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From State Veterinary Medical Institute, Stockholm, Sweden.

CEREBROSPINAL AND MUSCULAR NEMATODIASIS (ELAPHOSTRONGYLUS RANGIFERI) IN SWEDISH REINDEER

By

O. Ronéus and *M. Nordkvist*

Among the approximately 250,000 reindeer which live in the forest and highland areas of Sweden north of latitude 62° N, a disease characterised clinically by ataxia, paresis, and convulsions is fairly common. These clinical signs have long been familiar to the Lapps. *Qvigstad* (1941), for example, has listed several Lappish names for diseases which would seem to refer disorders of the central nervous system — "oaive-vuorre" (ataxia), "kinalkaesahtahka" (stiff neck), and "liwdsa-vikke" (posterior paresis).

Until recently the aetiology of this disease was unknown. A reindeer with typical clinical signs was autopsied in December 1960 and meningomyelitis in the lumbar region and a eosinophilic granuloma in one sciatic nerve were observed. The abundance of eosinophils in the inflammatory exudate aroused suspicion of a parasitic background to the lesions. To follow up this observation, the reindeer in the autopsy material of this institute were examined for the presence of parasites in the central nervous system. The reindeer in the autopsy material were not selected for locomotory disorders or any other particular disease.

MATERIALS AND METHODS

Altogether twenty reindeer were examined of which four were killed immediately before autopsy. Three others were killed be-

fore being sent to the laboratory and these together with the other thirteen animals which had died had some degree of cadaverous changes. Most of the animals were young, between six months and one year of age; two of them were four years old. Six were females and fourteen males.

At autopsy the brain was removed entire, the spinal cord was exposed along its whole length by removing the dorsal arches of the vertebrae and the sciatic nerves were dissected free as far as the stifle joints.

After being examined for the presence of nematodes the brain, spinal cord, and sciatic nerves were fixed in their entirety by immersion in ten per cent formalin. When fixed, usually after three days, blocks from particular areas were removed, labelled, and embedded in paraffin for sectioning. As a rule eight blocks were taken from the brain, eight from the spinal cord (at least three from the cauda equina), and six from the sciatic nerves. Sections were stained with haematoxylin and eosin, van Gieson, periodic-acid-Schiff, Luxol Fast Blue, and in some instances with Mahon's myelin stain.

RESULTS

Clinical observations

Eight of these twenty reindeer had shown distinct clinical signs — weekness and poor co-ordination of the hind legs. Posterior paralysis was complete in two animals (370, 8885, Fig. 1). The other six animals adopted an abnormal posture, the weight of the body was carried on the fore legs (Figs. 2 and 3), and the hind legs were somewhat extended with the metatarsi in an upright position. Some animals had a degree of cervical rigidity. Two animals carried their heads tilted towards one side and the ears asymmetrically.

No definite clinical signs referrable to the central nervous system were observed in nine of the animals examined. They had shown only increasing general weakness.

No anamnestic details were available for the three reindeer which had been found dead. One of these had apparently been killed by a lynx.

Autopsy findings

Nematodes were found in the central nervous system of practically all reindeer which had shown signs of locomotory dis-

turbances. The exceptions were the first two animals in the autopsy series which were not examined as carefully as the others. All but two of the other reindeer in the autopsy material also harboured nematodes in the central nervous system. Nematodes were also present in the skeletal musculature of several of the animals.

The nematodes were relatively long and narrow. Females had a mean length of 4.5 cm. and males 3.5 cm. The mean diameter for both sexes was 0.2 mm. In the central nervous system the nematodes were generally yellow-white in colour but in the musculature, often dark brown. Morphologically these nematodes fit the description of Elaphostrongylus rangiferi given by *Mitskevitch* (1958) with only minor differences in some measurements.

There was no discernible pattern in the distribution of the nematodes throughout the central nervous system although they were perhaps encountered most often on the ventral surface of the brain. The nematodes were most often found in the subarachnoidal space and only occasionally in the subdural space. In no instance was there evidence that the nematodes had penetrated into the adjacent nervous tissue.

In spite of their length, the nematodes were difficult to detect in the camouflage afforded by the meningeal blood vessels (Fig. 4). The number present in particular reindeer varied from only a few up to twenty.

After a number of animals had been autopsied the same nematode was encountered in the musculature. In this tissue they were very difficult to see and they had probably been overlooked in the earlier autopsies. Once aware of the possibility of their being present in this site, search of the musculature resulted in the demonstration of E. rangiferi in five of seven animals. In most instances the nematodes were found in the fascia of the superficial thoracis muscles, particularly beneath the latissimus dorsi and the deep pectoral. In the fascia they apparantly did not elicite any local reaction. Nematodes found beneath the epimysium were usually associated with haemorrhages about 1 cm. in diameter (Fig. 5).

Two of the animals had tilted their heads. In the one (no. 2380) which tilted its head towards the left, a living nematode was found lying on the mucous membrane of the left middle ear. The other animal (no. 1710) had tilted its head towards the right and a severe purulent otitis media was found on the right side.

Only slight macroscopical changes were observed in the meninges. On the ventral surface of the cauda equina at the level of the lumbosacral joint there were slight oedema of the leptomeninges and a few nodules about one or two mm. in diameter in the meninges in ten of the animals (Fig. 6).

Among the other autopsy findings can be mentioned the verminous pneumonia seen in many of the animals. The aetiology has not yet been definitely established. The larave found in the lungs were quite similar but not identical with Muellerius capillaris. No adult M. capillaris were found.

Histologically, nematodes were encountered in the meninges of the central nervous system (Figs. 7, 8) and under the epimysium of the skeletal musculature (Fig. 9). Erythrocytes were present in the intestinal lumen of some nematodes, a sign that they had ingested blood and an explanation of the brown colour.

A few larvae, presumably of E. rangiferi, were encountered in some reindeer in the leptomeninges of the brain and spinal cord, in a spinal nerve root (Fig. 10), and in the epimysium of the skeletal muscles.

Eggs, again presumably of E. rangiferi, were detected in the tissues of six reindeer. They were found in the leptomeninges (Fig. 11) of five animals and usually laid in groups. In a single cross-section of the spinal cord at the C_2 level 42 eggs could be counted. Eggs were also encountered in a section of the hypophysis (Fig. 12) lying in two groups of 17 and 30 respectively. Outside the central nervous system eggs were present in the epimysium and fascia of a few animals (Fig. 13). Some eggs were at the two-cell stage and a few had advanced somewhat further.

