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PATHOLOGY OF THE MIGRATION PHASE OF TAENIA HYDATIGENA (PALLAS, 1766) LARVAE

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Abstract. The pathological picture of the migration phase of *C. tenuicollis* in pigs is characterized by a haemorrhagia within the liver parenchyma and under the liver surface. The haemorrhagia, which represents a migrational canal, is induced by the destruction of liver sinuses by migrating larvae. Approximately on day 10 p.i. a serofibrinous peritonitis occurs and free cysticerci appear in the exudate. On days 14—16 p.i. the exudative peritonitis may increase. The cysticerci are localized under the serosas or on them. On about day 10 p.i. even the pulmonary form of the disease may occur. On day 13 p.i. the cysticerci are present in the lumen of lung arteries or they migrate out of them. The changes in the lungs and on the pleura, as well as their dynamic changes, are identical with the changes in the liver and on the peritoneum. The period on days 21—24 p.i. is characterized by extensive synechiae of serosas and the cysticerci are firmly attached to the serosas. On day 35 p.i. the connective tissue adhesions persist and many of the cysticerci exhibit dystrophic changes or are dead and often already calcified. The wall of the pseudocyst, in which the cysticercus is situated, consists of the fibrocytes and serosa, and its cavity is not lined with endothelium, as it is the case in *C. bovis* and *C. cellulosae*.

Cysticercus tenuicollis (the larval stage of *Taenia hydatigena* Pallas, 1766) is a common parasite of sheep, wild ruminants and omnivorous animals and it may sometimes occur also in cattle or goat. However, it has been reported even in dog, small rodents and primates (Sweatman and Plummer 1957, Smith and Jones 1963, Kuntz and Myers 1967). These findings are not considered to be of importance, since most often a single, fully morphologically differentiated and infective cysticercus occurs on the omentum and serosas of viscera and does not cause any pathological changes in the infected organ. Young developmental stages, however, before reaching their definitive localization migrate through the liver and lungs. In massive infections, the infected organs are seriously damaged, which results in a clinically apparent disease. The massive infections often cause the death of the host within a short time, particularly if young animals are infected (Jubb and Kennedy 1963, Joest 1967, Gradinarski 1982).

In the past, the acute form of visceral cysticercosis frequently occurred in grazed young pigs and sheep also in our country. However, some of the recent papers (Jurášek et al. 1981, Breza 1983) indicate that this infection may occur even at the present time, in both traditional and new technologies of breeding. Moreover it should be considered that even man may become infected with the eggs of *T. hydatigena* (Štěrba and Šlais 1972).

In comparative studies on the morphology of some cestode larvae the ultrastructure and histochemistry of *C. tenuicollis* at various stages of development have been dealt with (Hulínská 1978, 1979, 1980a, b). Attention was paid also to the pathological changes caused by the cysticercus and their dynamics in the course of the migration phase of infection. The knowledge and explanation of these changes are significant for the assessment of the pathogenicity of *T. hydatigena* larvae, differential diagnosis of parasitic diseases of liver and lungs of domestic animals, as well as for a model evaluation of the efficacy of new drugs used for cestode control which are being

developed by research institutes of the pharmaceutical industry. The present studies deal with the migration phase of the infection, since this period is the most important from the above points of view.

MATERIAL AND METHODS

Seven hybrid dogs of both sexes were infected with fully developed cysticerci recovered from the omentum of slaughtered sheep. The effect of the infection was verified by repeated examinations of dog faeces. The pigs were infected with mature proglottides excreted by the dogs after an anthelminthic treatment. The infectivity of eggs contained in the proglottids was tested after Silverman.

Five piglets weighing 10—30 kg and two piglets of China minipig, all at the age of 4 weeks, were infected perorally with 1—3 proglottids of *T. hydatigena* each. In all cases this was a massive infection. As it is reported in the literature, the number of eggs in mature proglottids varies from 700 to 27,000 (Sweatman and Plummer 1957). Another 8 piglets weighing 25 kg were infected approximately with 4,000 eggs of *T. hydatigena* in order to evaluate the pathological changes (on days 15 and 24) and to test newly developed drugs.

The pigs were bled to death by cardiac puncture after previous narcosis with Thiopental stunning. They were killed on days 7, 10, 13, 14, 15, 16, 21, 24, and 35 after infection and dissected. Their lungs, livers, intestines, mesenterial and portal lymph nodes and brains were examined histologically.

The samples for histological examinations (excised organs with cysticerci and cysticerci alone) were fixed with 10 % neutral formol and after Baker, embedded into paraffin using the common technique and cut. The sections were stained by haematoxyline-eosine, Van Gieson's method in combination with staining on elastine, by trichrome after Masson, and by the method after Mallory.

The excised organs with larvae and cysticerci destined for electron microscopical examination were fixed with 6 % glutaraldehyde in cacodylate buffer, postfixed with 1 % solution of OsO_4, dehydrated by an increasing acetone series and embedded into Vestopal. The sections were stained with uranyl acetate and lead citrate and examined with the JEM 100B electron microscope.

RESULTS

The macroscopy of the pathological changes

At the early phase of infection, on days 7—8, there is only a slightly increased amount of mostly transparent, light rose fluid in the abdominal and thoracic cavities and single fibrin fibres on the omentum and serosa of small intestine. The serosa of some small intestine loops is sometimes red. At this stage, even after a massive infection, the liver need not be enlarged (even if it contains 300—500 larvae), but it is penetrated by numerous dark red haemorrhagic canals visible through the Glisson's capsule. White-grey point-like foci are detectable under the serosa and within the liver parenchyma. These changes occur more frequently on the diaphragmatic than on the visceral flat of the liver.

On day 10 p.i. the amount of the red ascitic fluid in the abdominal cavity is markedly increased, while the pleura sometimes does not exhibit any pathological changes or fibrin fibres may be present on it. However, there are marked changes in the liver. It is increased and numerous haemorrhagic canals (migration canals) are visible through the capsule on its diaphragmatic and visceral sides. The surface of the liver is rugged and continuous fibrin depositions with cysticerci measuring about 2×3 mm are visible between the liver lobes. Free cysticerci are usually encountered in the exudate in abdominal cavity, on the serosa of stomach and intestines and on the omentum. They can be easily removed from these places.

In some cases, when even lungs are invaded by the larvae, pneumonic foci appear in the diaphragmatic lobes within the range of individual lobules at this time.

The changes found on days 14—16 p.i. are similar. There may be even 800 ml of the serofibrinous exudate in the abdominal cavity. The serosa of the colon is often markedly congested. In case of pulmonary cysticercosis, the changes in the lungs are more marked than on day 10 p.i. There are conspicuous dark red-brown foci within the range of a lobule and fields covered with a fibrin network. The cysticerci are visible through the lung serosa. They are situated mostly on the margin of diaphragmatic lobules and on cardiac lobules.

