A review of filarial infections in domestic ruminants: their importance and control

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**CONTENTS**

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Introduction</td>
<td>1</td>
</tr>
<tr>
<td>I. Parafilariasis</td>
<td>2</td>
</tr>
<tr>
<td>A. Aetiology</td>
<td>2</td>
</tr>
<tr>
<td>B. Occurrence and Species Affected</td>
<td>4</td>
</tr>
<tr>
<td>C. Transmission</td>
<td>7</td>
</tr>
<tr>
<td>D. Pathogenesis and Pathology</td>
<td>8</td>
</tr>
<tr>
<td>E. Importance</td>
<td>10</td>
</tr>
<tr>
<td>F. Diagnosis</td>
<td>11</td>
</tr>
<tr>
<td>G. Control</td>
<td>12</td>
</tr>
<tr>
<td>H. Discussion</td>
<td>14</td>
</tr>
<tr>
<td>II. Stephanofilariasis</td>
<td>16</td>
</tr>
<tr>
<td>III. Onchocerciasis</td>
<td>28</td>
</tr>
<tr>
<td>IV. Setariasis</td>
<td>42</td>
</tr>
<tr>
<td>V. Elaeophoriasis</td>
<td>54</td>
</tr>
<tr>
<td>VI. Discussion</td>
<td>62</td>
</tr>
<tr>
<td>Tables</td>
<td>(i)</td>
</tr>
<tr>
<td>Acknowledgements</td>
<td>(v)</td>
</tr>
<tr>
<td>References</td>
<td>(vi)</td>
</tr>
</tbody>
</table>
INTRODUCTION

Infections caused by parasites belonging to the superfamily Filarioidea are regarded as of clinical importance in the medical field. A considerable number of publications and reviews dealing with these infections in man has appeared during the past few decades. The more recent ones include a number of technical reports by the World Health Organization on onchocerciasis, Wuchereria and Brugia infections and human filariasis in general (WHO, 1966, 1967, 1974), and a treatise by Nelson (1970) on 'river blindness' of man caused by Onchocerca volvulus.

However, in the veterinary field, the situation appears to be reversed as there has been a paucity of such reviews especially on filarial infections of livestock. It seems necessary, therefore, that a review be initiated along this line in order that the role played by these infections can be elucidated.

The object of this paper will then be to consider the infections caused by a number of filarial nematodes in domestic ruminants. The species belonging to the genera Parafilaria, Stephanofilaria, Onchocerca, Setaria and Elaeophora will be reviewed in the light of morphologic and taxonomical, epidemiological and clinico-pathological information available on them. The significance of these parasites and the possible control measures which can be instituted against them will be discussed.
I. PARAFILARIASIS

Parafilariasis of cattle and water buffaloes is an affection of the skin and subcutis. It is characterized by diffuse oedema and formation of parasitic nodules which swell under the epidermis and rupture causing local hemorrhages.

Aetiology

Gulati in 1934 described the condition in Indian bullocks and was able to isolate from the haemorrhagic lesions worms which resembled Parafilaria multipapillosa of equines. They were white filariform nematodes, the male measuring 28 mm long with unequal spicules, and the female 40-70 mm long with the vulvar opening adjacent to the oral aperture. The anterior end of the female exhibited cuticular ornamentations in the form of elliptical and circular papillae. Tubangui (1934) and de Jesus (1934) noted a similar condition occurring in Philippine cattle and were able to provide a more detailed description of the parasite which Tubangui named Parafilaria bovicola. According to these authors, the oesophagus of the nematode is 0.23-0.25 mm long and the nerve ring is located near its middle. The intestine is prominent. The cervical papillae are small and are 0.28-0.30 mm from the anterior extremity. The uteri converge at a point 0.7 mm from the anterior end to form the vagina. Thin-shelled eggs found in the uteri are fully embryonated and measure 40-52 μ x 27-33 μ. The measurements
provided by Fain and Deramee (1949) and Fain and Herin (1950) for the adult forms were similar to those of Gulati (1934), and the female anterior end was described further by the former authors as being rounded with "transverse cuticular ridges which are interrupted at irregular intervals"; small rounded tubercles were also seen at the anterior extremity.

Srivastava and Dutt (1959) found similar lesions in Indian water buffaloes and established a new name, *P. sahaii*, (for the parasite which they isolated) on the grounds of differences to *P. bovicola* with regards to the host and the arrangement of cuticular ornamentations in the female anterior end. However, Patnaik and Pande (1963), in comparing female specimens of *P. bovicola* isolated from bullocks and "*P. sahaii*" from water buffaloes, found very insignificant differences in the arrangement, number and extent of these cuticular ornamentations and concluded that *P. sahaii* is not a valid species but, rather, a synonym of *P. bovicola*.

The microfilariae found in the lesions are unsheathed; the anterior ends are rounded while the posterior ends are curved and pointed (Lakra, 1964). According to Niilo (1968) they are active with flexing and curling motions. Different dimensions of these forms have been reported. Metianu (1949) and Alwar and Lalitha (1958) gave similar measurements which ranged from 196-249 μ in length and 9-13 μ in breadth. However, the measurements provided by Lakra et al. (1964) and Niilo (1968) were 164.5-172.6 μ x 6.9-8.0 μ and 154-215 μ x 9-13 μ respectively.
Occurrence and Species Affected

The clinical features of the disease and their relationship to a parafilarial parasite were first recognized in 1934 in India and the Philippines. Gulati (1934) noted its common occurrence in working cattle in several places in India especially during the summer months. He implicated *Parafilaria multipapillosa* (syn. *Filaria hemorrhagica*) as the causative parasite. However, Baylis (1939) suspected that he might be referring to a new species occurring in bovines. Lahiri (1934) also reported a similar disease which was locally called 'Pat futa' or 'skin hemorrhage' of cattle in Nawada, Gaya, Bihar and Orissa states of India. He also noted its common occurrence during the summer and rainy seasons and its frequent co-existence with another disease called 'nasal granuloma' (probably caused by *Schistosoma nasale*). Tubangui (1934) and de Jesus (1934) called the disease 'hemorrhagic filariasis' of cattle in the Philippines. Tubangui (1934) was able to describe a female parasite from a skin nodule collected from Tanauan, Batangas Province in Luzon and named it *Parafilaria bovicola*.

Further reports from India included those by Rai *et al.* (1960), Sahai *et al.* (1965), Khajuria (1966) and Srivastava *et al.* (1972). The occurrence of the disease was noted in the eastern regions and Mathura District of Uttar Pradesh, in the plains of Jammu Province and in the Bahr area of Patna. Khajuria (1966) again noted the seasonal occurrence of the disease which was locally known as 'Seraun Phutana'.
The occurrence of the disease in Indian water buffaloes was first reported by Srivastava and Dutt (1959) in a military farm in Namkum, Bihar State. Chauhan, Arora and Ahluwalia (1974) isolated the parasite from the eye of a water buffalo in Uttar Pradesh, India.

The disease has also been reported from several countries in Russia, Africa, Europe and North America. Masyukov (1941) reported it in cattle in the Caucasus (Russia). Fain and Deramee (1949) and Fain and Herin (1950, 1955) called the disease 'dermatorragie parasitaire' of cattle in Ruanda Urundi. In Rumania, Metianu (1949) reported 'parafilariose hemorrhagique des bovines' affecting a small percentage (3%) of cattle.

According to Soulsby (1965) the disease possibly occurred in the southern coast of France. This may have been confirmed by reports of Dryden (1967), Niilo (1968) and Webster and Wilkins (1970) on the occurrence of characteristic lesions in Charolais cattle recently imported from France to Canada. Clinical cases were reported from Saskatchewan and from Edmonton and Leithbridge, Alberta. The authors claimed that the disease did not spread from these places.

Pienaar and van den Heever (1964) first reported the disease in South Africa. While carrying out routine meat inspection in a government farm at Mara, Northern Transvaal, they discovered oedematous lesions in the dorsal aspects, loins, thoraces, humps and necks of beef carcasses. They were able to recover from these lesions filariform nematodes which Dr. Ortlepp of the Veterinary Research
Institute at Onderstepoort identified as *Parafilaria bovicola*. After seeing similar lesions in 2 cattle necropsied in a farm 30 miles west of Mara, the authors suggested that the disease may have a wide prevalence in South Africa. Carmichael and Koster (1978) carried out a preliminary survey of the disease in cattle slaughtered at abattoirs in Pretoria, Johannesburg and Durban. They traced these cattle to their farms of origin and were then able to cover a wider area and carry out extensive studies on the prevalence of the disease and its relationship to climate and other factors present in South Africa. According to them, the average prevalence rates were 35.9%, 13.5% and 12.2% in northern Transvaal, northern Cape Province and southwestern Transvaal respectively. Lesser infection rates were noted in Natal (6.2%), western Orange Free State (1.9%), eastern Orange Free State (1.8%) and South West Africa (0.7%). The highest rates (31.6%-47.0%) were recorded from the Bushveld areas of Northern Transvaal. In these areas, the optimum conditions for the occurrence of the disease were determined and included an annual rainfall of 400-700 mm, a frost period of less than 120 days, a mean annual temperature of 17.5-22.5 °C and an altitude of 800-1200 m. The number of bleeding and carcass lesions was greatest during the period August to January.

According to Gulati (1934) and Lahiri (1934), the disease affected mostly plough and cart bullocks in India; cows were rarely affected. The condition was not seen in water buffaloes until 1959 when Srivastava and Dutt claimed a "large number" of these animals were infected in Bihar.
The condition was later seen by Patnaik and Pande (1963). Metianu (1949) stated that the disease occurred mainly in animals between four and five years of age. However, Carmichael and Koster (1978) have shown in their extensive studies that the incidence of parafilariasis decreased in animals after about four years of age and that the highest rates recorded were in those animals older than about two years. No cases have been reported in young animals.

Metianu (1949) also stated that infections were unrelated to the sex or plane of nutrition of the host.

Transmission

The mode of transmission of Parafilaria bovicola has not been fully elucidated. Fain and Herin (1955) have suggested that Musca domestica, the domestic fly, may act as intermediate host. They found that the eggs and microfilariae ingested by this fly can undergo development in the intestines. Nevil (1975) collected a total of 10,093 flies from cattle in Transvaal, South Africa and dissected these to examine for the presence of P. bovicola larvae. Infective larvae were found in the probosces of 33/4347 Musca lusoria, 8/861 M. xanthomelas and 1/461 Musca n.sp. The natural infection rates for the two former species were 8.99% and 4.82% respectively. These flies were successfully infected in the laboratory, the rates found being higher and ranging from 40-52.8%. The larvae from artificially fed flies were found to be morphologically similar to those of naturally infected ones. Although these findings
indicated that development of *P. bovicola* can occur in species of *Musca*, the ability of these flies to transmit infective larvae remains to be proved. Nevil (1975) suggested that, since these flies feed mainly on the lachrymal secretions of cattle, there is a strong possibility of an orbital route of infection. Chauhan, Arora, Agrawal and Ahluwalia (1976) have also suggested this after finding an immature *P. bovicola* in the lacrimal duct and third eyelid of a water buffalo.

