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. tenuecolis in pigs is characterized by a haemorrhagic within the liver parenchyma and under the liver surface. The haemorrhages, which represents a migration canal, is induced by the destruction of liver sinuses by migrating larvae. Approximately on day 10 p.i. a serofibrinous peritonitis occurs and free cisticerci appear in the exudate. On days 14—16 p.i. the exudative peritonitis may increase. The cisticerci are localized under the serosa or on them. On about day 10 p.i. even the pulmonary form of the disease may occur. On day 13 p.i. the cisticerci 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 synchiae of serosa and the cisticerci are firmly attached to the serosa. On day 33 p.i. the connective tissue adhesions persist and many of the cisticerci exhibit dystrophic changes or are dead and often already calcified. The wall of the pseudocyst, in which the cisticercus is situated, consists of the fibrocytes and serosa, and its cavity is not lined with endothelium, as it is in the case in C. bovis and C. cellulosae.

Cysticercus tenuecolis (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 (Sweetman 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 cisticercus occurs on the omentum and serosa 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 (Jobb and Kennedy 1963, Joest 1967, Gradinarski 1968).

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 (Jurašek 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 (Štárba and Slais 1972).

In comparative studies on the morphology of some cestode larvae the ultrastructure and histochemistry of C. tenuecolis 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 cisticerci 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 carcass of slaughtered sheep. The effect of the infection was monitored by repeated examinations of dog faeces. The pigs were infected with mature proglottides excreted by the dogs after an anthelmintic treatment. The infectivity of eggs contained in the proglottids was tested after Silverman.

Five pigs 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, and by numerous dark red proglottids variegates from 700 to 27,000 (Sweetman 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 13 and 24) and to test newly developed drugs.

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

The results for histological examinations (excised organs with cestodec and cysticerci alone) were fixed with 10% neutral formal 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 elastin, 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 1% osm. osmium tetroxide, postfixed with 1% solution of Oso4, dehydrated by an increasing acetone series and embedded into Vestopal. The sections were stained with uranyl acetate and lead citrate and examined through 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 (except 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 acetic 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 cestodec are usually encountered in the excretate 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 focus 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 lobes and on cardiac lobules.

On days 21–24 p.i. the serofibrinous exudate in the abdominal and thoracic cavities (if free cestodec is involved) is resorbed and connective tissue is formed, consisting of parenchymatous organs and intestinal loops to the walls of abdominal and thoracic cavities, as well as areal synchiae of organs, are mostly found. The liver is grown together with the diaphragm and surrounding organs through the connective tissue, and if the synchiae 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 synchiae of both leaves of the pericardium may be encountered. At this stage, the liver is usually enlarged and rugged. Numerous transparent bladders with swelling of the polar turbidity (cysticerci) appear between the liver parenchyma, 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 x 2 mm and cysticerci of about 2 x 5 mm are localized within the liver parenchyma. Solid, yellow, barrel-like foci containing a calcified cysticercus are sometimes encountered under the serosa. Cysticerci measuring about 3 x 5 mm, either single or in clusters, occur on the parietal leaf 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 synchiae resulting from the passed peritonitis (or pleuritis). Often the liver is not enlarged and numerous bladders measuring 5 mm in diameter up to 8 x 10 mm promote on its surface. Some of the bladders are muddy. In the section through the liver, thin-walled canals with milky, thin contents, yellow nodules or migrated are seen. The cysticerci are present on the surface, and serosa reach the size of a pea and they are firmly attached, transparent, and with a conspicuous acoel. 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. tenuecuniculi*

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 proportionally increased 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 leukocytes. The surrounding tissue is without a marked inflammatory cellulization and only sometimes the periporal 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—haemorrhagiae—fill almost the whole space of the liver lobule and often extent up to the neighboring lobule. Haemorrhagiae either with cysticerci or without them or cysticerci filling almost the whole space and surrounded 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 leukocytes are accumulated around the cysticercus and they are more numerous at the periphery of the haemorrhagiae 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 haemorrhagiae in the alveoli and bronchi, and the alveoli are filled with the final elements of the edema fluid. The interlobular septa of the infected areas are widened by the edema.