On days 21—24 p.i. the serofibrinous exudate in the abdominal and thoracic cavities (if also pulmonary cysticercosis is involved) is resorbed and connective tissue synechiae of parenchymatous organs and intestinal loops to the walls of abdominal and thoracic cavities, as well as areal synechiae of organs, are mostly found. The liver is grown together with the diaphragm and surrounding organs through the connective tissue and if the synechiae are released, the liver parenchyma is severely damaged. The loops of the small intestine and threads of the large intestine adhere to the parietal leaf of the peritoneum through the connective tissue. Cord-like connective tissue adhesions occur also between the two leaves of the pleura and the lungs contain cysticerci measuring about 5 mm in the diameter and mostly localized under the serosa. Even synechiae of both leaves of the pericardium may be encountered. At this stage, the liver is usually enlarged and rugged. Numerous transparent bladders with marked polar turbidity (cysticerci) appear between the synechiae on the liver surface, particularly on the diaphragmatic portion. Some of them are spherical, others oval, and the migration canal at the stage of reparation, now of yellow colour, may be discerned in their proximity in the liver parenchyma. Yellow foci measuring up to 1×2 mm and cysticerci of about 2×3 mm are localized within the liver parenchyma. Solid, yellow, barley-like foci containing a calcified cysticercus are sometimes encountered under the serosa. Cysticerci measuring about 3×5 mm, either single of in clusters, occur on the parietal lèaf of the pleura which is rough and congested at some sites. Numerous cysticerci may be found also on the omentum, particularly near the liver, and on the serosa of intestines. Some of the bladders are slightly muddy at this time.

In the period around day 35 p.i. the section finding is not very changed. It is characterized by synechiae resulting from the passed peritonitis (or pleuritis). Often the liver is not enlarged and numerous bladders measuring 5 mm in diameter up to 8×10 mm prominate on its surface. Some of the bladders are muddy. In the section through the liver, thin-walled canals with milky, thin contents, yellow nodes or migration canals filled with a jelly-like matter are present. The cysticerci on the omentum and serosas reach the size of a pea and they are firmly attached, transparent and with a conspicuous scolex. If the serosa is carefully cut, they easily slip out of the cyst. Some of them are muddy or turn to yellow, hard nodes.

The histological picture and dynamics of the pathological reaction at the migration stage of *C. tenuicollis*

On about day 7 p.i. the migration canals in the liver appear histologically like a haemorrhagia occupying a greater part of the liver lobule. The canal usually contains the cysticercus, which is a bladder with a small central cavity. A large number of erythrocytes with a proportional number of lymphocytes and neutrophilic granulocytes are accumulated around the larva. The liver cells on the margin of the haemorrhagia are compressed and some of them are dystrophically changed. The cysticercus is sometimes surrounded directly by the liver cells and the migration canal filled with blood is situated in another part of the lobule (Plate I, Fig. 1). Between the body of

larva and the pressed liver parenchyma there is usually only a small number of leucocytes. The surrounding tissue is without a marked inflammatory cellulization and only sometimes the periportal fields are slightly infiltrated by lymphocytes. The lymphocytes may be accumulated also in the proximity of the central vein of the infected lobule.

On day 10 p.i. the histological picture of the liver is similar, but somewhat more complicated. Numerous migration canals—haemorrhagias—fill almost the whole space of the liver lobule and often extent up to the neighbouring lobule. Haemorrhagias with cysticerci or without them or cysticerci filling almost the whole acinus and surrounded not by a haemorrhagia but directly by liver cells with signs of pressure atrophy are visible in histological sections. Even at this period the inflammatory reaction around the larvae is very weak. Only a small number of leucocytes are accumulated around the cysticercus and they are more numerous at the periphery of the haemorrhagias and sometimes also in the sinusoids. Older areas of the migration canals left by the cysticerci are filled with connective tissue detritus, numerous neutrophilic granulocytes and a larger number of eosinophilic granulocytes; there are also conspicuous cells resembling young forms of leucocytes and erythrocytes (Plate II, Figs. 1 and 2). If the cysticerci penetrate into lungs (which occurred only exceptionally in our experiments on day 10 p.i.), they induce extensive haemorrhagias in the vicinity of larger bronchi, and the alveoli within the lobule are filled with an edema fluid. The interlobular septa of the infected areas are widened by the edema.

An almost dentical histological picture of the liver is characteristic also for the period of days 13—16 p.i. The leucocytes (neutrophilic leucocytes) are accumulated in numerous migration canals and the inflammatory infiltration of periportal spaces is stronger. The lymphocytes and leucocytes participate in the infiltration, but there is only a small number of eosinophilic granulocytes and plasma cells. Numerous mitoses of cells and activation of fibroblasts are encountered at the periphery of the lobule. The endothelial cells in the sinusoids are distinctly activated. The lymphatic spaces under the liver serosa are dilated and a strong inflammatory cellulization occurs there. The epithelial cells of the serosas are swollen and the serosa is covered with fibrin.

In case of pulmonary cysticercosis the histological picture of the lungs is very interesting at this time. The migration canals in form of extensive haemorrhagias are situated near the artery with an eosinophilic congested wall, numerous neutrophilic leucocytes are accumulated in the lumen of migration canals and a marked proliferation of cells of macrophage type and accumulation of lymphocytes occur in their vicinity. The interlobular connective tissue is edematous, the vessels and lymphatic capillaries beneath the serosa are dilated and their endothelium is swollen. Widened interalveolar septa in the region of the infected lobule exhibit an inflammatory infiltration; desquamated alveolar epithelia are accumulated in the alveoli. At that time, however, the cysticerci may be still found in the lumen of vessels of the arterial type and there occur also cysticerci which have destructed the vessel and are just leaving it at a simultaneous formation of an extensive haemorrhagia (Plates III, IV). Such arteries often contain a fresh wall thrombus, but even arteries almost completely filled with an arising thrombus may be found (on day 16 p.i.) (Plate V. Figs. 1.2).

Three weeks after infection the histological picture of the liver and lung changes is characterized by the signs of restitution. The migration canals are filled with a necrotic matter with nuclear detritus, around which the cells of histiocyte type proliferate and arrange in palisades. There are also multinuclear giant cells of the type of foreign bodies, even those of Langhans' type, and large macrophages with

phagocytized erythrocytes and leucocytes (Plate II, Fig. 2). Bands of connective tissue, sometimes with hemosiderin deposition, are situated at the periphery of the remnant of the migration canal. The cysticerci localized under the serosa and within the liver parenchyma are surrounded with a relatively thick layer of circularly arranged connective tissue (Plate I, Fig. 2). Sometimes the connective tissue capsule is without a marked cellulization, at other times it is strongly infiltrated with cells, particularly with lymphocytes. No inflammatory cellulization occurs in the parenchyma around the capsule. Single light macrophages are usually found in the cavity of this pseudocyst. Sometimes, however, it contains eosinophiles, neutrophilic leucocytes, a large number of macrophages and a necrotic mass indicating the death of the cysticercus. In the lungs, the traumas in the parenchyma are healed by the proliferation of cells of histiocyte type and by connective tissue scarring. The newly formed young connective tissue is congested. It is usually infiltrated by eosinophiles and contains nodular accumulations of lymphocytes and hemosiderin depositions.

Approximately on day 35 p.i. the residues of migration canals in the liver and lungs are encapsuled by the connective tissue. In the centre of the area demarcated by the connective tissue there is a nuclear detritus, leucocytes, even erythrocytes, and more to the periphery, there are lymphocytes and plasma cells and few eosinophiles. The eosinophiles are more numerous in the connective tissue of the demarcating rim. The residual necrotic masses of the former migration canals are often surrounded by macrophages with a light plasma and giant multinuclear cells, and at the periphery of this nodule, there are nodules formed by lymphoblasts.

At this time, the cysticerci in the liver are mostly surrounded with eosinophiles and they are encapsuled by a thick layer of connective tissue with clusters of epitheloid cells, sparsely diffusely infiltrated by eosinophiles. The cysticerci localized beneath the serosa are surrounded with a fine connective tissue capsule without cellular infiltration. The liver parenchyma contains even scars with pigment and the connective tissue is often hyalinized. The lungs contain connective tissue scars strongly infiltrated by eosinophiles and with frequent hemosiderin depositions or elongated necrotic foci with granulomatous reaction like in the liver. The pleura, particularly above the focal changes in the parenchyma, is usually thickened by the connective tissue and forms villous processes.