Pathogenesis and Pathology

Initial signs of infection, according to Gulati (1934) and de Jesus (1934), consist of the formation under the skin of nodules 5-7 mm high and 12-15 mm in diameter at the base. They appear slightly elevated and have a tough consistency on palpation.

Although these lesions are variably distributed on the body, the commonly affected sites are the back, neck, chest, quarters, legs and the perineum. According to Metianu (1955) the number of subcutaneous nodules varies from 1-10 per animal. The lesions in Philippine cattle are of variable duration, occurring during the period December to July (de Jesus, 1934). During this time some of the subcutaneous nodules swell up to a maximum of 40 mm base diameter and become painful on palpation and then rupture and bleed profusely. A fistulous tract may be seen from the apex of each bleeding nodule through which blood, necrotic tissue and the eggs and larval forms of the
parasite are extruded. Bleeding is manifested by the formation of coagulated blood streaks about 8-50 cm long on the flanks of the animal; this is considered one of the most characteristic signs of the disease by Metianu (1949) and Niilo (1968). Animals showing extensive bleeding may become debilitated (Gulati, 1934; de Jesus, 1934).

The pathogenesis of the infection has been elucidated by Metianu (1949) and Patnaik and Pande (1963). The bleeding which was preceded by the formation of subcutaneous nodules, was found to be due to the activity of gravid female worms. These migrate from the hypodermal connective tissue to the more superficial layers of the skin for oviposition. While doing so they pierce the dermis and epidermis and make an opening 0.5-1.0 mm in diameter through which blood containing eggs and microfilariae are released. Oviposition, according to Baumann (1946) and Webster and Wilkins (1970), takes place when the skin of the host is exposed to bright sunlight.

The movement of the parasite incites a marked inflammatory response from the host. Histopathologic evidence of these changes is given by Patnaik and Pande (1963) and Pienaar and van den Heever (1964). The former found 'tunnels' in the dermis containing gravid females and surrounded by an inflammatory reaction consisting mainly of lymphocytes and neutrophils. Eosinophils were rarely seen in this case. However, the latter author showed dense infiltration of eosinophils in 'tract-like' necrotic lesions seen in the subcutis.

Pienaar and van den Heever (1964), in examining
infected carcasses, also found oedematous plaques irregularly distributed in the subcutaneous tissue. These were greenish-yellow, irregularly circumscribed and measured from 7.5-15 cm in diameter. Histopathologic examination revealed dead parasites surrounded by zones of polymorphs and palisading macrophages. A thin layer of connective tissue surrounded the lesion and was infiltrated with lymphocytes and plasma cells.

Viljoen and Boomker (1977) studied the histopathology of lesions in animals treated with levamisole hydrochloride. At four to six weeks post-treatment, there was an acute inflammatory reaction surrounding dead parasites (presumably the effect of treatment). The worms appeared autolyzed with moderate mineralization. There were also oedema, fibrin deposition, perivascular infiltration as well as migratory tracts surrounded by eosinophils and hemosiderin-laden macrophages. An animal examined 8 weeks after treatment had developed a chronic response with marked fibrosis. At 9-12 weeks post-treatment, complete healing occurred and there were no parasitic granulomas observable.

**Importance**

Severe infestation leading to extensive hemorrhages may cause emaciation and debilitation of animals. As mostly bullocks or work oxen are affected in Asian countries, particularly India, the disease may have some effect on the agricultural economy (Khajuria, 1966).

Pienaar and van den Heever (1964) have suggested
that heavy infestation may also result in damage to carcasses and hides which may require trimming off of affected parts. This would further lead to disfigurement and early spoilage of affected carcasses and hides with subsequent reduction in their price. Carmichael and Koster (1978) found 33% of all carcass condemnations at the abattoir in Pretoria, South Africa were due to parafilariasis. Depending on the original carcass grades, downgrading and trimming off of affected parts were done in 0.2%-12.9% and in 11.2%-16.2% respectively, of affected carcasses. There was also a decrease in the market value of affected carcasses ranging from a difference of 0.72 cents to 1.20 cents per kilogram.

Diagnosis

Presumptive diagnosis is based on the seasonal occurrence of the condition, clinical signs and the presence of characteristic lesions. It is difficult to confirm this by isolating the parasite as very few can be found in the lesions especially older ones (Niilo, 1968; Webster and Wilkins, 1970). Where confirmation is attempted, the skin at the apex of the nodule should be shaven clean and then gently squeezed; any female worm appearing from the hole can be removed with a pair of forceps. It is important that this procedure be carried out as soon as the nodules start to bleed (Niilo, 1968).

Blood coming out from the nodules can also be examined in wet mounts for the presence of characteristic embryonated eggs and unsheathed microfilariae. The motility of the
latter is not observable when the sample collected has been refrigerated (Niilo, 1968).

Pienaar and ven den Heever (1964) suggested that a differential diagnosis should be carried out bearing in mind other conditions such as subcutaneous contusions, haemorrhages caused by biting arthropods, lumpy skin disease, urticaria and onchocerciasis of the skin.

**Control**

Control by chemotherapeutic treatment of clinical cases has been the only method tried. Antimonial drugs were the first to be used. Gulati (1934) and Lahiri (1934) claimed that potassium antimony tartrate (1% or 5% solution), injected intravenously at the rate of 100 ml (1%) or 20 ml (5%) per animal, can effect clinical recovery in the majority of cases with cessation of local haemorrhages, after one or two dosings. Sahai et al. (1965) tried sodium antimony tartrate 2% solution in a course of 6 intravenous injections given twice a week at the rate of 20 ml per animal; they also tried two other drugs, trichlorphon and antrypol. They found sodium antimony tartrate highly effective; of 11 cases given the full treatment clinical signs disappeared in 7 while the rest showed remissions because according to the authors, they were only given partial treatment (owing to their debilitated state). Khajuria (1966) and Srivastava et al. (1972) found antimosan to be highly effective. The latter authors reported that 23 of 30 animals recovered with disappearance of the nodules after being given 20–30 ml
of antimosan intramuscularly on alternate days for a total of six doses; 21 of these animals remained cured for three months.

Viljoen and Boomker (1977) reviewed the use of antimonial drugs by the Indian workers and tested the effect of sodium antimony tartrate in reducing the occurrence of parasitic nodules in a group of 9 infected animals. They noted a reduction of 24% in the number of lesions.

The same authors also tested trichlorphon, arsenic trioxide, levamisole hydrochloride and nitroxynil. Except for trichlorphon which was found ineffective, a greater percentage decrease in the number of lesions was noted with arsenic trioxide (38%), levamisole hydrochloride (76%) and nitroxynil (93%) than with sodium antimony tartrate. These drugs were used at high dosage rates and for nitroxynil, which is routinely administered at the rate of 10 mg/kg body weight, the authors suggested four successive injections in order to effect a cure. Treatment with levamisole hydrochloride did not result in immediate clinical recovery and the lesions took 8 weeks to heal. The authors recommended that 9 weeks is allowed after treatment before animals are slaughtered. Wellington (1978) confirmed the findings on the use of nitroxynil and showed that this could possibly be the drug of choice.
Discussion

Parafilariasis may be considered an important disease since it causes general debility in work animals in the Far East, and condemnation and downgrading of carcasses and hides in South Africa. However, no evidence of well-defined epidemiological studies in the Far East can be found in the literature. In India, there has been no accurate determination of what percentage of the cattle and buffalo population is affected and what number of draft animals suffer from extensive infestation and exhibit signs of debility. Consideration of the importance of this disease in this country cannot, therefore, be properly substantiated. It can only be surmised that, since conditions in the tropics allow for a large number of vectors to effectively transmit the parasite, a large proportion of the draft cattle population may be severely affected. However, it is known that microbial and nutritional diseases can also cause debility in animals. It is, therefore, necessary to establish in the first place the actual role of parafilariasis in draft animals by carrying out controlled studies to determine whether severe infestation is related to debility.

Prevention of this disease in the tropic necessitates determining what vectors are involved in transmission. The experiments of Nevil (1975) in South Africa have suggested that muscid flies may act as intermediate hosts for the parasite. It is possible that these flies may also be involved in the Far East. The control of Musca spp. as well as other arthropods has been reviewed in detail by Beesley.
The use of insecticides such as DDT and pyrethrum against the adult flies is generally recommended. Since these flies breed on moist organic matter, specific measures such as the proper disposal of manure, offal and spoiled feeds can be taken on the farm. The wide-scale application of insecticides can only be justified if the economic losses due to parafilariasis are found to be considerable.
II. STEPHANOFILARIAISIS

Stephanofilariasis refers to a number of chronic dermatitides commonly known as 'sores' occurring in various parts of the skin of ruminants notably the ox and water buffalo. Species of Stephanofilaria have been implicated as the causal agents, most of them distributed in the Far East.

Aetiology

Six species have been described and known to be pathogenic for domestic ruminants. They are Stephanofilaria dedoesi (Ihle and Ihle-Landenberg, 1933), S. stilesi (Chitwood, 1934), S. assamensis (Pande, 1936), S. kaeli (Buckley, 1937), S. zaheeri (Singh, 1958) and S. okinawaensis (Ueno and Chibana, 1977). These are typically small and slender nematodes. The male and female of S. dedoesi measure 2.3-3.2 mm and 6.1-8.5 mm long respectively. The mouth is circular and surrounded by a cuticular rim bearing a number of small spines. Posterior and adjacent to this rim is a row of 'cephalic' spines interrupted by amphids. The esophagus is short and not divided. The male tail bears a number of preanal papillae (Ihle and Ihle-Landenberg, 1933).

These species have largely been differentiated by their morphological characteristics. Lateral alae are present only in S. stilesi and S. assamensis (Chitwood, 1934; Pande, 1936). S. kaeli may be differentiated from
S. zaheeri in having a greater number of preanal papillae, 13 to 15 pairs as compared to 8 pairs only in the latter (Singh, 1958). No preanal papillae have been described for S. dedoesi and S. assamensis. Singh (1958), using data provided by previous workers, noted the absence of a distinct anus in the females of S. dedoesi and S. assamensis. Other anatomical criteria such as the size of the male left spicule (Pande, 1936; Singh, 1958) and the number of cephalic spines (Patnaik, 1964) were used by Ueno and Chibana (1977) to differentiate S. okinawaensis.