Inflammatory changes were regularly demonstrated in the central nervous system. On the whole, the presence of parasites and the distribution of inflammatory changes coincided. Inflammatory lesions were largely limited to the meninges and were regularly most severe at the level of the cauda equina.

The morphological changes are summarized in Table 1.

The leptomeninges of the brain were involved in thirteen animals with some extension of the inflammatory process to the superficial brain tissue in seven of these. The leptomeninges were obviously oedematous and diffusely but relatively sparsely infiltrated by lymphocytes and plasma cells. A slight increase in the number of fibroblasts was also evident. Some tendency for accumulation of these cells about the blood vessels could be

Table 1. Clinical, parasitological, and pathological observations.

Autopsy no.	Sex	Age	Nutritional state	Locomotory disturbance	E. rangiferi in CNS	E. rangiferi in skeletal musculature	Nematode eggs in CNS	Larvae in lungs	Macroscopical lesions in spinal meninges (cauda equina)	Encephalomeningitis	Myelomeningitis	Parasitic granulomas in CNS	Comments
8885/60	♀	6 months	poor	+	÷	÷	÷	+	÷	÷	+	+	Total posterior paresis
370/61	♂	6 „	moderately	+	÷	÷	÷	+	+	+	+	+	„ „ „
566/61	♂	8 „	inanition	÷	+	÷	—	+	—	+	+	—	
1155/61	♂	8 „	inanition	÷	+	÷	—	—	+	+	+	+	Killed by lynx
1198/61	♂	8 „	poor	+	+	÷	—	+	+	+	+	+	
1392/61	♂	9 „	inanition	—	+	÷	+	+	÷	—	+	—	
1430/61	♂	9 „	poor	+	+	÷	+	+	+	+	+	+	
1549/61	♀	9 „	inanition	—	+	÷	—	+	+	—	+	+	
1710/61	♂	9 „	inanition	+	+	÷	+	+	—	+	+	+	Torsion of the head towards the right. Purulent otitis media.
2105/61	♂	9 „	inanition	—	+	÷	—	+	+	+	+	+	
2108/61	♀	9 „	poor	—	+	÷	—	+	—	—	+	+	
2118/61	♀	9 „	inanition	—	+	÷	+	+	—	—	+	+	
2119/61	♀	9 „	inanition	—	+	÷	—	+	+	+	+	+	
2151/61	♂	4 years	inanition	+	+	+	+	+	—	+	+	+	Elaphostrongylus eggs in the hypophysis
2206/61	♂	10 months	poor	—	+	+	+	+	—	+	+	+	
2380/61	♂	10 „	poor	+	+	+	+	+	—	+	+	+	Torsion of the head towards the left. Elaphostrongylus in left middle ear
2561/61	♂	4 years	inanition	—	—	+	—	+	+	—	—	—	
3053/61	♀	1 „	inanition	—	—	—	—	—	+	+	+	+	
3205/61	♂	1 „	poor	÷	+	+	+	+	+	+	+	+	
3388/61	♂	1 „	inanition	+	+	—	—	+	—	—	+	—	

+ = present
— = not present
÷ = not examined

discerned. A few haemosiderin-containing macrophages could be identified among the cells. In five of the animals a moderate number of eosinphils were also present. The amount of exudate was often greatest at the depths of the sulci (Fig. 14). When present, changes in adjacent brain tissue consisted of a slight perivascular lymphocytic infiltration.

Similar inflammatory changes were observed in the leptomeninges of the spinal cord in nineteen of the twenty animals examined. Myelomeningitis was very irregularly distributed; some regions of a particular cord could be quite free from changes. The most severe changes were regularly encountered at the level of the cauda equina and fairly often at the junction of the cervical and thoracic regions of the cord. Sixteen of the animals had, in addition, multiple parasitic granulomas in the dura or the leptomeninges of the cauda equina, usually on the ventral surface.

The central portions of these granulomas often contained PAS-positive material, probably remnants of parasites. This material was surrounded by foreign-body giant cells and peripheral to these there was a zone containing lymphocytes and plasma cells and a large number of eosinophils (Figs. 15, 16). Even after careful search no signs of mechanical damage or foci of malacia or glia proliferation could be demonstrated. On the other hand, the inflammatory changes in the meninges sometimes extended into the roots of the spinal nerves (Figs. 17, 18).

A slight to moderate, often diffuse infiltration of the perineurium with lymphocytes, plasma cells and in most instances eosinophils was found in the proximal portions of the sciatic nerves of thirteen of the twenty reindeer. Sometimes eosinophils were the dominant cell type (Fig. 19). There were also a few haemosiderin-containing macrophages. In a few places there was a focal chronic neuritis with lymphocytic infiltration and induration of the peri- and endoneurium. A few parasitic granulomas were also encountered in the perineurium of these nerves. The inflammatory changes sometimes extended into the nerves themselves. In the sciatic nerves and the roots of the other spinal nerves there were demyelinisation and changes in the axons — swelling, fragmentation, and even lysis — in the regions adjacent to the inflammatory changes (Fig. 20). Similar degenerative changes were also observed in branches of the brachial plexus.

As already mentioned, a living nematode was found in the

middle ear of one reindeer. No microscopical changes, however, could be detected in the mucous membrane of this structure. In the animal with the macroscopically discernible otitis media, the mucous membrane was heavily infiltrated with neutrophils, lymphocytes, plasma-cells and a few esinophils. There was also a small particle of apparently foreign material which could not be identified, possibly of parasitic origin.

DISCUSSION

Cerebrospinal nematodiasis in reindeer is an example of a type of disease which in other animal species and human beings is probably better known in other parts of the world. A general survey of cerebrospinal and visceral nematodiasis may serve to place the reindeer disease in its proper perspective.

The earliest reference, according to *Sprent* (1955), was in 1706 to a round worm in the brain of a child.

A form of paralysis in horses, known as "Kumree" in India since at least the early 1800's, was described by *Place* (1911). He also referred to similar diseases in sheep, cattle and buffaloes. The clinical signs of posterior paralysis could be associated with foci of malacia and haemorrhage in the spinal cord. Filaria could be demonstrated in these foci and, in many of the affected animals, in the anterior chamber of the eye as well. The disease was evidently very common in Burma.

