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HAEMOSPORIDIOSIS AS A FATAL DISEASE IN MUSCOVY DUCKS (CAIRINA MOSCHATA) IN SOUTH BOHEMIA

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Abstract. A description is given of the disease afflicting muscovy ducklings (*Cairina moschata*) which repeatedly occurred in South Bohemia during the last decade. The symptoms and causative agent seem to be similar to the earlier described cases of illness in muscovy ducks reported from the Rhein region (Commichau and Jonas 1977). However, the disease was not caused by *Leucocytozoon simondi*, as the German authors erroneously supposed, because the schizogonic stages from the internal organs of the infected ducks considerably differ from the developmental stages of *L. simondi*. The causative agent is probably some species of haemosporidia acquired from the local populations of wild birds, a species able to attain partial development in the internal organs of muscovy ducklings. In this aberrant host, however, only schizogonic division takes place, the parasite is unable to form the blood stages and consequently complete its life cycle. As the morphology of blood stages is unknown, the causative agent cannot be identified. In the morphology of schizogonic stages the causative agent resembles the parasites of the genus *Haemoproteus* or *Plasmodium*, but it is less similar to *Leucocytozoon*. It is evidently not directly the species *Haemoproteus nettionis* which seems to have caused a similar disease in muscovy ducks in Canada (Julian and Galt 1980), because this species is missing in Central Europe.

In 1972—1979 an outbreak of a disease occurred in muscovy ducks raised in several localities in the South Bohemian region which showed high morbidity and mortality mainly in ducklings. The examination of dead and ill birds revealed that this disease was probably similar to that described by Commichau and Jonas (1977) in muscovy ducks in the Federal Republic of Germany. The mentioned authors considered *Leucocytozoon simondi* to be the causative agent of the disease in question. At the beginning we also thought it to be an infection with this parasite, but at closer comparison it became evident that the infection was caused by a haemosporidial agent which considerably differs morphologically from *L. simondi*. Julian and Galt (1980) described cases of a very similar disease in muscovy ducks from Canada and indicated *Haemoproteus nettionis* as the agent. Owing to the fact that this species does not occur in anatids in Central Europe (Kučera 1981 a, c) it was very likely another species of blood parasite. The present paper therefore describes the parasite found and discusses which species of blood protozoan it might be.

MATERIAL AND METHODS

Cases of an infection in muscovy ducks (*Cairina moschata* L. forma *domestica*) were reported by individual breeders from the following localities in the South Bohemia region:

1. Locality in the district of Blatná. The infection occurred in July and August 1972. A total of 24 ducklings, produced by two ducks, died. The first duck used to lead her young to the local pond since their hatching and at age 3 weeks all 11 ducklings died. The second duck which had produced 17 ducklings was kept away from the pond, but in its immediate vicinity and a few ducklings occasionally escaped to the pond. From this brood 13 ducklings died at age 4 weeks and the remaining four ducklings survived. In mid-September the first duck produced nine ducklings which were prevented from going to the pond at all and no one of them became infected.

2. Locality of Třeboň. The infection occurred in July 1973 among the ducks of a breeder and a few ducklings died at age 4—5 weeks.

3. Locality in the district of Prachatice. The infection occurred repeatedly in the summer of 1977 and in August 1978. The owner kept the ducks free on the water since hatching. In the former year a few ducklings died, in the latter year 12 out of 17 ducklings died at age 14 days.

4. Locality in the district of České Budějovice. In August 1980 about 15 ducklings aged 2—6 weeks died. From this locality the infection was already reported on 17 December 1979 when one young, not yet one-year-old duck died.

5. Locality in the district of Tábor. In October 1978 one two-month-old duck died.

While seeking in retrospect the data referring to the above mentioned cases we failed to find any more details.

Practically in all cases the ducks were kept in small flocks, could move freely since hatching and were allowed access to ponds or streams around the breeder's house. In some cases the ill ducklings were treated with Sulfadimidin and Furazolidon. Some of the ill ducklings from the České Budějovice locality were hospitalized at the State Veterinary Institute where they were treated with Stenorol (premix of Halofuginon) in a dose of 0.5 g/kg of feed.

