is a late tertiary period in which the condition is no longer amenable to treatment, irreparable tissue damage having resulted from continued toxaemia and gross fibrotic deformity of essential organs. It is not desired to carry analogy too far: toxic reaction is in general less evident than proliferative reaction.

As with other diseases, the ultimate result is to a certain extent dependent upon that indefinable quantity, host resistance. Consideration of this factor leads immediately to consideration of acquired immunity. Such acquired immunity/

immunity has been circumstantially established, but is never complete. And here again is a further point of resemblance between the schistosomiasis group and other granulomatous diseases. The reaction of the host to infection is governed not by this imperfect production of immune bodies in response to circulating toxins, but by intensity of infection and extent of tissue damage. Should he acquire a light infection and escape repeated infection, recovery is certain, and the disease may even be latent: a heavy infection, repeated, and possibly complicated by sepsis will inevitably produce all manifestations of the disease in its most extreme form.

On this conception of the disease in terms of body, or tissue, response, clinical description is reviewed under Primary, Secondary and Tertiary heads, each being related to a definite stage in the progress of the condition. So far as I am aware this classification has not previously been advanced, although BRUMPT and CHEVALIER[39] (in experimental work with schistosome infections in mice) classified lesions as nodular and toxic, and found that tubercle formation in the mouse was identical with that caused by Myco. tuberculosis in man. They noted further that no difference in bodily reaction was determinable according to the species of schistosome employed. The proliferative lesions occurred in relation to egg deposition; the toxic reaction in relation to/

to the adult worm, being indicated by necrotic foci or endophlebitis.

In support of this, BOURGUIGNON[40] has found that deposition, or rather fixation, of eggs in the omentum is followed by the development of tubercles with concentric fibrosis, fibroblasts and giant cells being in close relation to the ova. HOEPPLI[41](in the study of lesions produced by S. japonica) relates tissue change to actual secretion from the ovum: in the vicinity of this secretion appearances closely resemble those in the early tuberculous lesions - it is surrounded by a zone of degenerate polymorphs, or even by necrotic tissue, then by a band of epithelioid cells and peripherally by round cells, plasma cells and large mononuclears.

IV. 2.

Broadly speaking, the following classification is applicable to the Clinical Study of any of the schistosome infections:

PRIMARY (INVASIVE STAGE, assoc. with: Skin rashes, dermatitis
(followed by
PHASE (INCUBATION STAGE; " " : fever, malaise,
bronchitis, asthma,
urticaria.

SECONDARY/

SECONDARY PHASE	(DEFINITIVE STAGE; assoc. with:	frequency, strangury;	
		or	dysentery, fever, lieno-hepatitis, enlargement of liver and spleen, anaemia.
TERTIARY PHASE	(TERMINAL STAGE; assoc. with:	Profound anaemia, hepatic cirrhosis: splenomegaly, Ascites, Emaciation: cachexia, "Typhoid state."	

IV. 3.

In its primary and secondary phase the disease is amenable to treatment, and cure may be achieved. In the tertiary phase only temporary improvement can be expected, and specific treatment with Antimony Salts may be dangerous owing to much impaired liver function and to general debility. (Photograph,No. X shows a patient in this terminal stage.) In urinary infection (S. haematobium) spontaneous cure may take place: but intestinal infection (S. mansoni) is less liable to arrest, probably on account of the widespread vascular communications facilitating dissemination of ova.

There/

There is no agreement as to the incubation period of the disease - that is, the period between invasion and appearance of the first "definitive" symptoms - intervals of twenty days[42] to some months having been recorded. In one case of undoubted initial infection in my series, five weeks only had elapsed between arrival in the district (Abu Selim) and the appearance of dysentery. Examination of the stool at this time showed abundant ova of S. mansoni.

Symptoms and signs of invasion vary. Examination of the literature[42,43] suggests that such may be more obvious in affected Europeans than in dark skinned races. Itching, followed by an erythematous or more usually papular, and subsequently pustular, eruption is the sequel to penetration by the cercariae.[42,43]. This reaction is not peculiar to the cercariae of S. mansoni and S. haematobium but has been noted (1928) by CORT[44] in relation to penetration by C. ocellata La Valette 1855, and was again reported (1930) by TAYLOR and BAYLIS[45]. In my case series (entirely native) no instance of papular or pustular eruption was noted or complained of: but some (20%) complained of itching of the legs and arms after work in the flooded fields, and it is of interest to note that the majority of the complainants worked in close relation to the "take-offs" from the secondary canals. No specific eruption could be associated/

associated with the itching.

The invasive period, of indefinite duration, is associated with urticarial eruptions, cough, bronchitis, malaise and fever. JEWELL[42] has noted cyanosis and high eosinophilia. It is only recently that attention has been directed to the frequency of pulmonary symptoms at this stage of the disease. (A history of troublesome cough could frequently be obtained in my case series. In one instance, a boy aged 8, with S. haematobium infection, a severe bronchitis had occurred shortly before the onset of urinary symptoms, and the sputum had been faintly "rusty" in colour. There was only slight fever: and some patchy consolidation: he had contracted repeated bilharzial infection. Under treatment with Sodium Antimony tartrate improvement was rapid.) LE ROUX[18] found marked and constant changes (pigmentation and tubercle formation) in the lungs of sheep infested with S. mattheei, and remarked on the paucity of reference to lung lesions in the literature. TURNER[46] found that in 64% of 28 cases (S. African) dying of pulmonary disease ova of S. haematobium were present in the lung, while in 11 dying of other complaints the percentage was 33. SUAREZ[47] points out that the frequency of bronchitis associated with, or preceding, the intestinal symptoms of S. mansoni infestation has been remarked by many/

many authors. SULLIVAN[48] records a sporadic case in which there was a pertussis-like cough and strangury, the urine being found to contain terminal spined ova. Examination of sputa from cases - especially in those suspected of early initial infection - has so far failed to show ova or immature worms.

Malaise and fever are frequently complained of, particularly in those infested by S. mansoni. But it must be remembered that the majority of such were also infected with malaria. PONS and HOFFMAN,[49] however, record the occurrence of marked fever in the "invasive" stage in 7 cases of schistosomiasis mansoni. This fever was often associated with bronchial irritation or apical infiltration. In the course of treatment, I have noted the occurrence of irregular fever, frequently associated with the ingestion of Antimony Salt; and found that with preliminary antimalarial medication the numbers exhibiting febrile symptoms under treatment diminished. Acute malarial relapse following antimony tartrate injection has been noted in my own series. GIRGES[50] considers that there is a definite febrile or toxaemic period in what he terms the "intestinal" type (as opposed to "hepatic") of Schistosomiasis mansoni. This is of acute or insidious nature, with invariable enlargement of liver and spleen, with anaemic and leucocytosis, and lasts for three to six/

six weeks. I have not noted this fever in relation to Schistosomiasis: but I have constantly observed that antimalarial medication exerts an affect not only on the so called bilharzial fevers, but on the accompanying splenic enlargement in all stages of the disease. It is admitted that the majority of my series were infected with malaria, and that few occasions offered for the study of schistosomal infestation in the early stages: it is not clearly stated in GIRGES' report that chronic malarial infection had been positively excluded. KHALIL[51] and HACK,[52] among others, have reported on febrile reaction following administration of tartar emetic.

Following upon the development of infestation, and the maturation and localisation of the parasite in its definite site a further train of symptoms of more obviously schistosomal origin follow. They coincide with the appearance of ova in urine or in faeces according to species as a rule - though as noted in a previous section S. haematobium may produce dysenteric symptoms and bowel lesions, while S. mansoni is not necessarily restricted to a purely intestinal locale. Frequency, strangury and haematuria in the one case, and dysentery and lienohepatitis in the other, are the obvious concomitants of developed urinary and intestinal schistosomiasis.

But they are not necessarily present to extreme degree/

degree, and in a certain proportion may not be evident at all. Locally, this has been found the case much more commonly in urinary than in rectal schistosomiasis: and this condition, in which eggs in considerable number may be voided notwithstanding absence of clinical symptoms, must be distinguished from latent disease, in which ova are from time to time entirely absent from the excretions, in that the former are active carriers of infection. Even in such, however, the egg output from day to day necessarily shows great variation.

Some 30% of children infected with S. haematobium and 15% of adults infected with S. mansoni were found to show no obvious signs of disease, the condition being detected on routine survey. In the latter instance, however, a story of intermittent dysentery, slight and infrequent, could invariably be obtained on enquiry. This agrees with the experience of GOPSILL[6,53] in Nyassaland: but it is doubtful if such history should be considered of such proved diagnostic importance as he suggests.