The morphology of the parasite at various stages of the migration phase

One week after infection the larvae are spherical or oval bladders with differentiating inner cavity partitioned by protoplasmic processes. The bladders measure 0.8—1.0×0.3—0.7 mm and their wall is 23—26 μm high. On day 10 p.i. the bladders measure 1.0—2.6×0.9—1.7 mm and germinative cells of the scolex anlage are accumulated on one of their poles. In massive infections, however, the liver lesions contain also elongate larvae of smaller size and without the cellular anlage of scolex. On day 13 p.i. the size of the cysticerci is 1.3—1.8×1.4—1.9 mm. The cysticerci in liver and lung lesions are smaller than those swimming freely in the exudate from abdominal cavity. At this time the cysticerci have a scolex anlage with a canal invaginated up to the depth of about 0.03 mm. On day 14 p.i. the scolex anlage is somewhat larger (0.12×0.18 mm), the invaginated canal is longer and the bladders measure 3.5—4.9×1.8—3.9 mm.

On days 15 and 16 p.i. the cysticerci measure 3.6—4.8 mm in length and 1.9 to 3.2 mm in width on the average. The development of the scolex proceeds in both the cysticerci from the migration canals and those from the exudate. The scolex anlage

measures 0.21×0.46 mm and has a conspicuous invaginated canal with rostellar cone projecting from its bottom.

On day 21 p.i. the size of the cysticerci is 4.5—6.7×3.4—4.5 mm. The differentiating scolex has a spiral canal and the rostellar cone further differentiates into a bulb and prebulbar region. Sucker anlages are already formed. On day 24 p.i. the prebulbar region grows intensively and the bulb sinks. The suckers continue to differentiate.

On day 35 p.i. the larvae are 10.0—12.0 mm long and 6.0—9.0 mm wide. The cysticerci from the pseudocysts on the omentum have a fully differentiated scolex with spiral canal, rostellum with hooks and morphologically differentiated suckers. The scolex parenchyma contains calcareous bodies. In the cysticerci from the liver, the scolex with rostellum is not yet completely differentiated; it is divided into bulb and prebulb, the tegument of which contains forming hooks. The morphological characters of the larvae at individual stages of development on days 7—35 p.i. are summarized in the table.

Another of the characters helping to determine the age of the larva is the fact that the microtriches with differentiated base and point appear on the bladder in the 3rd week (on 21st day) after infection, whereas in younger larvae, finger-like microvilli with rounded distal ends are formed (Plate VI, Figs. 1, 2). From the practical point of view, however, this is not very important, since the configuration of the bladder surface is visible only in the electron microscope.

The differential diagnosis of the migration phase of *C. tenuicollis*

The pathological changes arising during the migration of *T. hydatigena* larva in the liver are very conspicuous. These are the haemorrhagias affecting one liver lobule or several neighbouring ones. Their extent is much wider than that of haemorrhagia induced by nematode infection. The inflammatory infiltration of the liver tissue is minimal or completely lacking. The migration canals, which are essentially of the sa me character, arise even during the migration of young trematodes through the liver before reaching the site of definitive localization (*Fasciola hepatica*, *F. gigantica*) or trematodes migrating through the liver even after attaining sexual maturity (*Fascioloides magna*). The damage of the vessel system induced by the migration of trematodes (Blažek 1973) is similar to that occurring in pigs after the infection with *C. tenuicollis*. The migration of trematodes, however, is slower and therefore the haemorrhagias are often bordered by cellular infiltrates of various types. In some cases, the typical dark brown pigmentation in the liver lesions indicates what species of trematodes is involved (*F. magna*) (Blažek and Gilka 1970).

A decisive factor for the exact determination of the etiology of the detected changes is the finding of the larva in histological sections. The body structure of cestode larvae is typical. A very young larva is an elongate compact structure consisting of mutually connected cells, the nuclei of which are only slightly stained with haematoxyline-eosine. It is covered with a fine but distinct tegument. At a later stage, the larva is a bladder with the wall differentiated into tegument and subtegumental cellular layer and with the central cavity. *C. tenuicollis* at the beginning of migration is always already differentiated, in contrast to larvae of other cestode species, in which the central cavity is lacking mostly even 14 days after infection (e.g. *C. bovis*). The larvocyst of *Echinococcus granulosus* at the early stage of development differs from *C. tenuicollis* in the size. Two or three weeks after infection it measures approximately 0.2 mm, whereas *C. tenuicollis* has 5 mm in diameter at the same time (Nemeséri and Holló 1965 ex Jurášek et al. 1981).

The young stages of trematodes may be easily distinguished from the cestode

larvae. In the sections through their bodies, the intestinal branches and often also the typical ventral suckers are always visible. Also the configuration of the trematode cuticle is quite different from the tegument of cestode larvae and usually bears typical spines. Even the pathologist with a limited erudition in parasitology should not mistake these two parasites during the examination of the material.

It is rather easy to distinguish the pseudocysts in the liver with *C. tenuicollis* at the end of the migration phase from the pseudocyst of *E. granulosus*, since the hydatid pseudocysts have a typical wall structure (Joest 1967). The tissue reaction to young, developing larvocysts of *E. granulosus* in pigs is usually strong and it is characterized by a three-layered envelope surrounding the larva. Particularly typical is the layer of high, slender, epitheloid cells which often turn to multinucleate cells. This epitheloid rim is often necrotic (Šlais and Vaněk 1980). In fertile cysts, the diagnosis is quite unambiguous if scoleces are found.

DISCUSSION

The pathological changes observed in our experimental animals in general conformed to the known findings in this larval cestodosis. Some deviations occurred in some details and time, which is understandable considering the species and individual variability of the biological material.

The migration canals were found already on day 7 p.i. As it was reported in the literature, the macroscopical changes in the liver may be found already on days 4—7 p.i., and from day 10 p.i. still more migration canals are found, particularly in the pig (Joest 1967). Other authors (Heath and Smyth 1970) found migration canals in the liver on days 7—10 p.i. The oncospheres of *T. hydatigena* penetrate into the villus of intestinal mucosa in the jejunum region (Heath 1971) and get to the organ of definitive localization within several hours. Then begins the period of the postoncospheral reconstruction. The larva starts to move after the formation of the muscle system (Heath and Smyth 1970). It was found that the muscles in the oncosphere of *T. taeniaeformis* disappear within 3 days after infection of the intermediate host and new muscles are formed beneath the tegument of larva. It is improbable that the muscles formed up to day 5 p.i. would enable the larva a larger moving. On day 6 p.i., however, the muscle bundles are already developed and they are still more marked on day 7 p.i. (Engelkirk and Williams 1982).

It may be supposed that this is the case also in *T. hydatigena* larvae. The bundles of muscle fibrils were found in the larvae on day 7 p.i. (Hulínská 1980a, b) and therefore the potential beginning of the migration may be considered to occur on day 7 p.i. At the time when the larva starts to migrate through the liver, its bladder wall is already well differentiated and the cavity is either outlined or completely distinct. We do not think that the haemorrhagic canals could arise only after the penetration of the oncospheres into small vessels, as it was reported by Migaki and Zinter (1974).