The ill or dead birds were histologically investigated and some of them were tested microbiologically. However, only histological investigations yielded positive results. If possible, also blood smears and bone marrow impressions were made at autopsy; after fixation in methylalcohol the impressions were stained with Giemsa. The lungs, liver, gut, spleen, brain, heart, kidneys and samples of skeletal muscle of the dissected birds were fixed in 10 % formol and the histological preparations were stained with hematoxylin-eosin. The preparations were examined under microscope by means of oil immersion objective (100-fold magnification). A few healthy adult ducks from contaminated localities were also examined by means of blood smears. These birds, however, were found to be negative.

Histological preparations with hepatic schizonts and megaloschizonts of *Leucocytozoon simondi* were loaned as comparative material from the collections of the Department of parasitology, Natural Sciences Faculty at the Charles University in Prague by courtesy of Doc. RNDr. Ž. Černá. The histological preparations with megaloschizonts of parrot aberrant leucocytozoonosis were obtained by courtesy of M. Lávička, M.V.D., of the Central State Veterinary Institute in Prague.

RESULTS

In the infected muscovy ducklings torpidity, even lethargy and reluctance to movement were noted. The birds breathed with difficulty (had laboured breathing) especially when they were forced to move and refused to drink and feed. The symptoms lasted about a week and the majority of the infected ducklings died during this period. The surviving ducks were in a very bad condition and were lagging behind their development for a long time. The drugs administered to some ducks (see Material and Methods) had no essential effect on the course of the illness. The drugs, however, were administered only after the appearance of clinical symptoms, when the pathological changes in the organs of the ill ducklings were more or less of irreversible character, so that the treatment was of no avail.

The dead and ill birds were dissected and most organs revealed important pathological changes. The congested epicardium was with numerous petechiae and in some cases hydropericardium was observed. The lungs were heavily filled with blood, with oedematous infiltrates and firm to touch. The liver was enlarged and congested, sometimes with petechiae under the liver sheath. The kidneys were pallid, anemic, only locally congested and penetrated by petechial haemorrhages. Sometimes, evidently due to the thirst of ducklings, the kidneys were swollen, enlarged, containing stagnant urates in the distended renal tubules. Likewise the spleen was sometimes enlarged, with dilated blood vessels under the capsule. In some cases congestion in the mesentery and serosity of gut were observed.

In all cases the histological sections of organs showed, under microscope, schizogonic formations located partly in the endothelial cells of capillaries, blood vessels and sinusoids and partly in the closely undetermined macrophages. As a rule, masses of schizonts were found in the lungs, deposited in blood extravasates and in the mixed inflammatory infiltrate with a marked admixture of eosinophils. Likewise the spleen, and propria mucosae of intestines were infected, showing identical inflammatory infiltration. The

schizonts were less frequently found in the endothelium of the blood vessels of brain, kidneys, heart and the skeletal muscle as well as sinusoids. In addition, dystrophic even necrotizing focal changes and mixed inflammatory infiltrates in portobiliar fields with marked admixture of eosinophils were noted in the liver parenchyma. The schizogonic formations in less changed tissue parts currently occurred in the endothelial cells along the blood vessels. The schizonts in the inflammatory infiltrates were detected in undetermined macrophages. The size of the host cell was somewhat enlarged, the nucleus however did not, as a rule, differ from the nuclei of uninfected cells.

