HISTOPATOLOGY AND CYTOLOGY OF POULTRY DISEASES

A COLOUR ATLAS

IVAN DINEV &

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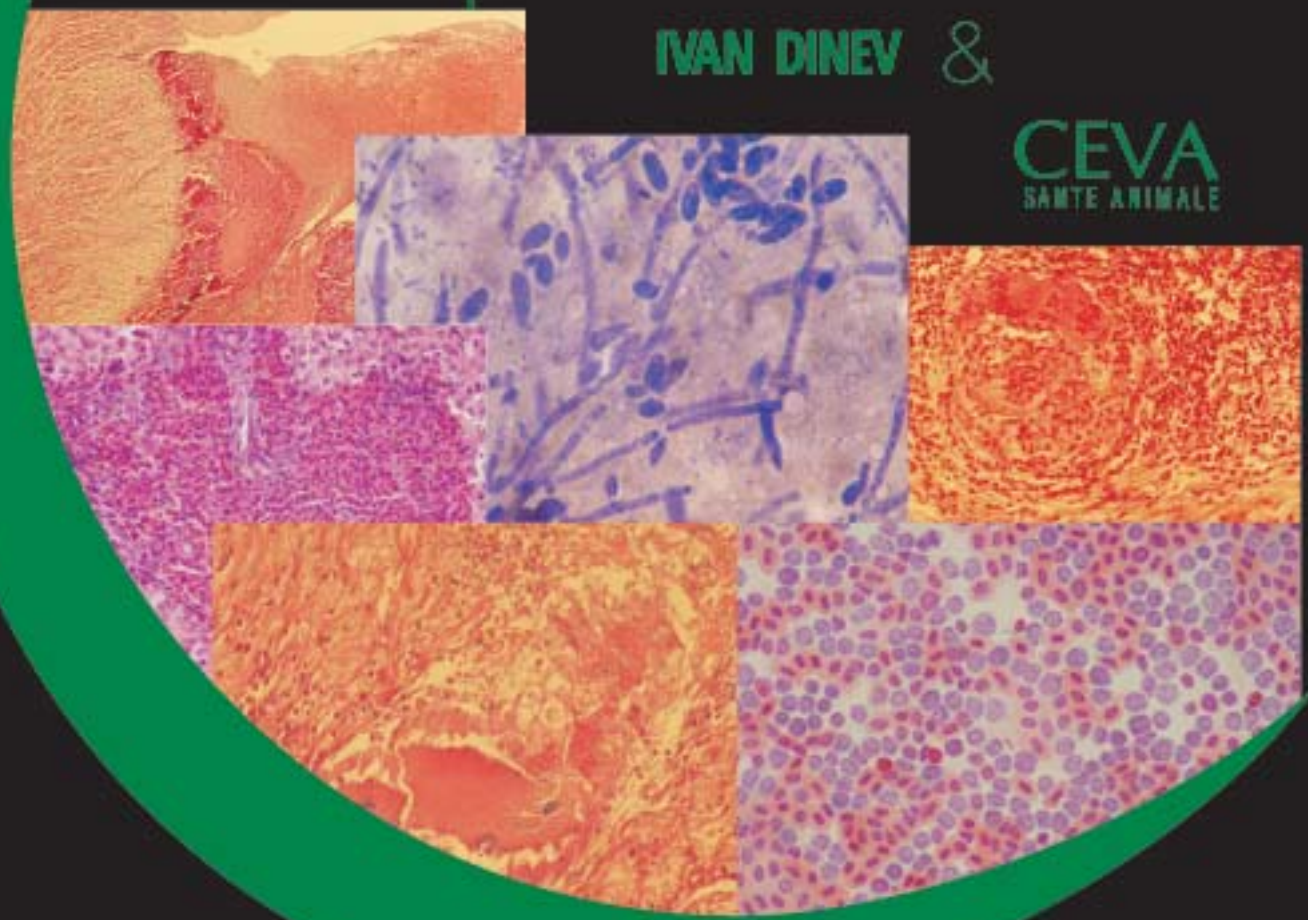
SANTE ANIMALE



A COLOUR ATLAS

HISTOPATHOLOGY AND CYTOLOGY OF POULTRY DISEASES

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A COLOUR ATLAS

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Acknowledgements

First and foremost, I would like to express my heartfelt gratitude towards the team of CEVA SANTE ANIMALE, for their understanding, cooperation, and financial support for the publishing of this book. Thanks to our mutually beneficial cooperation, through this publication we managed to maintain and enrich, on a more professional level, a part of the necessary materials for diagnostical practice in the field of industrial poultry breeding.

