Malignant Catarrhal Fever in Texas Cervids

Authors: CLARK, KEITH A., ROBINSON, R. M., MARBURGER, R. G., JONES, L. P., and ORCHARD, JOHN H.

Source: Journal of Wildlife Diseases, 6(4) : 376-383

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-6.4.376

Malignant Catarrhal Fever in Texas Cervids

KEITH A. CLARK and R. M. ROBINSON
Department of Veterinary Pathology
Texas A & M University
College Station, Texas

R. G. MARBURGER
Texas Parks and Wildlife Department
Kerrville, Texas

L. P. JONES
Texas Veterinary Medical Diagnostic Laboratory
College Station, Texas

and

JOHN H. ORCHARD
Kingsville, Texas

Introduction

Malignant Catarrhal Fever (MCF) is a fatal, sporadic, infectious viral disease of world-wide distribution.[20] It has been reported in cattle, buffaloes[11] and Père David's deer *(Elaphurus davidianus),*[6] and has been experimentally transmitted to cattle,[8,13,14,19,20] red deer *(Cervus elaphus),*[6] *rabbits,*[8,13,17,19,20] and possibly other species.[8] The causative virus has been isolated in tissue culture, where it caused cytopathic changes,[20] but has not been well characterized. The virus is extremely fragile,[16] but some strains can be maintained by serial passage through rabbits or by low temperature storage in tissue culture. Similarities to the herpes viruses have been described.[20]

Clinical signs of MCF are quite variable,[14] and the following clinical forms have been described: peracute, alimentary, head and eye, and mild forms.[8] The peracute form is manifest by fever, hemorrhagic gastroenteritis, and death in one to three days after the first appearance of clinical evidence of disease. The alimentary form is characterized by fever, diarrhea, diffuse exanthema, lacrimation, and enlargement of lymph nodes.[7] The head and eye form, which is the typical form observed in South Africa,[14] is characterized by neurologic disturbances, enlargement of superficial lymph nodes, mucopurulent nasal discharge, corneal opacity, and edema of the head and neck.[8,14] The mild form is characterized by watery nasal and lacrimal discharges which may become mucopurulent, exfoliative dermatitis, and swollen lymph nodes.[5] Recovery usually occurs in the latter form. This form is observed most often in experimental transmissions of the disease.[8,6]

A contribution of a cooperative project between the Texas Parks and Wildlife Department and the Department of Veterinary Pathology, College of Veterinary Medicine and the Texas Agricultural Experiment Station, Texas A&M University; supported by Texas Pittman-Robertson Project W-93-R.

In view of the overlapping of clinical signs between forms of the disease and the wide variation of the magnitude of such signs, these subdivisions have little practical value.[7] The clinical syndrome has been summarized as being "characterized by severe inflammation of the upper respiratory tract, eye and buccal cavity, and less often of the remainder of the alimentary tract". Neurologic disturbances, dermatitis and enlargement of the superficial lymph nodes also were associated with the disease.[9]

The role of carrier animals in the transmission of MCF is well established,[3,4,12,24] but the method of transmission remains a mystery. Insect transmission has been suspected but has not been proven.[12] There is considerable disagreement among authors as to the role of sheep as carrier animals,[3,4,5,12,23,24] but little doubt exists as to the importance of the blue wildebeest *(Connochates taurinus)*[20] and the black wildebeest *(Connochates gnu)*[12] as carriers in Africa. Transplacental transmission has been demonstrated in the blue wildebeest,[20] and outbreaks of MCF in cattle have long been recognized to occur concurrently with the wildebeest calving season in Kenya and Tanzania.[3] Intravenous inoculation of blood from an apparently healthy black wildebeest produced the disease in oxen.[12]

Attempts at experimental transmission of MCF have yielded variable results. It has been transmitted to susceptible animals by injections of infected blood and/or lymphoid tissues,[3,6,12,13,14] but it has also been produced by the injection of 0.05 milliliters of buffy coat leukocytes,[15] suggesting that the virus may be attached to lymphocytes. Other attempted transmissions to healthy animals have failed.[3,6,14] Reports of transmission by direct contact are rare,[9] and the disease generally is not considered to be contagious.