An almost identical histological picture of the liver is characteristic also for the period of days 13–16 p.i. The leucocytes (neutrophilic leukocytes) are accumulated in numerous migration canals and the inflammatory infiltration of periporal spaces is diffuse with the infiltration of the arteries and veins the central ones. The cysticerci are often completely surrounded by 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. On day 10 p.i. the bladders measure 1.0–2.5 × 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 viscera could also degenerate because of the growing number of scolecis. Usually at 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 × 1.5–3.9 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 rostellum conic 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 spatulate canal and the rostellar cone further differentiates into a bulb and prebulbar region. Sucker anlagen 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 10–12.0 mm long and 6.0–9.0 mm wide. The cysticerci from the pseudocysts on the omentum have a fully differentiated scolex with a well-developed calyceal canal with hooks and a well-developed rostellum differentiated suckers. The scolex parenchyma contains calcareous bodies. In the cysticerci, the scolex with rostellar cone is not yet completely differentiated; it is divided into bulb and prebulb, the tegument of which contains hooking hooks. The morphological characters of the larvae at individual development stages 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 microscopich 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 *T. tenuiscolitis*

The pathological changes arising during the migration of *T. hydatigena* larva in the liver are very conspicuous. These are the haemorrhages affecting one liver lobule or several adjacent ones. Their extent is much wider than that of haemorrhagic induced by nematode infection. The inflammatory infiltration of the liver tissue is minimal or completely lacking. The migration canals, which are essentially of the same 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 achieving sexual maturity (*Fascioloides magna*). The damage of the vessel system induced by the migration of trematodes (Blázek 1973) is similar to that occurring in pigs after the infection with *T. tenuiscolitis*. The migration of trematodes, however, is slower and therefore the haemorrhages 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 (Blázek and Gílka 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 the cercariae 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 haematoxylin-safranin. 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 subterminal segmentular cellular layer and with the central cavity. *C. tenuiscolitis* at the beginning of migration is always already differentiated, in contrast to larvae of other cercariae species, in which the central cavity is lacking mostly even 14 days after infection (e.g. *C. bovis*). The cercariae of *Echinococcus granulosus* at the early stage of development differs from *C. tenuiscolitis* in that it is smaller and it measures 0.2 mm, whereas *C. tenuiscolitis* has 5 mm in diameter at the same time (Nemeséri and Holó 1965 ex Jurášek et al. 1981). The young stages of trematodes may be easily distinguished from the cercariae 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 cercariae 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. tenuiscolitis* at the end of the migration phase from the pseudocyst of *E. granulosus*, since the hydatid pseudocyst bears a typical wall structure (Joy and East 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 subserous connective tissue cells which often turn to multinucleate cells. This epithelial rim is often necrotic (Slaia and Vanček 1980). In fertile cysts, the diagnosis is quite unambiguous if scolecities are found.

**DISCUSSION**

The pathological changes observed in our experimental animals in general conformed to the findings in this larval cestodes. 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 macroscopic 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 (Joy and East 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 postoncospheric recovery interaction. 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. tenuiscolitis* 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 movement. By 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. (Hulinska 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 Migas 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 than that larva capable of active migration got from the liver into lungs through the blood circulation and immediately migrated out from the lung arteries. The latter hypothesis is more probable after 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 postembryopetal 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 15–20 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 fibers 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 bigger 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 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 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.](image-url)
demonstrated a serious functional damage of liver caused by C. tenuissima 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 serosa. 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 of other authors (Joest 1967, Sweatman and Plummer 1957). The underdeveloped cysticerci remain within the organism for a rather long time without being destroyed. 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 cysticerci 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 cysticerci 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.

**REFERENCES**


ŠTĚRBA J., SLAIS J., To the diagnostics of nodular structures in the liver. Železniční 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, 300×).

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 eosinophils at some foci. (HE, 155×).

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 haemorrhage 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 x).

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 x).

Fig. 1. The tegument of C. tenella forms finger-like microvilli (MV) on day 7 p.i. and pyriform neurocytes (arrow) with electron-dense plate are visible in the distal cytoplasm. Muscles (SV) are developed in the subtegumental tissue. (10,000 x).

Fig. 2. The bladder tegument contains microvilli 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 x).