The schizogonic stages in the endothelial cells of blood vessels and capillaries were lining the vessel (Plate I, Figs. 1, 3, Plate II, Fig. 9) and frequently filled up the entire lumen of the capillary, this being particularly visible on cross sections (Plate I, Fig. 4, Plate II, Fig. 10). The length of these formations ranged from 20 to 40 μm and the width varied between 3.7 and 8.1 μm depending on the thickness of the capillary. When more cells in a row were infected, the length of schizonts seemed to be much greater (Plate II, Fig. 9). Schizogonic formations of the second type in macrophages (Plate I, Figs. 2, 5, 8, Plate II, Figs. 11, 13) were 11.1×7.3 μm large on the average, the smallest measuring, 4.7×3.9 μm and the biggest 24.7×9.5 μm. In the clusters of infected cells the separate schizonts were difficult to distinguish, so that the schizogonic formations seemed to have even larger dimensions (Plate I, Fig. 8, Plate II, Fig. 13). In histological preparations the merozoites inside the schizonts appeared to be spherical, oval or even crescent-like corpuscles of dark colour, frequently with a lighter ring around. Sometimes they were very minute, measuring 0.5×0.5 μm, the biggest reaching the size up to 1.9× ×1.4 μm. Judging from the size of the merozoites two types of schizogonic stages seemed to be present here: schizonts with tiny micromerozoites and schizonts with big macromerozoites. However, there were also various transient forms of merozoites and their average size was 1.4×1 μm. The schizogonous formations were also found on the bone marrow impressions stained by Giemsa. Likewise, they occurred here in macrophages and the merozoites were almost always of ring-like form with dark red nuclei and a band of pink-blue cytoplasm around a small vacuole (Plate I, Figs. 6, 7, Plate II, Fig. 12). The dimensions of these schizonts as well as merozoites agreed with the formations observed in histological preparations.

Merozoites were far less frequently found in the blood smears from the infected birds. They occurred either in the regressively changed or partly desintegrated cells of the leucocyte type and their morphology was of the same ring-like type as that observed in the bone marrow impressions, or they occurred freely in the blood plasma. These free merozoites were most frequently of oval shape, measuring 0.5×1 μm on the average, with a red-stained nucleus and a thin band of blue cytoplasm. Most frequently they occurred in clusters (Plate II, Fig. 14). Apart from these merozoites no other developmental stages of haemosporidia were present in the peripheral blood of the ill or dead birds.

DISCUSSION

The disease observed in muscovy ducks, which was caused by the above described parasite, may be designated as haemosporidiosis. It is evidently haemosporidiosis despite the fact that we have not found the blood stages of these parasites. The schizonts found in the internal organs of the infected ducks are similar to typical developmental stages of a number of haemosporidia (see e.g. Garnham 1966) not only in their morphology, but in the localization in the endothelial cells and in the macrophages and in the distinct spread in the blood stream to all internal organs. The infected ducklings were given access to free nature since their hatching and apparently became infected

there by the blood-sucking insects. The disease occurred most frequently in June to August, namely in the period when an intensive transmission of avian haemosporidia by insect vectors takes place (Kučera 1981b, c). The sporadic case reported from December 1979 may be regarded as a relapse of the previously acquired infection or explained by the fact that November and December of that year were extraordinary warm, so that the transmission by blood-sucking insects cannot be ruled out even in this period.

The incidence of the disease is supposed to be far higher than that revealed by the number of cases related by us. The muscovy ducks are raised in South Bohemia exclusively by individual breeders and their flocks are not under regular veterinary control. Only a few breeders submitted their dead or ill birds to veterinary examination, so that many cases went unnoticed. The first finding of the parasites discussed was made by one of the authors (K. M.) shortly after her arrival at her working place in České Budějovice in 1972 and it may be surmised that the disease in question probably occurred in South Bohemia much earlier.