Involvement of liver and spleen is much more evident with infestation with S. mansoni than with S. haematobium This is considered not as a specific action of the parasite per se, but as the result of the vascular communications between these organs and the mesenteric venules: they are involved by the passive conduction of worms and ova through the portal/

portal and systemic circulations. Both liver and spleen are "filters" in the circulation. The lung, to a less obvious degree, shares in constant involvement. (Diagram No. IX constructed from the papers of Faust, Hoffman and Jones shows the route by which larval forms reach definitive sites: and mature forms and ova may be similarly distributed.) In addition to the more commonly favoured sites of accumulation, eggs have been reported from Spinal Cord (S. haematobium and S. mansoni)[54,55] conjunctiva,[56] lung[18,47,57] associated with generalised pulmonary endarteritis obliterans - and involvement of supra renals has been suggested by development of pigmentation and emaciation.[58] Diabetes has been caused, though rarely, by schistosomiasis. ERFAN[59] cites a case reported by DAY and describes another (of S. mansoni infection) in which cure was accompanied by a fall of urinary sugar from 10% to Zero.

From this, it is evident that schistosomiasis is not to be considered as a disease purely of the intestinal and genito-urinary systems, and so narrowly partitioned as "urinary", "hepatic" or "intestinal" - such divisions, convenient as they may be, being indicative only of a particular phase of the disease, and centering attention on the later and frequently incurable stages.

Advancing anaemia marks the transition of the secondary/

secondary into the early tertiary stage. It was noted in my cases of S. haematobium infection, but was both marked and more constatly present in the S. mansoni infections, and advanced with the progressive involvement of liver and spleen. The anaemia may become severe, but is always of secondary type. Time did not permit of a detailed study of the blood picture in my own cases, but there is no reason to believe that such would not agree with the findings recorded in the literature. ERSPAMER[60] has noted that anaemia is not a constant feature of vesical infections. GIRGES[50] has noted "chlorotic anaemia" both in his "intestinal" and "hepatic" types of schistosomiasis mansoni. The fully developed picture is that of profound secondary anaemia, frequently of the toxic aplastic type. In my own series the contributory factors of chronic malaria, and (less frequently) amoebic dysentery, in the production of anaemia are not overlooked: and it is considered that malaria may play a considerable part in the production of anaemia in cases infested with S. haematobium.

Analysis of my case series showed that, while anaemia (in Schistosomiasis mansoni) was almost constantly present, the degree of involvement of liver and spleen could not be directly related to the duration of symptoms. This is considered to be due to the intensity of infestation initially contracted, and not to any specific peculiarity of infective agent/

agent or degree of resistance or "immunity" on the part of the host. As an example, one case gives a history of over two years disturbance but presents no obvious gross lesion as yet, though moderate splenomegaly (probably malarial in origin) and slight anaemia, and diarrhoea with passage of blood and mucus are present: on the other hand, a case giving a history of about 7 months sojourn in the neighbourhood shows gross splenomegaly, diminution of liver and severe anaemia with commencing ascites, but without any severe dysenteric symptoms. This notwithstanding that both show ova in the stools.

The numbers of ova passed from day to day not only vary, but bear little or no relation to the severity of symptoms. In three cases in the late tertiary period of Schistosomiasis mansoni eggs were not detected until 8, 10 and 21 days respectively after the admission to hospital, although repeated daily examinations of faecal smears had been carried out.

Lieno-hepatitis was an almost constant finding in the late secondary stage, and excellent results were obtained when treatment was initiated at this stage. Once marked enlargement of liver and spleen has taken place, results are not so wholly satisfactory. 71% of all persons examined in the first survey showed splenomegaly: but among those/

those infected (35.1% of total) 75.6% had enlarged spleens. For the second survey (Zone S2) the corresponding figures were 25% of total examined and 35% among the infected group. (Graph No. VII shows the enlargement in its incidence according to age-group). It was found that splenic enlargement diminished in 85% of cases after the administration of 9 gr. to 11 gr. of Sodium Antimony Tartrate. 70 cases were subsequently taken at random and given Atebrin and Quinine (1 tablet Atebrin and 5 gr. Quinine Sulphate t.d.s. for five days) and this was found to result in a further diminution of spleen size.

Dysentery, however, continued in intermittent form in many cases both during and for some time after treatment, though attacks were less severe: and in a few it has been persistent. On examination of 32 persons who had completed treatment 6 months previously, 28 were found still to pass abundant ova. The numbers per smear were, however, less than formerly and in all but two cases the ova did not appear viable, - the miracidia being shrunken, granular and showing occasional vacuolation, and the inner envelope retracted. In all cases the general condition was greatly improved by treatment, and return to work has been the rule.

The outlook in Schistosomiasis mansoni is always grave/

grave. Fortunately, in so far as Berber Province is concerned, a much lighter view can be taken of S. haematobium infections. In the latter, no case has yet been found to show the gross lesions so commonly reported from Egypt: and spontaneous cure (which need not be looked for in rectal bilharziasis) results in the great majority of cases. Attention has already been drawn to the marked difference in the age-incidence of S. mansoni and S. haematobium infection.

The associated conditions commonly noted in conjunction with urinary schistosomiasis in this Province are Splenomegaly and Anaemia: but as formerly emphasised, it is difficult to assess to what degree they are the result of concomitant malaria, since almost all cases in which they occur have chronic malarial infection. A few cases showed evidence of hepatitis, the liver being enlarged and tender. In one case, in which abundant ova (S. haematobium) were being passed in the urine - incidentally without obvious haematuria - very marked enlargement of liver and spleen was present. The late results of Schistosomiasis mansoni present a very different picture. Following upon progressive hepatic cirrhosis, ascites appears, and may reach an extreme degree. (In the case shown in Photograph No. X the abdomen was tapped at 14 day intervals, and 300 oz., 288 oz., 242 oz., of fluid were/

were withdrawn on respective tappings.) DAY[61] is of opinion that the splenomegaly is dependent on venous stagnation, and may be influenced by involvement of the splenic vein. It is not proportional to the degree of hepatic involvement. GIRGES[62] gives two years as the expectation of life after appearance of ascites: while it has not yet proved possible to confirm this locally, appearances seen fully justify a grave prognosis at this stage.

No note on splenomegaly in association with Bilharzial disease would be complete without mention of GIRGES' papers[63, 64] on the etiology of Egyptian Splenomegaly. He holds that "Egyptian splenomegaly is a disabling endemic parasitic syndrome caused by male Schistosoma mansoni infestation of the liver and portal vein...There is very little or no alimentary disturbance or implication of the gut, the brunt of the infection being inflicted on the viscera. Thus, besides the febrile symptoms, anaemia and other blood changes there is at first a definite hepatic enlargement and an increasing hypertrophy of the spleen and abdominal lymphatic glands, and later on there occur hepatic cirrhosis, marked splenomegaly and ascites."

No such cases have come under my notice: some few have closely resembled the syndrome described, but all have in time been found to pass ova in the stool. Day (loc. cit/

cit.) ascribes the condition to retention of ova in the solid viscera, with ultimate periportal cirrhosis, and splenic enlargement. Such retention, and progressive involvement, is considered the primary cause of the Lieno-hepatic changes as they occur in the present series.

Emaciation rapidly developes subsequent to the appearance of ascites, and may proceed to a toxic cachexia. Death results from pneumonia or is ushered in by terminal diarrhoea, or may occur suddenly from cardiac failure.

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53. GOPSILL (L.W.) Schistosomiasis in Nyassaland and its Treatment by means of Fuadin. JL. MED. ASSOC. SOUTH AFRICA 1931. V. 7. pp. 222-223.

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58. KIRKLAND (H.T.) A case of Schistosomiasis presenting Some Unusual Features JL. TROP. MED. and HYG. 1928. XXXI. 7 pp. 78-79.

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62. GIRGES (R.) Prognosis of Schistosomiasis mansoni. JL. TROP. MED. & HYG. 1930. XXXIII 5. pp.65-68.

63. Do. Causation of Egyptian Splenomegaly by Male Schistosoma mansoni alone. JL. EGYPTIAN MED. ASSOC. 1931. XIV. 5. pp.282-304 35 refs.

64. Do. The Aetiology of "Egyptian Splenomegaly". JL. TROP. MED. & HYG. 1932. XXXV. 6. pp. 86-90: 7. pp. 99-105. 37 Refs.

V. 1.

TREATMENT.

It is considered that Sodium Antimony Tartrate is still the drug of choice in treatment of Schistosomiasis in this Province; and analysis of recent literature has not disturbed this conviction. For routine administration the following method and dosage have been found successful, and free from complication:

Initial dose: gr. ¼, in fresh solution, intravenously:

Increase by gr. ¼ till maximum of gr. 2 is given as single dose:

The total amount given, for an adult, is gr. 24 - gr. 28.

The course may be repeated after one month interval.

The first 3 injections are given daily; and subsequently injection is given on alternate days.

The period of time involved in treatment is a difficulty, and makes for slight irregularity in attendance especially among cultivators: but the introduction of an individual card system, (by which date and time of injection, and does given are recorded) has facilitated supervision, and relatively few toxic reactions have been noted. Nothing more serious than cough, nausea, bone pains and slight fever have occurred, and could be alleviated with mild sedative or aspirin in the great majority of cases. In a few instances -

of/

of debilitated patients receiving treatment in hospital - glucose and glucose saline, have been used as adjuvants, and morphine hydrochloride gr. ¼ has been required for the control of cough.