Because of the variability of the clinical syndrome and irregular transmissibility, the diagnosis of MCF is difficult. However, the demonstration of characteristic histopathological lesions is considered adequate grounds to substantiate the diagnosis.[3,10,19] The lesion which is considered by some authors to be pathognomonic is a fibrinoid necrotizing vasculitis,[7,10,20] accompanied by massive lymphocytic or mononuclear infiltration around vessels of the brain, kidney, liver and lung.[2,3,10,12,20] Hyperplasia of free macrophages in lymphoreticular organs is also considered to be a constant lesion.[19] Corneal opacities result from leukocytic infiltration,[19] and the many variable lesions observed elsewhere are considered a result of the primary necrotizing vasculitis.[7,19] A direct necrotizing effect on smooth muscle of the spleen and lymph nodes has also been described.[10,20] The present report describes the pathological findings in a herd of axis deer and one white-tailed deer affected with MCF.

History and Clinical Signs

In late 1968, a disease which had been observed sporadically during the previous 12 months in a herd of axis deer *(Axis axis)* in southern Texas was investigated. The disease had killed all except fourteen of the original forty animals in the herd. Eighteen animals died before one sick animal was captured and submitted to a veterinarian for examination.

The axis deer were held in a 1,000 acre pasture along with domestic sheep and cattle, mouflon sheep *(Ovis musimon)*, fallow deer *(Dama dama)* and white-tailed deer *(Odocoileus virginianus)*. Nilgai antelope *(Boselaphus tragocamelus)* also inhabit the general area. None of these species have been observed to have been affected.

The first sick animal was necropsied in November, 1968; the second in January, 1969; and a third in June, 1969. On September 12, 1969, another sick female deer, approximately one-year old, was captured and transported to the Wildlife Disease Laboratory at Texas A&M University. No further losses have occurred since that time and no other sick animals have been observed.

The owner of the herd of axis deer reported that the affected animals became progressively weaker over a period of about one week, strayed from the herd,

(axis deer are quite gregarious) and invariably stood with an arched back. Without exception, they appeared to be blind, and all had crusty skin lesions. Those animals examined were lethargic, emaciated, weak, ataxic, and apparently blind. Encrusted lesions were observed on the skin around the mouth, eyes, anus, along the back and sides and around the feet. One animal had involuntary rhythmic movements of the head, excess salivation, and constantly turned to one side. Its corneas were slightly cloudy. (Figure 1)

In April, 1969, in another part of southern Texas, a one-year-old female white-tailed deer was observed to be flailing a prickly pear plant with her front feet. She was down on her carpi and made sporadic jumps and lunges. After two or three minutes of such activity, she lunged forward, falling onto her side, gave a few convulsive jerks of the rear legs and died. The deer was in good physical condition, and except for its unusual behavior, appeared normal. No gross lesions were found at necropsy and selected tissues were saved for histopathologic evaluation.

Gross Lesions

The following observations were made from two animals, but on the basis of field necropsies, are considered to be typical of the affected axis deer. The cutaneous lesions were areas of alopecia encrusted with thick, dried exudate. The crust was brittle and serum oozed from it when it was cracked. The lymph nodes and lymphatics, particularly those in the mesentery, were greatly enlarged and appeared as white cords radiating from the lymph nodes. The mesentery was gelatinous and clear fluid ran freely from the tissue when it was incised.