Practically identical infection in muscovy ducks with similar symptoms and morphologically very similar causative agent was described previously by Commichau and Jonas (1977) from the Rheinland in the Federal Republic of Germany. Although these authors did not detect gametocytes in the blood of the surviving ducks either, they determined the causative agent only on the basis of schizogonic stages in the internal organs as *Leucocytozoon simondi* Mathis et Leger, 1910. This blood parasite of the suborder Haemosporina causes an infection with similar symptoms in anatids and is considerably pathogenic primarily for domesticated species. *L. simondi* causes high losses primarily in North America (e.g. Herman 1963 etc.), but also occurs in Europe, though it is quite rare in Anseriformes in Central Europe and seems to be rather a northern species (Kučera 1981a, b, Herman 1968). The comparison between the morphology of schizonts observed in the internal organs of muscovy ducks and the morphology of internal stages of *L. simondi* shows that the parasites found in muscovy ducks cannot be considered as developmental stages of *L. simondi*. While making this comparison we not only used published data on the development of *L. simondi* (Garnham 1966, Fallis and Eide 1970, Eide and Fallis 1972, Desser 1973, Fallis and Desser 1974, 1977, Desser and Ryckman 1976, Herman et al. 1977 etc.), but also the comparative material of hepatic schizonts and megaloschizonts of *L. simondi* in histological preparations (see Material and Methods). In their size the hepatic schizonts of *L. simondi* resemble the stages found by us in internal organs of the muscovy ducks. However, in the *L. simondi* infection these stages are encountered only in parenchymous cells of liver, while the schizonts observed by us in muscovy ducks occurred in macrophages and endothelial cells of all organs examined and never in the parenchymous cells of liver. Likewise megaloschizonts, which are so typical of the infection caused by *L. simondi*, were never found in our material. Although Commichau and Jonas (1977) consider some stages from internal organs of muscovy ducks to be megaloschizonts, they did not give their description in detail. The photographs published by them, however, show that the stages are probably identical with the stages found by us and are not the true megaloschizonts. After Fallis and Desser (1974) the megaloschizonts of *L. simondi* are characterized primarily by the fact that: 1. they originate from the hepatic schizonts and develop in reticuloendothelial cells primarily of the vascular endothelium, 2. they cause extreme hypertrophy of the host cell nucleus and 3. grow to considerable size (100 or more than 400 μm), producing over one million of merozoites. The schizonts observed by us in the organs of muscovy ducks were also present in the reticuloendothelial cells (endothelium, macrophages), but they never caused hypertrophy of the host cell nucleus; their average size was 11.1×7.3 μm and the maximum length of elongated forms

was 40 μm. They also contained only a few scores of merozoites and never had the typical habitus of megaloschizonts, as could be verified directly from the comparative material. Commichau and Jonas (1977) probably confused the two organisms due to the fact that the schizogonic stages in the heavily infested organs of muscovy ducks are sometimes so closely packed one on another that they appear to be bigger than the individual schizonts (see e.g. Plate I, Fig. 8 and Plate II, Fig. 13).

Another proof that the infection in question was not caused by *L. simondi* is the fact, that no gametocytes typical of this species were ever encountered in the blood of the ill ducks by us or by Commichau and Jonas (1977). It is true that haemosporidian gametocytes appear in the blood of their hosts only after a certain prepatent period, which lasts about 4 to 5 days in *L. simondi*. If it were an infection by *L. simondi*, we should have found gametocytes in ducks which survived the infection. Briggs (1960) also compared the course of infection caused by *L. simondi* in domestic ducks and in muscovy ducks and detected gametocytes in the blood of both duck species, though in muscovy ducks gametocytaemia was lower. Unfortunately this author paid no heed to the morphology of the internal stages of *L. simondi* in the ducks in question.

The muscovy duck is also known to be the host of *L. simondi* in other regions (Lapage 1961, Garnham 1966, Hsu et al. 1973), so that the *L. simondi* infection in our case to be atypical owing to unusual host is improbable.

A very similar infection in muscovy ducks was also observed in Canada (Julian nad Galt 1980). Neither these authors found any gametocytes of haemosporidia in the blood of the ill birds, and described schizonts of identical morphology in their internal organs as in our case. Moreover, their paper shows that the causative agent is *Haemoproteus*, most likely a species occurring in Anseriformes — *H. nettionis* (Johnston et Cleland, 1909) Coatney, 1936. This parasite has insofar been considered to be a non-pathogenic species (Levine 1973). After Julian and Galt (1980) its increased pathogenicity to muscovy ducks and inability of forming gametocytes in this host is due to the fact that muscovy ducks (genus *Cairina*) are unusual hosts for this parasite. Muscovy ducks originally come from South America and in the North America *Haemoproteus nettionis* occurs in ducks of other genera (primarily *Anas*) systematically less related to the genus *Cairina*.