It will be seen that the above method, in its essentials, follows the recommendations of CHRISTOPHERSON[65,68]. Careful examination for, and treatment of, concomitant disease is carried out in all cases. Patients from certain districts receive routine Antimalarial treatment (Atebrin & Quinine) prior to commencement of Antimony injection even in absence of signs or symptoms of malarial infection. In the latter part of the course, specific treatment is supplemented by Iron and Arsenic medication, (including the occasional administration of "914"); and in certain instances Calcium lactate and alkaline glucose saline have been used with good effect.

V. 2.

CHRISTOPHERSON[68] stresses the importance of the blood picture - particularly of eosinophilia - as indicative of the patient's response to treatment. "Eosinophilia, however, has/

has no relation to the number of parasites present - it registers the effort of the organism to combat the parasite." It is stated that eosinophilia increases as treatment proceeds and if present "three months after the termination of antimony injections argues the patient uncured". This interpretation agrees with the experimental findings of OZAWA[83], who found that eosinophils disappeared rapidly when the disease became grave. CAWSTON[69] draws the opposite conclusion, being of opinion that persistence, or increase, of eosinophilia during treatment is indicative of persistence of parasites. Observations on in-patients of the present series support the view taken by Christopherson.

V. 3.

HACK[52] has drawn attention to the occurrence of delayed toxic reaction following Antimony therapy, and points out that his case was associated with use of the potassium salt. The sodium salt is locally employed, and great care is exercised to ensure that all solutions used are freshly prepared. No instance of delayed toxic reaction has yet been brought to my notice. (One apparent case of toxic reaction, cited previously, was found to be suffering from an acute relapse/

relapse of Malignant Tertian Malaria.)

It has been suggested[70] that dosage should be regulated according to age and severity of infection rather than by patient's weight: for it is claimed that experience has shown that those with heavy infection tolerate heavy dosage. Experience in this Province shows precisely the reverse, and I am in emphatic disagreement.

Fouadin, ushered in in 1928 with rather extravagant claims, is now no longer advised with the same enthusiasm. Recommended by KHALIL and others in Egypt, it appears to have little advantage over sodium or potassium antimony tartrate. There is considerable diversity of opinion as to its toxicity. CAWSTON[71] reported favourably on antimosan (of which fouadin, or "neo-antimosan", is a modification having 1% more tri-valent antimony), but noted (1929) that the use of Fouadin "frequently results in severe hepatitis"; and again[72] (1933), that "fouadin often produces late vomiting and insidious symptoms of hepatic disturbance". ORENSTEIN[73] and GOPSILL[74] take a more favourable view and the latter claims that a more rapid cure may be obtained with fouadin than with tartar emetic. MACIEL[75] notes toxic action on the liver and comments on the need for careful supervision during administration: he finds that it is well borne by children.

V. 4.

Expense has prevented trial of fouadin on a large scale in my series. Given to six patients under supervision in hospital it showed no advantage over routine treatment, but gave no toxic reaction.

Following on the favourable reports of TSYKALAS[79] and REIGL[76], FAIRLEY (cited by former), PELTIER and RAYNAL[77] and others[78] on the use of emetine, this drug (emetine hydrochloride) was used on selected cases; both alone and combined with Sodium Antimony Tartrate in alternate injection.

STAUDT[80] claimed (1931) excellent results from administration of 4, 6 and 8 cgm. of emetine followed by 6, 8 and 8 cgm. of tartar emetic: but in the present instance results were in all cases disappointing.

BOUSQUET[78] prefers emetine to tartar emetic, antimosan or fouadin: but for out-patient treatment methylene blue. This last is said to be very popular with the patients!

FISHER[81] reported (1934) promising results with Acriflavine, though actual cure was not claimed. There were no toxic sequelae. KHALIL and SALAH[82] subsequently found that acriflavine had no curative effect on either S. mansoni or S. haematobium: and that toxic symptoms followed the dosage employed. This drug has not been used locally, and is not considered to have any specific effect though in cases with secondary/

secondary septic involvement it might be employed as an adjuvant to antimonial medication.

65. CHRISTOPHERSON (J.B.) The Successful Use of Antimony in bilharziasis. LANCET, 1918 Sept. 7.

66. Do. Intravenous Injections of Antimonium Tartratum in Bilharziasis.
BRIT. MED. JL. 1918 Dec. 14.

67. Do, and NEWLOVE (J.R.) Laboratory and Other Notes on Seventy Cases of Bilharzia Treated at the Khartoum Civil Hospital by Intravenous Injections of Antimony Tartrate.
JL. TROP. MED. and HYG. 1919 (July 15.)

68. Do. and OGIER WARD (R.) Bilharzia Disease in England: Cystoscopic appearance of the Bilharzia Bladder before and after Intravenous Injections of Sodium Antimony Tartrate.
BRIT. JL. OF SURG. 1934 XXI, 84, pp. 632-636 (Reprint received from Author).

69. CAWSTON (F.G.) Evidence of the Successful Destruction of Schistosomes.
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70. Do. A Consideration of the Doses and Solution used in the Treatment of Schistosomiasis.
JL. TROP. MED. and HYG. 1930 XXXIII. 1. pp. 7-8.

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IBID. 1929 XXXII. 4. p. 47.

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74. GOPSILL (W.L.) Schistosomiasis in Nyassaland, and its treatment by means of Fuadin.
JL. MED. ASSOC. SOUTH AFRICA 1931 v. 7. pp. 222-223.

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REV. MED-CIRURG. DO BRAZIL. 1931. XXXIX, 2, pp. 43-52 - ABS. TROP. DIS. BULL. 1931, XXVIII, 670.

76/

76. TSYKALAS & REIGL (R) Emetine and Papaverine in the Treatment of Schistosomiasis
REV. MED. ET HYG. TROP. 1929. XXI. 5.
pp. 137-157. 32 Refs.

77. PELTIER (M) & RAYNAL (J) Emetine Hydrochloride subcutaneously in the Treatment of Vesical and Intestinal Schistosomiasis.
BULL. SOC. PATH. EXOT. 1929. XXII. 3
pp. 168-173. 2 Refs.

78. BOUSQUET (A) Schistosomiasis in the Nefzaoua
ARCH. INST. PASTEUR DE TUNIS XIX. 4. pp. 438-450.
9 Refs.

79. TSYKALAS (D) L'emetine contre les affections schistosomiques (1933) Decalogue de l'application de l'emetine (1932) ABS. TROP. DIS. BULL.
1934. XXXI 2. p. 113.

80. STAUDT (H) Schistosomiasis at a School in the Katanga Province ANN. SOC. BELGE DE MED. TROP. 1931.
XI. 1. pp. 67-73.
ABS. TROP. DIS. BULL. 1932. XXIX 1. p. 42.

81. FISHER (A.C.) The Treatment of Schistosomiasis with Acriflavine. LANCET. 1934. APRIL 28. p. 897.

82. KHALIL (M) & SALAH (M) Treatment of Schistosomiasis with Acridine Compounds. IBID. OCT. 20. pp. 862-863.

83. OZAWA (M) Experimental Study on the Anaemia caused by Schistosomiasis Japonica.
JAPANESE JL. EXPERIM. MED. 1931. IX. 1.
pp. 39-45.

VI.

SANITARY CONTROL: PREVENTIVE MEASURES.

As a measure for the suppression, or even control, of the disease it is obvious that treatment of the individual alone is comparatively futile. Attention has already been drawn to the relatively high proportion of the infected in the population who do not complain of the disease, and who are not accessible to treatment.

It therefore follows that general sanitary measures, supported by widespread propaganda afford the only means of control.

Measures must be directed against:-

1. Fouling of water by infected persons.
2. Spread of the intermediate host.

The first essential is the provision of wells. Until such time as villages are provided with an adequate supply of well water, and the use of water from irrigation channels is prohibited no diminution in incidence need be expected.

At present, the treated and presumably cured individual is only too frequently re-infected almost immediately on return to his native village.

The second essential is the provision of some effective/

effective form of latrine. In the dry and sandy soil of this Province it should be possible to devise some form of trench latrine from purely local materials. The difficulty is to enforce its use: and local custom at present contributes both to spread and persistence of infection. It is hoped, in time, to familiarise the people with the dangers attendant on bathing, washing and defaecating in irrigation ditches. Great importance is attached to persuasive propaganda, and encouraging response has already been obtained.

Energetic measures are required against the intermediate hosts, and the following recommendations are put forward:-

1. All canals and ditches to be cleared of weed to one foot below low water level.

2. Canals to be dried off when possible, and to be treated with "Ialine", or "Sizoline", prior to complete drying.*

3. Pools near sluices and bridges should be so treated every 30 days, or preferably baled out. Subsidiary canals should be baled out at "scour" within 3 days of watering, or treated with fuel oil.