I would also like to thank all my colleagues and practitioners, who submitted materials for microscopic study in a number of cases throughout the demanding process of diagnosing poultry diseases.

I am grateful to the staff of the histopathological laboratory at the Department of General and Clinical Animal Pathology at the Faculty of Veterinary Medicine, Trakia University, Stara Zagora, for the high professional level of the materials prepared as part of our diagnostical and research activities in the field of poultry pathology, from which the microscopic photos in this atlas were taken.

Thanks to all who took part in the writing and publishing of this book!

May, 2008 I. Dinev

Preface

After publishing our first book, „A Colour Atlas of Poultry Diseases", our next challenge was to produce a more in-depth and specialized book on the matter. As we noted, the purpose of our first book was to present, in a broader and more accessible way, the macroscopic lesions, characterizing specific diseases and directing towards a specific diagnosis in a pathoanatomical study. The aim of the current book is to present the microscopic lesions on tissue or cellular level that have a diagnostic value for detection of specific diseases in birds.

The book is divided into units on an etiological basis, with the idea of more convenient laboratory diagnostics of poultry diseases. The primary approach is the demonstration of lesions through photographic material. The book contains 214 colour photos total. All images are original and obtained during our research and diagnostic consultant activities.

The demonstrated lesions on the cytological level are relevant for the quick diagnostics of some poultry illnesses, such as neoplastic diseases, some clostridioses, etc. Apart from diagnostics, the histological lesions can be taken into consideration for the understanding of the disease's histogenesis and mechanism. Of course, for some diseases in birds, structural lesions have a decisive diagnostic value, while for others only complementary. Cytological and histopathological changes have a crucial diagnostic value in neoplastic diseases, some viral infections with inclusion bodies (adenoviral, laryngotracheitis), infectious encephalomyelitis, etc. In the majority of cases, the essence of histopathological or cytological studies is confirming a tentative diagnosis based on observed gross lesions.

We hope that the current publication will be readily accepted by avian pathologists working in the field of laboratory diagnostics of poultry disease, as well as by veterinary professionals and students interested in this aspect of pathology.

May, 2008 I. Dinev

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HISTOPATHOLOGY AND CYTOLOGY ASSOCIATED WITH BACTERIAL DISEASES

ESCHERICHIA COLI INFECTIONS

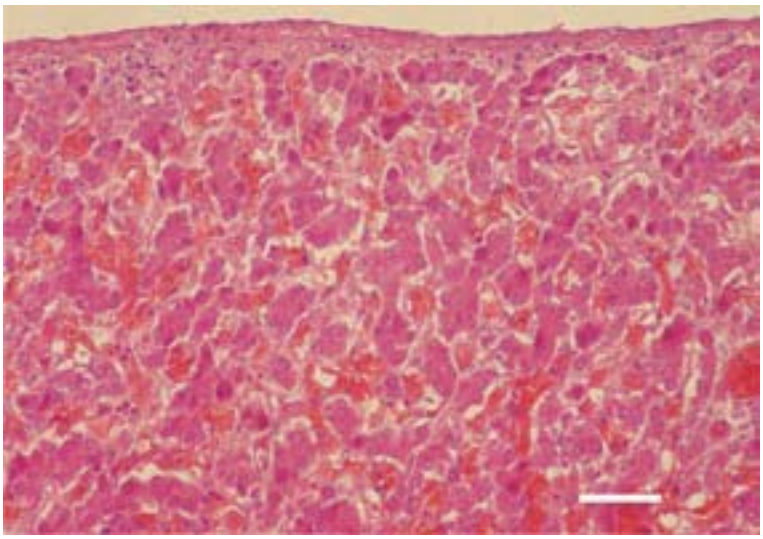
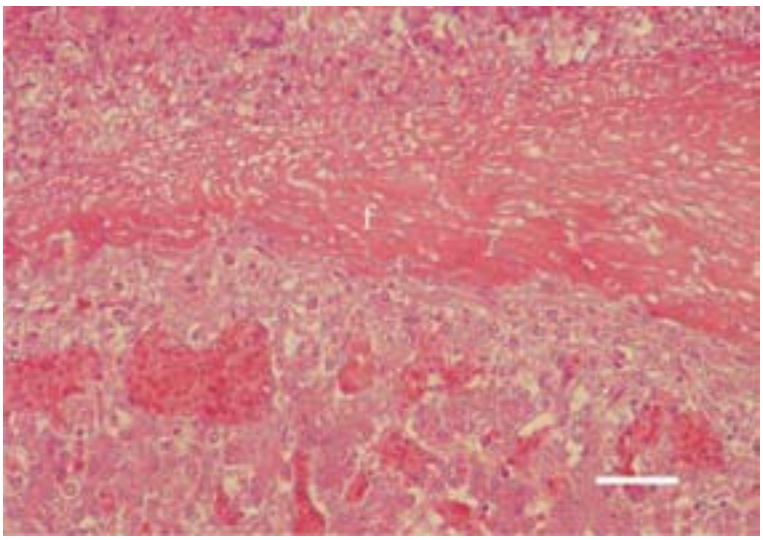


