

to see if savage races are likewise involved. At present no man knows the far-reaching effect of tonsillar infections, but it is quite certain that they are responsible for untold ills.—I am, etc.,

SYDNEY PERN, M.R.C.S., L.R.C.P.Lond.
Melbourne, July 7th.

INTERMEDIARY HOSTS OF SCHISTOSOMA HAEMATOBIIUM AND MANSONI IN NYASALAND.

SIR,—I have received a letter enclosing two tubes containing specimens of five species of fresh-water molluscs from Captain W. H. Dye, R.A.M.C., medical officer, Karonga, Nyasaland, British Central Africa. He writes: "I think I can say that the enclosed specimens represent all the fresh-water molluscs to be found in this district as I have searched most thoroughly."

Captain Dye was able to infect two of the species experimentally with *Schistosoma haematobium* and *Schistosoma mansoni* respectively. The molluscs have kindly been identified as follows by Mr. G. C. Robson, Zoological Department, Natural History Museum, South Kensington.

1. *Lanistes affinis* Smith (full-grown and young).
2. *Vivipara robertsoni* Frauenfeld.
3. *Limnaea natalensis* Krauss.
4. *Physopsis sc. globosa* Morelet.
5. *Planorbis*, sp. near *sudanicus* Martens.

Captain Dye writes of 4 (*Physopsis sc. globosa* Morelet):

"They are very common in the marshy pools, although rather difficult to find owing to their predilection for the muddy undersides of reeds, etc., and their habit of dropping off when the plant is touched. They appear to attract *S. haematobium* readily, and large numbers of miracidia disappear out of the tube in which they are put, against the control."

He goes on to describe in detail the experiments he made. He finds that the mollusc dies in two days when heavily infected with the miracidia of *S. haematobium* after taking precautions to keep the water as free from decomposition matter as possible. The experiments were repeated several times with the same results. The snails were "not killed by *S. mansoni*, but one cannot get such concentration of eggs from faeces as from urine."

In regard to 5 (*Planorbis*, sp. near *sudanicus* Martens) he writes: "The one and only species of planorbis in this part of the world (I have most thoroughly searched)." This species is not killed by a heavy infection of *S. haematobium*; but they were "infected from a good heavily infected stool with *S. mansoni*."

Captain Dye sent infected snails, but they died on the journey and were too decomposed for sectioning on arrival. He appears to have discovered that *Physopsis sc. globosa* Morelet is the intermediary host of *S. haematobium* in Nyasaland, and possibly he has also found the intermediary host for *S. mansoni* in Nyasaland (*Planorbis*, sp. near *sudanicus* Martens).

The other snails which he sent had, he wrote, no attraction for either *S. haematobium* or *mansoni*.—I am, etc.,

J. B. CHRISTOPHERSON, C.B.E.,
London, W., Sept. 1st. M.D., F.R.C.P.

SCHISTOSOMA INFESTATION.

SIR,—The account by Drs. Sinderson and Mills in the BRITISH MEDICAL JOURNAL for June 9th (p. 968) of rectal papillomata in a patient at the Baghdad Hospital harbouring *Schistosoma haematobium* raises several points of interest.

First, the recent recognition of cases in Palestine, Mesopotamia, and Turkish Arabia suggests the possibility of the introduction of bilharzia disease to new countries, although the thoroughness with which this disease is now being studied would account for the recognition of cases in countries where until recently it was unknown. Severe manifestations of *Schistosoma haematobium* in a comparatively early stage of the disease might suggest that the parasite has but recently been introduced on to new soil. I have noticed slight manifestations of *Schistosoma mansoni* in two patients in Natal who have never left the country. It may have been that the condition had only recently been acquired, for both of them came complaining

rather from the effects of *Schistosoma haematobium* which complicated the condition. Nevertheless, the finding of *Segmentina kanisaensis* Preston at Merebank, Natal, a shell which was previously known only from the Upper Nile, supports the assumption that *Schistosoma mansoni* infestation has been introduced from Egypt.

Secondly, a very high eosinophilia may occur in the acute stage of bilharzia disease. In my own case it rose to 39.5 per cent. during the hepatic invasion of the flukes, but fell to 12 per cent. in the next few months without treatment. It is not common to find such high eosinophilia in chronic cases, even where the infestation is a high one; a relatively high eosinophilia may persist even after the parasites have been successfully destroyed, although it generally returns practically to normal.

Thirdly, it is to be regretted that intravenous antimony could not have been employed before surgical measures were adopted in the case at Baghdad; for extensive bilharzia tissue rapidly subsides as the adult parasites and their ova are destroyed.—I am, etc.,

Durban, Natal, July 27th. F. G. CAWSTON, M.D. Cantab.

ORAL HYGIENE.

SIR,—The views of Sir John O'Connor on the use of the tooth-brush reiterated in your issue of August 25th are so diametrically opposed to the opinion and experience of the great majority of dental surgeons that they must not be allowed to pass unchallenged.

Sir John states his conclusion that the cause of "dental caries, pyorrhoea, gastric pollution, appendicitis, and organic decomposition in general"—a catalogue reminiscent of the "cancer, cataract, and consumption" of the quack medicine vendor—is to be found in the "inane daily scrubbing of teeth with bristles and by the insane use of chemical dentifrices."

Regarding "gastric pollution, appendicitis, and organic decomposition in general," whatever that may mean, I express no opinion, but the experience of thirty-five years has convinced me that the thorough and intelligent daily scrubbing of the teeth, and more particularly of the gums, and the reasonable use of a dentifrice, though this is far less important, reduces the incidence of dental caries and pyorrhoea to an enormous degree; and every day my belief is strengthened by results.

Dental caries is produced by the action of fermenting food particles on the teeth, which, with the progress of civilization, have acquired an increased susceptibility to this form of attack. The debris of the soft food of the present day lodges around and between the necks of the teeth, and in the crevices of their crowns, and until the cause of this susceptibility and the means of combating it shall have been discovered, or until the diet of the people shall have reverted to a more primitive type, thorough and careful removal of these particles with the tooth-brush remains the most efficient means of preventing caries and pyorrhoea; for pyorrhoea, like caries, begins as a dirt disease.

Débris lodging upon the gum margin infects it and sets up a marginal gingivitis. The gum margin becomes tender, swells, and stands a little way from the tooth instead of being closely applied. This affords lodgement for more debris, while the tenderness leads to avoidance of the contact of any cleansing agent; more accumulation increases, and a vicious circle is established.

Friction of the gum margin, provided by Nature when man lived on uncooked hard food, is essential to the health of teeth and gums alike; it removes the debris and stimulates the circulation in the gum margin and keeps it firm, pale, and healthy. As the cooked artificial diet of civilization no longer supplies it, it must be provided artificially. Carefully persevered with in spite of some soreness at first it will cure even a long-standing gingivitis, while it will prevent or arrest caries on all surfaces to which it is applied. For the application of this friction nothing is so good as a bristle brush, because nothing else penetrates so well between the teeth and reaches the interdental papillae, though when the gums are tender a rubber brush may be used until they are hard enough to tolerate bristles. A badger-hair brush is altogether too soft to effect this.