4. All stakes, branches and brushwood should be cleared from canals.

5. Aprons on either side of bridge regulators should be carried beyond foundation width, and the brickwork rendered with cement.

Snails are carried into subsidiary (tertiary) canals in great numbers, and the use of palm fronds, implanted at points/

* In suitable dilution, use of these preparations ensures destruction of intermediate hosts.

points 10', 20' and 30' distant from the inlet has greatly assisted,

1. In reducing the numbers of snails escaping to the lower and more stagnant reaches of subsidiary canals.

2. In investigation into the predominant snail types, and the incidence of cercarial infestation of such snails.

3. In determining rate of spread of snails throughout canals.

Success in the elimination of Schistosomiasis will only be achieved by elimination of the intermediate host: in short the key to the problem as a whole, is <u>sanitation, not medication</u>.

VII.

CONCLUSION AND SUMMARY.

On the findings of the above study it is concluded:

1. That the Clinical Aspect presented is dependent on:
   (a) intensity of infection.
   (b) repeated infection.
   (c) may be modified by concomitant infection, especially by Malaria and Dysentery.

2. That Modification of the disease is not dependent to appreciable degree on a personal Immunity to infection.

3. That co-incident Malaria has not been sufficiently studied in relation to splenic enlargement of Schistosomiasis - particularly that of Schistosomiasis mansoni.

4. That the existence of "Carriers" is not yet fully appreciated in the planning of measures against the disease: detectable only on Survey, they nullify attempts to suppress infection through treatment of the individual.

5. That infections show a marked Seasonal fluctuation, especially that due to S. haematobium: but that such fluctuation is less marked in areas of pump-fed irrigation.

6. That pumps should on no account draw from other than the free running stream: and that greatest snail concentration is found in end-canals.

7. That a sufficient distinction is not always drawn between infestation of the river and canals by snails, and infection of these areas with Bilharzia.

8/

8. That lack of Survey of the "Snail Population" of such localities may lead to much unnecessary expenditure; many snails show evidence of Trematode infection other than Bilharzia, and failure to appreciate this may lead to error.

9. That the propagation of infection from season to season is not dependent upon the survival in the canal bed of the intermediate host through the dry period.

10. That re-infestation of "cleared" irrigation zones by the intermediate host takes place via pump and main canal, and that the greatest snail concentration is to be found in end-canals.

11. That chemical properties of water may limit the range of appropriate intermediate hosts.

12. That infection of man takes place from contact with the water in pools, drains and terminal canals, and that the greatest incidence in infection is found in villages in relation to such collections of water.

13. That Animals do not form a reservoir of Schistosomal infection transmissible to man.

14. That schistosome species affecting man and animals cannot invariably and positively be identified by the characters of ova found in excreta.

15. That Urinary Schistosomiasis is a disease of Children, and (locally) shows a tendency to spontaneous cure, and seldom produces lesions of grave character.

16. That Rectal, or visceral, Schistosomiasis occurs irrespective of age; and becomes more common with advancing age: and that the resulting lesions are extensive, and the ultimate outlook in the majority of cases grave.

17. That Schistosomiasis in its essential pathology belongs to the granuloma-group, and should not be considered as a "local" disease of the genito-urinary or intestinal systems.

18/

18. That cure may be achieved in the Primary or Secondary phase of the disease, but that only temporary improvement can be looked for in the Tertiary phase.

19. That Specific (Antimonial) Treatment in the Tertiary phase is not without danger; and that failure to appreciate the generalised and progressive nature of the disease has led to the many conflicting views on the action of the Antimony-group of drugs.

20. That ova may be passed long after the conclusion of Antimony-medication in effective dosage; and that viability of ova, and percentage eosinophilia should determine the need for additional medication.

21. That concurrent disease, especially malaria and dysentery should be eliminated prior to commencement of Specific medication.

22. That retention of ova in the solid viscera, and toxaemia, and not infection by male S. mansoni alone (as claimed by GIRGES), are considered the cause of the so-called "Egyptian Splenomegaly".

23. That the spread of the disease, and its propagation year to year, can only be effectively combatted by Sanitary Measures directed against the intermediate host, and by propaganda among the affected population.

SUMMARY.

1. The Endemiology of Schistosomiasis in Berber Province is discussed.

2. Attention is drawn to the occurrence of "Carriers" who constitute a little appreciated source of dissemination of infection.

3. Incidence among Population groups is mentioned. The settled Agricultural group is principally studied.

4. The effect of irrigation on the spread of the disease is discussed.

5. Seasonal fluctuation in incidence is noted. March, April and May are the months of highest incidence.

6. Probable snail hosts are described, with reference to literature. Melanoides tuberculata is held - on circumstantial evidence - to be a local host to S. haematobium, and possibly also to S. mansoni.

7. The occurrence of trematode infestation, other than Bilharzial, in the snails is noted, and instances of "double infection" of snails are described.

8. The possibility of aberrant infections in animals and man is discussed. It is concluded that Animals do not form reservoirs of schistosomal infection transmissible to man.

9. Double infection with S. haematobium and S. mansoni had not been noted in the Province. This may be due to chemical reaction of localised collections of water limiting range of appropriate intermediate host.

10. Surveys were carried out in February and March, 1933 and 1934. 2690 persons were examined for urinary schistosomiasis and 88 found infected. Infection is not common between 5 and 15 years of age. Albuminuria is a constant and valuable sign in the detection of infection.

11. A circumscribed zone of endemic Schistosomiasis mansoni was detected. 547 persons were examined and 202 found infected./

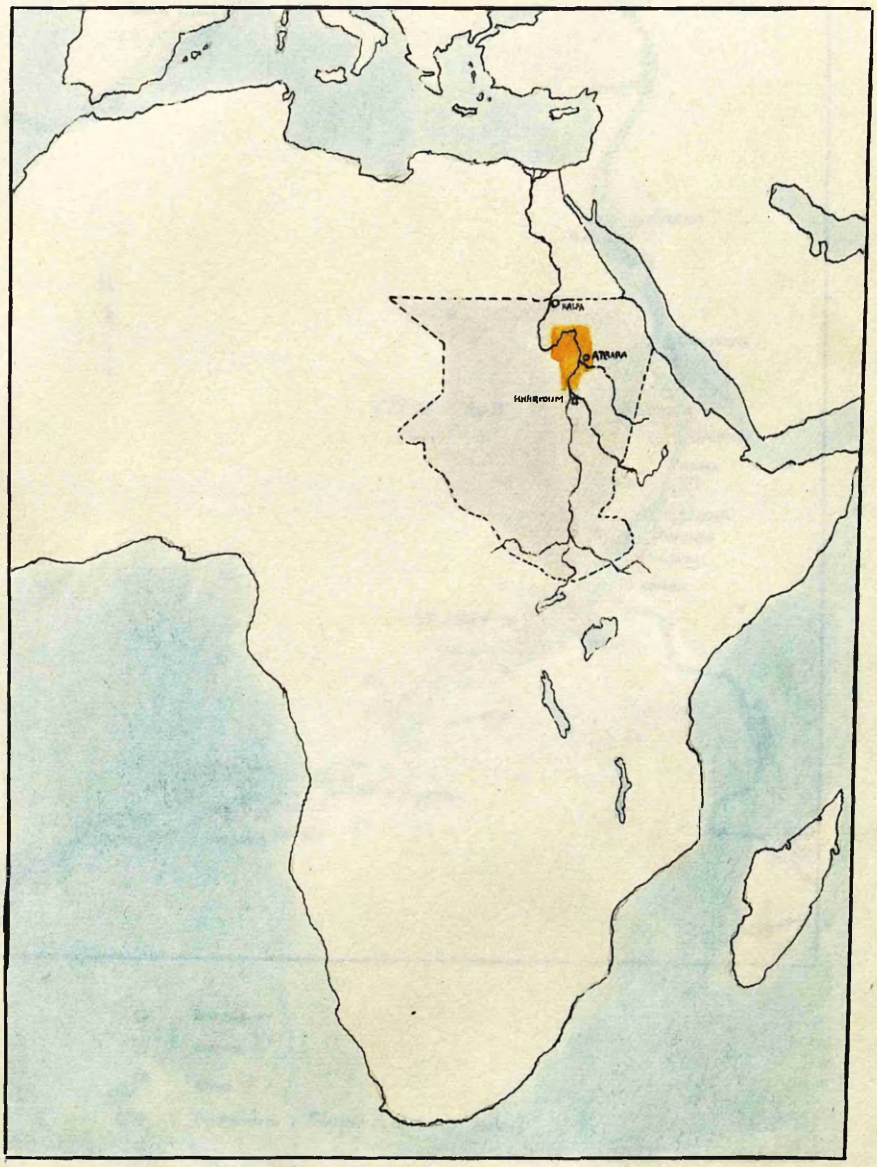
infected. The infection has not been found elsewhere in the Province. Incidence was found to rise with age.

12. Methods of study are detailed, with reference to recent literature.

13. A new schema of Clinical Classification is put forward. The essential pathology is discussed, and clinical progress reviewed. The effects of concurrent malaria, in particular, are emphasised.