In Central Europe, however, *Haemoproteus* does not occur in Anseriformes (Kučera 1981c) and from other parts of Europe only two sporadic cases of infection by these parasites were reported from ducks in the Paris and London zoos (Kowarski et al. 1937, Hammerton 1931). After Williams and Bennett (1980) the *Haemoproteus* species in anatids are distributed throughout the world except the Palaearctic region. Consequently, in our case the infection was not caused by *Haemoproteus nettionis* directly, but most likely by another species of haemosporidia.

Having their site in endothelial cells of capillaries and sinusoids of internal organs of muscovy ducks and due to their morphology as well, the described schizonts very much resemble not only the schizogonic stages of some known *Haemoproteus* species (see Aragao 1908, Baker 1966, Garnham 1966, Khan and Fallis 1969, Burtikashvili 1973, Peirce 1976), but similar stages of *Plasmodium*, primarily the species of the subgenus *Haemamoeba* (see e.g. Garnham 1966 etc.). However, *Plasmodium* does not occur in free-living Anseriformes in Central Europe either (Kučera 1981a, c) and even from other parts of Europe not a single finding of these parasites in Anseriformes is known. On the other hand, different species of *Haemoproteus* and *Plasmodium* as well as *Leucocytozoon* are abundant in many other wild birds in Central Europe (Kučera 1981b, c).

It may be gathered from the above said that muscovy ducks in our case must have been infected by some unspecific parasite species acquired from local populations of wild birds, apparently other than Anseriformes. Consequently, in our case the infection

may be considered an aberrant infection in muscovy ducks, because it was caused by an unusual parasite species in an exotic host. The causative agent is virulent to such an extent that it is able to develop in the internal organs of muscovy ducks and to cause death of these hosts. It is, however, unable to complete its life cycle in this unusual host and to form blood stages (gametocytes) capable of further transmission.

A similar aberrant haemosporidiosis in Europe occurred in the last two decades as the so-called aberrant leucocytozoonosis of parrots kept in open-air cages (see e.g. Frank 1965, Walker and Garnham 1972, Minárik and Dyml 1972). This disease affecting the youngs of Australian parrots shows similar symptoms as those described by us in the case of infection of muscovy ducks. In the internal organs of the infected parrots, however, typical megaloschizonts were present which are an evidence that in this case the infection was caused by some undetermined *Leucocytozoon* species (Frank 1965, Walker and Garnham 1972). However, these megaloschizonts are absolutely different from the stages present in the internal organs of muscovy ducks. We have verified this fact directly in the comparative material from the parrots.

The morphology of schizonts present in the internal organs of muscovy ducks shows that in our case the causative agent of the disease is most probably some species of *Haemoproteus* or *Plasmodium*. In view of insufficient knowledge on the morphology of similar stages in the majority of avian species of haemosporidia in Central Europe also the related *Leucocytozoon* may be involved. This genus namely contains species known for the absence of megaloschizonts in their development (Khan and Fallis 1970, Clark 1965). The differentiation of genera and species of avian haemosporidia, however, is mostly based only on the morphology of blood stages (Garnham 1966 etc.), but in our case this morphology is unknown and therefore it is very difficult to identify the parasites described in muscovy ducks.

The above discussion shows that the infection in muscovy ducks was not caused by *Leucocytozoon simondi* as had been supposed earlier (Commichau and Jonas 1977). It was apparently an aberrant infection caused by an undetermined haemosporidian, whose reservoir are probably the populations of local wild birds. In agreement with Julian and Galt (1980), the most probable causative agent seems to have been *Haemoproteus*, but some other species than *H. nettionis* typical of ducks, which does not occur in Anseriformes in Central Europe. The morphology of the parasite stages found indicates that *Plasmodium* might be involved and *Leucocytozoon*, for the time being, cannot be ruled out, either.