The morphology of the larval forms of S. assamensis, S. kaeli, S. stilesi and S. okinawaensis is described in detail by Sen, Sinha, Chowdhury and Ray (1956) and Patnaik (1973); Fadzil (1975); and Hibler (1966); and Ueno and Chibana (1977) respectively. The microfilariae found in the dermis of the affected skin is enclosed in a sheath or egg membrane. When they migrate to the more superficial layers of the skin, their sheaths are lost. The microfilariae of S. assamensis and S. kaeli and S. okinawaensis are similar in appearance and measure about 109 μm x 6 μm. The anterior end is blunt while the posterior end tapers gradually. The microfilariae of S. stilesi is much smaller, measuring only 52 μm x 3 μm. No description of the microfilariae of other species could be found in the literature.

Occurrence and Species Affected

S. dedoesi causes 'cascado' or prurient dermatitis in cattle, goats and water buffaloes. It was initially
recorded from two large Indonesian islands, North Celebes and Sumatra (Bubberman and Kraneveld, 1933) and thence in Sumbawa (Kraneveld, 1935). It is now considered to be widespread in Indonesia. Bubberman and Kraneveld (1933) reported the occurrence of the disease in 90% of cattle during the rainy season. Muchlis and Soetijono (1973) claimed a 100% infection rate in adult cattle in endemic areas of Sulawesi.

*S. stilesi* causes 'summer sore' of cattle in many parts of the world. Infections have been reported from North America by Dikmans (1934) and Chitwood (1934) and from Russia by Ivashkin, Khromova and Shimotova (1963). The possible occurrence of the parasite has also been noted in Colombia (Guzman and Morales, 1976). Maddy (1955) and Hibler (1966), working in the United States of America, noted the high prevalence of infection among cattle managed under rangeland conditions and in irrigated pastures. Maddy claimed that infection rates can be as high as 80-90% in beef herds in the western parts of the United States.

*S. assamensis* infects the hump, hence the disease 'humpsore', of zebu cattle in India and East Pakistan. It has been reported from several low-lying areas of India including Assam, West Bengal, Orissa, Andhra Pradesh, Bihar and Guharat (Srivastava and Malviya, 1968; Pal and Sinha, 1971). A quarter to a third of the cattle population in these areas may be affected at one time (Srivastava and Malviya, 1968; Ahmed and Ali, 1973). In the Mymensingh District of East Pakistan, 'humpsore' is considered to be a commoner disease than either mange or warts (Mia and
S. kaeli produces a dermatitis known as 'Krian sore' in the legs of cattle and goats in Malaysia. Fadzil (1977) listed 8 endemic foci of the infection on the west coast of the Peninsula. He noted that its occurrence was related to climatologic factors such as high relative humidity and ambient temperature but not to the terrain nor the types of vegetation present.

S. zaheeri affects the ears ('earsore') of water buffaloes in the southern parts of India. A study by Ahmed (1961) in the Hyderabad District showed 70-80% infection rates in adult water buffaloes. The same prevalence in dairy buffaloes was found by Agrawal and Dutt (1977).

S. okinawaensis causes dermatitis of the muzzle and teats of cows in Japan. It was first reported by Kono in 1965 from the Amami and Ryukyu Islands. Ueno, Chibana and Yamashiro (1977) have also noted it in the island of Nansei and reported incidences of 25% in 1,678 Japanese Black cattle and Holstein hybrids reared on pasture, and 23% in 1,396 semi-grazing cattle.

Infections by the above parasites are mostly observed in adult animals (Ahmed, 1961; Mia and Haque, 1967; Fadzil, 1973, 1977; Ueno et al., 1977). S. zaheeri has been found to infect mostly adult buffaloes. 'Humpsere' in East Pakistan was not seen in animals less than two years old, while clinical cases of 'Krian sore' in Malaysia were recorded only in animals from one and a half years of age. A field survey in Japan also showed that cattle less than 16 months of age were rarely infected with S. okinawaensis.
Transmission

Climatologic factors such as high rainfall, ambient temperature and relative humidity are associated with stephanofilariasis and indicate the essential role of insect vectors as transmitters of the disease (Bubberman and Kraneveld, 1933; Fadzil, 1977).

Rahman (1957) stated that stephanofilarial infections are transmitted mechanically by muscid flies. However, Ivashkin, Khromova and Shimotova (1963), Hibler (1966), Patnaik (1973) and Fadzil (1975) have shown that these parasites require prior development in an intermediate host into the infective stage. Microfilariae present in the peripheral part of skin lesions are taken in by an insect vector (Musca conducens for S. assamensis and S. kaelii; Haematobia irritans and Lyperosia titillans for S. stilesi) and develop as first stage larvae either in the abdominal fat or haemocoel of the insect. Here, they transform into broad crescentic forms known as 'sausage' stages, then undergo two ecdyses to become third stage larvae. They consequently migrate to the thorax as infective stages and finally to the proboscis through which they can be deposited onto the skin of the host. Further development into adult forms in the host was studied by Hibler (1966) who found that the prepatent period was 6-8 weeks in S. stilesi.

Blood-sucking flies are suspected to be the biological vectors for S. dedoesi (Bubberman and Kraneveld, 1933). Although no well-defined transmission experiments have
been carried out with *S. zaheeri*, larval forms were claimed to be present in *Musca autumnalis* by Patnaik and Kumar (1972). No data is available on the mode of transmission of *S. okinawaensis*.

**Pathogenesis and Pathology**

The entry of infective larvae into the skin of the susceptible host is made possible by a pre-existing lesion which is often caused by biting flies or by some mechanical means, e.g. damage to the skin of the hump brought about by pressure from the yoke (Dewan and Rahman, 1970). A notable exception to this is *S. stilesi* which is injected directly into the skin by the vector. Initiation of lesions is also believed to be by pre-infection of the wound by *Staphylococcus aureus* and *Staphylococcus albus* (Dewan and Rahman, 1970; Fadzil, 1977).

The lesions of *S. dedoesi* have been described in detail by Bubberman and Kraneveld (1933) and Kraneveld (1935). They commonly occur on the skin of the neck, withers, dewlap, shoulders and around the eyes of cattle and goats and on the ears of water buffaloes. They appear initially as small papules and, later, they coalesce to form larger encrusted lesions which may reach a diameter of 25 cm. The hair falls out of the affected area and the skin becomes inflamed and oedematous. Intense pruritus brought about by these lesions may occasionally cause the animal to rub the affected parts against hard surfaces, thus aggravating the lesions. The adult worms cause inflammation of the
rete Malpighii and destruction of the hair follicles, skin glands and epithelial cells. These lesions may resolve spontaneously.

The lesions of *S. stilesi* occur commonly on the mid-ventral surface of the body. They have been described by Dikmans (1934), Levine and Morrill (1955) and Turk and Batte (1963). They vary in size from 2.5-15 cm in diameter. The affected skin is thickened due to proliferation of the stratum corneum, and becomes encrusted. Degeneration of the hair follicles and sebaceous glands leads to intense pruritus which may cause the animal to rub off the crusts; ulcers are thus formed which later become suppurative. The adult worms are found in cyst-like structures at the bases of hair follicles, surrounded by a cellular infiltrate consisting of lymphocytes, neutrophils and eosinophils. The microfilariae are located in the more superficial layers of the lesions.

The lesions of *S. assamensis* occur mostly on the hump area but may also be found in other parts of the body. They begin as multiple infiltrations in the dermis which cause intense itching. The lesions coalesce and dry off at the surface forming scabs which are later rubbed off by the animal. A healing ulcer is thus formed with exuberant granulation tissue which gives it a cauliflower-like appearance. In extreme cases, the sore may involve the entire region of the hump. It may attract large numbers of flies and birds which may do further damage and keep it fresh, active and bleeding (Pande, 1935 and Mohan, 1945). The sore dries up during cold weather but may become active
again during the warm rainy season.

The lesions of *S. kaeli* have been described in the works of Buckley (1937) and Loke and Ramachandran (1967). The sores appear mostly on the lower legs but may be found in the ears and teats. They vary in size from simple lesions about 2.5 cm in diameter to fungating growths approaching 10-15 cm, usually occurring on the legs. The smaller sores are slightly raised, firm in consistency, and are usually devoid of hair. The large sores may greatly increase the diameter of the leg by as much as 5 cm. They have a dry, crusty surface with cracks and indentations and they bleed profusely when cut. The affected animal frequently licks the sores which are also attractive to flies. Microscopically, there is proliferation of the stratum corneum with formation or rete-peggs. Cyst-like structures containing adult worms may be seen in the dermis adjacent to the tip of these epidermal prolongations. The dermis is remarkable in having an increased number of lymphatic vessels and a cellular infiltrate consisting of lymphocytes and macrophages which may be dense in the perivascular areas.

The lesions of *S. zaheeri* have only been described in the ears of water buffaloes (Agrawal and Dutt, 1977, 1978). They are essentially similar to those described for other species. These develop also in several stages from macules and papules, which coalesce to form encrusted scabs, to ulceration. The dermis is also thickened with oedema and cellular infiltration. In advanced stages, active ulcers may be seen.
The lesions of *S. okinawaensis* have been described as 'leucoderma' of the muzzle and teats of cattle (Kono, 1965; Ueno, Chibana and Yamashiro, 1977). Initially, the affected skin becomes swollen and depigmented and shows varying degrees of hyperkeratosis. The surface becomes covered with blood and cellular exudates which later dry up to form scabs. The lesions are histologically similar to those previously described in other species.

**Importance**

No quantitative data are available on the economic losses brought about by infections with these parasites. Mia and Haque (1967) considered 'humpsore' as the major cause of condemnation and downgrading of carcasses and hides in East Pakistan.

Ueno et al. (1977) have suggested that infection of the teats of milking cows by *S. okinawaensis* may disrupt normal milking procedure and affect milk yield.

**Diagnosis**

Dikmans (1934) suggested the use of skin scrapings for the diagnosis of *S. stilesi*. The lesions can be processed in a 'meat grinder' and the worms and larvae collected with a Baermann apparatus. Deep skin biopsies can also be treated in the same manner. Ueno et al. (1977) described a procedure in which pieces of tissue, 1.0-1.5 cm by 0.3-0.4 cm may be removed surgically, placed in a petri
dish containing physiological saline and left overnight at room temperature; worms moving out of the tissue may be collected with a pipette.

According to Griffiths (1974), there should be no difficulty in recognizing the gross lesions of stephanofilariasis especially if they occur in animals from known endemic areas.

Control

Chemotherapy has been the only method described for the control of stephanofilariasis. For 'cascado', Muchlis and Soetijono (1973) suggested the application of 2% coumaphos (Asuntol, Bayer AG Leverkusen) in vaseline; they claimed that healing is rapid especially in rested animals, taking on the average two weeks post-treatment.

Several authors have also claimed successes in the treatment of 'humpsore'. Srivastava and Malviya (1968) and Patnaik (1970) showed that topical applications of 6-8% trichlorphon (Neguvon, Bayer AG Leverkusen) in oil base have a curative effect; however, a long period of treatment (up to 40 days) may be required in order to prevent remission of lesions. Dewan and Baki (1976) have claimed that 20% trichlorphon ointment with added sulphanilamide cures all cases within 10-20 days.