Fig. 1. Congestion (overfilling of blood vessels with red blood cells) of the liver as an initial manifestation of ***E. coli*** septicaemia in a broiler chicken. H/E, Bar = 40 μm.

Fig. 2. Serofibrinous perihepatitis consequent to E. coli septicaemia in a broiler chicken. Huge fibrinous pseudomembrane (f), coating the liver surface. H/E, Bar = 50 μm.



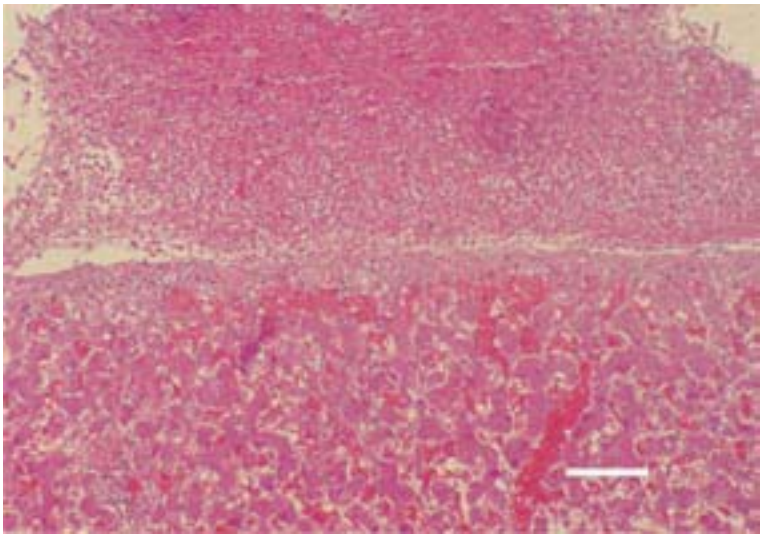


Fig. 3. Serofibrinous perihepatitis. Organization of pseudomembranous deposits. H/E, Bar = 50 μm.

Fig. 4. ***E. coli*** septicaemia. Massive perivascular liver necrosis. H/E, Bar = 30 μm.

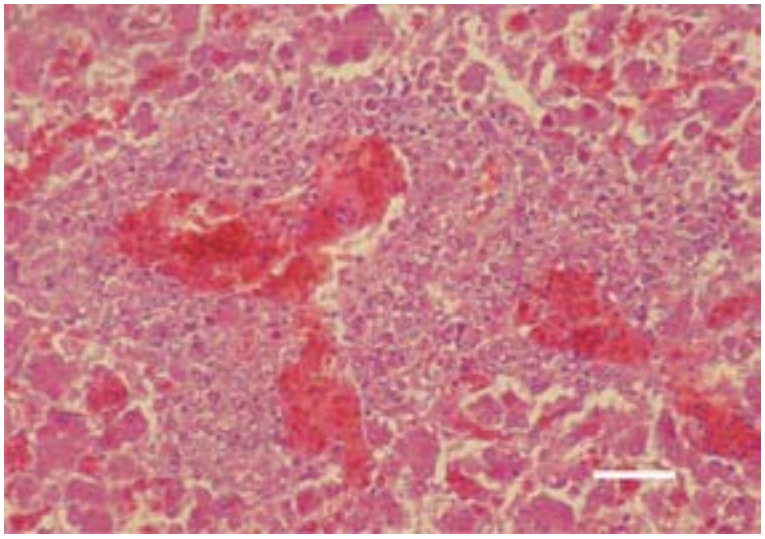
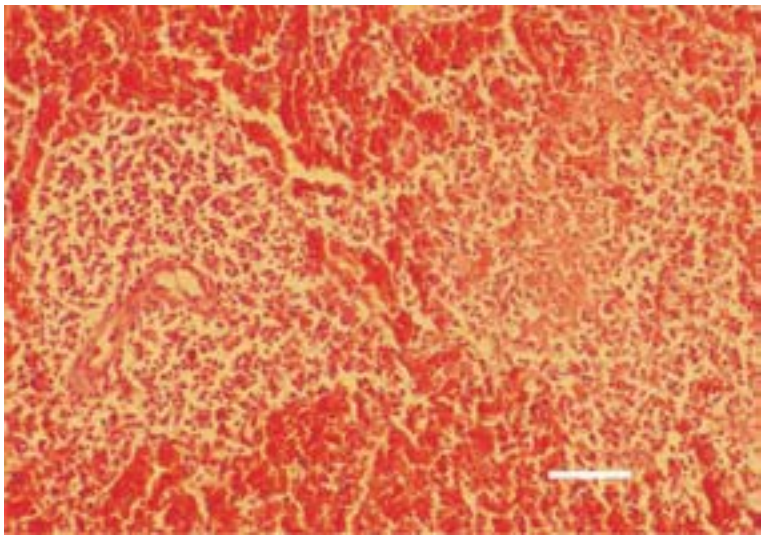


