

## TISSUE REACTION TO CYSTICERCUS BOVIS IN THE LUNG OF ARTIFICIALLY INFECTED CATTLE

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**Abstract.** The tissue reaction to *Cysticercus bovis* in the lung of cattle with an experimental infection was an inflammatory rim originating in the immediate vicinity of the cysts. The cysts recovered at days 83 and 102 p.i. contained living cysticerci. The rim was composed either of a layer of high histiocytes organized in palisades (at day 83 p.i.), or a layer of flat histiocytes (at day 102 p.i.). The outer layer of the rim consisted of fibroblasts, reticular cells and a different number of eosinophil- and neutrophil leukocytes. On the periphery, the rim was formed by granulation tissue infiltrated with lymphoplasmacytes. At the border between the layers of the inflammatory rim there were conspicuous foci of a necrotic appearance typical of a tissue reaction to *C. bovis*.

Apart from typical sites of location of *C. bovis* in cattle, i.e., the skeletal muscles and the heart, cysticerci were found in the lung. Various authors reported an occasional finding of cysticerci in this organ (Graber et al. -ex Leinati et al. 1962). Out of a total of 269 animals examined, Sgambati (1959) found an occasional cysticercus in the lung of two of these animals. Similar results were obtained by Leinati et al. (1962), Koudela (1965), Romboli et al. (1959 b), Mango and Mango (1972), and recently by Kozakiewicz (1977, in press). These authors made their studies both on animals with spontaneous and artificial infection. However, none of these gave a description of the histopathology of cysticercosis of the lung except for Romboli et al. (1965 a) in a short communication without figures. The authors distinguished three stages of a tissue reaction to *C. bovis* in the lung (infection, migration, location).

Differences in the intensity of tissue reactions in the individual organs (liver, heart, skeletal muscles) have been pointed out in earlier papers (Štěrba 1978, Štěrba and Dyková 1978 a, b, Štěrba et al. 1979). In the present study, these have also been found in the lung. In an attempt to obtain a better understanding of these differences, we have evaluated the tissue reaction in relation to the location, morphology and morphogenesis of the cysticercus.

### MATERIALS AND METHODS

We obtained 4 cysts from calves infected experimentally with *C. bovis*: one at day 83 p.i., 3 at day 102 p.i. A description of the experiment has been given in an earlier paper (Štěrba 1974). Although we examined most carefully the lungs of animals spontaneously infected with *C. bovis*, we never found a cysticercus in this organ.

For histological tests, the material was fixed with 10 % formal. A major part of the material was treated with standard paraffin techniques, the rest with the freeze technique. A complete series of histological sections was made with methods suggested by Šlais (1960, 1970), and modifications of these methods (Štěrba and Šlais 1972, 1974). Sections were stained with haematoxylin-eosin, van Gieson's elastic method, Masson and Goldner's trichrome, Gomori's impregnation method. Kossa's method was used for the detection of calcium. Of histochemical methods employed, several were essentially similar to those described in earlier papers (Štěrba 1974, 1978).

## RESULTS

The location of cysts was shown to be subpleural in the lung of experimentally infected calves (Plate I, Fig. 1). The pleura displayed no macroscopic signs of a fibrinous pleuritis. All cysts contained living cysticerci (Plate I, Fig. 2). In three instances, the parenchymal portion of the bladder worm was partly enclosed in the bladder, in one case it was almost completely enclosed in it.

Dilated, lymphatic, spaces were always the site of location of the parasite. A local inflammation originated close to the parenchymal portion of the parasite. Endothelium was absent in part of the lymphatic vessel affected by inflammatory changes, but was normally present outside this area (Plate II, Figs. 1 A, B).

The parasite inside the cyst from day 83 p.i. was surrounded by a fine, granular, substance, which was proteinaceous in nature and contained an occasional eosinophil leukocyte. The inflammatory rim consisted of a layer of high histiocytes organized in palisades, an outside, narrow, layer made up of fibroblasts and reticular cells infiltrated by numerous eosinophils and a few neutrophils (Plate II Fig. 2), and a peripheral layer composed of granulation tissue with infiltrating lymphoplasmocytes.