Dutta and Hazarika (1976) have found that tobacco ointment containing tobacco leaf decoction and creosote in vaseline base was highly effective; of 10 animals treated, 9 were cured after 12 days treatment.
For the treatment of 'ear sore', Roychowdhury and Chakrabarttz (1969) suggested the use of 4–10% formalin in order to produce necrosis and sloughing off of the proliferative lesions. Agrawal and Dutt (1977) found 6% malathion ointment moderately effective; of 12 severe cases treated, 7 totally recovered while the rest showed signs of improvement.

Fadzil (1977) found that trichlorphon, at either 6% or 10% concentration combined with vaseline, coconut oil or linseed oil and applied topically, cured 100% of cases of 'Krian sore'.

Discussion

No reports could be found in the literature of specific studies on the effects of *S. assamensis*, *S. dedoesi* and *S. kaeli* on the performance of draft cattle. However, it may be safely assumed that these parasites cause pain and loss of condition which may put these animals out of work, especially during the rainy season when they are needed to plough the fields.

Similarly, very few studies could be found on *S. stilesi* in North America and Russia. In countries like these where resources are available for extensive studies to be carried out, it is surprising that no data is available on the economic effects of this parasite on livestock. It is therefore likely that its prevalence is so low that it has received little attention.

Ueno *et al.* (1977) have suggested that infections with *S. okinawaensis* may be important as it affects the teats of dairy cows. Further studies are needed to quantify this and to determine whether control measures are necessary for this parasite.

The control of these parasites has only been carried out through the chemotherapy of clinical cases. The cost of this can be very high considering the wide prevalence of infections in certain areas, the high rate of transmission by vector flies, and the relatively expensive drugs. Preventive measures may also be possible against some vector species. Measures described against muscid flies have been briefly described and these can be applied similarly to hornflies (Beesley, 1973).
III. ONCHOERCIASIS

Onchocerciasis of domestic ruminants refers to several infections occurring in the nuchal, gastro-splenic and joint ligaments, subcutaneous and intermuscular connective tissues, and aortae caused by species of Onchocerca. Although striking granulomas may be seen in these sites, generally no significant signs are manifested. These infections are thus regarded of little veterinary importance (Nelson, 1970; Eichler and Nelson, 1971).

Aetiology

A large number of onchocercal species appeared in the comprehensive list of Skrabin (1969). These included Onchocerca caprae (von Linstow, 1883), O. lienalis (Stiles, 1892), O. armillata (Railliet and Henry, 1909), O. gibsoni (Cleland and Johnston, 1910), O. bovis (Piettre, 1912), O. indica (Sweet, 1915), and O. cebet (Gaillard and Cebe, 1937). Four additional species have been described recently, O. ochengi (Bwagamoi, 1969), O. sweetae (Spratt and Moorhouse, 1971), O. dukei (Bussieras, Ameguee and Bain, 1974), and O. dermata (Bussieras et al., 1974).

There has been wide controversy as to the taxonomical validity of some of these species. Skrabin and Shikhobalova (1945) found O. caprae, O. bovis and O. indica incompletely described and regarded these as species inquirendae. Caballero (1945) likewise questioned the validity of O. caprae. The reasons for the superficial description of
these species may have been provided earlier by Baylis (1939) who found great difficulty in obtaining complete specimens of these parasites "owing to their great length and fragility and to the density of the tissues that they commonly inhabit." Further, the lack of constant taxonomical characters causes difficulties in differentiating these species. Eichler (1973b) found that the differences in the morphology of *O. gutturosa* and *O. lienalis* were variable and insufficient to justify considering them as distinct species.

Caballero (1945) established the synonymy of these species and placed *O. lienalis* and *O. bovis* under *O. gutturosa*, and *O. cebei* and *O. indica* under *O. gibsoni*. Bain, Bussieras and Amgeee (1976) considered *O. dermata* identical to *O. ochengi*. Following these authors, the valid species are then *O. armillata*, *O. gibsoni*, *O. gutturosa*, *O. ochengi*, and possibly *O. sweetae* and *O. dukei*.

The morphological description established by Diesing in 1841 for the members of the genus *Onchocerca* appears in the works of Baylis (1939) and Skrabin (1969). The nematodes referred to are elongate, filiform or thread-like in appearance. The mouth is simple and devoid of lips. The female cuticle is thick with transverse or annular striations or thickenings which may be spirally arranged. The oesophagus is short and somewhat divided into an anterior muscular and a posterior glandular portion. The male tail is spirally coiled and devoid of caudal alae (except in *O. armillata*); the spicules are
unequal, the left being longer, and dissimilar. The female tail is conical and bears a pair of small papillae. The vulva is situated in the region of the oesophagus. The female is viviparous, producing microfilariae which are unsheathed.

**Occurrence and Species Affected**

*O. armillata* has been recorded in Asia including Asia Minor, and Africa. According to Levine (1969), the distribution in Asia includes India, China, Malaysia, Indochina and Indonesia. Most reports have come from India. Prevalence rates ranging from 90-95% were found in cattle in the districts of Mukteswar, Mathura and Bihar (Bhalerao, 1936; Varma, 1953). Patnaik (1962) found the parasite in 146 (99%) of 148 aortae examined at an abattoir in Orissa; he also found microfilariae in the blood of 68 pre-slaughter cattle. The parasite has also been isolated from the aortae of pigs in Madras (Ramanujachari and Alwar, 1953), water buffaloes in Uttar Pradesh (Singh and Singh, 1970), and goats from the Indian Veterinary Research Institute at Izatnagar and from a slaughterhouse at Patna–Danapur (Prasad, Rajya and Mohanty, 1975). Very high infection rates were initially reported from Africa. Chodnik (1957) found 95% of cattle infected in Ghana, while Abdel-Malek in 1958 reported a prevalence of 94–96% in cattle in Sudan. However, Clarkson (1964) found the parasite in only 2.1% of 280 aortae of cattle examined at abattoirs in Kenya; he confirmed an
earlier observation by Nelson, Heisch and Furlong (1962) that the parasite does not occur in the coastal provinces of Kenya. *O. armillata* has recently been reported from Togo (Bain, Denke, Amegee and Chabaud, 1977). Recent surveys carried out in Asia Minor included those by Alibasoglu, Golesuk, Erturk and Guler (1969) who found the parasite in 86% of cattle in southern Turkey; Al-Zubaidy (1973) who reported it in 92% of cattle in Iraq; and Cheema and Ivoghli (1978) who found it in 28% of cattle examined in Shiraz, Iran.

*O. gibsoni* infects cattle and water buffaloes in Australia, the Far East and some parts of Africa. Seddon and Albiston (1967) have mapped out the distribution in cattle in Australia. The highest prevalence rates have been observed in the northeastern states of Australia, particularly in Queensland and the northern parts of the Northern Territory. Endemic areas included New South Wales, the northern parts of South Australia, Victoria, and the northern parts of Western Australia. In the Far East, Buckley (1937) found that the parasite was common in Malaysia, Thailand and Bali, and reported an incidence in cattle of 90–95%. Isshiki (1963) collated reports of the occurrence of the parasite in water buffaloes of Burma, the Belgian Congo, Formosa, Indonesia and Pakistan. He also studied the parasite in the southern provinces of Korea and reported a prevalence rate of 9.8% in cattle. He considered Southeast Asia the reservoir area for infections with the parasite.

*O. gutturosa* occurs in cattle and has a wide distribution. Reports have come from practically every
inhabited continent. The earliest surveys which were carried out in Europe showed varying rates of infection. In Great Britain, Webber, Crisp, Wright and Williams (1957) reported a 25% infection rate in cattle from an abattoir in North Wales, while Venkataratnam and Kershaw (1959) noted a similar prevalence in an abattoir in Liverpool. A recent study by Eichler and Nelson (1971) showed much higher figures; 58% of 1583 cattle were found infected at an abattoir in Reading, and 84% of 50 cattle at an abattoir in London Colney. In Denmark, Kolstrup (1975) reported an average infection rate of 9.4% in cattle from four localities. Elsewhere in Europe, no survey reports are available, but, according to Kolstrup (1975) the parasite has been recorded in Austria, Rumania and the USSR. According to Levine (1969), bovine onchocerciasis does not seem to be common in North America. Scholtens, Adams, and Broderson (1977) found it only in 12.4% of 1000 cattle slaughtered in Georgia, U.S.A. Very few reports are available from Central and South America. In Guatemala, Gibson (1952) reported an infection rate of 87% in 148 cattle examined. According to Levine (1969) the parasite has also been reported from Puerto Rico, Argentina, Venezuela and Uruguay. Seddon and Albiston (1967) considered *O. gutturosa* to be highly prevalent in New South Wales and Queensland in Australia. Ottley and Moorhouse (1978) quoted infection rates of up to 100% in abattoirs in the northern parts of the country. The parasite has also been seen in Victoria and is suspected to be present in Western Australia (Seddon and Albiston,
In Africa, interest in *O. gutturosa* was initially from the taxonomical viewpoint and epidemiological studies were relatively scarce (Neumann, 1910; Fain, Herin and Thienpont, 1955). It was not until the last decade that prevalence surveys were carried out in Kenya, Uganda and Sudan. Studies have also been carried out in Ethiopia, Chad and the Central African Empire, according to Elbihari and Hussein (1978). Clarkson (1964) surveyed the Athi River, Mombasa and Kisumu abattoirs in Kenya and found the infection rates to be 26/623 (4.2%), 13/199 (6.5%) and 71/898 (7.9%), respectively. An undetermined number of infected cattle came from Somalia via Lamu and were slaughtered at Mombasa. Bwangamoi in 1970 reported the results of a survey which was carried out in municipal abattoirs located at Kampala, Mbarara, Gulu and Lira in Uganda. An average infection rate of 82% was noted in these places although very few animals (only 50 cattle) were examined. Surveys carried out by Hussein, Abdel Nur, Gassouma and Nelson (1975) in Khartoum, Sudan showed 170 of 248 (68.5%) cattle infected at the Omdurman Central Abattoir. There are relatively few available reports from Asia. Gaiger (1915) and Baylis (1939) described the parasite under the name *O. lienalis* from specimens collected from India and Ceylon. In the Mathura District of India, Pande and Ahluwalia (1964) claimed that infections are commoner with *O. gutturosa* than with *O. gibsoni* although they did not quote any figures to substantiate this. In Iran, Cheema and Ivoghli (1978) found the parasite in 82 (28.8%) of 284 nuchal ligaments
and 11 (7.85%) of 140 rumenosplenic ligaments examined at Shiraz.