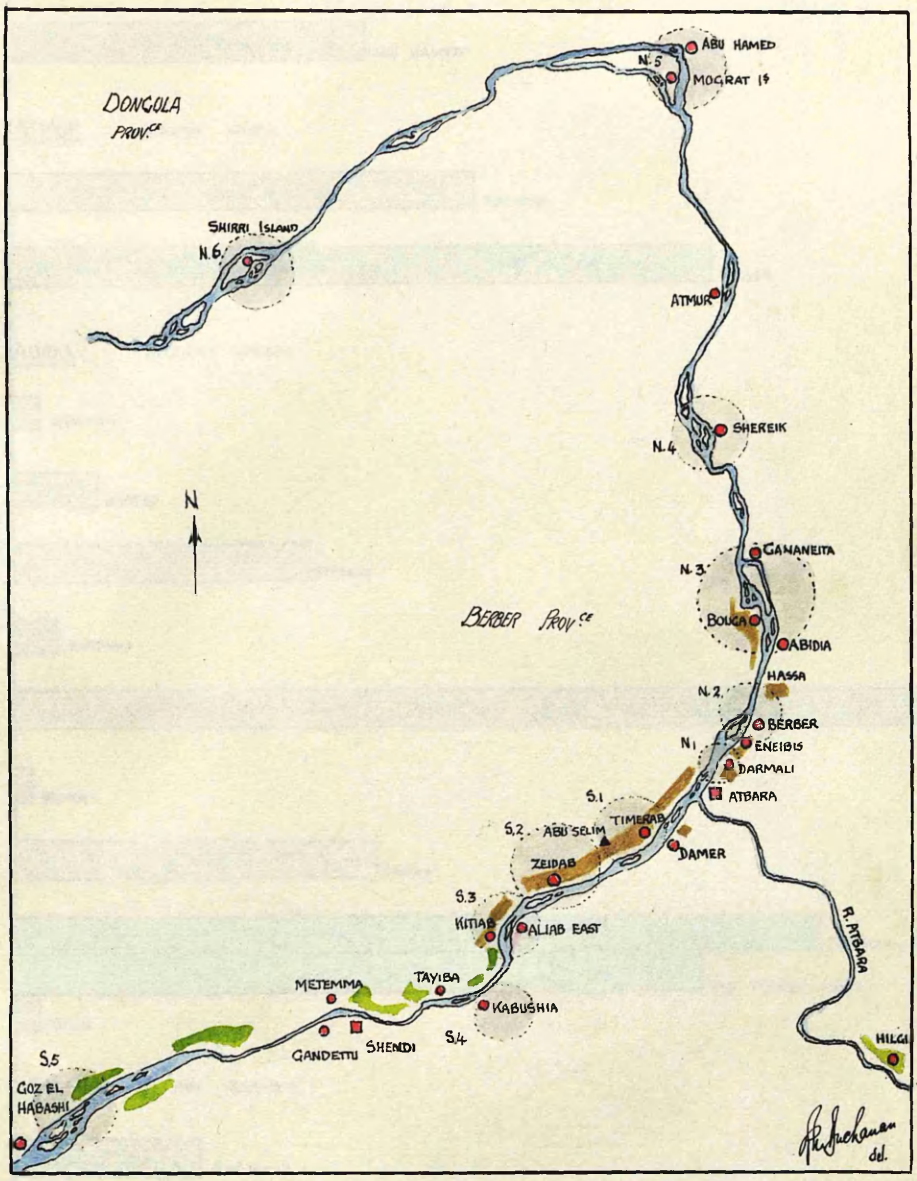
14. Treatment is described. Antimony, in the form of Sodium Antimony Tartrate is considered the only established specific. Other treatments are discussed in brief.

15. Sanitation, rather than medication, is considered the essential in the control of the disease. Measures advised are described in brief.



ANGLO-EGYPTIAN SUDAN: *BERBER PROVINCE* INDICATED IN YELLOW.

II.



DISPENSARY

HOSPITAL

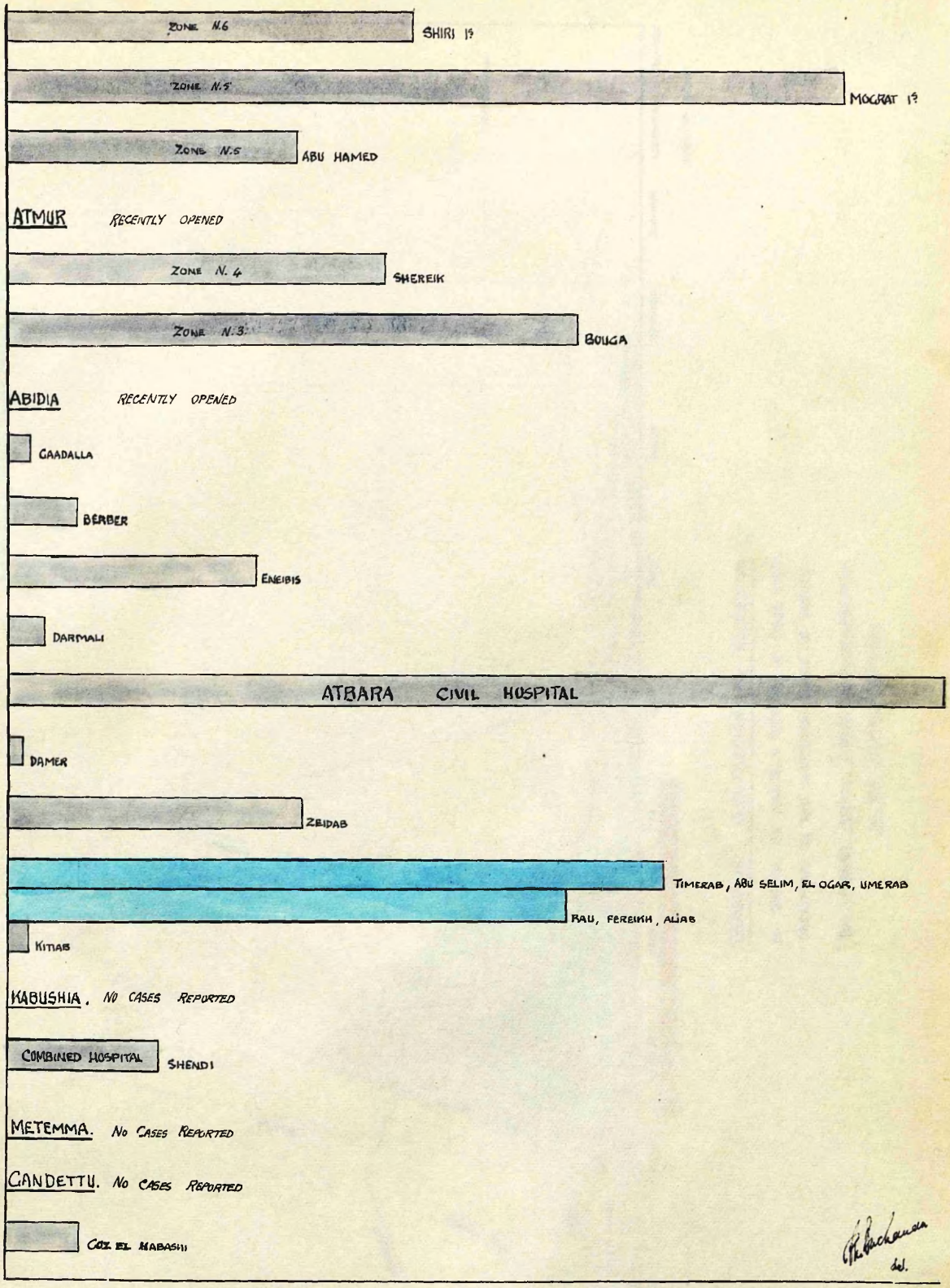
BASIN

PUMPSCHEME (*Principal Schemes only, shown.*)

N.1 SURVEY-ZONE WITH REF. NO.

THE COURSE OF THE NILE THRO' BERBER PROVINCE:

showing also zones of Survey and the associated dispensaries.