The parasites inside cysts recovered at day 102 p.i. were embedded in a homogeneous substance, which also was proteinaceous in nature but contained neither eosinophil leukocytes nor other infiltrating cells. Again, the inflammatory rim was confined to the immediate vicinity of the parenchymal portion of the parasite. It consisted of a very narrow layer of flat histiocytes passing into an outer, narrow layer of fibroblasts and reticulate cells containing an occasional eosinophil leukocyte. The periphery of the inflammatory rim was made up of a thin layer of mature connective tissue with an occasional nodular accretion of lymphoid tissue, (Plate III, Fig. 1).

Between the individual layers of the inflammatory rim, we observed conspicuous, necrotically appearing foci in the lung (Plate III, Fig. 2, Plate IV, Figs. 1, 2) which were similar to those found earlier in the skeletal muscles and the heart. These foci seemed to be typical of a tissue reaction to *C. bovis* (Štěrba and Dyková 1978 b, Štěrba et al. 1979), particularly as these have never been found in tissue reactions to other larval cestodes or other parasites. The lung tissue abutting the cyst was atelectatic, and once we found in it minute foci of a postpneumonic carnification. No changes were observed in lymphatic vessels in the neighbourhood.

## DISCUSSION

A comparison of tissue reactions showed, that even as short a period as 19 days influenced remarkably the character of the pathological process.

We did neither observed signs of infection nor haemorrhages, necrosis, diffuse edema, eosinophil alveolitis and interstitial pneumonia of a leukolymphocytic nature which, according to Romboli et al. (1965 a) are caused by the migration of the parasite.

Changes observed in the lung at days 83 and 102 p.i. are conform to changes during the so-called stage of the location of the parasite described by Romboli et al. (1965 a) in their short communication. These authors maintained that the extent of the initial, heavy, inflammatory, reaction decreased as the cysticercus developed until it became confined to the immediate vicinity of the cyst. The cavity containing the parasite was separated from the remaining parenchyma by a thick capsule consisting of an inner layer made up by densely arranged histiocytes resembling a mesothelial layer (we believe that it was the endothelial lining of the dilated capillary), and of an outer layer composed of fibroblasts. The surrounding tissue contained always sparsely distributed leuko- and lymphocytes. In the description of this stage, the authors emphasized

vesicular changes which we did not observed in our material. However, it might well be that the parasites described by them were obtained from deeper parts of the parenchyma.

Also we observed a reduction in the extent of the tissue reaction in material from the skeletal muscles and the heart of animals spontaneously infected with *C. bovis*. However, this applied only to cysts containing a live cysticercus, and lasted for the period of development of the cysticercus. As soon as this was complete, there followed a characteristic development of inflammatory changes at the site facing the opening of the spiral canal onto the surface of the parenchymal portion of the cysticercus as this was observed with cysts recovered at days 83 and 102 p.i.

Having regard to the limited number of cysts examined, we are unable to explain the finding of live cysticerci in the cysts, while other authors (Sgambati 1959, Leinati et al. 1962, Kozakiewicz 1977, in press), reported to have found dead ones only. Features characteristic of a tissue reaction to dead cysticerci are an exudation, resorption and a gradual cicatrization. According to Kozakiewicz (1977), the number of dead cysticerci recovered from the heart and the lung was always higher than that of dead cysticerci recovered from the skeletal muscles.

The death of a cysticercus, irrespective of its location, might be caused by a number of factors (allergy, local disturbance of the circulatory system, state of the lymphatic vessel, disposition of the infected animal, infective dose, etc.). In order to disclose the factor responsible for the death of the cysticercus, detailed morphological studies will have to be made on a more extensive material together with a complex evaluation of all factors influencing the location of the cysticercus, and the origin and development of the tissue reaction.

The pathological character both of changes in the parenchyma of the lung and in the vicinity of the cyst appears to be too general as to be regarded as typical of a lung cysticercosis. On the basis of the material examined so far, we are unable to give an opinion as regards their etiology. However, an important piece of information appears to be our first confirmation of the location of the cysticercus in the lymphatic capillaries of the lung.

## ТКАНЕВАЯ РЕАКЦИЯ В ЛЕГКИХ СКОТА ПРИ ЭКСПЕРИМЕНТАЛЬНОМ ЗАРАЖЕНИИ ЦИСТИЦЕРКОМ *CYSTICERCUS BOVIS*

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**Резюме.** Изучали тканевую реакцию в легких теленков, экспериментально зараженных цистицерками *Cysticercus bovis*. На 83-й и 102-й день после заражения все цисты содержали живые цистицерки и в их окрестности образовалась характерная воспалительная реакция. Воспалительная кайма состояла или из слоя высоких, расположенных в форме палисада гистиоцитов (на 83-й день после заражения) или из слоя плоских гистиоцитов (на 102-й день после заражения). Наружный слой состоял из фибробластов, ретикулярных клеток и разного количества нейтрофильных и эозинофильных лейкоцитов. Периферический слой каймы состоял из грануляционной ткани с инфильтрацией лимфоплазмоцитов. На границе между отдельными слоями воспалительной каймы обнаружены резкие очаги некротического вида, типичные для тканевой реакции на *C. bovis*.