Infections with *O. ochengi* (syn. *O. dermatata*) have only been recorded in cattle from East and West Africa. Bwangamoi (1969a, 1969b) noted the occurrence of nodules caused by *O. ochengi* in raw hides collected from Monduli, Korogwe and Dar es Salaam in Tanzania; Gulu in Uganda; and Addis Ababa in Ethiopia. At the Gulu abattoir, he examined pre-slaughter cattle and found the nodules in 50% of these. Bain *et al.* (1977) also reported the occurrence of *O. ochengi* in cattle from Togo.

*O. sweetae* has only been recorded in water buffaloes from the Northern Territory of Australia (Spratt and Moorhouse, 1971).

The occurrence of *O. dukei* in West African cattle was first reported by Bussieras *et al.* (1974). Bain *et al.* (1977) studied this species in detail and noted its more limited distribution. He found the parasite in North Togo, Benin, and Upper Volta; at the Lama-Kara focus, he found 50% of cattle infected.

**Transmission**

Steward (1937) found that *Simulium ornatum* and, to a lesser degree, *S. erythrocephalum* can act as vectors for *O. gutturosa* in England. *S. ornatum* was subsequently found to be also the vector in Russia, Austria and Denmark (Gnedina, 1950; Supperer, 1952; Kolstrup, 1975). The development of *O. gutturosa* larvae in this fly has been described by Steward (1937) and Eichler (1973).
Ingested larvae begin their development in the midgut and then migrate in a few days time to the thoracic muscles. Here, they become sausage-shaped measuring 200 μ x 18-22 μ after about 10 days. They then undergo a rapid phase of growth after which they migrate to the proboscis as infective larvae in 19-22 days.

In England, Eichler (1971) studied the behaviour of S. ornatum in relation to its ability to transmit the microfilariae of O. gutturosa. He found that the fly fed mostly in the umbilical area of the skin of cattle where, fortuitously, the largest concentration of microfilariae are present. He considered this as an adaptive form of transmission. Kolstrup (1975) and Scholtens et al. (1977) also noted the same finding in Denmark and in the U. S.A. However, parallel studies carried out in Africa by Bain et al. (1977) and Elbihari and Hussein (1978) showed that the microfilariae of O. gutturosa were located mostly in the back region (hump and withers) of the skin of cattle (which suggested that, possibly, different species of vectors are involved in the transmission of the parasite in Africa).

Buckley (1938) found that Culicoides pungens, C. oxystoma, C. shortii, and C. orientalis can act as experimental vectors for O. gibsoni in Malaysia. However, he considered only C. pungens as the natural vector. He found that the development of O. gibsoni larvae in this fly requires 6-12 days.

Spratt, Dyce and Standfast (1978) found that a midge which they identified as Culicoides sp. "M" can act as a natural intermediate host of O. sweetae.
They found that the development of the parasite in this fly requires more than 8 days at 24°C.

No data are available on the modes of transmission of *O. armillata*, *O. ochengi* and *O. dukei*.

**Pathogenesis and Pathology**

*O. armillata* causes atheromatous lesions in the aorta. They have been described by Chodnik (1958), Patnaik (1962) and Cheema and Ivoghli (1978). The inner wall of the aorta is thickened and corrugated by the formation of tortuous tunnels and nodules containing the parasite. The external surface may present small hollow nodules containing the anterior ends of the females. The worms are found tightly coiled in the middle layer of the aorta. They do not penetrate into the intimal lining, but may cause punctate haemorrhages on it. Patnaik (1962) found that the degree of sclerosis or thickening of the aortic wall depends on the density and duration of the infection. No clinical signs are generally attributed to these lesions. However, Patnaik (1962) claimed that they may cause blindness and nervous signs.

The nodular lesions or 'worm nests' of *O. gibsoni* have been described in detail by Ishiiki (1963), Seddon and Albiston (1967) and Ottley and Moorhouse (1978). They are mostly seen in the subcutaneous connective tissue of the ventral region of the brisket near the fourth and fifth junctions of the sternal and costal cartilages. They may also be seen in the lateral and posterior
aspects of the femoro-tibial joint and the lateral aspect of the buttock. They are roughly globular, firm in consistency, and freely movable in loose tissue; they measure 2.0-3.5 cm in diameter. Microscopically, the lesion consists of an inner zone of tunnels in which the adults are intricately coiled, and an outer zone of dense fibrous encapsulation.

Viable adults of *O. gutturosa* have not been found to produce lesions in the nuchal and gastroplenic ligaments. However, degenerate or dead worms may incite nodular inflammatory reactions. The lesions have been described by Eichler and Nelson (1971), Hussein et al. (1975) and Cheema and Ivoghli (1978). The nodules are flat and 0.4-2 cm in diameter; they are surrounded by a thick encapsulation of fibrous tissue. Microscopically, they contain dense infiltrations of eosinophils, lymphocytes, and plasma cells surrounding the remnants of the parasite. However, no clinical manifestations have been attributed to these lesions (Eichler and Nelson, 1971). Lesions have also been seen in the lateral ligaments of the femoro-tibial joint (Herin and Fain, 1955; Webster, Dukes and Bundza, 1977).

Bwangamoi (1969b) described the nodules of *O. ochengi*. They are mostly found beneath the skin of the scrotum and udder, but they may also occur on the flanks and the head. The nodules are creamy white, roughly spherical and measure about 3-12 mm in diameter; they may aggregate into lobular lumps. Microscopically, they are encapsulated by fibrous tissue in the reticular
layer of the corium. Each nodule contains tortous cavities in which both male and female worms may be seen.

The nodules of *O. sweetae* occur in the subcutis of the pectoral region. They may resemble those of *O. gibsoni* (Spratt and Moorhouse, 1971).

The lesions of *O. dukei* occur in the intermuscular connective tissues. They resemble those of *Cysticercus bovis* (van den Heever, 1971).

**Importance**

Infections with *O. gibsoni* were, at one time, considered economically important in Australia. Removal or 'cropping' of affected briskets which was required during the period 1910-1928 by the British market caused enormous losses in meat exports. Regulations have since been modified which allow for excision of only the nodules present in briskets and removal of only the affected tissues and tendons of the femoro-tibial joints. These have greatly reduced economic losses (Seddon and Albiston, 1967).

*O. ochengi* is regarded by Bwangamo (1969b) as the most economically important filarial species affecting cattle in East Africa. Nodules caused by this parasite aggregate into masses under the skin which, after tanning, leaves craters or 'pock marks' on the underside. Affected skins are thus rendered useless for making into suedes.

There appears to be no significance attached to the infections by the other species.
Diagnosis

Clinical diagnosis of onchocercal infections of the subcutaneous tissues (*O. gibsoni*, *O. ochengi* and *O. sweetae*) can be carried out by palpating specific areas on the body for characteristic nodules. Infections with *O. armillata*, *O. gutturosa* and *O. dukei* are rarely recognized in the live animal and are encountered only at necropsy.

Laboratory diagnosis of all these infections, except *O. armillata*, can be achieved by examining skin biopsies or 'snips' for the presence of microfilariae. The method of Gibson (1952) consists of surgically removing small portions of skin about 2 mm² and placing these in several drops of physiological saline. These are left to stand for some time to allow microfilariae to migrate into the saline which is consequently examined for their presence. A modification of this method which was introduced by Kolstrup (1975) consists of placing the skin biopsies wrapped in gauze in a 10-ml syringe containing saline.

The microfilariae of *O. armillata* can be examined from the jugular blood. Patnaik (1962) claimed that they exhibit a certain degree of nocturnal periodicity.

Measurements of these microfilariae are provided in Table 3.

Control

As most of these parasites rarely cause ill-health
in their hosts, control measures against them would not be cost-effective and could hardly be recommended.

Klesov (1964) reported an effective control measure against bovine onchocerciasis in Russia. He stated that "as a result of our experiments, destruction of the pre-imaginal stadia of simuliidae in streams and small rivers prevent the adult form and reduce the disease in animals. The most effective insecticides are concentrated emulsions of nicochloran and a concentrated emulsion of polychloran: nicochloran 0.000014% and polychlorpinep 0.00014%. A series of measures against onchocerciasis, elaborated during the year 1962-1965, in unfavourable conditions of maintenance are effective prophylactic means to protect cattle against onchocerciasis."
Most of the surveys on the prevalence of onchocercal infections have been carried out in abattoirs. This probably indicates that these infections are not easily recognized in the field. Nelson (1970) and Eichler and Nelson (1971) have considered the species of *Onchocerca* as generally "unobtrusive" parasites and, indeed, this review has shown most of them to be so.

Of the species reviewed here, probably *O. ochengi* is the only parasite which can be considered at the present time to have some economic importance. The abattoir survey carried out by Bwangamoi (1969) probably indicates that the prevalence of this parasite in East Africa is high. However, field surveys must be carried out to confirm this. The life cycle of this parasite is still not known; recognition of its vectors is necessary in order that preventive measures can be properly instituted.
IV. SETARIAISIS

Setariasism in domestic ruminants occurs in two forms—firstly, as a silent infection by adult setarial nematodes of the peritoneal cavity of the natural hosts (cattle and water buffaloes) and, secondly, as a clinically manifest infection brought about by migration of immature forms into the central nervous system chiefly of aberrant hosts such as sheep and goats. The latter form, known as epizootic cerebrospinal nematodiasis or 'goat paralysis' in some countries of the Far East, is characterized by ataxia and lumbar paralysis. Lesions are often due to a single worm migrating into the brain or spinal cord.

Aetiology

Two species are involved in setariasism, Setaria labiato-papillosa Allesandrini, 1838 (syn. S. cervi) and S. digitata von Linstow, 1906 (syn. S. marshalli). These are natural parasites of the peritoneal cavity of bovines. The latter species has been dealt with in majority of the studies reviewed here, and so will be preferentially treated as a matter of convenience.

The morphological description of Viborg (1795) for the genus Setaria in which these parasites are members, appears in the works of several authors including Boulenger (1921) and Yeh (1959). The parasites referred to are short but stout nematodes. The mouth is surrounded by a protruding cuticular rim which extends into dorsal, ventral and lateral prominences or lips. The cuticle is
transversely striated though less prominently. The cephalic end is provided with four spine-like papillae. The esophagus is divided into (short) anterior and (much longer) posterior parts. The posterior end of the male is elongate, spirally twisted and bears a pair of small lateral appendages. The female posterior end is likewise elongate and spirally twisted but terminates roundly; a pair of lateral processes may be seen at the tip. The vulva is located in the esophageal region. The female is ovoviviparous, the microfilariae being enclosed in a transparent sheath.