ГЕМОСПОРИДИОЗ КАК СМЕРТЕЛЬНОЕ ЗАБОЛЕВАНИЕ УТОК ВИДА *CAIRINA MOSCHATA* В ЮЖНОЙ ЧЕХИИ

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Резюме. Дано описание заболевания утят вида *Cairina moschata*, повторно появившегося в южной Чехии в последнем десятилетии. Признаки и возбудитель этого заболевания оказываются сходными с ранее описанным заболеванием уток указанного вида в Рейнской области (Commichau et Jonas 1977). Однако, это заболевание не вызвано организмом *Leucocytozoon simondi*, как ошибочно полагали авторы из Федеративной Республики Германии, т. к. шизогонные стадии из внутренних органов пораженных уток вида *Cairina moschata* морфологически значительно отличаются от стадий развития *L. simondi*. По всей вероятности возбудителем заболевания является какой-то вид гемоспоридий от местных популяций диких птиц, который способен частично развиваться в внутренних органах этих уток. В этом необычном хозяине, однако, протекает лишь шизогонное разделение и паразит неспособен образовать кровяные стадии и таким образом завершить свой цикл развития. Так как морфология кровяных стадий не известна, нельзя пока возбудителя ближе определить. По морфологии шизогонных стадий возбудитель больше всего походит на паразитов родов *Haemoproteus* или *Plasmodium*, менее на *Leucocytozoon*. Очевидно дело не касается вида *Haemoproteus nettionis*, который вероятно вызывает подобное заболевание у уток вида *Cairina moschata* в Канаде (Julian et Galt 1980), т. к. этот вид в Средней Европе отсутствует.

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A CONTRIBUTION TO THE KNOWLEDGE OF LOUSE FLIES (DIPTERA, HIPPOBOSCIDAE) FROM AFGHANISTAN

The research workers of the Institute of Parasitology, Czechoslovak Academy of Sciences, studied the parasites of domestic animals in Afghanistan during an expedition organised in the autumn (September—October) of 1976. The collections and studies were carried out at the Kabul abattoir and in some other localities. The reported results follow up the line of the previous expedition in 1974 (Minář J. et al., Folia parasit. (Praha) 24: 92—93, 1977). The authors thank Doc. Dr. K. Blažek and Dr. A. Amin for their collaboration.

Hippobosca longipennis Fabricius, 1805

Material: 1 ♀, Jalalabad, 3. 10. 1976, in a human dwelling (hotel).

A species parasitic on carnivores, distributed in the southern part of Palaearctic region, in the Ethiopian region and in the western part of the oriental region (Chalupský J., Hippoboscidae. In: Fauna ČSSR, Blood-sucking flies and warble flies, Academia, Praha: 447—478, 1980, in Czech, Doszhanov T. N., Louse-flies (Diptera, Hippoboscidae) of Kazakhstan, Nauka, Alma-Ata, 206 pp., 1980, in Russian). It was reported from Iran and Pakistan by Maa (Studies in Hippoboscidae (Diptera) Part 2. Pacific Insects Monograph 20. Ent. Dept. B. P. Bishop Mus., Honolulu, 312 pp., 1969). It is the first finding from the territory of Afghanistan.

Lipoptena capreoli Rondani, 1878

Material: 6♂♂, 18 ♀♀, Kabul 27. 9. 1976. Host: domestic goat (*Capra hircus* L.).

This species was found in Afghanistan by the previous expedition of the Czechoslovak Academy of Sciences (Minář et al. 1977). The hitherto found specimens were wingless and collected in the autumn period.

Pseudolynchia canariensis (Macquart, 1840)

Material: 3♂♂, 5♀♀, Karezimir, 6. 10. 1976. Host: domestic pigeon (*Columba livia* L.).

This species is distributed in the south of Europe, in subtropics and tropics of Asia and Africa. It was reported from Afghanistan by Maa (Studies in Hippoboscidae (Diptera). Pacific Insects Monograph 10, Ent. Dept. B. P. Bishop Mus., Honolulu, 148 pp., 1966).