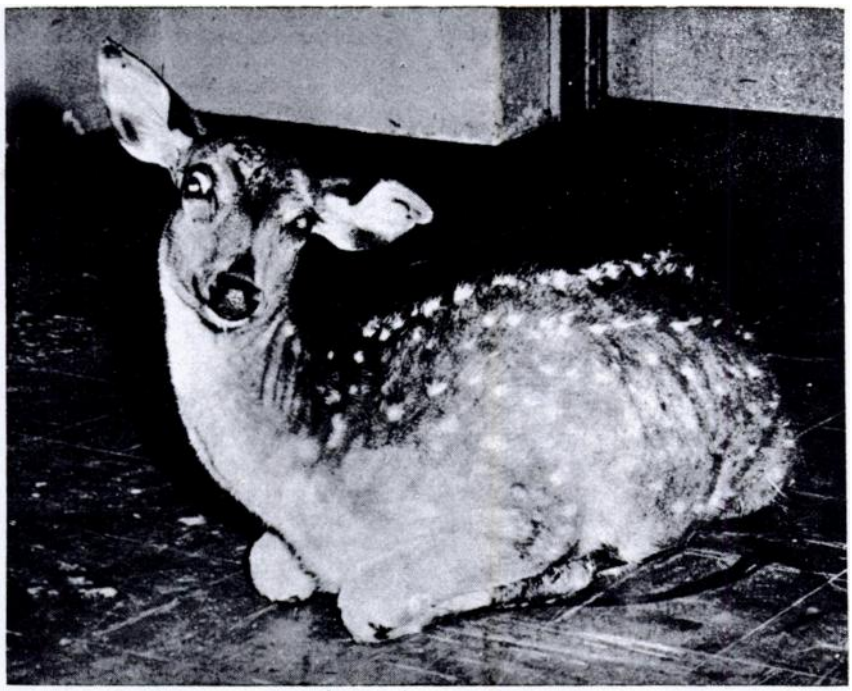

FIGURE 1. *Clinical appearance of an axis deer with signs of Malignant Catarrhal Fever. Note opaque corneas, encrusted skin on sides, excess salivation, attitude of head and neck.*

The arteries of the last animal examined were greatly enlarged, some appearing three times as large in diameter as those of normal animals. This was particularly true of the coronary arteries which were exceedingly prominent (Figure 2). Oral reports of other necropsies suggested this vascular change was present in the other deer. The nasal mucosa was bright pink and a clear serous fluid coated its surface. The oral and gastrointestinal mucosa was not remarkable in appearance.

Histopathology

Microscopic examinatien of tissues from four of the affected axis deer revealed an arteritis, phlebitis, and lymphangitis which was severe. The affected vessels had a marked intimal proliferation, almost to the point of luminal obliteration. The hyperplastic endothelium and arterial media were infiltrated with lymphocytes, and the adventitia was infiltrated with lymphocytes and macrophages (Figure 3). Fibroblastic proliferation had occurred within the tunica media and had replaced smooth muscle fibers. Perivascular cuffing by lymphocytes and macrophages was pre-

FIGURE 2. *Heart showing great increase in size of coronary vessels and perivascular edema.*

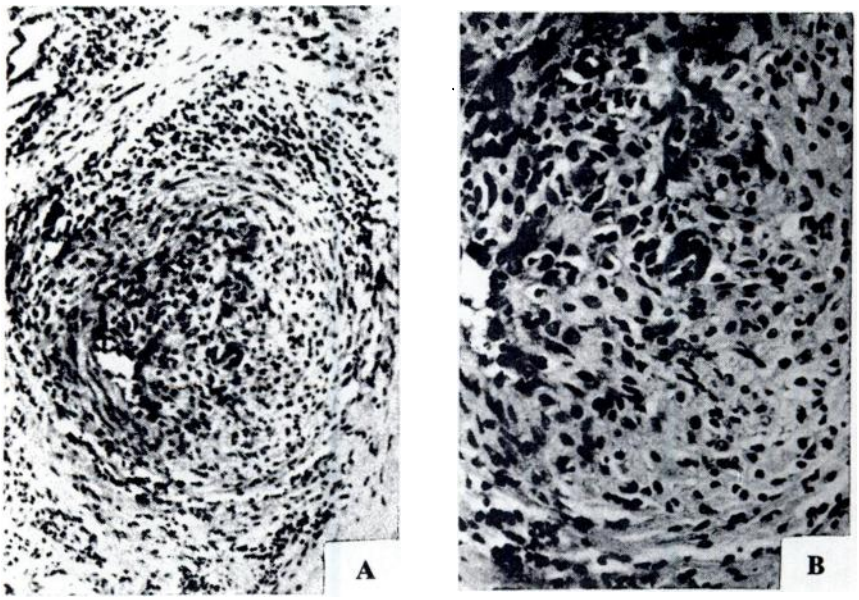

FIGURE 3. *Arcuate artery. Lumen is almost obliterated by proliferation of reticuloendothelial cells; Adventitia is infiltrated by lymphocytes and macrophages. A. H&E x 200. B. H&E x 400.*