Of interest is the finding of the cysticerci in lung arteries on day 13 p.i., when the cysticerci were found already in the abdominal cavity. At that time, however, the wall of the invaded artery was damaged and the larva immigrated into the lung parenchyma. This may mean that the larvae in the lungs developed somewhat more slowly than those in the liver and that they remained (at least some of them) in the arteries for a longer time or that larvae capable of active migration got from the liver into lungs through the blood circulation and immediately migrated out from the lung arteries. Although the latter hypothesis is more probable, the finding of wall thrombi in the arteries with cysticerci and thrombosis of vessels at the stage of the organization

of thrombus with a residual lumen suggests that the period of postoncospheral reconstruction might occur even in vessels.

As it was demonstrated, the migration of the cysticerci through the pig liver is very rapid and already on day 10 p.i. they may be found in the exudate from abdominal cavity. It is possible that they have got there earlier, as it is indicated by the great amount of the serofibrinous exudate. Abuladze (1964) (ex Jurášek et al. 1981) reported that the cysticerci migrate from the pig liver into the abdominal cavity from day 9 p.i., whereas other authors (Sweatman and Plummer 1957) never found cysticerci in the abdomen of pigs.

In the lambs, the migration seems to be slower, since only single cysticerci were found by Sweatman and Plummer (1957) in the abdominal cavity on day 10 p.i. The cysticerci appeared there mostly on days 18—20 p.i. and it is interesting that, according to these authors, the pulmonal infection started only at that time or still later, on day 43 p.i. Also the character of the pathological changes in the lamb liver indicates that the migration of cysticerci is slower, as it was found in our comparative histological studies of spontaneous lamb infections. The activation of fibroblasts and formation of collagenic fibres were distinct even in the immediate proximity of young larvae, which has never been observed in pigs. Also in goats, the migration seems to be slower. Pathak et al. (1982) did not observe any pathological changes in experimental kids on day 7 p.i. and wide subcapsular migration canals were found in the liver still on day 60 (!) post infection.

A striking fact in the histological picture of liver lesion in pigs is the absence of a marked cellulization which also indicates that the migration is rapid. Haemorrhagias with and without cysticerci, as well as cysticerci surrounded directly with the liver tissue, are regularly found in the same histological section. A more pronounced exudation or cell proliferation occurs only in older parts of the canals during their reparation. Tissue eosinophilia, i.e. a marked proportion of eosinophiles in the cellular infiltrate, is not typical for the migration of *C. tenuicollis*. A larger number of eosinophiles appear only in young scars around the bladder of dying larvae and in the connective tissue wall of pseudocyst around them. At the same time there is a marked hyperplasia of the sessile lymphatic tissue with a blastic transformation of lymphocytes in these cases. This is probably a morphological reaction of the host organism to the elimination of a larger amount of antigens from the disintegrated tissue of larvae.

On the basis of the pathological changes in the organs even the phase of the disease induced by *C. tenuicollis* may be determined, as it was described already by Anishchenko (1954) (ex Jurášek et al. 1981). According to this author, the latent period is on days 1—6 p.i., which is the period from the penetration of the oncosphere into the intestinal wall up to the beginning of migration in the liver. At that time, no pathological changes can be detected in the liver and the disease is clinically silent. The period of the development of traumatic hepatitis induced by cysticercus (on days 6—12 p.i.) is limited by the beginning of cysticercus migration into liver parenchyma and beginning of its immigration into the abdominal cavity. The third period (on days 12—20 p.i.) is characterized by the origin and development of serofibrinous peritonitis. A restitution of the changes in the liver and healing occur on days 20 to 30 p.i.

Anishchenko's division of the course of the disease is in agreement with our own results. It corresponds to the present knowledge of the activity of *C. tenuicollis* during the infection and to logical deduction made from the observed morphological changes. Most important from the clinical viewpoint is undoubtedly the period of the massive destruction of liver produced by migrating larvae, as it was described by Pathak and Gaur (1981). During their studies of serum enzymes, the authors

Table 1. Morphological characters of larvae at individual stages of development

		Time of infection in days									
		7	10	13	14	15	16	21	24	34
Scolex part	Stage of scolex differentiation									
	Only bladder developed	+								
	Cellular scolex anlage		+	+	+	+	+			
	Cellular scolex anlage with invagination			+	+	+	+			
	Scolex anlage with rostellar cone					+	+			
	Cellular scolex anlage with differentiating rostellar cone							+	+	
	Scolex with hooks and suckers									+
Bladder	Microvilli	+	+	+	+	+	+			
	Microtriches							+	+	+
	Measurements in mm	0.8—1.0 × 0.3—0.7	1.0—2.6 × 0.9—1.7	1.3—1.8 × 1.4—1.9	3.5—4.9 × 1.8—3.9	3.8—4.9 × 2.4—3.8	3.5—4.8 × 1.5—2.6	4.5—6.7 × 3.1—4.5		10.0—12.5 × 6.0—9.0
	Calcareous body							+	+	+

+ = presence

demonstrated a serious functional damage of liver caused by *C. tenuicollis* in goats. Even the residues after a massive infection, the healing of which is never complete, may induce a chronic disease decreasing the profitability of the breeding.

Many of the cysticerci die during the migration phase without reaching the serosas. The cysticerci which remained in the liver still at the time when those under the serosa were already morphologically differentiated are retarded in their development and most probably do not attain a complete morphological differentiation. This is supported also by the findings by other authors (Joest 1967, Sweatman and Plummer 1957). The underdeveloped cysticerci remain within the organism for a rather long time without being destructed. Underdeveloped cysticerci (measuring 1—9 mm) were found in the liver parenchyma of lambs and pigs still 9 months after infection (Sweatman and Plummer 1957). In the elk and reindeer, however, some cysticerci terminated their development even within the liver parenchyma (Sweatman and Plummer 1957), which may be significant for maintaining of *T. hydatigena* in the nature. It was found that in the frost the cysticerci on serosas die very rapidly, whereas those localized in the liver parenchyma remain infective for at least 2 days. From the general biological point of view it would be of interest to assess whether also in other animal species the cysticercus which has not penetrated up to the serosa of the organs may complete its development. It is possible, particularly in massive infections, that the development of the cysticercus may be completed later than it is generally supposed. Although the fibrous capsule of the pseudocyst in the liver spatially limits the cysticercus, we know that the wall of some pseudocysts around relatively developed cysticerci consists of several layers of fibrocytes without any further cellular infiltration indicating an active process at the death of the larva.

ПАТОЛОГИЯ ФАЗЫ МИГРАЦИИ У ЛИЧИНКИ ЦЕСТОДЫ *TAENIA HYDATIGENA* (PALLAS, 1766)

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Резюме. Патологическая картина фазы миграции *C. tenuicollis* от свиньи характеризована геморрагией в паренхиме печени и под поверхностью печени. Геморрагия, представляющая собой канал миграции, причинена разрушением лакун печени мигрирующими личинками. Около 10-го дня после заражения встречается серофиброзный перитонит и в эксудате появляются свободные цистицерки. На 14-й—16-й день после заражения эксудативный перитонит может усилиться. Цистицерки локализованы под серозами или на них. Около 10-го дня после заражения может появиться также легочная форма заболевания. На 13-й день после заражения цистицерки встречаются в просвете легочных артерий или мигрируют из них. Изменения в легких и на плевре и их динамическая перемена в основном те же самые как и в печени и на брюшине. Период от 21-го до 24-го дня характеризован обширными сращениями сероз и цистицерки крепко прикреплены к серозам. На 35-й день после заражения адгезии соединительной ткани переживают, многие из цистицерков проявляют дистрофные изменения или они мертвы и часто уже кальцифицированы. Стенка псевдоцисты, в которой находится цистицерк, состоит из фиброцитов и серозы и ее полность не выстлана эндотелием, как у *C. bovis* и *C. cellulosae.*

REFERENCES

BLAŽEK K., Pathological changes in the vessel system during fascioloidosis. Vet. med. 18: 25—38, 1973. (In Czech.)