According to Yeh (1959), the male of *S. digitata* measures 35-46 mm x 0.3-0.5 mm while the female is 65-75 mm x 0.5-0.7 mm. *S. labiatopapillosa* is slightly longer, measuring 40-51 mm x 0.38-0.45 mm and 60-94 mm x 0.6-0.9 mm for the male and female respectively. At one time these bovine species were regarded as separate from *Setaria equina* of horses and given the genus name *Artionemma* (Yeh, 1959). However, Nelson (1962) and Becklund and Walker (1969) considered the similarities in the morphology and biology of these species binding and sufficient to place them back to the genus *Setaria*.

Purvis (1931) and Baylis (1936) considered *S. digitata* synonymous to *S. labiatopapillosa*. However, Sarwar (1946), Anantaraman and Victor (1957) and Yeh (1959) have stated that these are separate species. The differential characters used by these authors included the distance between dorsal and ventral elevations which was found to be 50-100% longer in *S. labiatopapillosa*; the shape of the oral aperture, which is rounded in *S. digitata* but
elongate in *S. labiatopapillosa*; the lateral lips which are less prominent and triangular in *S. labiatopapillosa*; the terminal knob on the female tail which has large papillae in *S. labiatopapillosa* but may be smooth or only slightly papillated in *S. digitata*; and the distance of the caudal appendage from the extremity which in *S. labiatopapillosa* is twice that in *S. digitata*.

**Occurrence and Species Affected**

The distribution of *S. digitata* is probably restricted to countries in Asia (Yeh, 1959; Levine, 1969). Reports have come from Japan, Korea, India, Ceylon and the USSR (Innes, Shoho and Perumal Pillai, 1952; Innes, 1953; Ishii, Yaguria, Sugawa, Ishiwara, Ogata and Hashiguchi, 1953; Kadenatsii, 1956). From 1945 to 1949, Bush (1951) carried out a survey of 'lumbar paralysis' in domestic animals in Japan and found 65 (2.7%) cases in 2,389 sheep examined and 53 (9.5%) cases in 553 goats. These figures may not be totally representative of the cases due to setarial infection since Bush (1951) has suggested that osteomalacia or phosphorus deficiency may be the major cause of the condition. However, Innes *et al.* (1952) regarded setarial larvae as the primary agents involved and found clinical cases in 6% of 100,000 sheep examined in Fukoshima. Yoshikawa, Oyamada and Yoshikawa (1976) examined the abdominal cavities of 105 cattle slaughtered at an Aomori abattoir and found the adult worms in 58 (55%) of these animals.
There are relatively few available reports regarding the situation in countries other than Japan. However, Innes et al. (1952) have indicated that the disease is 'quite common' in sheep and goats in Ceylon; 23 cases have been recorded in goats in a number of government farms in this country. Most of these occurred during the warm months from March to October. Much of the work done in Korea has been carried out by Japanese workers who published their five-volume Special Korean Research Commission Report (1939-43); this work has not been fully translated into English (Innes et al., 1952). Mohan (1977) recovered *Setaria digitata* from the peritoneal fluid of 7 out of 9 cattle and 6 of 11 buffaloes examined in Andhra Pradesh, India.

*Setaria labiatopapillosa* is more widely distributed, having been described from China (Canton), USSR, Malaya, India, Ceylon and Northern Rhodesia (Yeh, 1959). Frickers (1948) reported an infection rate of 42% in cattle slaughtered in Surinam while in Gambia, McFadzean (1955) reported a similar rate in cattle. In Georgia, U.S.A., Becklund (1959) found 10 of 29 adult cattle infected. Pryadko (1961) found 1.3% of cattle infected in southeast Kazakhstan. Ansari (1977) considered the parasite to occur commonly in cattle especially during the rainy season in Aligarh District, Uttar Pradesh, India. The role of larvae of *Setaria labiatopapillosa* in the causation of cerebrospinal nematodiasis has not actually been determined. However, in India, Anantaraman and Victor (1957) suggested it may cause a similar nervous disorder in the bovine.

The definitive hosts for *S. digitata* are cattle.
(ox and zebu) and water buffaloes while the aberrant hosts include sheep, goats and horses.

Yeh (1959) claimed that *S. labiatopapillosa* is host-specific for cattle and water buffaloes only. However, Becklund and Walker (1969) have reported the parasite in the horse as well as in several wild animals including the antelope (*Antilocapra americana*), bison (*Bison bison*), deer (*Odocoileus homeanus*), moose (*Alces alces*) and caribou (*Rangifer caribou*) of Canada and the U.S.A. Earlier, Bhalero (1933) described a parasite in a hill goat (*Capra sibirica*) which he named *S. buxi*, and which Baylis (1936) considered synonymous to *S. labiatopapillosa*.

**Transmission**

Setarial parasites are transmitted indirectly. The microfilariae of *S. digitata* invade the bloodstream and are ingested by mosquitoes (Innes et al., 1952). In Japan and Russia, *Anopheles hyrscarus*, *Armigeres obturbans* (syn. *A. subaltus coquillellette*), *Aedes togoi*, *Aedes sp.* and *Culex pipiens* have been incriminated as vectors (Ishii et al., 1953; Kadenatsii, 1956). The Japanese workers have shown that the microfilariae develop in the thoracic muscles and then migrate to the labium and salivary glands as infective forms after 12-14 days. In cooler areas development may take a longer period, 32-35 days, as reported by Kadenatsii (1956). The life cycle is normally completed when the infective larvae are inoculated by the insect vector onto another bovine host where they develop into adult stages in the peritoneal cavity in 8 to
10 months. However, it is never completed in aberrant hosts such as the sheep or goat; the infective larvae meander through the tissues and find their way to the cranio-vertebral canal where they remain or, occasionally, migrate as immature forms to the tissues of the central nervous system.

Early reports cited by Yeh(1959) regarded *Stomoxys calcitrans* as the intermediate host of *S. labiatopapillosa*. The role of this insect as a vector has never been confirmed according to Yeh (1959) and Nelson (1962) and appears unlikely that this insect is involved in transmission. Nelson (1962) found that the mosquito, Aedes aegypti can be infected experimentally with microfilariae of *S. labiatopapillosa*. However, he did not consider it an important vector in nature; earlier, he dissected more than 5,000 Ae. aegypti from the coastal region of Kenya and found no infective setarial larvae in these mosquitoes. He pointed out that the natural vectors could possibly be *Mansonia africanus* and *M. uniformis* which he found associated with infected cattle near a river in Kenya.

The development of microfilariae of *S. labiatopapillosa* in Ae. aegypti was found by Nelson (1962) to be similar to that of *S. digitata*. This occurred initially in the midgut of the mosquito and then in the thoracic muscles. In the latter site, the microfilariae thicken and shorten into the sausage stage; they then undergo a rapid phase of growth after which they moult twice and become infective forms. They mature at 75°C and 70-80% relative humidity in 8-10 days then migrate to the haemocoel and abdomen;
by the 11th day, most of them will be located in the head and proboscis. The infective microfilariae of *S. labiato-papillosa* are probably the largest forms that have been recorded in mosquitoes, according to Nelson (1962) who recorded their measurements as more than 2,200 μ in length and 36 μ in breadth.

Innes et al. (1952) believed that intrauterine infection by *S. digitata* may also occur in cattle and produce blindness and paralysis in newborn calves. Refuerzo (1952) claimed that prenatal infection with *S. labiatopapillosa* occurred in a calf in the Philippines.

**Pathogenesis and Pathology**

A. In bovine hosts. Adults forms of *S. digitata* and *S. labiatopapillosa* do not apparently produce lesions in the peritoneal cavity of cattle and water buffaloes. However, young worms may be found in the anterior chamber of the eye where they cause verminous opthalmia (Yeh, 1959, Levine, 1969); the lesions include cloudening of the aqueous humor, iritis, keratitis, conjunctivitis and corneal opacity. This condition, according to Levine (1969), occurs only in the Far East and caused more often by *S. digitata* than by *S. labiatopapillosa*.

Ohbayashi (1953) and Yoshikawa et al. (1976) have found *S. digitata* in nodular granulomas infiltrated with eosinophils in the urinary bladder of cattle.

While the majority of cases of cerebrospinal nematodiasis occur in abnormal hosts, nervous manifestations have been reported in the bovines, but the incidence is probably
much less. Place (1911) has reported the occurrence of Setaria sp. in the spinal cord of buffaloes and cattle. Mohiyudden (1957) and Anantaraman and Victor (1959) have described enzootic bovine paraplegia and paralysis in cattle in India in which the clinical signs and lesions were similar to those in sheep and goats.

B. In sheep and goats. Emoto (1927) first recognized a form of "lumbar paralysis" in goats in Japan. He examined more than 40 cases in which he noted the characteristics of the disorder. He noted its seasonal occurrence, being more frequent during late summer and autumn. Paralysis in both young and adult animals occurred either in all four limbs or only in the hind ones. The onset and course of the disorder were acute, subacute or insidious. Crawford (1939-42) also recognized the seasonal occurrence of this disorder in a number of breeds of goats in Ceylon. He considered typical cases showed muscle incoordination and weakness in the hind limbs only. Nystagmus may also be observed. The onset was either sudden or subacute. The disease was non-febrile. About 10% of all the cases died; a few recovered with or without limb weakness and ataxia.

Innes et al. (1952) have also given a detailed account of this disorder in sheep and goats. They noted that following infection, there is an incubation period of about a month before the appearance of neurological disorders.

The migration route taken by the larvae from the site of inoculation has not been determined. Innes et al. (1952) have postulated a "direct tissue-wandering path" after discounting the possibility of transport via the circulatory
system (the lesions were found not to be of an embolic nature but rather, of a traumatic one). These authors also suggested that the immature larvae enter the cerebrospinal canal via the intervertebral foramina along the nerve roots. These forms may become static in the cerebrospinal canal producing only a mild meningitis and radiculitis with eosinophilic infiltration which may or may not be associated with transient clinical signs of weakness and incoordination. However, some larvae may migrate farther from these sites and invade the nervous tissue producing focal malacia. If lesions occur in the lower regions of the spinal cord, they may be associated with severe paralysis which frequently terminates in death of the animal. A single worm is usually associated with the lesions. However, several worms may enter the central nervous system causing multiple lesions in more than one site. Lesions may occur anywhere in the central nervous system but in Japan, the cerebellum, thalamus and spinal cord are frequently affected, while in Ceylon only the cord is affected (Innes et al., 1952).

Ishii et al. (1955) produced the disease experimentally in goats. They extracted infective larvae from mosquitoes and injected these subcutaneously to these animals. Typical nervous signs and lesions were observed and the parasites were found.

Shoho and Tanaka (1955) noted that reinfection of animals can occur. Old lesions may be found in one part of the spinal cord and in another lesions of acute malacia with secondary degeneration may be seen.
Anderson (1968) has reviewed the histologic changes occurring in cerebrospinal nematodiasis. The microscopic lesions are essentially those of mirocavitation, perivascular infiltration with lymphocytes and eosinophils, and degeneration and disappearance of axis cylinders and myelin sheaths. Extravasation of red blood cells may be seen where worms have caused damage to blood vessels.