sent in the brain in all cases examined. (Figure 4), and infiltration of the ependymal lining of the choroid plexus by lymphoid cells was sometimes seen. Infiltration by lymphoid cells was also commonly observed in hepatic portal areas, in and around vessels of the kidneys, lungs, myocardium, spleen, lymph nodes, intestine, and in the aorta. The skin lesions were ulcerative, with necrotic debris adhering to the dermis which was infiltrated by lymphocytes, plasma cells, and neutrophils. Bilateral panophthalmitis was present. The cornea was edematous and infiltrated by lymphoid cells (Figure 5). Increased vascularity was also present in affected areas. A fibrin clot was present in the anterior chamber, and the ciliary body was edematous and infiltrated with lymphoid cells. Anterior or posterior synechia and cataracts were present, and lymphocytes and macrophages were present within the vitreous body. Perivascular lymphoid cuffs surrounded retinal vessels, and optic neuritis was manifest by perivascular lymphoid infiltration. Hyperplastic reticuloendothelial tissue filled the sinusoids of lymph nodes (Figure 6). In some cases, few active germinal centers were present in the spleen and lymph nodes. Foci of necrosis with concomitant mineralization were present in the aortic wall and in splenic trabeculae (Figure 7). The vasculitis was most severe in the brain of the white-tailed deer (Figure 8), but was also present in its lymph nodes and spleen.

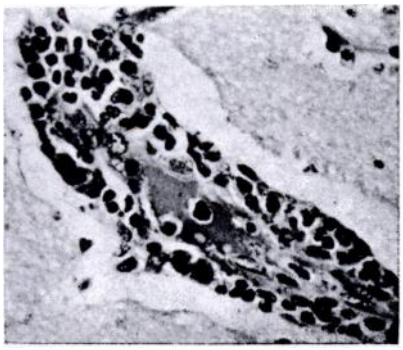

FIGURE 4. *Perivascular cuffing by lymphocytes and macrophages in the cerebral cortex. H&E x 400.*

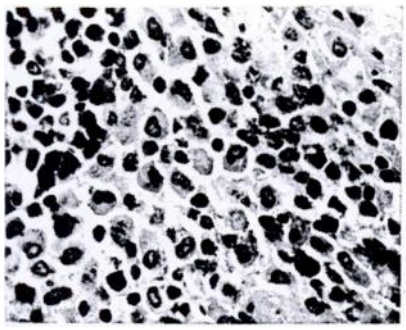

FIGURE 6. *Lymph node. Sinusoids are filled with macrophages. H&E x 400.*

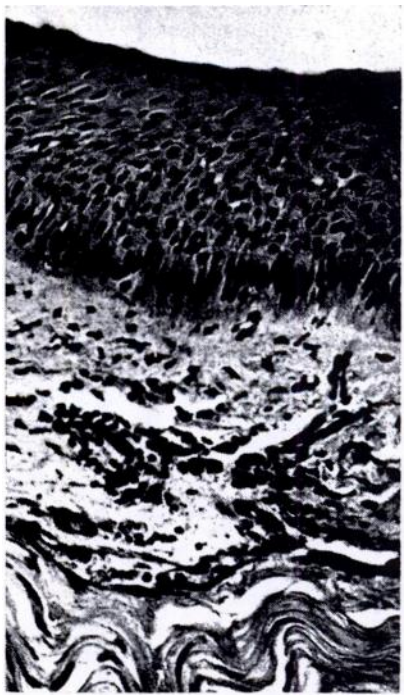

FIGURE 5. *Cornea. Increased vascularity. infiltration by lymphocytes, and edema of the lamina propria. H&E x 200.*

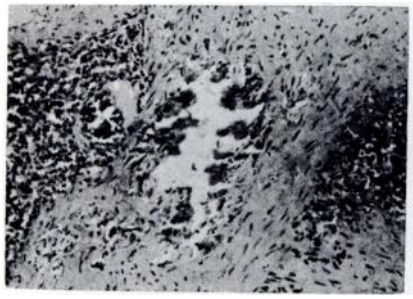

FIGURE 7. *Necrosis and mineralization of smooth muscle of splenic trabeculus. H&E x 200.*

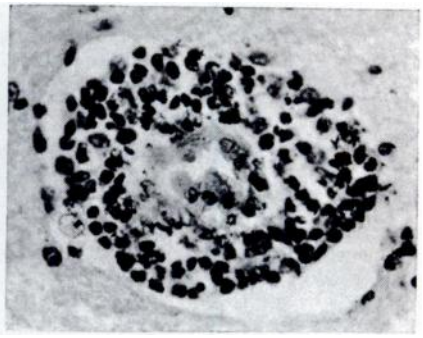

FIGURE 8. *Perivascular cuffing by lymphocytes, macrophages, and a few neutrophils in the cerebrum of a white-tailed deer. H&E x 400.*

Discussion

The clinical signs observed during this disease outbreak are consistent with those described for MCF. However, the mucopurulent nasal discharge usually seen in cattle was absent, there being a clear serous nasal discharge in all cases observed. One must bear in mind, however, that the clinical signs of MCF are outward manifestations of the primary vasculitis, and different species might reasonably be expected to show different clinical signs. It has been reported that catarrhal signs disappeared with repeated experimental passages in cattle,[18] and since the described signs were present late in the disease outbreak, the virus may have been modified.