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Received 17 October 1978.

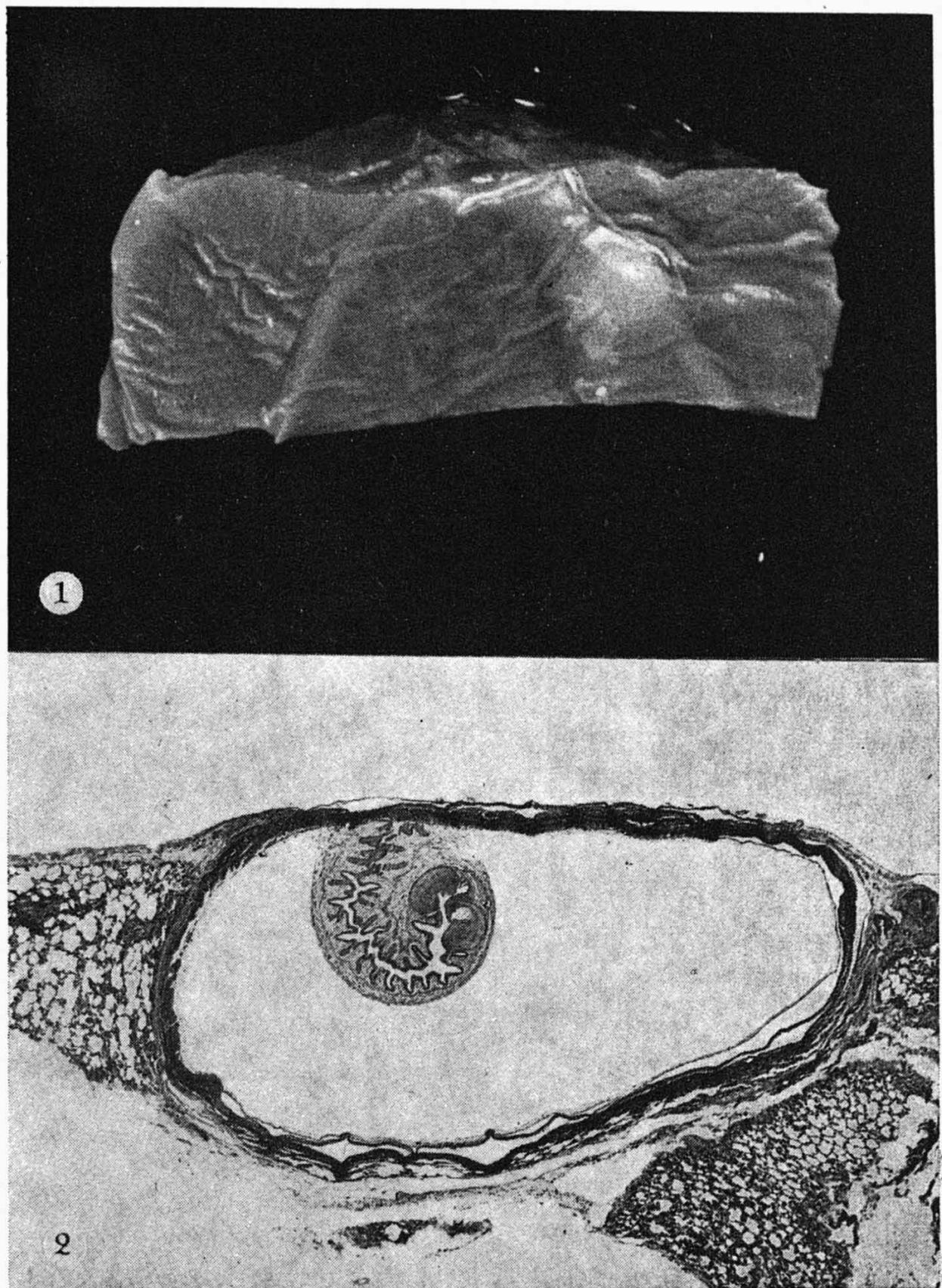


Fig. 1. Subpleural location of *C. bovis* in the lung of a calf with an artificial infection ( $\times 9$ ).