Since then several others working in the Far East have associated certain types of common or even epizootic paralysis of sheep, goats, horses, cattle, and buffaloes with the presence of nematodes in the central nervous system. The parasite which has been implicated in that part of the world is the setaria of cattle, Setaria digitata. A number of experiments carried out in Japan during the second World War demonstrated that an epizootic posterior paralysis of sheep, goats, and horses in Japan and Korea was caused by immature Setaria digitata (*Shoho* 1952). *Innes* (1951) and *McGaughey* (1951) described "goat paralysis" in Ceylon and the latter succeded in finding nematode larvae in the brain and spinal cord lesions. *Innes & Shoho* (1952), *Innes, Shoho & Pillai* (1952), *Shoho* (1953, 1954) and *Shoho & Tanneka* (1955) supported the Japanese opinion. They considered that Setaria microfilariae, which live in the blood stream, have a tendency to wander to the central nervous system if they happen

to be transferred to a host animal of another species by the bite of mosquitoes. Migration within the central nervous system would then produce mechanical damage and cause the focal encephalomyelomalacia. These mechanical lesions in turn caused clinical paralysis. A meningitis with predominantly eosinophil infiltration was also seen.

Mohiyuddeen (1956, 1958) in India has also described an enzootic bovine paraplegia associated with the presence of larvae and immature form of an unidentified nematode in the brain and spinal cord. *Katiyar & Varshney* (1961) found nematodes in the central nervous system of six of twenty-five sheep and goats with posterior paralysis.

In recent years it has become apparent that in a foreign host nematode larvae can migrate but not attain sexual maturity. During migration they can damage various organs including the central nervous system. Perhaps the most widely known example is that of the dog ascarid, Toxocara canis, and the lesions associated with it in children. *Beaver et al.* (1952) identified larvae of Toxocara canis in the liver of human beings and coined the phrase "visceral larva migrans" to cover the clinical syndrome. In a later paper, *Beaver* (1956) mentioned that apart from Toxocara canis other nematodes from dogs and rats can cause the syndrome, sometimes with fatal results. Visceral larva migrans has been produced experimentally in various animals using a number of different ascaris species. As early as 1921 *Fülleborn* demonstrated that feeding guinea pigs with a large number of ascaris eggs resulted in migration of larvae to various organs including the brain. Similar results were obtained by *Sprent* (1951) using mice and various ascaris species. *Tiner* (1951) mentioned that larvae of the ascaris species infecting racoons regularly caused the death of exposed rodents by migrating through the central nervous system. *Done, Richardson & Gibson* (1960) experimentally produced larva migrans in pigs by feeding them eggs of Toxocara canis capable of invasion. They observed that the presence of larvae in the central nervous system could produce clinical signs.

While all these examples of cerebrospinal nematodiasis have concerned migration of parasites in foreign hosts or only doubtfully specific hosts we are dealing with quite other circumstances in the types of cerebrospinal nematodiasis known to occur in reindeer and Cervidae generally. In these species it is with all

probability a parasite in its specific host which migrates to the central nervous system.

As early as 1931 *Cameron* mentioned that a nematode about 5 cm. long had been observed in the fascia beneath the latissimus dorsi of a Scottish deer (Cervus elaphus). This nematode was given the name Elaphostrongylus cervi gen. et sp. nov. What are apparently related nematodes have since been found in the central nervous system of various Cervidae. In Germany *Schwangart* (1940) found a large number of 2.3 to 5 cm. long nematodes in various stages of maturation in the brain and spinal cord of paralysed deer (C. elaphus). *Burg, Baudet & Verwey* (1953) working in Holland, also found nematodes classified in the genus Elaphostrongylus in the leptomeninges of the brain and spinal cord of two deer of the same species. In one of the deer there was extensive subdural haemorrhage and clumps of cells resembling eggs in early division were found close to the nematodes. Some, at least, of the parasites were considered to be sexually mature. In the other deer there were no haemorrhages but a fibrinopurulent meningitis and focal myelitis. Nematodes identified as Elaphostrongylus odocoilei were encountered in the cranial cavity of eleven of thirty-one Virginia deer (Odocoileus virginianus) examined in Canada.

In the USA, *De Guisti* (1955) has reported the presence of immature Protostrongylus nematodes in the leptomeninges of thirty-six of seventy-nine Virginia deer examined in Michigan. From Pennsylvania, *Alibasuglo, Kradel & Dunne* (1961) reported that some sixty of eighty-one Virginia deer had Elaphostrongylus tenuis in the leptomeninges of the brain and a chronic meningitis with eosinophil infiltration. Most of these animals were apparantly normal when shot during hunting season but a few had shown signs of nervous abnormalities. Much earlier, *Hobmaier & Hobmaier* (1934) had recorded the finding of Elaphostrongylus odocoilei in black-tailed deer (Odocoileus columbianus) in California. The nematodes were observed in the musculature and connective tissue in various parts of the body and in one animal, in the right cardiac atrium. No mention was made of nematodes in the central nervous system.

The life-cycle of the genus Elaphostrongylus in Cervidae is not known. There is a possible parallel in the life-cycle of a rat lungworm, Angiostrongylus cantonensis, as described from Australia by *Mackerras & Sanders* (1955). This nematode in its

natural host, the rat, has during its life-cycle a migratory phase which includes the central nervous system.

Elaphostrongylus tenuis has also been reported to cause paralysis in lambs in the USA (*Kennedy, Whitlock & Roberts*, 1952, and *Whitlock*, 1952, 1953). Haemorrhages, focal malacia, and an eosinophil meningitis were observed in the brain and spinal cord.

As far as reindeer (Rangifer tarandus) are concerned, *Mitskewitch* (1958) reported that Elaphostrongylus larvae could be demonstrated in twenty to sixty-one per cent of focal samples collected from various herds in the northern USSR. Reindeer were experimentally infected with Elaphostrongylus larvae in the invasive stage. One animal which received one hundred larvae had at autopsy thirty nematodes, 50 mm. long, in the leptomeninges of the brain. Several species of terrestial and aquatic molluscs could serve as intermediate hosts for larvae development. A detailed morphological description was given. Because of differences from other species described, the name Elaphostrongylus rangiferi sp. nov. was proposed.

The nematodes observed by us in the central nervous system of the reindeer autopsied here could be identified as Elaphostrongylus rangiferi (Mitskevitch). In some reindeer we have also seen eggs and larvae which probably originated from nematodes. None of the nematodes examined, however, were unmistakably sexually mature. Eggs with a structure which is compatible with their origin from Elaphostrongylus nematodes have been encountered in the central nervous system of many of our reindeer and in the skeletal musculature of some. The eggs have usually laid in large clumps, sometimes adjacent to nematodes, and this suggests that the particular species of Elaphostrongylus infecting these animals can attain sexual maturity in the central nervous system. The finding of nematodes and large collections of eggs in the skeletal musculature suggests that the nematodes can also attain sexual maturity there. Since E. rangiferi were found as often in the skeletal musculature as in the central nervous system, both these sites seem to serve as its habitat.