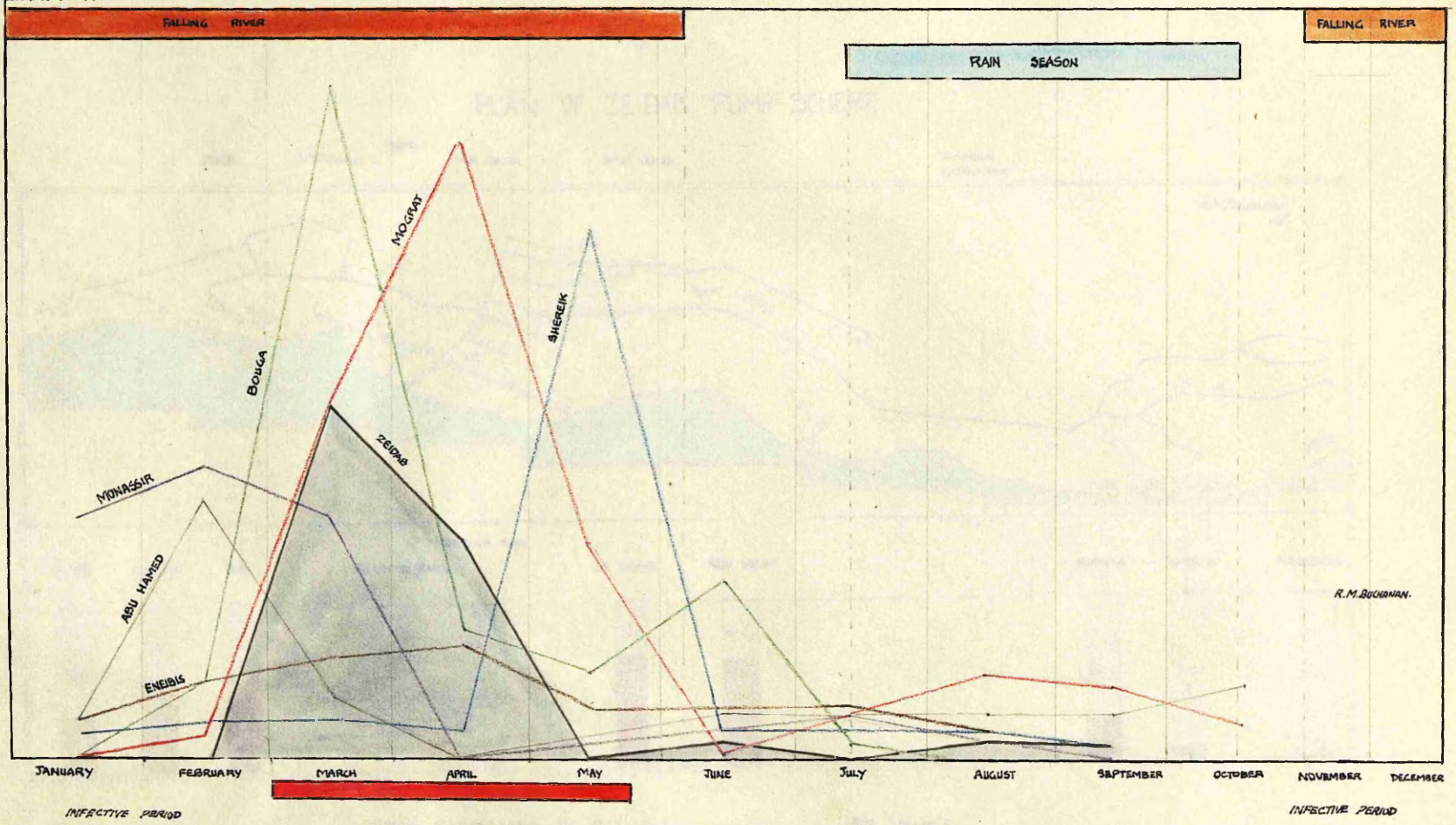
RETURN OF CASES TREATED AT THE DISPENSARIES

INCIDENCE THROUGOUT PROVINCE

GREY *Schistosaminsis haematobium*

BLUE *Schistosomiasis mansoni*

GRAPH No IV.

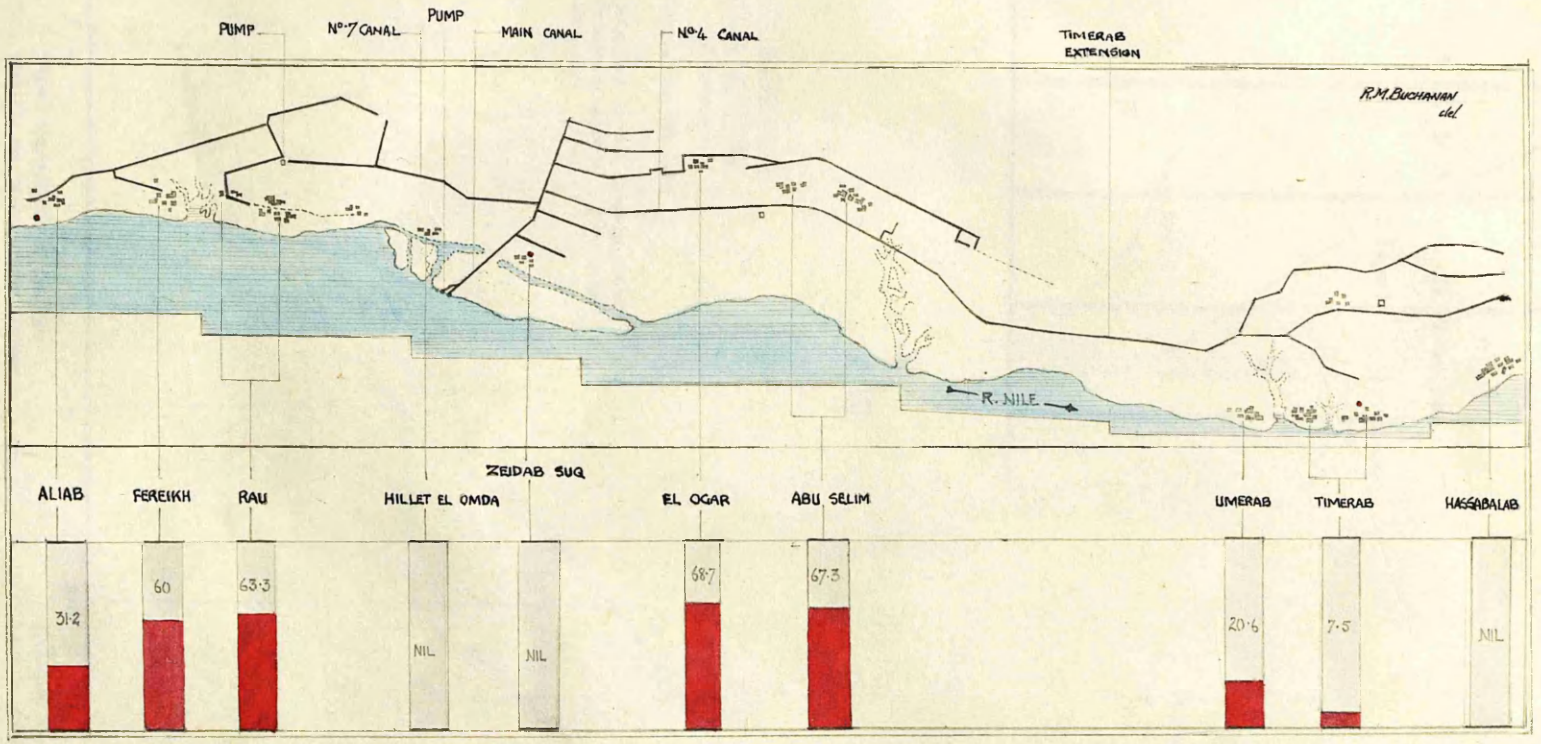


SEASONAL FLUCTUATION IN INCIDENCE

AS SHOWN BY MONTHLY RETURNS OF CASES UNDER TREATMENT IN THE NORTHERN ZONES OF SURVEY.

THE SHADED PORTION INDICATES INCIDENCE NOTED IN THE SOUTHERN DISTRICTS

PLAN OF ZEIDAB PUMP SCHEME

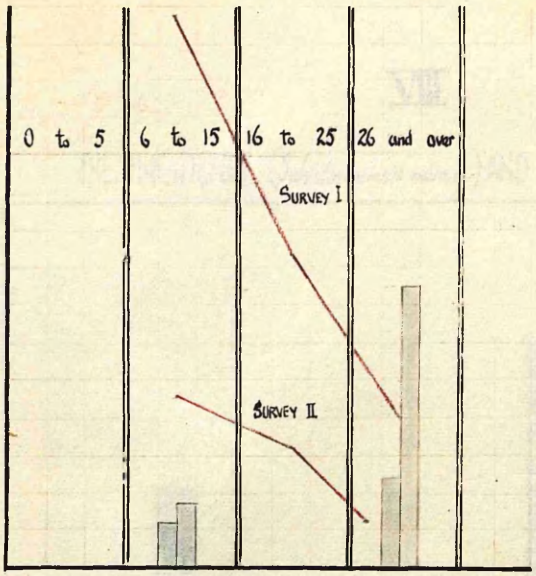


THE INCIDENCE OF Schistosomiasis mansoni PER VILLAGE

shown as percentage of
population examined.

V.