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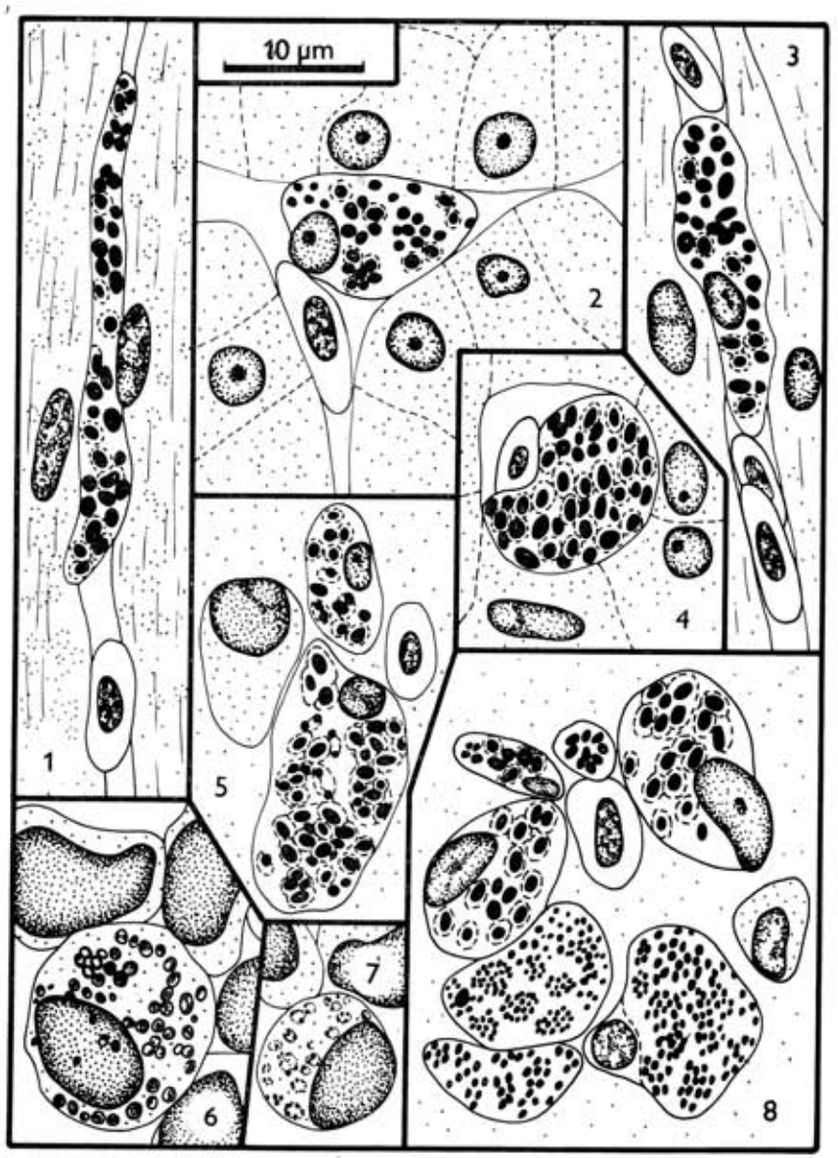

Fig. 1. Elongated schizont in the endothelium of capillary in the heart muscle. **Fig. 2.** Schizont in the liver sinusoid. **Fig. 3.** Schizont in the endothelium of capillary in propria mucosae of intestine. **Fig. 4.** Cross section of capillary in kidney almost blocked by schizont located in the endothelium. **Fig. 5.** Two schizogonic stages in the inflammatory infiltrate in the lungs. **Fig. 6.** Schizonts with ring-like formations on the bone marrow impression. **Fig. 7.** Distorted ring-like forms in the macrophage from bone marrow. **Fig. 8.** A cluster of schizogonic formations in the inflammatory infiltrate in propria mucosae of intestine. Figs. 1—8 have been drawn half schematically from the preparation by means of Abbé's drawing apparatus in the uniform scale of magnification (see the abscissa 10 µm in Fig. 2).