The lesions in the central nervous system — a lymphohistiocytic and eosinophilic meningitis and the granulomas — are of a type which can reasonably be attributed to parasitism. Since there was no definite evidence of mechanical damage in the central nervous system, the lesions are probably less a reaction to

the physical presence of the nematodes than to the effects of nematode toxins or metabolites. Whether living E. rangiferi or only dead nematodes elicite the reactions remains to be determined.

Some of the reindeer in which Elaphostrongylus could be demonstrated in the central nervous system had shown clinical signs of ataxia or paralysis. Available anamnestic details for many of the animals, on the other hand, have mentioned only general weakness. Most of the animals were emaciated, some to the point of inanition. As mentioned above, two of the reindeer carried the head in an abnormal position. Paralysis can undoubtedly be explained by the lesions in the brain, spinal cord and spinal nerves. The extent and severity of the lesions varied widely from animal to animal. Animals with slight lesions may have had correspondingly slight locomotory disability, so slight that it may not have been noticeable but still sufficient to have prevented the animals from ranging widely enough to gather an adequate amount of feed.

In the animal which carried its head tilted towards the left the living Elaphostrongylus found in the left middle ear can undoubtedly be considered the cause even although local inflammatory changes could not be observed. The other example of an abnormal position of the head may also have a parasitic origin for the purulent inflammation of the middle ear.

Verminous pneumonia was also observed in many of our reindeer but the identity of the responsible parasite could not be established. The larvae found in the lungs resembled but were not identical with Muellerius capillaris. *Hobmaier & Hobmaier* pointed out the close resemblance between the larvae of Muellerius capillaris and of Elaphostrongylus. There is, then, the possibility that the larvae in the lungs of our reindeer were actually Elaphostrongylus larvae. More work is needed on this and other parasitological aspects of the reindeer disease which we have observed. In this report we have concentrated upon the disease itself.

It is conceivable that the common clinical signs of some degree of posterior paresis in reindeer may be produced by different aetiological entities. In our opinion, many if not most animals with these signs are examples of cerebrospinal migration of the Elaphostrongylus species we have encountered.

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SUMMARY

Twenty reindeer, most between eight and twelwe months old, were autopsied during the first half of 1961. Eight of these animals had shown degrees of ataxia or posterior paresis and the other, signs of general weakness. Most of the animals were in a state of inanition.

Nematodes belonging to the genus Elaphostrongylus (E. rangiferi) were found in the leptomeninges of the brain and spinal cord in

sixteen of these animals. E. rangiferi was also found in the skeletal musculature of five of seven animals examined and in the middle ear of one animal. Nematode larvae apparently of E. rangiferi, were present in various tissues of some animals. Eggs, often in large clumps, were found in the central nervous system and musculature of six animals and in the hypophysis of one animal.

The presence of the nematodes could be associated with a lymphohistiocytic and eosinophilic meningitis, particularly at the level of the cauda equina, the junction between cervical and thoracic regions of the spinal cord, and over the brain. In some animals inflammatory exudate was also present in the superficial layers of the brain. Parasitic granulomas were also found in the meninges about the cauda equina in many animals. No definite signs of mechanical damage could be observed in the brain and spinal cord which could be attributed to the nematodes. The meningitis was often associated with degenerative changes in the spinal nerve roots. Inflammatory and degenerative changes were observed in the proximal portions of the sciatic nerves.

The presence of Elaphostrongylus in the central nervous system has been considered to be the cause of the lesions and the locomotory disability observed. This form of cerebrospinal nematodiasis is apparently quite common among Swedish reindeer.

ZUSAMMENFASSUNG

Cerebrospinal und muscular Nematodiasis (Elaphostrongylus rangiferi) in schwedischen Renntieren.

Während des ersten Halbjahres 1961 wurden 20 Renntiere obduziert, von denen die meisten 8—12 Monate alt waren. 8 Renntiere hatten variierende Grade von Bewegungsstörungen gezeigt, während sich bei den übrigen allgemeine Schwäche wahrnehmen liess. Die Mehrzahl der Tiere war in einem Zustand von Inanition gestorben.

Bei 16 Renntieren fanden sich zirka 5 cm lange, zur Gattung Elaphostrongylus gehörige Nematodenwürmer. Diese Würmer lagen oft frei im Subduralraum sowohl des Gehirns als auch des Rückenmarks. Bei 5 von 7 Renntieren wurden dieselben Parasiten auch in der Körpermuskulatur und in einem Fall im Mittelohr angetroffen. Nematodenlarven wurden in einzelnen Fällen wahrgenommen. Eier, oft in Haufen gelegen, liessen sich in 6 Fällen im zentralen Nervensystem und in der Körpermuskulatur sowie in einem Falle im Hypophysengewebe feststellen.

Infolge des Vorkommens dieser Parasiten waren bei fast sämtlichen Renntieren die pathologisch-anatomischen Veränderungen in Form nonpurulenter lymphohistiozytärer, eosinophiler Meningitis vorzugsweise auf die Cauda equina, den Uebergang zwischen Hals- und Brustrückenmark sowie das Gehirn lokalisiert. In einer Reihe von Fällen war die Meningitis mit einer gelinden Encephalitis kombiniert. Ringsum die Cauda equina wurden in der Mehrzahl der Fälle multiple parasitäre Granulome in den Meningen wahrgenommen. Die Meningitis verursachte oft die Entstehung degenerativer Veränderungen in den

Spinalnervenwurzeln. In den proximalen Teilen des N. ischiadicus liessen sich ferner sowohl entzündliche als auch degenerative Prozesse nachweisen. Durch die Parasiten verursachte mechanische Schäden konnten im Gehirn und in den Rückenmarksgeweben nicht ermittelt werden.

Die Anwesenheit von Elaphostrongylusparasiten im zentralen Nervensystem wird als die Ursache der pathologischen Veränderungen und der beobachteten Bewegungsstörungen betrachtet. Diese Krankheit hat vermutlich eine ziemlich grosse Bedeutung für die schwedische Renntierzucht.

SAMMANFATTNING

Cerebrospinal och muskulär nematodiasis (Elaphostrongylus rangiferi) på svenska renar.