No. VI



AGE INCIDENCE IN URINARY SCHISTOSOMIASIS

(S. haematobium)

Compiled from Surveys I and II

I. Gadawab - Bouga

II Zeidab

The Grey Columns indicate incidence of Albuminuria not due to Schistosomiasis

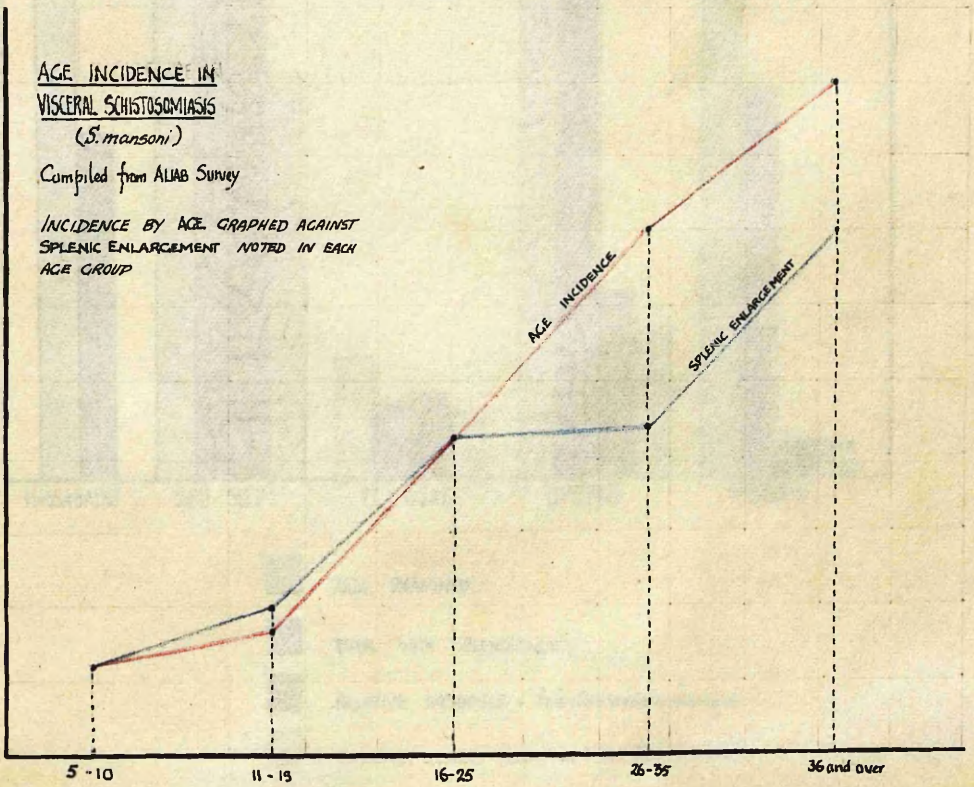
No. VII

AGE INCIDENCE IN VISCERAL SCHISTOSOMIASIS

(S. mansoni)

Compiled from Aliab Survey

Incidence by age graphed against Splenic Enlargement noted in each age group

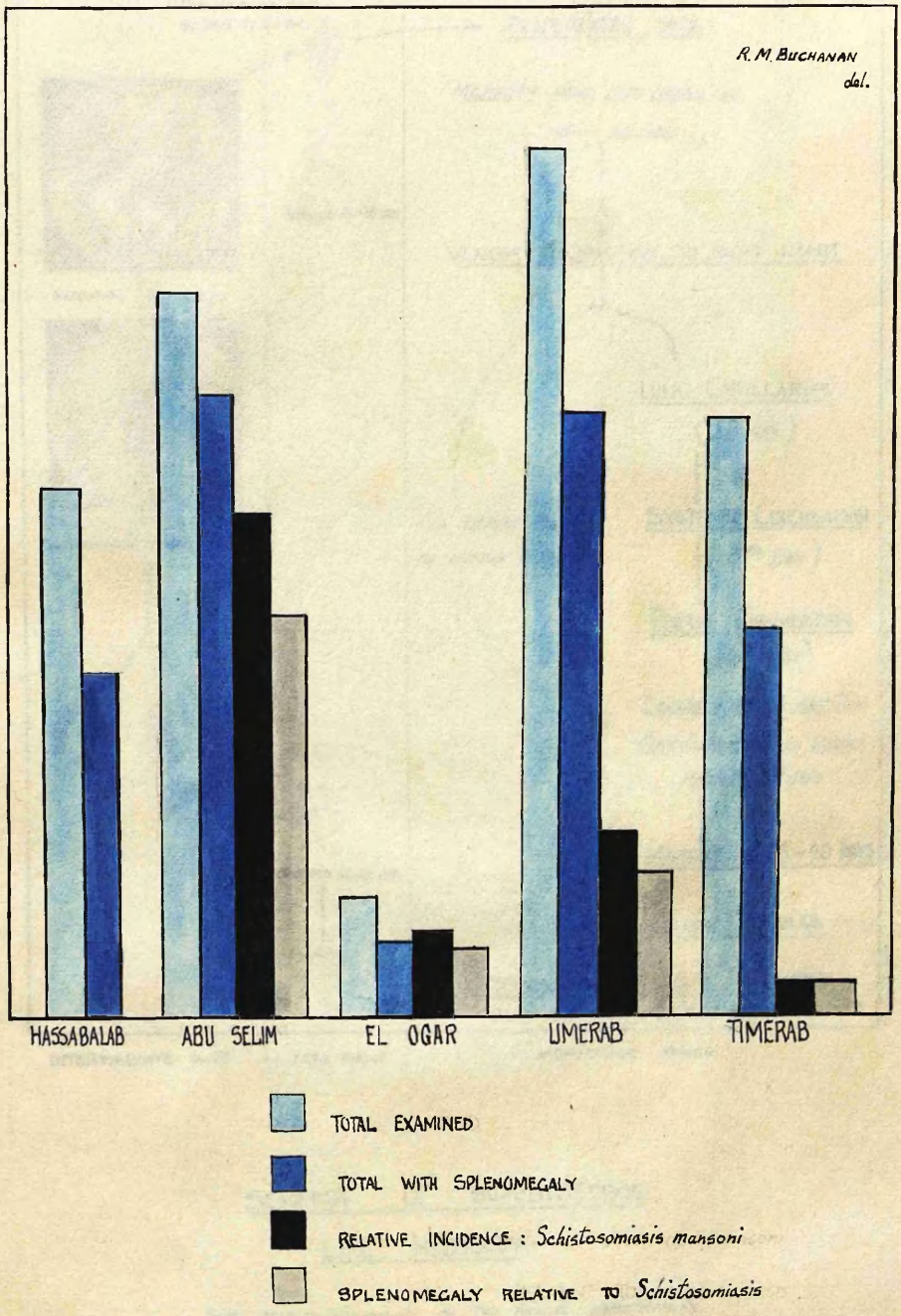


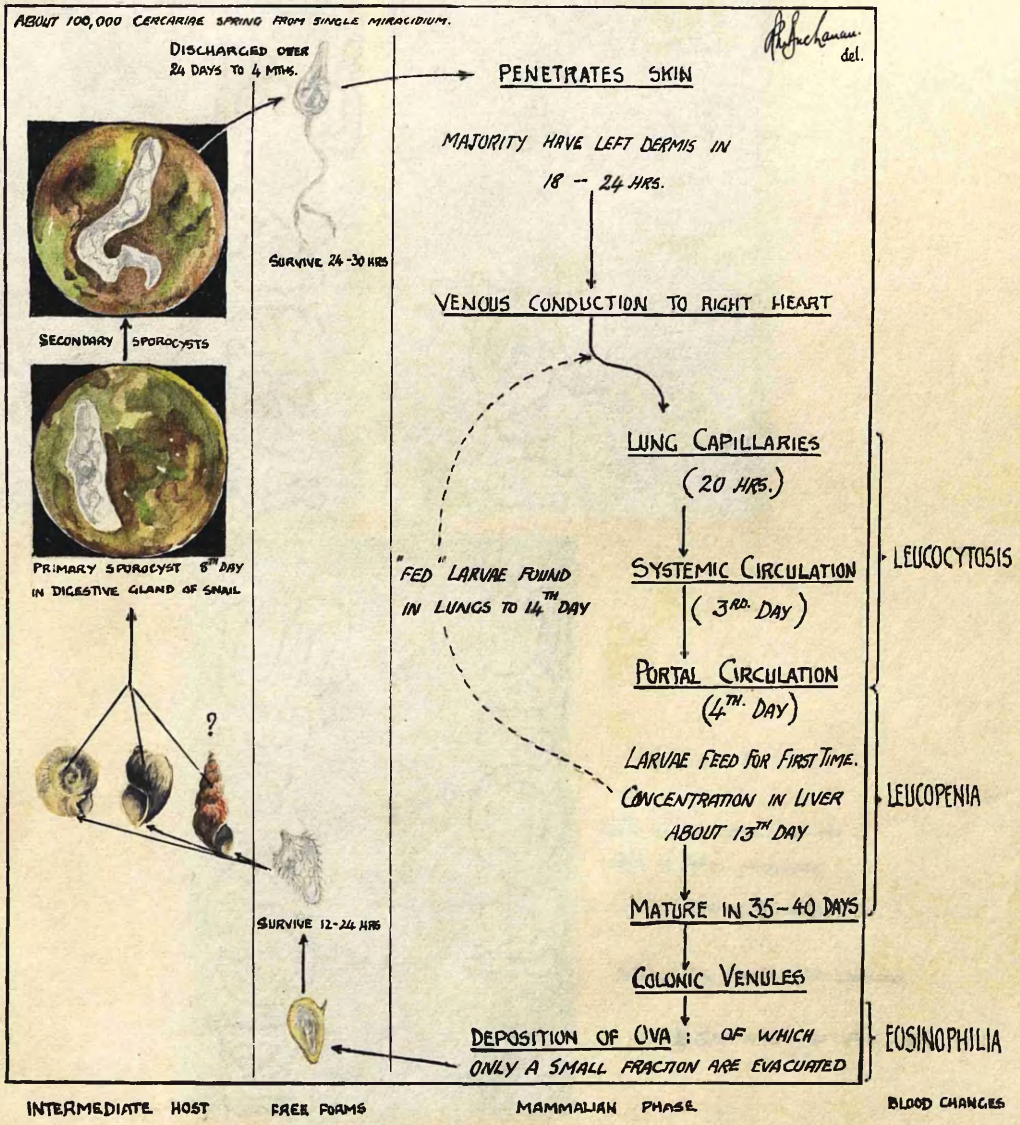
Splenic enlargement in children probably due to coincident *Malaria*