Importance

In discussing the importance of cerebrospinal nematodiasis, reference should be made to the earlier sections, clinical signs and pathology, of this chapter. Innes et al. (1952) has found that the mortality rate in sheep and goats is 10%. Losses could be considerable as 600 sheep may have perished in the outbreak that occurred in Fukoshima.

Shoho (1953) has drawn attention to the possibility of the disease occurring in many other countries outside the Far East where suitable vectors and species of Setaria are present. Further, he suggested that the disease may have also occurred in human beings.

Diagnosis

Diagnosis of cerebrospinal nematodiasis can be based on the clinical signs which, however, may vary according to the site of the lesion in the central nervous system. The seasonality of the condition should be considered. Innes et al. (1952) have suggested that differential diagnosis
should include other helminth diseases such as "gid" in sheep, caused by *Coenurus cerebralis*, and microbial infections such as listeriosis and Japanese B encephalitis. They have also recommended the use of allergic and complement-fixation tests utilizing setarial antigen.

Necropsy may provide the best means of diagnosis. The malacic lesions may not be visible on gross examination, but they can be seen on histopathologic examination of representative specimens.

**Control**

There are no data available on control measures applicable to setarial infections in the bovine. Shoho (1952) and Perumal Pillai and Perera (1955) have recommended the use of 1-diethylcarbamyl-4-methyl-piperazine citrate (Caricide) for the prophylaxis and treatment of epizootic cerebrospinal nematodiasis. The former showed that the oral administration of the drug at a dose of 40 mg/kg can prevent the occurrence of the disease during the summer months. The latter found that the same dose can have a curative effect if given in 2-3 consecutive daily administrations during the early course of the disease.
Discussion

*Setaria digitata* is clearly the more important species involved in infections of the normal and aberrant hosts. It is probably the only species responsible for cerebrospinal nematodiasis of sheep and goats and all the clinical cases have been recorded within the supposed distribution area of this parasite. If the more widely distributed *S. labiatopapillosa* were also involved, then clinical cases could have occurred in countries outside Asia.

The term 'epizootic cerebrospinal nematodiasis' used for the disease in sheep and goats is probably a misnomer. A morbidity rate of only 6%, as has been reported in Japan, does not constitute an 'epizootic'. The rate might be higher in Ceylon and India but, then, there is no available evidence that this is so. In the meantime, because of the seasonal occurrence of this disease the term 'enzootic' or 'endemic' may be more appropriate.

More epidemiological studies are needed in countries other than Japan to determine the actual losses caused by this disease in sheep and goats. Until these are carried out, little justification can be made for its control and prevention. However, no such studies can be found in the literature during the last 20 years.
V. ELAEOPHORIASIS

Elaeophoriasis refers to infections of the major arteries of cattle, water buffaloes and sheep caused by adults of Elaeophora. As a clinical entity, it may also refer to a circumscribed form of dermatitis in sheep caused by microfilariae of E. schneideri.

Aetiology

Two species are involved in arterial infections; these are Elaeophora poeli (Vryburg, 1897) and Elaeophora schneideri (Wehr and Dikmans, 1935). The general description by Railliet and Henry (1921) for these species appears in the works of Skjrabin (1969) and Levine (1969). These nematodes resemble species of Onchocerca in having a simple mouth which is devoid of lips. The cuticle of the male and the anterior part of the body of the female are also transversely striated. The anterior end of the female is narrow and attached to the wall of the artery, while the posterior end is expanded and lies free in the lumen. The oesophagus is long and cylindrical. The tail of the male is curved ventrally; it is devoid of caudal alae, but has a number of pre- and post-anal papillae which are located near the cloaca. The spicules are unequal. The vulva is located at the level of the oesophagus. The uterus divides into four parallel branches, each ending in an ovary. The microfilariae are unsheathed.

E. poeli measures 45-70 mm x 200-260 μ and 40-300 mm
x 350 µ in the male and female respectively. *E. schneideri* is typically shorter but thicker, the male being 60–65 mm x 730 µ and the female 110–120 mm x 750–810 µ. The microfilariae of *E. poeli* are found in the blood and measure 340–360 µ x 7.0–7.5 µ, while those of *E. schneideri* are located in the skin and measure 207 µ x 13 µ (Soulsby, 1965; Levine, 1969).

Occurrence and Species Affected

*E. poeli* occurs in cattle and water buffaloes in the Far East (India, Indo-China, Malaysia, Indonesia, Philippines) and possibly in some parts of Africa including the Belgian Congo (Tubangui, 1947; Varma, 1953; Douglas, Cordy and Spurlock, 1954; Soulsby, 1965; Levine, 1969). It has also been recorded in the African buffalo (*Syncerus caffer*) in Uganda (Bindernagel, 1971). No infection rates for this parasite are available in the literature.

Mesina (personal communication) carried out a survey in the Philippines and found the parasite in 327 (65.4%) of 500 water buffaloes examined in abattoirs near Manila.

*E. schneideri* infects sheep in the Western United States of America. Clinical signs are attributed to the microfilariae of this species, the disease being known as 'filarial dermatosis'. Cases have been recorded in New Mexico, Arizona, Colorado, Montana, Nebraska, Utah, North Dakota, and California (Kemper, 1938; Dikmans, 1948; Douglas, *et al.*, 1954; Smith, Lovell and Ray, 1954). Infections occur in about 0.2–0.3% of sheep in ewe flocks moved during summer to mountain ranges with altitudes
from about 2,135 m to 3,050 m (Kemper, 1938, 1957; Douglas et al., 1954). The parasite has also been reported in the deer (Odocoileus homeonous), elk (Cervus canadensis), and moose (Alces alces) from these states, particularly New Mexico (Kemper, 1957; Hibler, Gates, White and Donaldson, 1971). Kemper (1957) suggested that the deer is the natural host of the parasite and that it may act as the reservoir of infection for sheep.

Transmission

No data are available on the mode of transmission of *E. poeli*.

Hibler, Adcock, Gates and White (1970) carried out experimental studies in New Mexico on the transmission of *E. schneideri* in the sheep and deer. They found that species of *Hybomitra* and *Tabanus* can act as intermediate hosts for the parasite. The infection rates of horseflies in the Gila forest of this State were determined by Hibler, Gates, White and Donaldson (1971). They were found to be 15.0%, 23.3% and 8.6% in the northern and northwestern, central and southern, and eastern and southeastern areas, respectively. Worley, (1975), working in Montana, considered the following insect species as potential vectors: *Hybomitra rhombica osburni*; *H. tetratica*; *H. metabola*; *Chrysops noctifer pertinax*; and *Atylotus incisuralis*.

Pathogenesis and Pathology

*E. poeli* is responsible for atheromatous lesions
occurring in the aorta. The description of these lesions appears in the works of Soulsby (1965) and Levine (1969). Nodules are formed primarily in the thoracic portion of the aorta. They begin as irregular caseous areas which develop into granulation tissue. They thus form thrombi which partially obliterate the vessel. The nodules vary between 5-15 mm in diameter and may reach a height of 13 mm from the base (Mesina, personal communication). Old lesions calcify and regress into fibrotic plaques on the inner wall of the aorta. There are no recognizable symptoms due to these lesions.

The adults of *E. schneideri* are found in the carotid, mesenteric and iliac arteries. They apparently do not produce lesions in these sites. Abdelbaki and Davis (1972) have suggested that the adults of this species may also lodge in the arteries associated with the eye and cause lesions of pigmentary chorio-retinal degeneration.

The presence of the microfilariae of *E. schneideri* in the skin incites an inflammatory reaction which is manifested by intense pruritus. This occurs commonly in the head. Frequent rubbing and scratching of the involved areas with the hindfoot results in laceration and formation of raw bleeding lesions which have a granular appearance. Microscopically, these are infiltrated with polymorphonuclear cells. The lesions in the head are seen mostly in the poll region; they appear as small circumscribed areas varying from 5 to 10 cm in diameter. However, these may extend over the face and involve the nostrils, lips and mouth (Kemper, 1938; Micozzi, 1956).
Lesions are also found on the abdomen and hindfeet. Kemper (1957) observed that these are associated with the presence of the adult worms in the mesenteric and iliac arteries.

The irritation of the skin may temporarily subside for several days; this allows for the formation of raised encrustations over the affected area. These crusts are later rubbed off, exposing a raw bleeding surface. A cycle or recurrent scab formation may occur over a period of 7 months or longer. When the lesions finally heal, new wool grows over the area and the skin returns to its normal appearance.

Importance

E. poelii is probably not an important parasite as it has no clinical significance.

Tancours, Roddy and O'Flaherty (1957) have claimed that filarial dermatosis causes considerable damage to the skin, making it unsuitable for processing into leather.

Diagnosis

Infection with E. poelii can be diagnosed by finding the characteristic microfilariae in the blood. The adults can be recovered from the aorta at necropsy.

Infections of the eye by adult E. schneideri can be diagnosed by ophthalmoscopic procedures. The funduscopic changes include diffuse patches of edema and areas of
dark pigmentation in the tapetum (Abdelbaki and Davis, 1972).

Presumptive diagnosis of filarial dermatosis can be based on the history of recently pasturing sheep at high altitudes and on characteristic signs and lesions. Confirmatory diagnosis can be achieved by finding the microfilariae in the skin lesions. The maceration technique proposed by Kemper (1957) is considered effective and practical. This consists of finely shredding the skin samples, placing these in warm physiological saline for two hours and centrifuging the suspension to examine the deposit for motile microfilariae. Davis and Kemper (1951) suggested that diagnosis can also be obtained by histopathologic examination of the affected skin, but they admit that this takes a longer period of time to carry out.

Control

Control is probably not warranted for in infections by *E. poelii*.

For filarial dermatosis of sheep, control by chemotherapy has been the only method described. Kemper and Roberts (1946) have claimed successes in the use of antimony compounds. The drugs they used included tartar emetic administered alone or in combination with emetine hydrochloride at the rate of 324 mg per animal for 8 injections at weekly intervals; lithium antimony thiomalate (Anthiomaline) given at a total dose of 75 ml over a period of
10 weeks; and sodium antimonyl tartrate in a solution of trypan blue (Trichicide) given at doses ranging from 15-40 ml for a total of 6 injections. These compounds were found effective against both the adults and microfilariae. Sheep so treated showed clinical recovery with complete healing of lesions soon after the period of administration.
Filarial dermatosis may be considered of minor importance only as it occurs at a fairly low rate (0.2-0.8%) and affected sheep usually recover without treatment. The wool is shed in areas of the skin where the lesions occur, but returns on complete healing.

