The Epizootiology of an Outbreak of Bilharziasis in Zululand

BY

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A massive outbreak of bilharzia occurred in the lower Mkuzi in July and August, 1964, causing the death of large numbers of sheep and a fair number of cattle.

History.—Sheep died at an alarming rate in the winter followed by cattle. Bilharzia was diagnosed at autopsy and the parasite was subsequently identified as *Schistosoma mattheei*. Affected cattle were treated with tartar emetic and antimosan, but the mortalities continued.

Clinical Signs.—In sheep a progressive wasting disease, emaciation, in advanced cases diarrhoea and an anxious expression in the eyes are noted. Cattle show additional signs of salivation. Mortalities in sheep are heavy, for example, a flock of 56 was reduced to 14 animals in two months. Cattle also die, but not at the same rate, although many may succumb after treatment (see below).

Post-mortem Examination.—The pathology has been well described by McCully and Kruger (1969) and only a few points will be emphasised.

Marked emaciation with hydropericardium, hydrothorax and ascites is common in cattle and sheep. A consistent finding in sheep is the light to dark grey lungs, which appear to get darker the longer they are exposed to light. The liver is usually dark brown to black in colour and atrophied with an uneven surface. Most of the worms are in pairs in the mesenteric veins of the jejunum, while the odd parasites are situated on the surface of the caecum.

With the exception of the lungs, which are normal in colour in cattle, the macroscopic appearance is similar to that in sheep. Some cattle were treated either with tartar emetic or antimosan. Six to 14 days later some of these treated animals died while others were killed *in extremis*.

The lesions of these treated animals are alarming. In addition to the lesions in untreated animals, there is a massive "liver shift" after treatment of dead worms from their normal habitat in the mesenteric veins to the portal vein and its branches. Many white spots, particularly on the periphery of the liver, prove on section to be worms which adhere to the knife blade if it is wiped across the cut surface. Numerous red spots in the blood vessels caused by dead worms

are a consistent feature (McCully and Kruger, 1969). At the edge of the liver there may be a wedge-shaped infarct. The marked pathological lesions which follow treatment and lead to "liver shift" may cause the death of an animal already weakened by bilharzia.

Differential Diagnosis.—The clinical signs resemble nagana (trypanosomiasis). Bilharzia can, however, be differentiated from nagana in that the sheep die while the cattle survive and dogs show no clinical signs. In nagana cattle die, dogs show clinical signs, but sheep, although susceptible, are rarely affected. Examination of faeces for eggs in bilharzia or blood smears for trypanosomes will confirm the diagnosis.

Epizootiology.—To our knowledge this is the first time that cattle have died on any scale, although mortalities in sheep have been reported (Le Roux, 1929; Strydom, 1963; Hurter and Potgieter, 1967; Lawrence, 1968).

These mortalities may be explained as follows: *S. mattheei* is enzootic in the lowveld and as many as 80 to 90 per cent. of cattle and goats may be infested (Pitchford, 1959). The intermediate host *Bulinus (Physopsis) globusus* is present in every stream, pan, dam, etc., in large numbers.

In the last 20 years, State lands have been leased to farmers in areas previously unsuited for humans or animals, due to malaria and nagana respectively. These diseases were eliminated by the eradication of the insect vectors and a thriving livestock industry established.

The definitive and intermediate hosts are present in large numbers and *S. mattheei* is present in most of the livestock, albeit in moderate numbers. It merely needs some trigger mechanism for this parasite to increase to outbreak proportions.

The Umsundizi and Mkuzi rivers overflow their banks every December in this flat country. When the floods subside, numerous pans are left in the surrounding areas which dry up and disappear in the winter. In July, 1963, however, unseasonal rains caused the rivers to overflow their banks, filling the pans, which were refilled in December. Thus large areas of water highly suitable for the multiplication of snails were present almost continuously for 18 months, instead of a mere five or six months each year.

The water in the rivers is very low in the winter and the banks lined with thorn trees and similar shrubs. Sheep and cattle are only able to drink or cross the river at shallow drifts which, being limited and rather narrow, are unable to accommodate large numbers of animals at a time and livestock do not spend long periods standing in or drinking water at these drifts. Conversely, the pans, which are extensive and shallow, are ideal drinking places and almost all the animals enter the water and stand in it for long periods, covering their lower limbs with mud up to their hocks and knees. Possibly they stand in the water for long periods because of the extreme heat. Moreover, the grazing on the edge of the pans is greener and more succulent compared with the veld, which is dry, particularly in the winter.

The animals defaecate in the water, eggs of *S. mattheei* hatch and the hatched miracidia infest the large populations of *B. (P.) globusus*, which shed vast numbers of cercariae after a few months. The life-cycle is completed and animals are subjected to a massive invasion of parasites, causing sheep to succumb followed by cattle.

Treatment of animals already weakened causes a massive "liver shift." The dead parasites aggravate the lesions already present in the liver, with fatal consequences.

Discussion

Dr. Pitchford asked what clinical signs dogs showed in nagana to differentiate it from bilharzia.

Prof. Reinecke: In nagana the cattle die first, the dogs show a keratitis and the sheep are last affected.

Prof. Elsdon-Dew: To those of us who are familiar with the pathology of bilharziasis in humans, the infection of the liver in this epidemic is a fantastic phenomenon which we do not often see in humans. We cannot, like the vets., study the anatomy of the disease in its human host.

Prof. Reinecke: Surely here is an indication for study of the disease in primates. Get on with the job and tell us what happens. After all, there are very few helminthic diseases of humans, compared with what the veterinarian has to deal with.

Mr. Mansfield: I should like to ask Prof. Reinecke whether any habitat such as he described on the Mkusi occurs on the flood plains of Pongola and Usutu.

Prof. Reinecke: Dr. Pitchford would know the country better than I. I referred to the outbreak described by Strydom, which was controlled through following his advice.

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