Splenomegaly in adults ? due to *Schistosomiasis*.

VIII

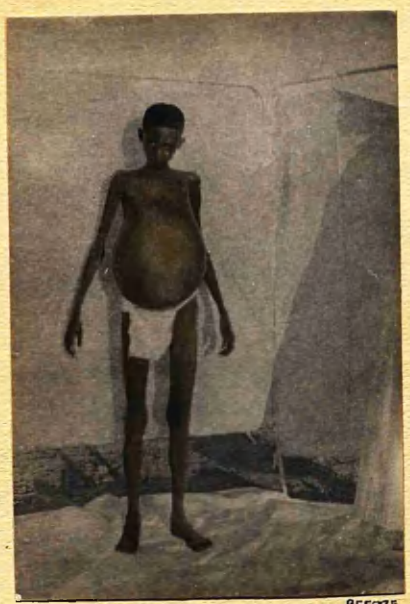
THE INCIDENCE (*Schistosomiasis mansoni*) AND RELATED SPLENOMEGALY.





SCHEMA OF SCHISTOSOME LIFE HISTORY.

WITH ACKNOWLEDGEMENT TO THE RECENT INVESTIGATIONS OF FAUST, HOFFMAN and JONES.



BEFORE TAPPING

AFTER TAPPING.

White line on abdomen indicates extent of Splenic enlargement.

There is marked cirrhosis of liver

Note wasting, and stunted appearance.

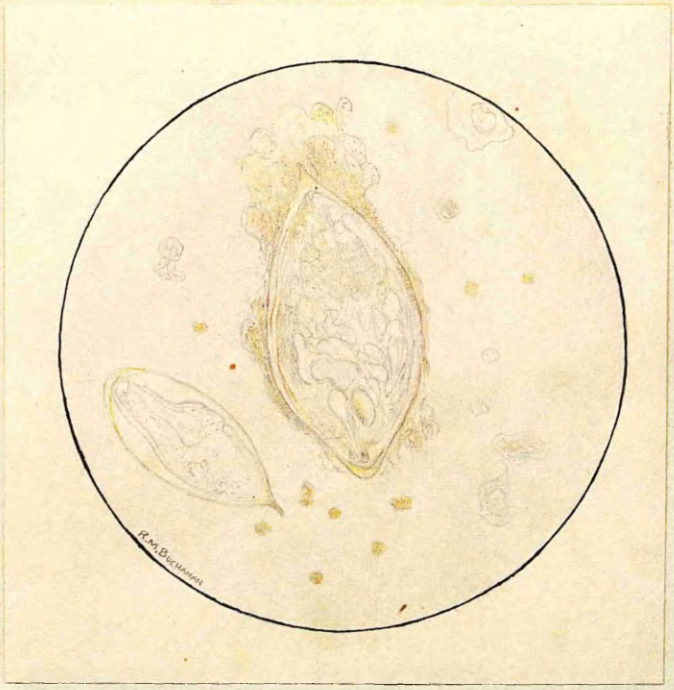
◆ SEE ALSO: pp. 43 and 44.

M.O. 19 yrs.

CASE OF VISCERAL SCHISTOSOMIASIS : (*S. mansoni*)

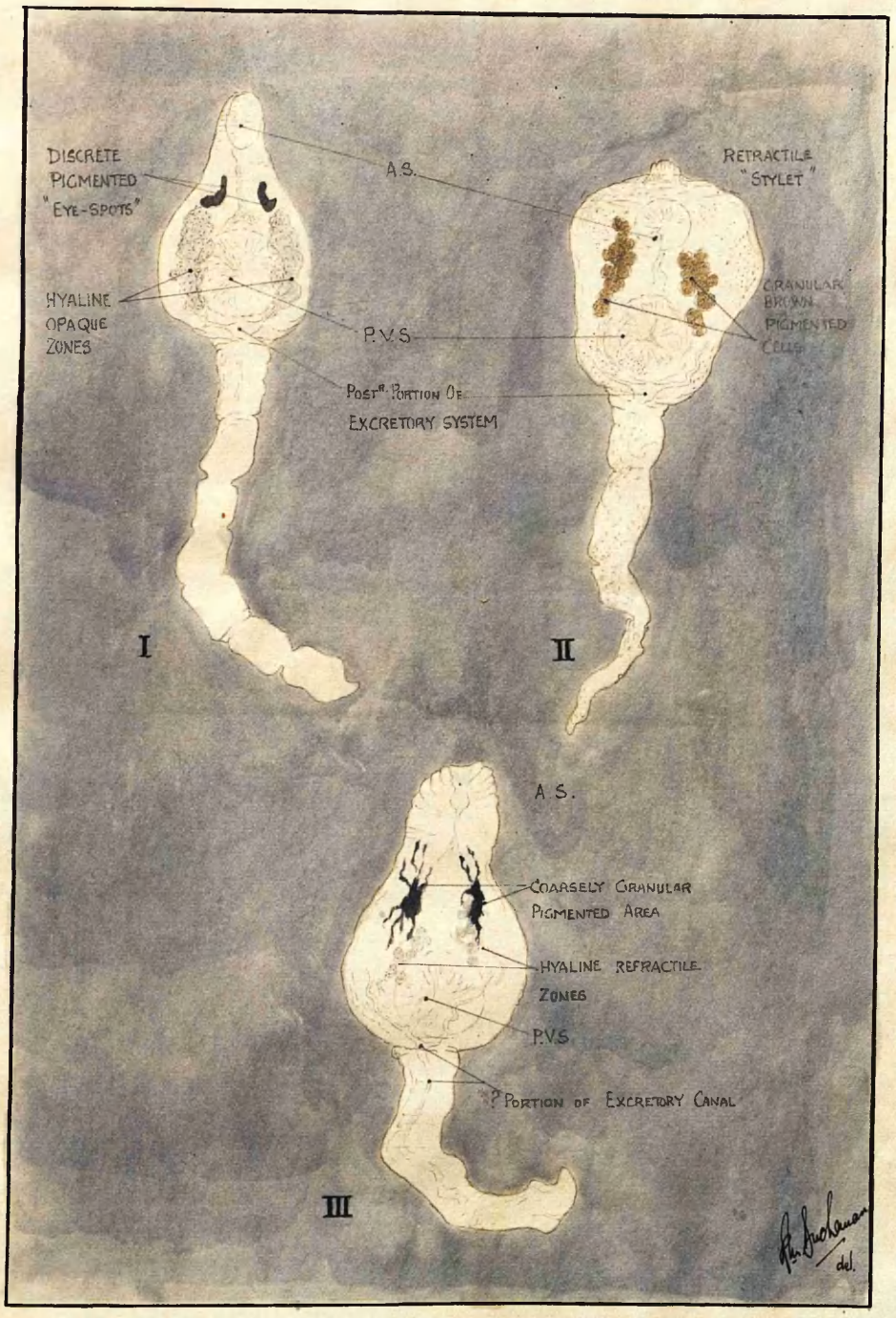
LATE "TERTIARY" STAGE.

PHOTOGRAPHED AND COLOURED
BY R.M. BUCHANAN, CIVIL HOSPITAL, ATBARA.



OVA OF S. haematobium : note disparity in size, and outline.

A SAGIA : this photograph is included to show typical river side of waterwheel. The sagia "bay" affords no suitable harbourage for snails.



CERCARIAE OF NON-FURCOCERCOUS TYPE

OBTAINED FROM SNAILS OF
Melanoides, Cleopatra and *Bulinus* spp.

NB: TYPES I AND II WERE NOTED IN ASSOCIATION WITH NON-PIGMENTED FURCOCERCOUS TYPES: CONSTITUTING A MIXED INFECTION OF INTERMEDIATE HOST.