Under första halvåret 1961 obducerades 20 renar, de flesta 8—12 månader gamla. 8 renar hade visat varierande grader av rörelsestöranden medan de andra visat allmän svaghet. Flertalet hade dött i ett inanitionstillstånd.

På 16 av renarna fanns ca 5 cm långa nematodmaskar tillhörande släktet Elaphostrongylus. Maskarna lågo ofta fritt i subduralrummet på såväl hjärna som ryggmärg. Hos 5 av 7 renar påträffades samma maskar även i kroppsmuskulaturen, och i ett fall i mellanörat. Nematodlarver har påträffats i enstaka fall. Ägg, som ofta lågo hopade, kunde i 6 fall upptäckas i centrala nervsystemet och i kroppsmuskulaturen samt i ett fall i hypofysvävnaden.

Som en följd av parasitförekomsten hade så gott som samtliga renar patologisk-anatomiska förändringar i form av nonpurulenta lymfohistiocytära, eosinofila meningiter företrädesvis lokaliserade till Cauda equina, övergången mellan hals- och bröstryggmärg samt hjärnan. I en del fall var meningiten kombinerad med en lindrig encefalit. Multipla parasitära granulom i meningerna omkring Cauda equina iakttogs i flertalet fall. Meningiten gav ofta upphov till degenerativa förändringar i spinalnervrötterna. I N. ischiadicus proximala delar kunde även såväl inflammatoriska som degenerativa processer påvisas. Några mekaniska skador orsakade av parasiterna har i hjärna och ryggmärgsvävnader ej med säkerhet kunnat konstateras.

Förekomsten av Elaphostrongylusparasiter i centrala nervsystemet har ansetts vara orsaken till de patologiska förändringarna och till de iakttagna rörelsestörandena. Sjukdomen förmodas ha tämligen stor betydelse för den svenska rennäringen.

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Fig. 1. Female reindeer calf, 8 months old. Total paraplegia. (O 370/61).

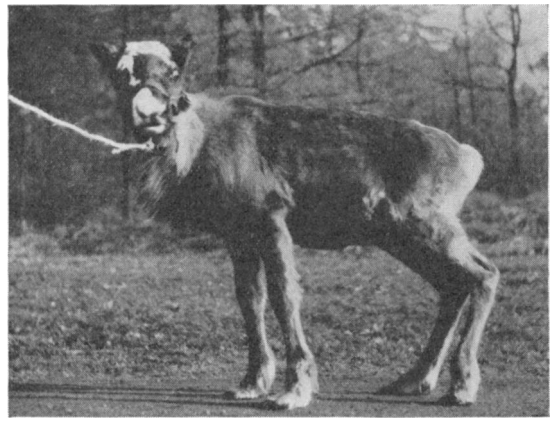

Fig. 2. Male reindeer calf, about 10 months old. Posterior weakness, note knuckling over of the near hind pastern. (O 1430/61).

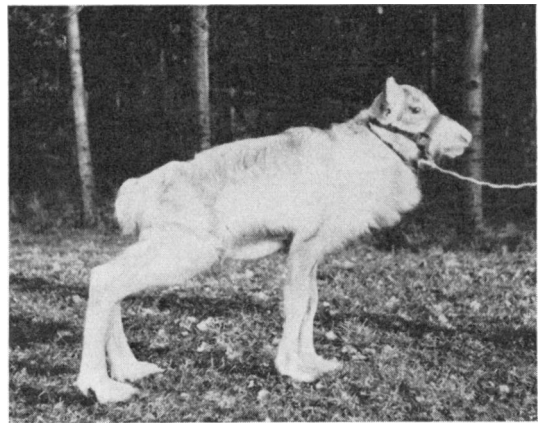

Fig. 3. Male reindeer calf, about 10 months old. Obvious posterior weakness; most of the weight is borne by the fore legs. (O 1430/61).

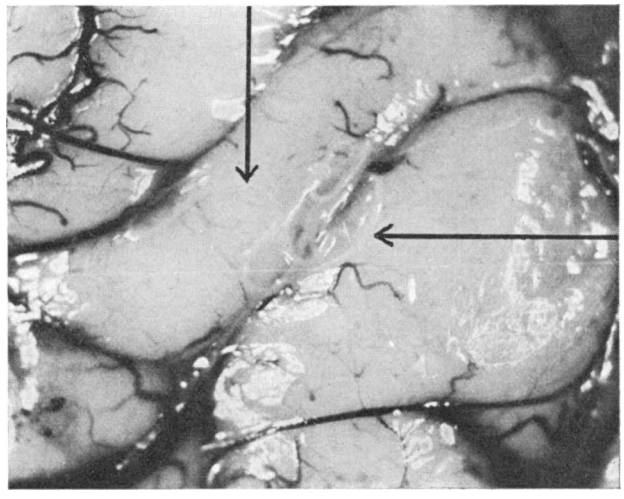

Fig. 4. Cerebrum, with a nematode of the genus Elaphostrongylus lying under the leptomeninges. The nematodes can be difficult to detect because of their light colour (arrows) and the camouflage afforded by the meningeal blood vessels. (O 7180/61, about 4 ×).

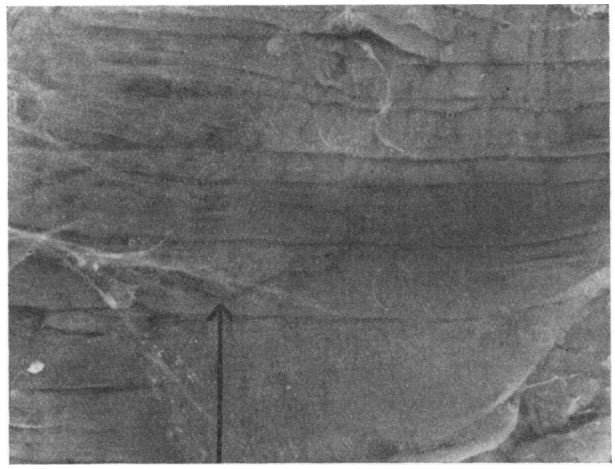

Fig. 5. Skeletal musculature. A brown-coloured E. rangiferi (arrow) is visible through the epimysium. Recent haemorrhage to the right of the nematode. (O 3053/61, about 4 ×).