Fig. 2. Subpleural location of *C. bovis* in the lung of a calf with an artificial infection. Haematoxylin-eosin ( $\times 24$ ).

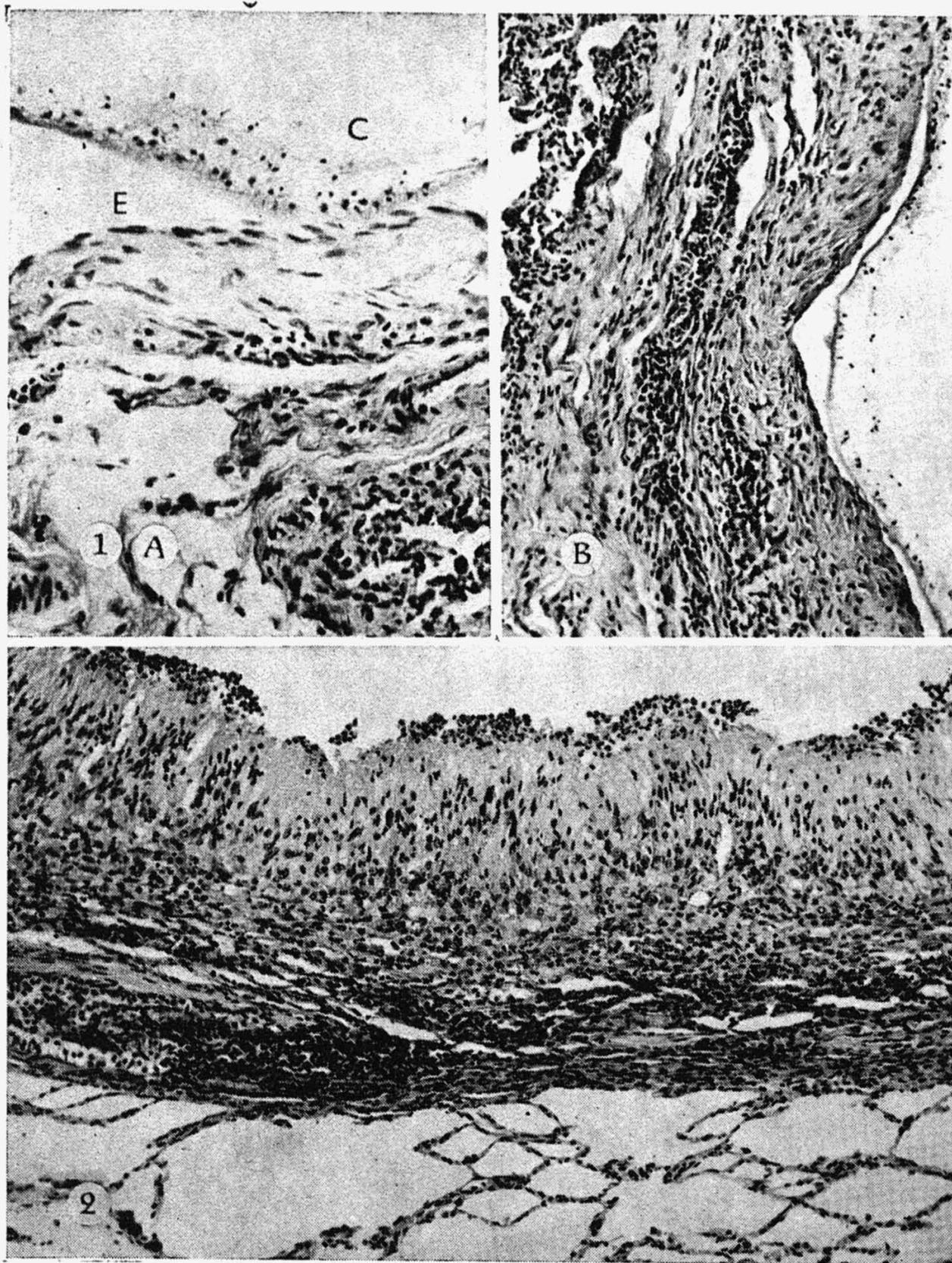


Fig. 1A. Endothelium preserved in a lymphatic capillary (E) containing a *C. bovis* (C) ( $\times 230$ ).