—, GILKA F., Contribution to the knowledge of the pigment found in infection with *Fascioloides magna*. Folia parasit. (Praha) 17: 165—170, 1970.

BREZA M., Occurrence of *C. tenuicollis* and *C. bovis* in traditional and new technologies of breeding of domestic animals. Veterinářství 33: 121—122, 1983. (In Slovak.)

ENGELKIRK P. C., WILLIAMS J. F., *Taenia taeniaeformis* (Cestoda) in the rat: ultrastructure of the host-parasite interface on days 1 to 7 post-infection. J. Parasitol. 68: 620—633, 1982.

GRADINARSKI I., Patologoanatomični izmenenija pri tenuikolisnata cisticerkoza. Vet. sb. 80: 27—29, 1982.

HEATH D. D., The migration of oncospheres of *Taenia pisiformis, T. serialis* and *Echinococcus granulosus* within the intermediate host. Int. J. Parasitol. 1: 145—152. 1971.

—, SMYTH J. D., In vitro cultivation of *Echinococcus granulosus, Taenia hydatigena, T. ovis, T. pisiformis* and *T. serialis* oncosphere to cystic larva. Parasitology 61: 329—343, 1970.

HULÍNSKÁ D., Fine structure of cells and tissues of *Cysticercus tenuicollis* aged 13 and 16 days. Folia parasit. (Praha) 25: 339—345, 1978.

—, The distribution and activity of enzymes in larvae of *Taenia hydatigena* Pallas, 1766. Folia parasit. (Praha) 26: 315—324, 1979.

—, The fine structure of larvae of *T. hydatigena* aged seven and ten days. Folia parasit. (Praha) 27: 237—242, 1980a.

—, The morphogenesis of microtriches in the tegument of *Taenia hydatigena* throughout its larval development. Folia parasit. (Praha) 27: 329—336, 1980b.

JOEST E., Handbuch der speziellen pathologischen Anatomie der Haustiere. Verlag Paul Parey, Berlin, vol. 6, 744 pp., 1967.

JUBB K. V. F., KENNEDY P. C., Pathology of domestic animals. Academic Press, New York—London, vol. 2, 613 pp., 1963.

JURÁŠEK V., BREZA M., ANDRAŠKO H., BEŇUŠKA N., Finding of young forms of *Cysticercus tenuicollis* in lambs fed with milk (system of early weaning). Veterinářství 31: 114—117, 1981. (In Slovak.)

KUNTZ R. E., MYERS B. J., Primate cysticercosis: *Taenia hydatigena* in Kenya vervets (*Cercopithecus aethiops* Linnaeus, 1758) and Taiwan macaques (*Macaca cyclopis* Swinhoe, 1864). Primates 8: 83—88, 1967.

MIGAKI G., ZINTER D. E., Hepatic lesions caused by *Cysticercus tenuicollis* in sheep. J. Amer. Med. Assoc. 164: 618—619, 1974.

PATHAK K. M. L., GAUR S. N. S., Serum levels of GOT, GPT and OCT enzymes in goats infected with *Cysticercus tenuicollis*. Vet. Parasitol. 8: 95—97, 1981.

—, —, SHARMA S. N., The pathology of *Cysticercus tenuicollis* infection in goats. Vet. Parasitol. 11: 131—139, 1982.

SMITH H. A., JONES T. C., Veterinary Pathology. Lea and Febiger, Philadelphia, 1192 pp., 1963.

SWEATMAN G. K., PLUMMER P. J. C., The biology and pathology of the tapeworm *Taenia hydatigena* in domestic and wild hosts. Canad. J. Zool. 35: 93—109, 1957.

ŠLAIS J., VANĚK M., Tissue reaction to spherical and lobular hydatid cysts of *Echinococcus granulosus* (Batsch, 1786). Folia parasit. (Praha) 27: 135—143, 1980.

ŠTĚRBA J., ŠLAIS J., To the diagnostics of nodular structures in the liver. Plzeňský lékařský sborník 37: 75—78, 1972. (In Czech.)

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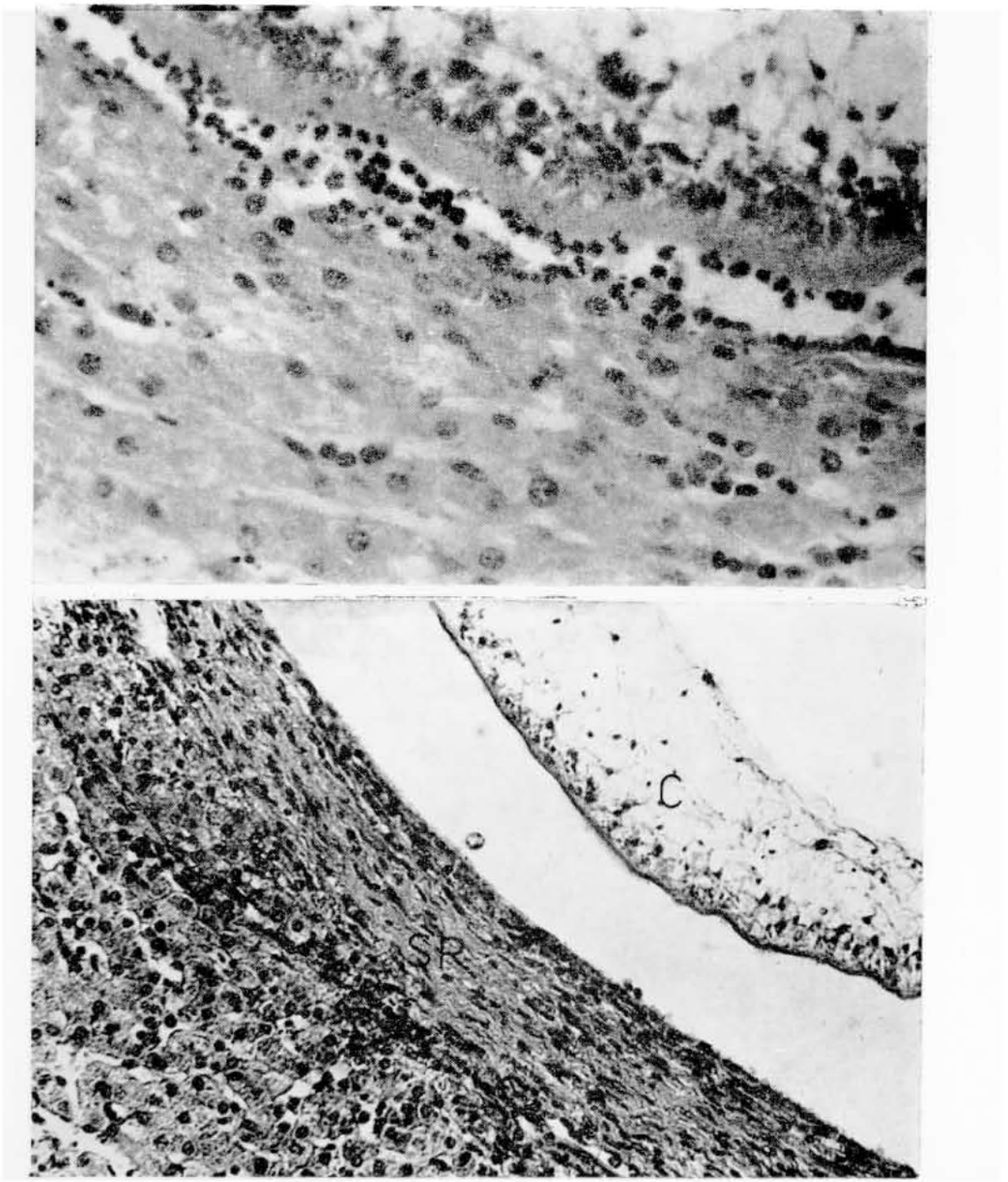
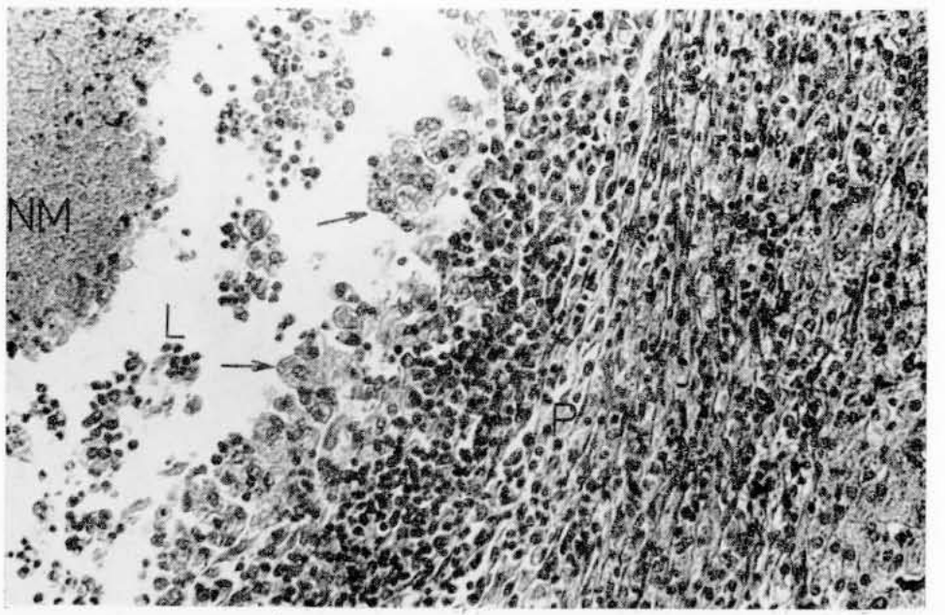