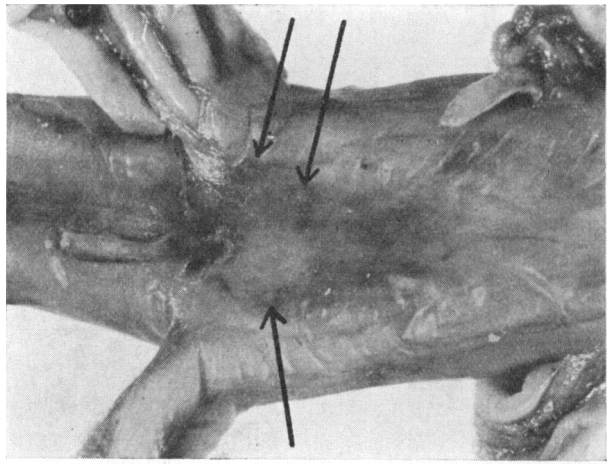

Fig. 6. Ventral surface of the spinal cord at the level of the lumbosacral joint. Three small nodules (arrows) can be seen in the ventral dura of the Cauda equina. (O 1198/61, about 3 ×).

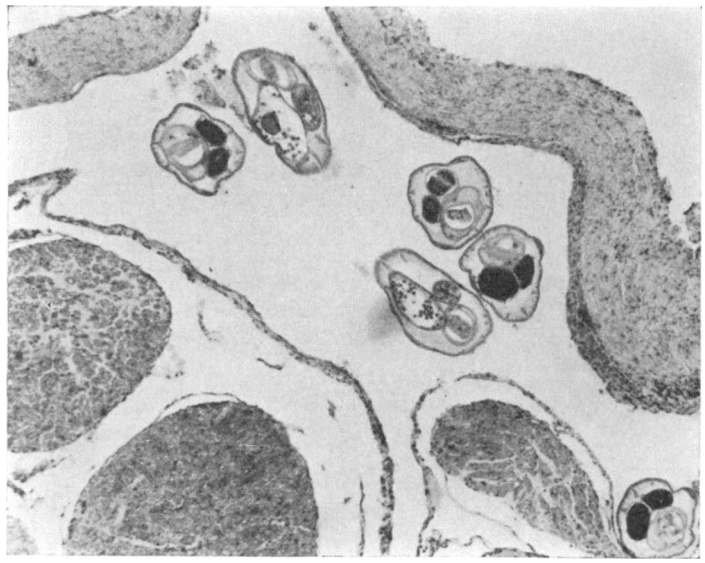

Fig. 7. Spinal cord, cervical region, with an Elaphostrongylus nematode cut in cross-section at several levels. No reaction in tissues adjacent to living nematodes. (O 1198/61 haematoxylin and eosin, 300 ×).

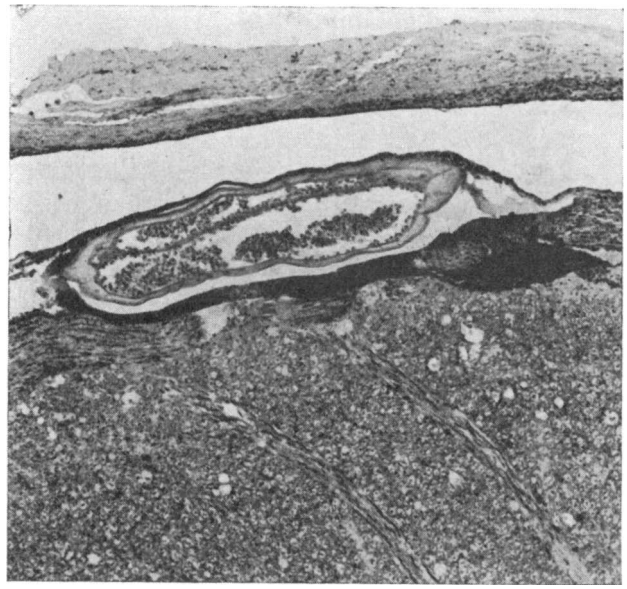

Fig. 8. Spinal cord. An Elaphostrongylus nematode, here cut in sagittal section, in the leptomeninges and surrounded by haemorrhage. (O 1198/61, haematoxylin and eosin, 50 ×).

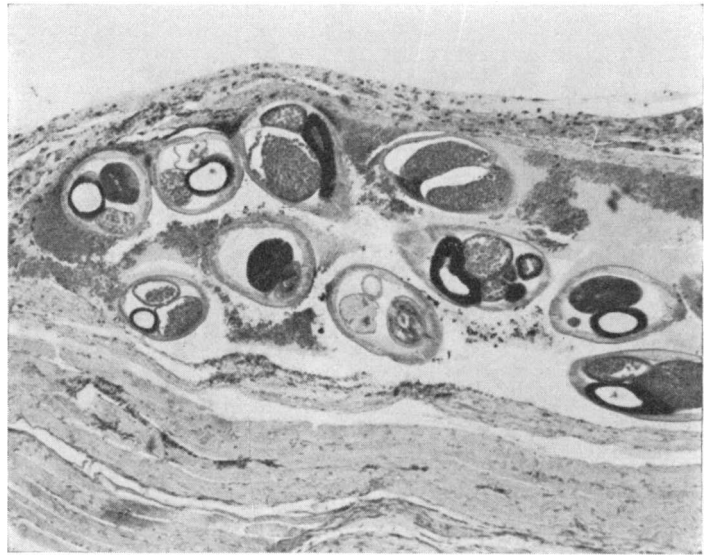

Fig. 9. Immediately under the epimysium of the latissimus dorsi is an Elaphostrongylus nematode cut in cross-section at several levels. Note the surrounding haemorrhage and the ingested blood. (O 2151/61, haematoxylin and eosin, 50 ×).

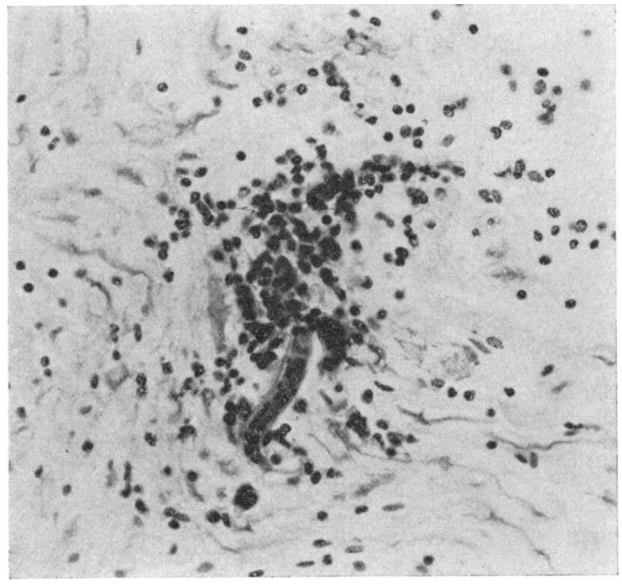

Fig. 10. Root of a spinal nerve. Nematode larvae surrounded by lymphocytes. (O 1710/61, haematoxylin and eosin, 500 ×).