Differential Diagnosis

Several diseases other than MCF have been considered in differential diagnosis:

Bluetongue: Histologically, the only infectious disease of ruminants other than MCF which resembles the described syndrome is bluetongue, in which the primary lesion is a vasculitis, with thrombosis being common. However, ocular and neural lesions are not reported, and the severity of the vascular change is not as great.[22]

Foot and Mouth Disease: The usual form of this disease is vesicular, affecting epithelial surfaces; in young animals, a form manifest by acute myocarditis and gastroenteritis occurs, but in neither form is the primary lesion vascular in origin.[7]

Vesicular Stomatitis: In deer, this disease is one of vesicle formation on the oral epithelium, and recovery is rapid.[8]

Mucosal Disease — Virus Diarrhea Complex: This disease produces signs of enteritis in cervids, and the primary tissue affected is oral and gastrointestinal mucosal epithelium[21] and lymphatics.

Periarteritis Nodosa: A white-tailed deer shot by a hunter in New York State is reported to have had typical lesions of periarteritis nodosa in the soft tissues around a year-old gunshot wound and fracture. These lesions are intense arteritis; thickening and infiltration of all layers of the arterial walls by lymphocytes, plasma cells, and a few neutrophils. The media was disorganized, and about one-fourth of the lesions had areas of fibrinoid necrosis.[1] Unfortunately, a review of the medical literature reveals that the name "periarteritis nodosa" has been applied to many syndromes in which there is widespread necrotizing arteritis with obscure or unknown etiology. There have been several reports of periarteritis nodosa in animals, and it is especially interesting that it has been described in an epizootic form in only one species — axis deer. This report by a German pathologist in 1906, described a disease, which he identified as periarteritis nodosa, in a

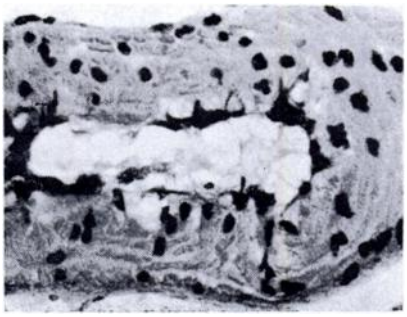

FIGURE 9. *Usual appearance of pulmonary vessels from apparently healthy axis deer. There is apparent increase in size and numbers of nuclei of endothelial cells, vacuolation of the tunica media, and hyperchromasia of smooth muscle nuclei. H&E x 400.*

single herd of axis deer in Germany. This disease had reportedly existed in the herd for over 100 years, and had killed about 100 animals. Diagnosis was apparently based on gross lesions, but later there was a report of histologic conformation. Vasculitis was present in the aorta, the spleen, liver, kidneys, and brain. Catarrhal gastroenteritis was consistently present.[1] These lesions apparently are identical with those observed in the Texas syndrome.

Because of the striking similarity between two disease syndromes in axis deer widely separated by time and space, and because periarteritis nodosa does not appear in epidemic or epizootic form in any other species, the diagnosis of periarteritis nodosa in either outbreak is highly questionable, and there is the possibility that both diseases were MCF.

At this point, it is worthwhile to consider the vessels usually observed in axis deer. Arteries from twenty-five apparently healthy axis deer which have been examined vary in their histologic morphology from those of most other species. These arteries frequently have eccentric lumens, more endothelial cells than usual, and acidophilic media (Figure 9). Endothelial cells appear swollen, and frequently are vacuolated. Vacuolation of the arterial media is also common. Inflammatory infiltrations and necrosis of vascular walls, however, characterize the vascular lesions of the axis deer with MCF.