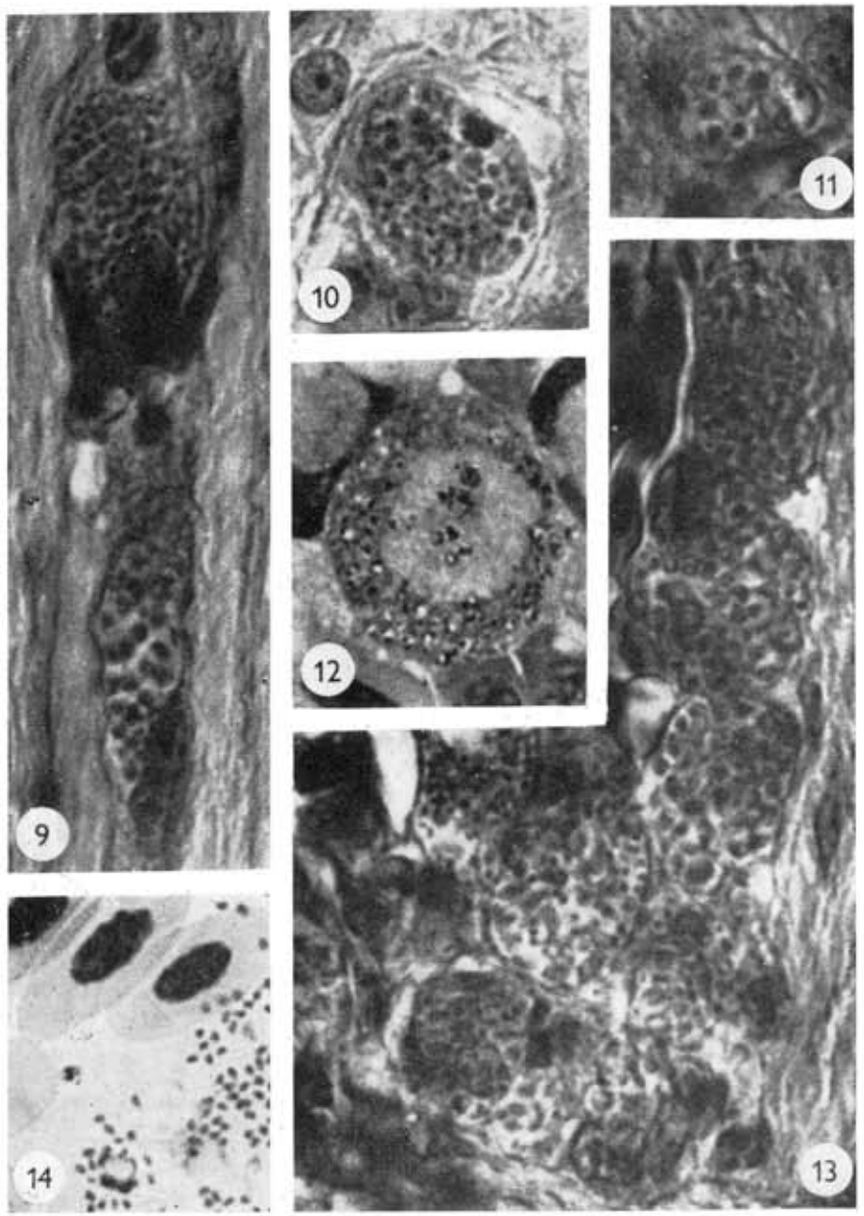

Fig. 9. Photomicrograph of two schizogonic formations located in a row in the endothelium of capillary in muscle layer of intestine. **Fig. 10.** Schizont in kidney capillary (cross section). **Fig. 11.** Schizont in liver sinusoid. **Fig. 12.** Schizogonic stage with ring-like forms from the bone marrow impression. **Fig. 13.** Inflammatory infiltrate in propria mucosae of intestine with numerous schizonts. **Fig. 14.** A cluster of merozoites in the blood plasma on blood smear.
All photomicrographs are in the same scale of magnification, corresponding with the scale of Figs. 1—8 in Plate I. (see abscissa 10 µm in Fig. 2).