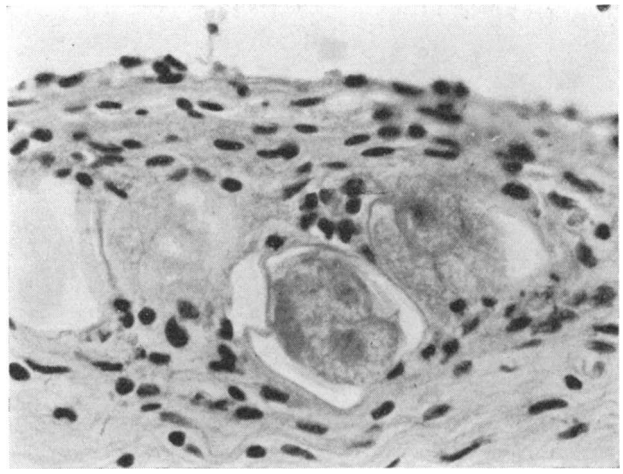

Fig. 11. Leptomeninges of the spinal cord containing a clump of nematode eggs in multiple cell stages. (O 1710/61, haematoxylin and eosin, 500 ×).

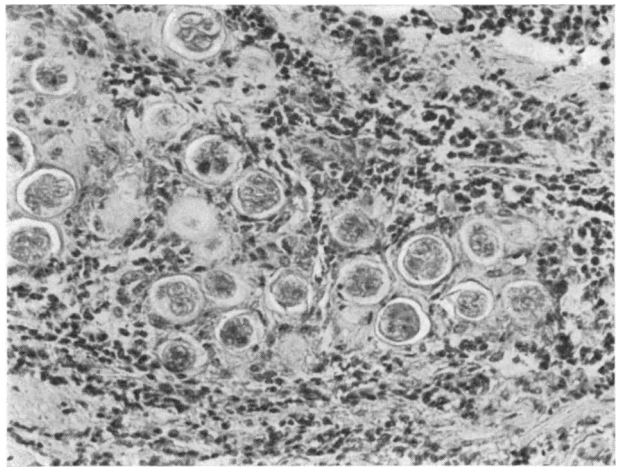

Fig. 12. Hypophysis. A large clump of nematode eggs, many at multiple cell stages. (O 2151/61, haematoxylin and eosin, 150 ×).

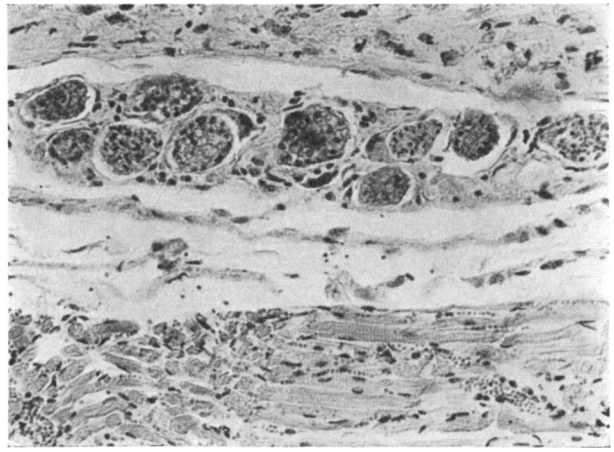

Fig. 13. Epimysium of the latissimus dorsi, nematode eggs in multiple cell stages. (O 3205/61, haematoxylin and eosin, 150 ×).

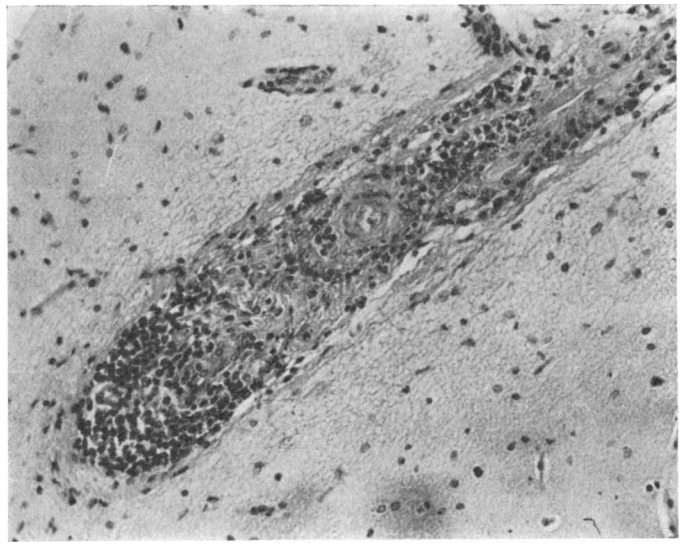

Fig. 14. Cerebrum with a lymphohistiocytic and eosinophilic meningitis in a sulcus. (O 1198/61, haematoxylin and eosin, 150 ×).

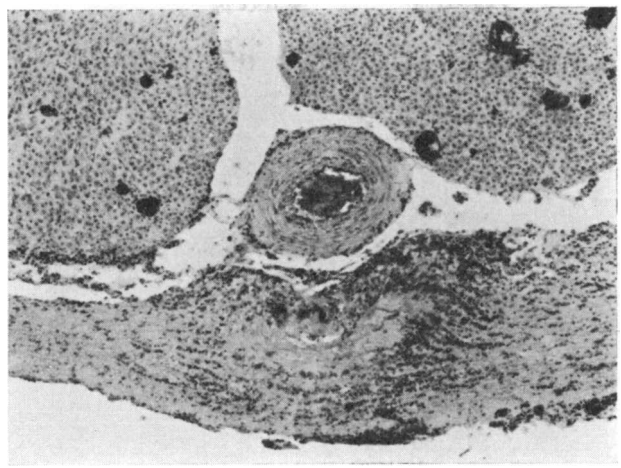

Fig. 15. Cauda equina with a parasitic granuloma in the dura. In the centre of the granuloma are small parasite remnants surrounded by lymphocytes, plasma cells, eosinophils, and some giant cells. (O 1155/61, haematoxylin and eosin, 50 ×).