Because this disease syndrome was observed only in the herd of axis deer on a ranch where many species ranged together, and because of the importance of carrier animals in the spread of MCF, it is necessary to consider the other species present as possible carriers of the disease. This disease outbreak points out two important management considerations: First, the importation of exotic animals may expose our native animal populations to potential disease problems due to the possibility of latent or inapparent infections, and secondly, MCF may be a limiting factor in the development of the axis deer for sporting use in enzootic areas. This potentially is of great economic significance, since the axis is one of the more popular exotic game species in Texas.

Literature Cited

1. BOHROD, M. G. 1956. Periarteritis nodosa in an American deer. Arch. Path. 62: 17-22.
2. DAUBNEY, R. 1959. Suspected bovine malignant catarrh. Letter. Vet. Rec. 71: 493.

3. DAUBNEY, R. and J. R. HUDSON. 1936. Transmission experiments with bovine malignant catarrh. J. Comp. Path. 49: 63-89.
4. DE KOCK, G. and W. O. NEITZ. 1950. Sheep as reservoir host of snotsiekte (or bovine malignant catarrhal fever) of cattle in South Africa. S. Afr. J. Sci. 46: 176-180.
5. DU TOIT, P. J. and R. A. ALEXANDER. 1938. Malignant catarrhal fever and similar diseases. Proc. 13th Inter. Vet. Cong. Pt. 1: 551-557.
6. HUCK, R., A. SHAND, P. J. ALLSOP, and A. B. PATERSON. 1961. Malignant catarrh of deer. Vet. Rec. 73: 457-465.
7. JUBB, K. V. F. and P. C. KENNEDY. 1963. *Pathology of Domestic Animals.* Vol. 2. Academic Press, New York and London, 613 pp.
8. KARSTAD, L. and R. P. HANSON. 1957. Vesicular stomatitis in deer. Am. J. Vet. Res. 18: 162-166.
9. MACKINNON, M. M. 1956. Malignant catarrhal fever of bovines. N. Z. Vet. J. 4: 91-96.
10. MACKINNON, M. M. and D. H. LE SOUEF. 1956. Bovine malignant catarrhal fever in New Zealand. N. Z. Vet. J. 4: 86-91.
11. MANJOER, M. 1957. Contribution to the knowledge of the "Penjakit ingusan" of cattle and buffaloes in Indonesia, especially on the Island of Lombok. Vet. Bulletin, 1958; 2144.
12. METTAM, R. W. M. 1923. Snotsiekte in cattle. 9th and 10th Onderstepoort Reports. pp 395-432.
13. PATTISON, I. H. 1946. Observations on bovine malignant catarrh in Palestine. J. Comp. Path. 56: 254-265.
14. PIERCY, S. E. 1952. Studies in bovine malignant catarrh. I. Experimental infection of cattle. Brit. Vet. J. 108: 35-47.
15. PIERCY, S. E. 1952. Studies in bovine malignant catarrh. II. Experimental infection of cattle (continued). Ibid. 108: 214-220.
16. PIERCY, S. E. 1953. Studies in bovine malignant catarrh. III. The effect of storage and temperature on the viability of the virus. Ibid. 109: 59-65.
17. PIERCY, S. E. 1955. Studies in bovine malignant catarrh. IV. Adaptation to rabbits. Ibid. 111: 484-491.
18. PIERCY, S. E. 1953. Investigations into infectious bovine malignant catarrh. Proc. 15th Inter. Vet. Cong. Pt. 1: 328-332.
19. PLOWRIGHT, W. 1953. The pathology of infectious bovine malignant catarrh in cattle and rabbits. Ibid. 1: 323-328.
20. PLOWRIGHT, W., R. D. FERRIS, and G. R. SCOTT. 1960. Blue wildebeest and the aetiological agent of bovine malignant catarrhal fever. Nature, Lond. 188: 1167-1169.
21. RICHARDS, S. H., I. A. SCHIPPER, D. F. EVELETH, and R. F. SHUMARD. 1956. Mucosal disease of deer. Vet. Med. 51: 358-362.
22. STAIR, E. L., R. M. ROBINSON, and L. P. JONES. 1968. Spontaneous bluetongue in Texas White-tailed deer. Path. Vet. 5: 164-173.
23. STENIUS, P. I. 1953. Bovine malignant catarrh (a statistical, histopathological and experimental study). Proc. 15th Inter. Vet. Cong. Pt. 1: 320-323.
24. WYSSMAN, E. 1938. *Bosartiges katarrhalfieber und Ahnliche Krankheiten.* Proc. 13th Inter. Vet. Cong. Pt. 1: 560-570.