Fig. 1. At the beginning of the migration through the liver tissue there are only few leucocytes between the cysticercus surface and compressed liver cells. Liver, day 7 p.i. (HE, 360×).
Fig. 2. A pseudocyst is formed after the penetration of the cysticercus (C) under the serosa of liver. The pseudocyst wall consists of several layers of fibrocytes (SP). No pathological changes occur in the neighbouring liver tissue. Day 21 p.i. (HE, 180×).



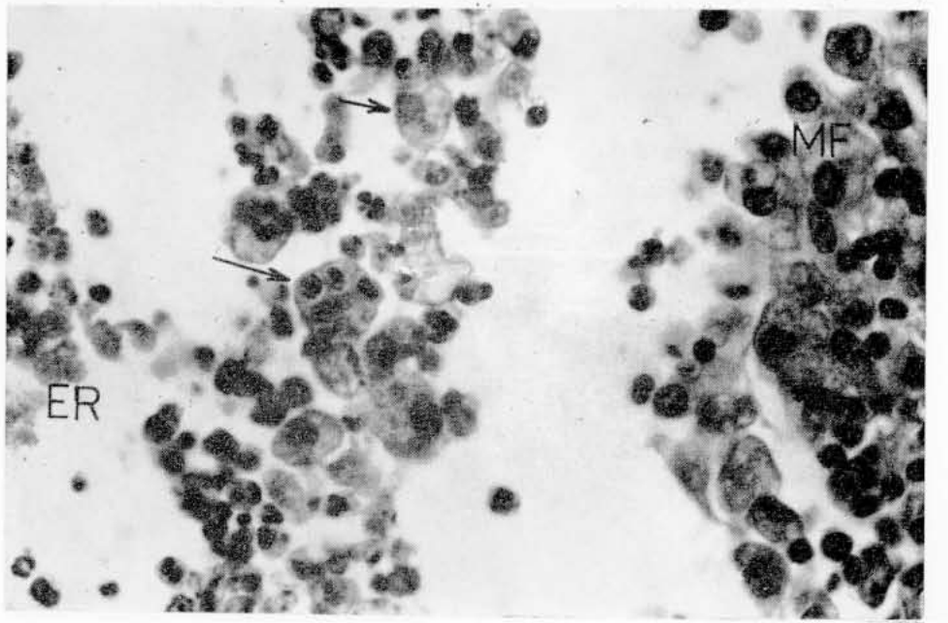


Fig. 1. The migration canal left by the cysticercus contains in the centre necrotic masses (NM), neutrophilic and eosinophilic leucocytes (L) and large macrophages migrating from the periphery (arrow). The connective tissue encapsulation at the periphery (P) is strongly infiltrated by lymphocytes and eosinophiles at some foci. (HE, 135×).
Fig. 2. Detail of the cavity of migration canal (Fig. 1) showing individual erythrocytes (ER), macrophages, the plasma of which contains phagocytized erythrocytes and leucocytes (arrows), and macrophages proliferating from the periphery (MF). (HE, 540×).