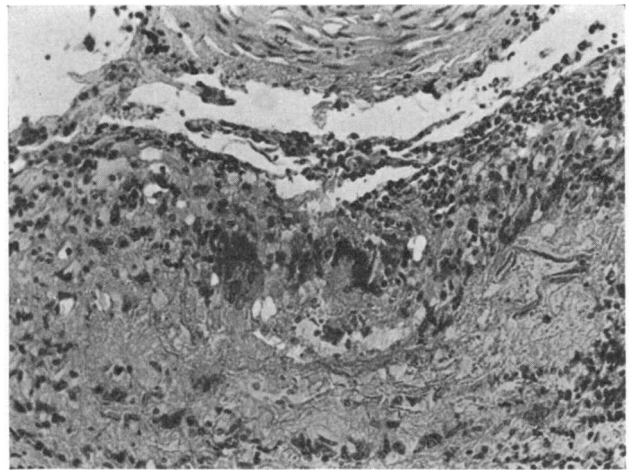

Fig. 16. Detail of Fig. 15 at a greater magnification. (150 ×).

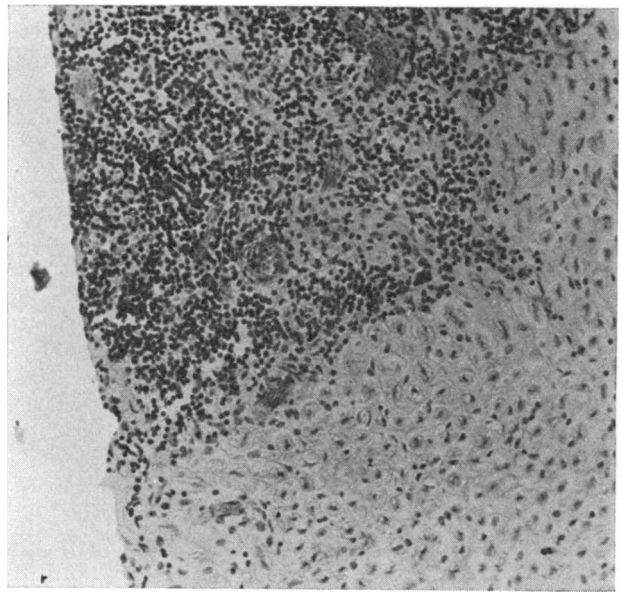

Fig. 17. Spinal nerve root arising from the cauda equina. Focal lymphocytic infiltration. (O 1155/61, haematoxylin and eosin, 150 ×).

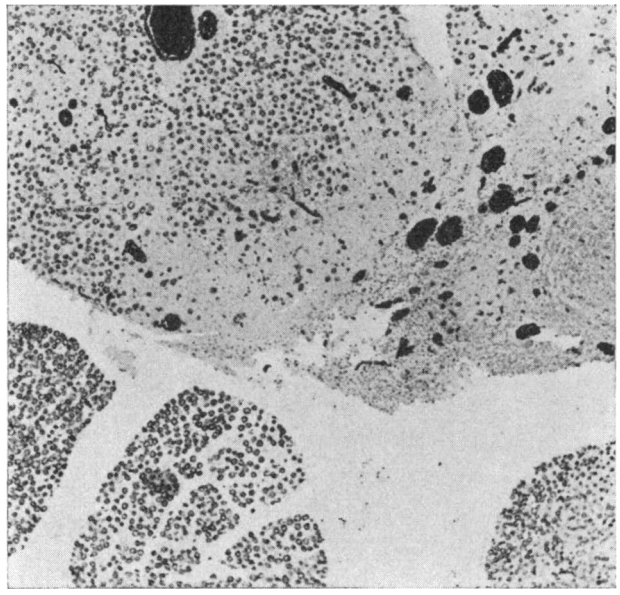

Fig. 18. Spinal nerve root arising from the cauda equina. To the right, complete demyelinisation. Lymphocytic infiltration of the perineurium. (O 1155/61, Mahon, 50 ×).

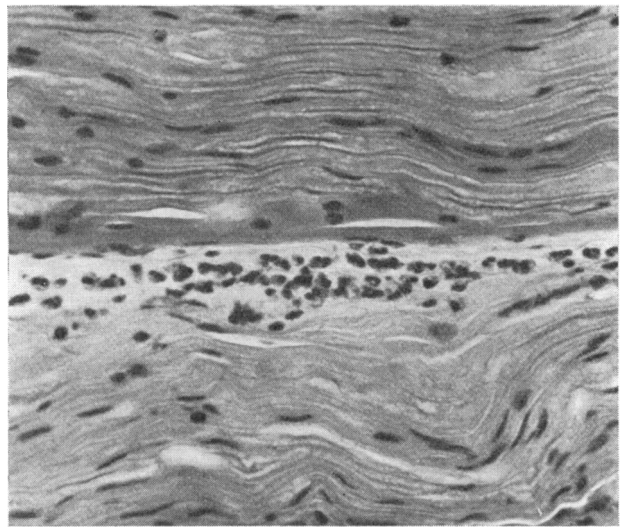

Fig. 19. Proximal portion of a sciatic nerve. Infiltration of the perineurium with eosinophils. (O 1198/61, haematoxylin and eosin, 300 ×).

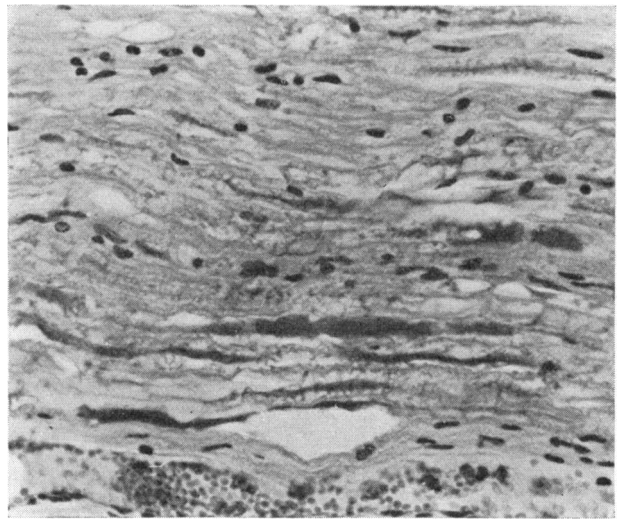

Fig. 20. Proximal portion of a sciatic nerve. Extensive demyelinisation and degeneration of the axon cylinders. (O 2105/61, haematoxylin and eosin, 300 ×).