Fig. 1A. Inflammatory rim in the vicinity of a *C. bovis*. Haematoxylin-eosin ( $\times 150$ ).

Fig. 2. Inflammatory rim consisting of palisade-like organized histiocytes. Haematoxylin-eosin ( $\times 130$ ).

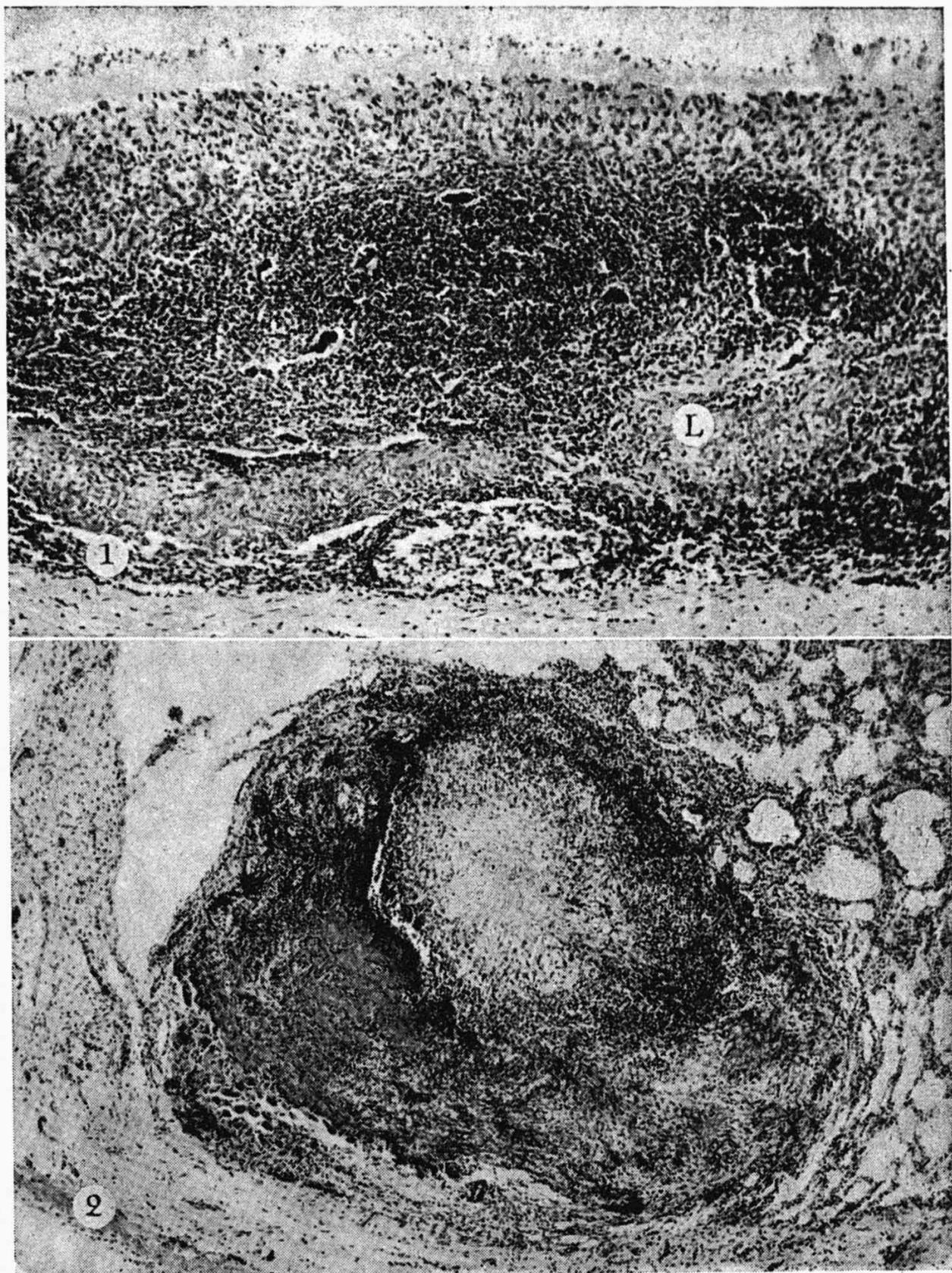


Fig. 1. Nodular accretion of lymphoid cells in the inflammatory rim. Focus of necrotic appearance (L). Haematoxylin-eosin ( $\times 130$ ).