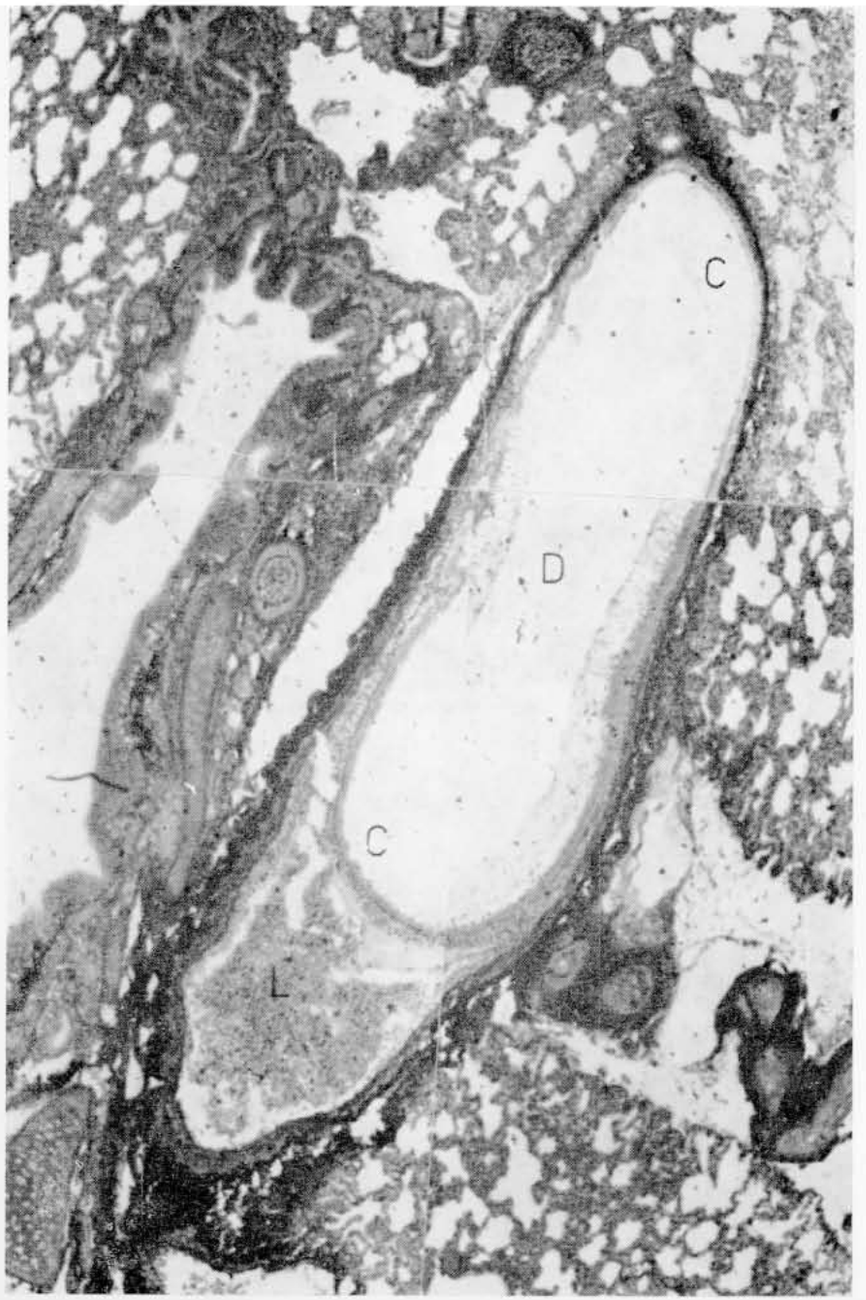


Fig. 1. In pulmonary cysticercosis, some cysticerci may be found in the lumen of vessels (L) even on day 13 p.i. The neighbouring lung tissue is not very changed. The cysticercus in the vessel C) has a well developed central cavity (D). (Van Gieson + elastica, 40×).

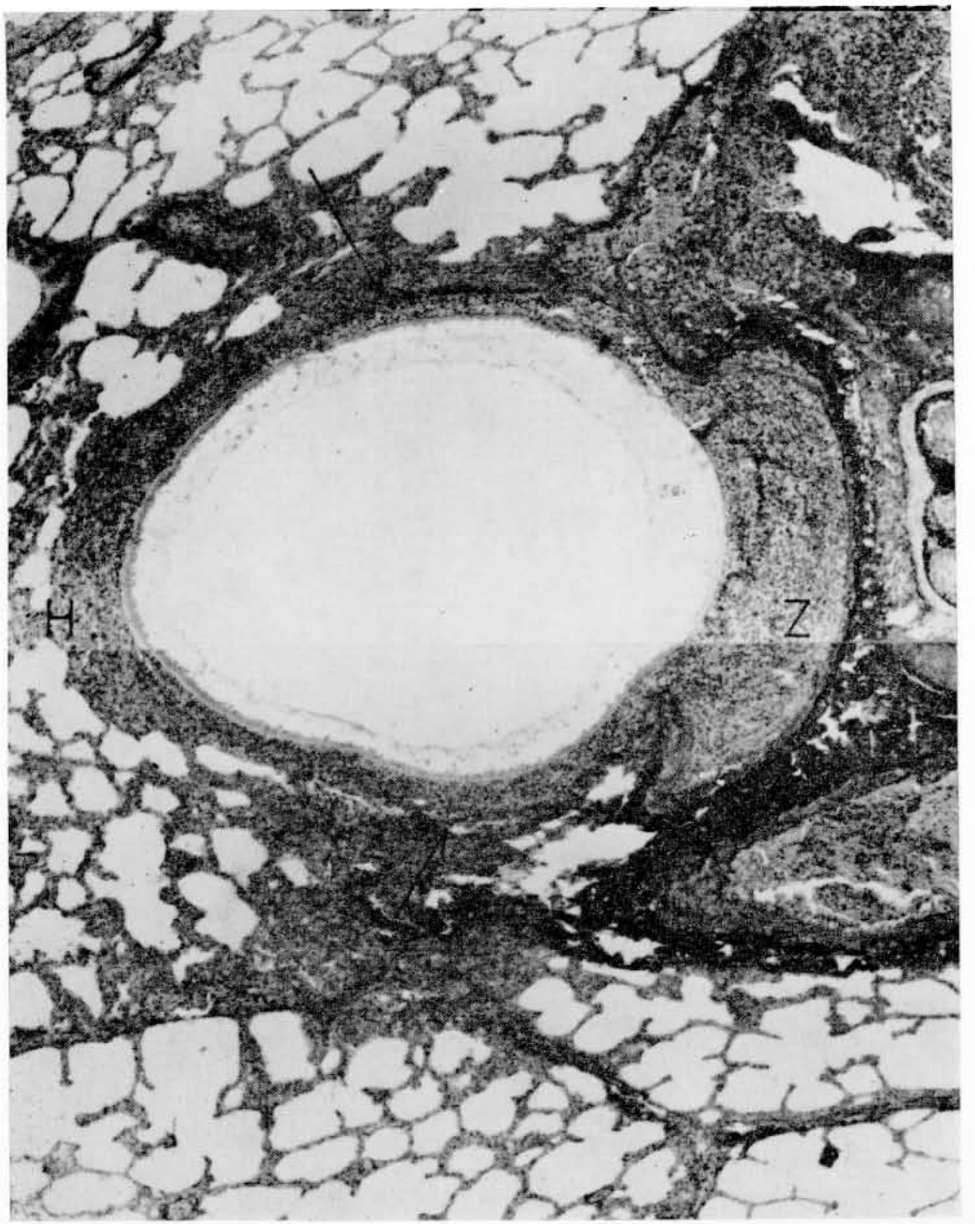
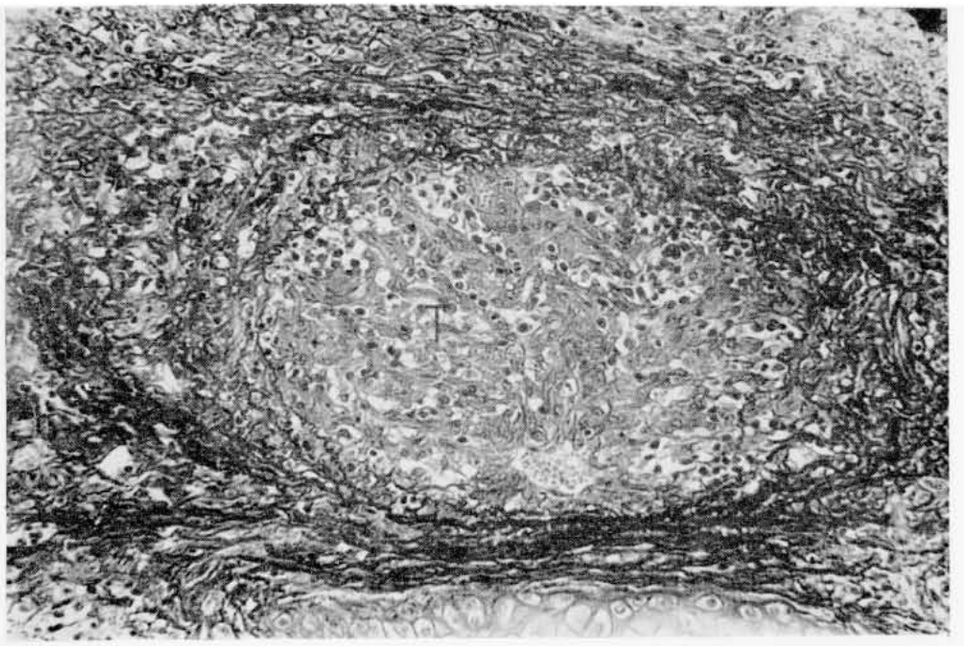


Fig. 1. At the time when the cysticercus leaves the vessel, there occur inflammatory changes in some parts of its wall (Z), destruction of the elastic layer (arrows) and haemorrhagia into lung tissue arrows). (Van Gieson + elastica, 50×).