Control of the tabanid vectors of this disease can hardly be recommended. Destruction of wildlife species which may act as reservoir hosts for the parasite is also hardly feasible. Avoidance of pastures at high altitude is not a practical way of preventing the disease as it is contrary to the farming practices established in the western USA.
DISCUSSION

The foregoing chapters have dealt with filarial diseases of domestic ruminants. Data available in the literature on the parasites involved in these diseases are summarized in Tables 1 and 2. It may be stated that the important filarial diseases of domestic ruminants in the tropics are parafilariasis and stephanofilariasis. Few of the other filarial parasites are considered to be important since they rarely cause clinical disease. However, it is considered by this author that at least some of them may be found to have clinical and economic significance.

A large part of this review has been devoted to the morphology and the taxonomy of the nematodes involved in these diseases. This is because there are still different views regarding the systematics of some species, for example, *Onchocerca gutturosa* and *Setaria digitata*.

Many of the filarial infections have a limited geographic distribution. The majority have been recorded in tropical countries, particularly those situated in the Far East and Africa. The prevalence of infections in these countries should be higher than in temperate zones inasmuch as climatic factors such as rainfall, temperature and relative humidity are optimal for the multiplication and survival of disease-transmitting vectors. However, this could not be supported in this review as there have been relatively few available reports and surveys carried out in the tropics. Nevertheless, comparison of the infection rates of *O. gutturosa* gathered from temperate countries
in Europe and North America and tropical countries situated in Central and East Africa show generally higher figures for the latter.

Members of the superfamily Filarioidea require arthropods as biological vectors. The vectors of Stephanofilaria dedoesi, S. zaheeri, S. okinawaensis, Onchocerca armillata, O. ochengi, O. dukei, and Elaeophora poeli have yet to be determined. There have been reports that setarial species can also be transmitted by the intra-uterine route (Innes et al., 1952; Refuerzo, 1952). These should require further confirmatory studies. There is also a possibility that wild animals may be involved and act as reservoirs for infections by Setaria labiato-papillosa and Elaeophora schneideri.

Of the 17 filarial species reviewed here, perhaps only 9 are recognized clinically in the field (Table 2). All of these, except Setaria digitata, occur in the skin. Although internal lesions have been observed with some of the other species, rarely are any clinical signs attributed to them. The nervous signs due to Setaria digitata have mostly been observed in sheep and goats.

Diagnosis of these recognizable infections can be based on their seasonal occurrence and clinical manifestations. The presence of haemorrhagic streaks on the flanks of cattle is indicative of parafilariasis. On the other hand, the occurrence of sores and ulcers on specific sites of the body can be diagnostic for stephanofilariasis. Confirmatory diagnosis is usually carried out by examining appropriate specimens for the presence of the parasite. The characteristics of the microfilariae are presented in Table 3.
<table>
<thead>
<tr>
<th>Parasite</th>
<th>Geographic Distribution</th>
<th>Species Affected</th>
<th>Location of Adults</th>
<th>Vector</th>
</tr>
</thead>
<tbody>
<tr>
<td>Parafilaria bovicola</td>
<td>India, Philippines, North &amp; South Africa, France</td>
<td>Cattle, water buffaloes</td>
<td>Skin &amp; subcutis</td>
<td>Muscid flies</td>
</tr>
<tr>
<td><em>Stephanofilaria</em> dedoesi</td>
<td>Indonesia</td>
<td>Cattle, water buffaloes, goats</td>
<td>Skin</td>
<td>?</td>
</tr>
<tr>
<td><em>S. stilesi</em></td>
<td>North America, Russia, Colombia</td>
<td>Cattle</td>
<td>Skin (abdomen)</td>
<td>Hornflies</td>
</tr>
<tr>
<td><em>S. assamensis</em></td>
<td>India, East Pakistan</td>
<td>Zebu cattle</td>
<td>Skin (hump)</td>
<td>Muscid flies</td>
</tr>
<tr>
<td><em>S. kaeli</em></td>
<td>Malaysia</td>
<td>Cattle, goats</td>
<td>Skin (legs)</td>
<td>Muscid flies</td>
</tr>
<tr>
<td><em>S. zaheeri</em></td>
<td>India</td>
<td>Water buffaloes</td>
<td>Skin (ears)</td>
<td>?</td>
</tr>
<tr>
<td><em>S. okinawaensis</em></td>
<td>Japan</td>
<td>Cattle</td>
<td>Skin (muzzle &amp; teats)</td>
<td>?</td>
</tr>
<tr>
<td><em>Onchocerca armillata</em></td>
<td>Asia, Asia Minor, Africa</td>
<td>Cattle, water buffaloes, goats</td>
<td>Aorta</td>
<td>?</td>
</tr>
<tr>
<td><em>O. gibsoni</em></td>
<td>Far East, Australia, Africa</td>
<td>Cattle, water buffaloes</td>
<td>Subcutis, joints</td>
<td>Midges</td>
</tr>
<tr>
<td><em>O. gutturosa</em></td>
<td>Worldwide</td>
<td>Cattle</td>
<td>Nuchal, gastro-splenic &amp; joint ligaments</td>
<td>Blackflies</td>
</tr>
<tr>
<td>Parasite</td>
<td>Geographic Distribution</td>
<td>Species Affected</td>
<td>Location of Adults</td>
<td>Vector</td>
</tr>
<tr>
<td>------------------------</td>
<td>------------------------------</td>
<td>------------------------------</td>
<td>----------------------------</td>
<td>------------</td>
</tr>
<tr>
<td>Onchocerca ochengi</td>
<td>East Africa, Togo</td>
<td>Cattle</td>
<td>Subcutis</td>
<td>?</td>
</tr>
<tr>
<td>O. sweetae</td>
<td>Australia</td>
<td>Water buffaloes</td>
<td>Subcutis</td>
<td>Midges</td>
</tr>
<tr>
<td>O. dukei</td>
<td>West Africa</td>
<td>Cattle</td>
<td>Intermuscular tissues</td>
<td>?</td>
</tr>
<tr>
<td>Setaria labiato-papillosa</td>
<td>Asia, Russia, Africa</td>
<td>Cattle, water buffaloes, goats</td>
<td>Peritoneal cavity</td>
<td>Mosquitoes</td>
</tr>
<tr>
<td>S. digitata</td>
<td>Asia</td>
<td>Cattle, water buffaloes, sheep, goats</td>
<td>Peritoneal cavity</td>
<td>Mosquitoes</td>
</tr>
<tr>
<td>Elaeophora poeli</td>
<td>Southeast Asia, Africa</td>
<td>Cattle, water buffaloes</td>
<td>Aorta</td>
<td>?</td>
</tr>
<tr>
<td>E. schneideri</td>
<td>Western U. S. A.</td>
<td>Sheep</td>
<td>Arteries</td>
<td>Tabanids</td>
</tr>
</tbody>
</table>
### Table 2. List of the clinically recognizable infections of domestic ruminants

<table>
<thead>
<tr>
<th>Parasite</th>
<th>Common Name(s) of Infection</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Parafilaria bovicola</em></td>
<td>Haemorrhagic parafilariasis</td>
</tr>
<tr>
<td></td>
<td>'Seraun Phutana'</td>
</tr>
<tr>
<td></td>
<td>'Pat futa'</td>
</tr>
<tr>
<td></td>
<td>Dermatorragie parasitaire</td>
</tr>
<tr>
<td><em>Stephanofilaria dedossi</em></td>
<td>'Cascado'</td>
</tr>
<tr>
<td><em>S. stilesi</em></td>
<td>'Summer sore'</td>
</tr>
<tr>
<td><em>S. assamensis</em></td>
<td>'Humpsore'</td>
</tr>
<tr>
<td><em>S. kaeli</em></td>
<td>'Krian sore'</td>
</tr>
<tr>
<td><em>S. zaheeri</em></td>
<td>'Ear sore'</td>
</tr>
<tr>
<td><em>Onchocerca gibsoni</em></td>
<td>'Worm nests'</td>
</tr>
<tr>
<td><em>Setaria digitata</em></td>
<td>Epizootic cerebrospinal nematodiasis</td>
</tr>
<tr>
<td></td>
<td>Lumbar paralysis</td>
</tr>
<tr>
<td></td>
<td>'Goat paralysis'</td>
</tr>
<tr>
<td><em>Elaeophora schneideri</em></td>
<td>Filarial dermatosis</td>
</tr>
</tbody>
</table>
Table 3. Characteristics and location of some microfilariae found in domestic ruminants

<table>
<thead>
<tr>
<th>Species</th>
<th>Length (μm)</th>
<th>Width (μm)</th>
<th>Sheath</th>
<th>Location</th>
</tr>
</thead>
<tbody>
<tr>
<td>Parafilaria bovicola</td>
<td>196-249</td>
<td>9-13</td>
<td>Absent</td>
<td>Skin</td>
</tr>
<tr>
<td>Stephanofilaria stilesi</td>
<td>52</td>
<td>3</td>
<td>Absent</td>
<td>Skin</td>
</tr>
<tr>
<td>S. assamensis</td>
<td>109</td>
<td>6</td>
<td>Absent</td>
<td>Skin</td>
</tr>
<tr>
<td>S. kaeli</td>
<td>109</td>
<td>6</td>
<td>Absent</td>
<td>Skin</td>
</tr>
<tr>
<td>S. okinawaensis</td>
<td>100-110</td>
<td>5-7</td>
<td>Absent</td>
<td>Skin</td>
</tr>
<tr>
<td>Onchocerca armillata</td>
<td>300</td>
<td>6</td>
<td>Absent</td>
<td>Blood</td>
</tr>
<tr>
<td>O. gibsoni</td>
<td>220-350</td>
<td>3-4</td>
<td>Absent</td>
<td>Skin</td>
</tr>
<tr>
<td>O. gutturosa</td>
<td>189-251</td>
<td>3</td>
<td>Absent</td>
<td>Skin</td>
</tr>
<tr>
<td>O. ochengi</td>
<td>180</td>
<td>5</td>
<td>Absent</td>
<td>Skin</td>
</tr>
<tr>
<td>O. sweetae</td>
<td>222-258</td>
<td>4-5</td>
<td>Absent</td>
<td>Skin</td>
</tr>
<tr>
<td>O. dukei</td>
<td>225-265</td>
<td>6</td>
<td>Absent</td>
<td>Skin</td>
</tr>
<tr>
<td>Setaria labiato-papillosa</td>
<td>140-230</td>
<td>Present</td>
<td>Blood</td>
<td></td>
</tr>
<tr>
<td>S. digitata</td>
<td>154-204</td>
<td>Present</td>
<td>Blood</td>
<td></td>
</tr>
<tr>
<td>Elaeophora poeli</td>
<td>340-360</td>
<td>7</td>
<td>Absent</td>
<td>Blood</td>
</tr>
<tr>
<td>E. schneideri</td>
<td>207</td>
<td>13</td>
<td>Absent</td>
<td>Skin</td>
</tr>
</tbody>
</table>
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