Fig. 2. Inflammatory rim with foci of necrotic-like appearance. Haematoxylin — eosin ( $\times 65$ ).

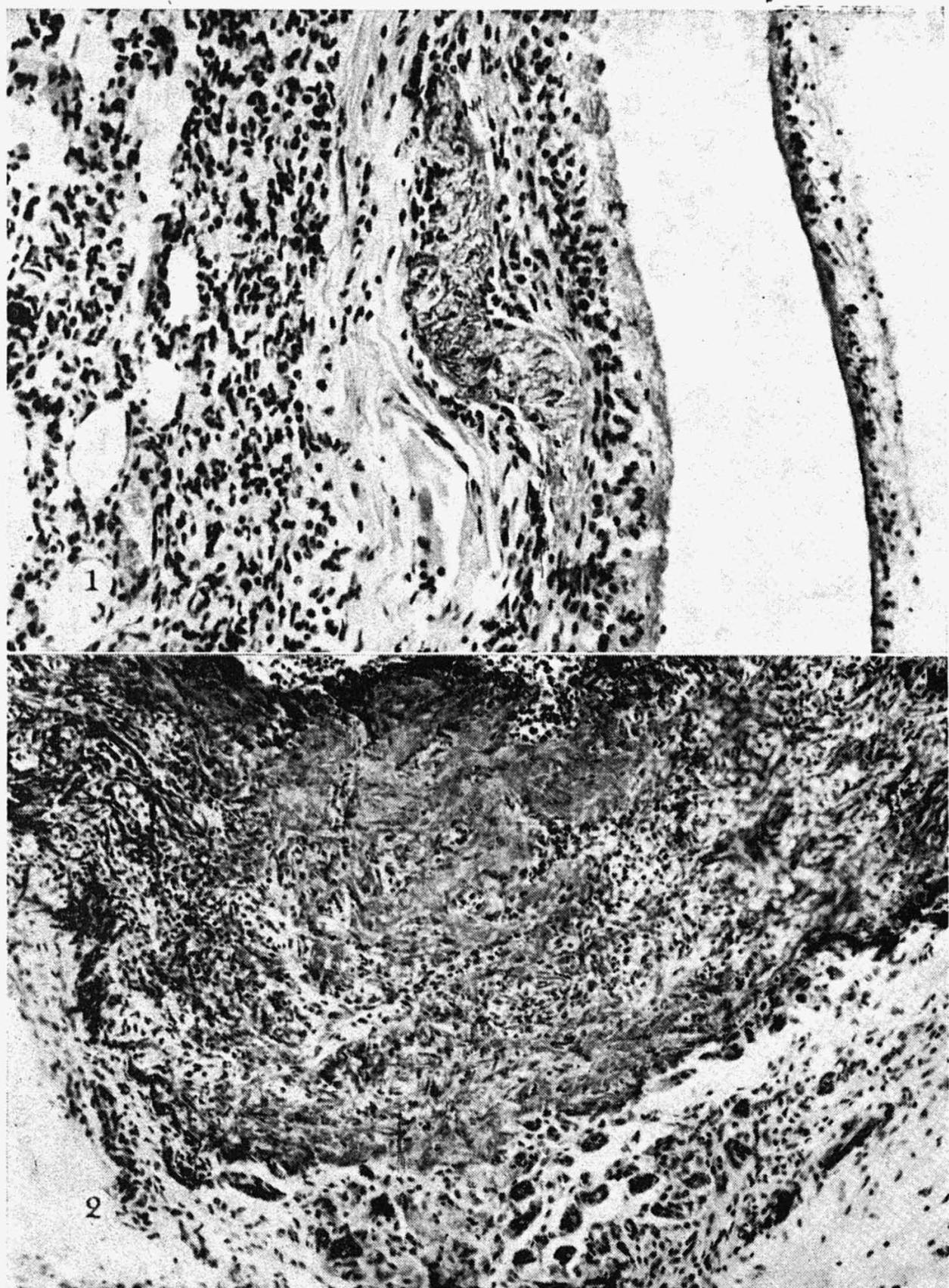


Fig. 1. Inflammatory rim surrounding the parenchymal portion of a *C. bovis*, with a focus of necrotic appearance. Haematoxylin-eosin ( $\times 250$ ).

Fig. 2. Hyaline focus necrotic-like in appearance. Haematoxylin-eosin ( $\times 160$ ).