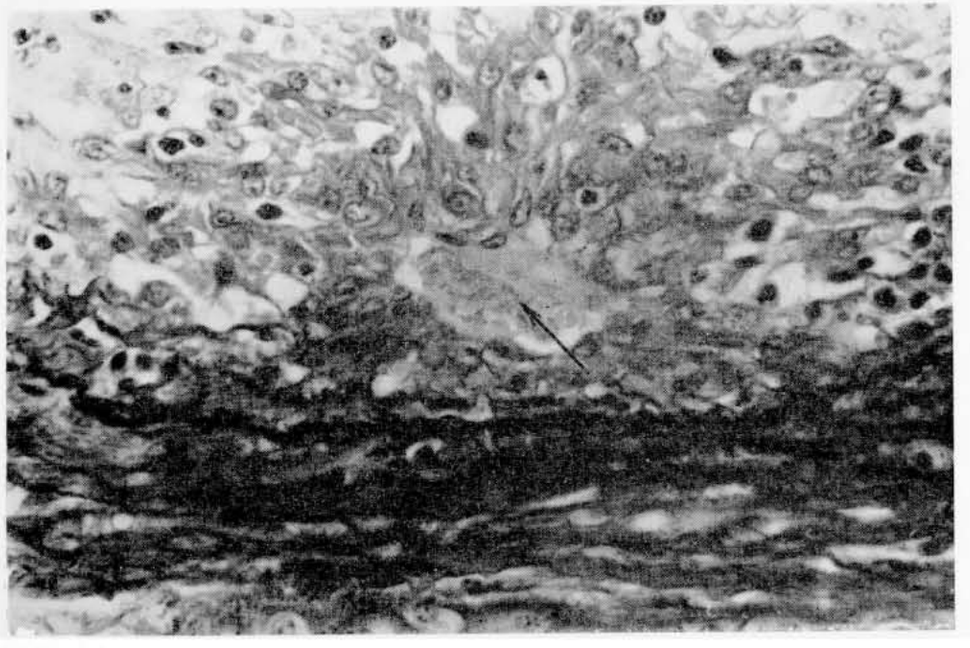
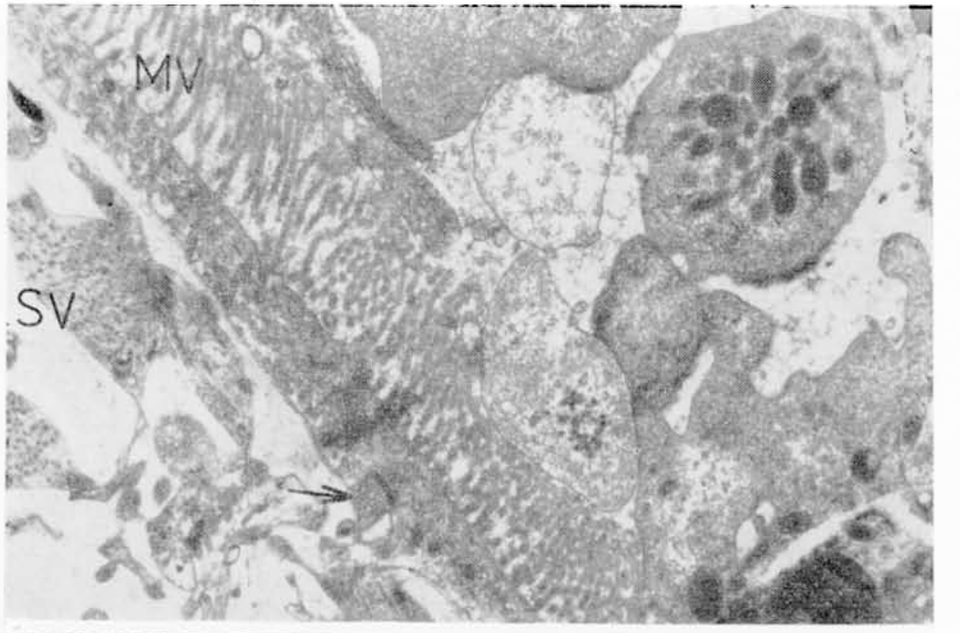


Fig. 1. On day 16 p.i. the lungs may contain arteries, the lumen of which is almost completely filled with a partly organized thrombus (T). (Van Gieson + elastica, 125×).
Fig. 2. Detail from Fig. 1 showing a minute residual lumen of vessel with erythrocytes (arrow) and the character of the proliferation. (Van Gieson + elastica, 385×).



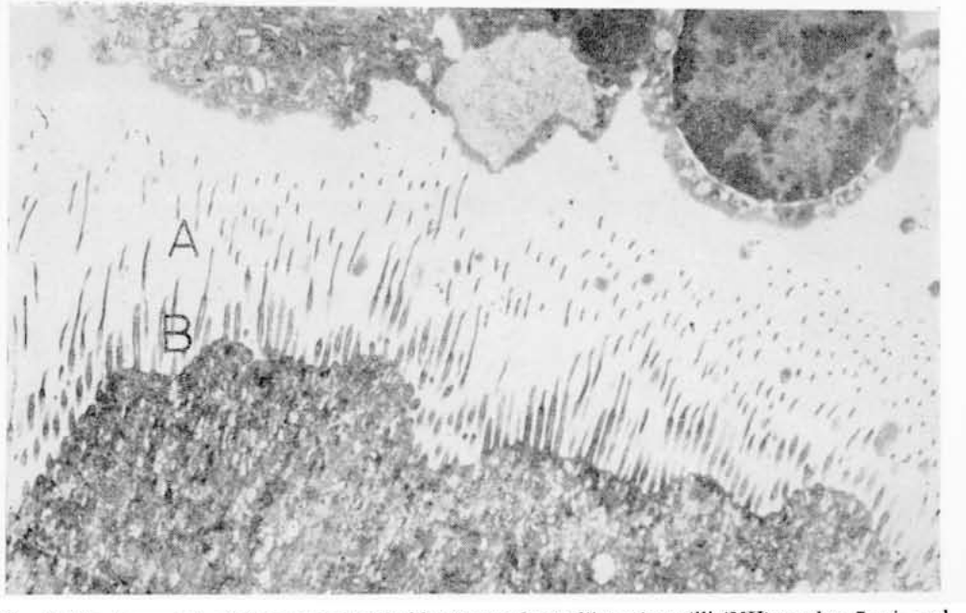


Fig. 1. The tegument of *C. tenuicollis* bladder forms finger-like microvilli (MV) on day 7 p.i. and pyriform nerve endings (arrow) with electron-dense plate are visible in the distal cytoplasm. Muscles (SV) are developed in the subtegumental tissue. (10,200×).
Fig. 2. The bladder tegument contains microtriches differentiated in a wider base (B) and pointed distal part (A) on day 21 p.i. Numerous vesicles and vacuoles are visible in the distal cytoplasm. (7,200×).