Fig. 5. Periarteriolar reaction, fibrinoid necrosis and congestion of the spleen in E. coli septicaemia. H/E, Bar = 30 μm.



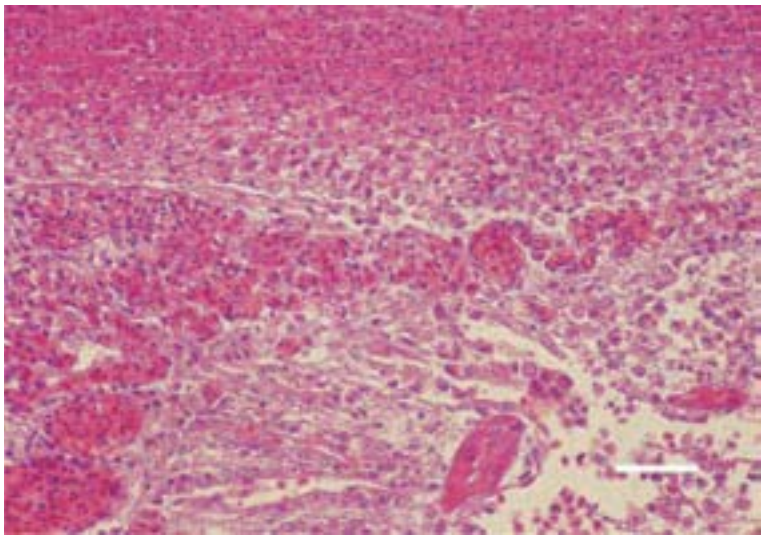


Fig. 6. Croupous pleuropneumonia - one of the commonest findings in ***E. coli*** septicaemia of respiratory origin. H/E, Bar = 25 μm.

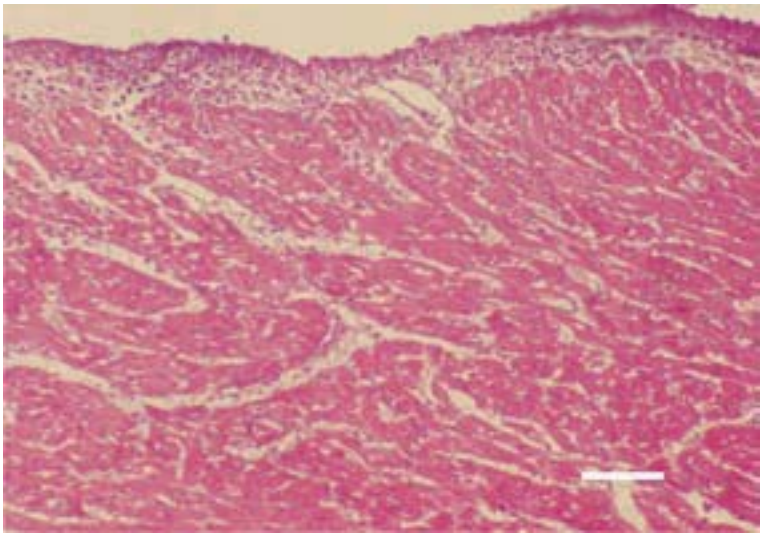
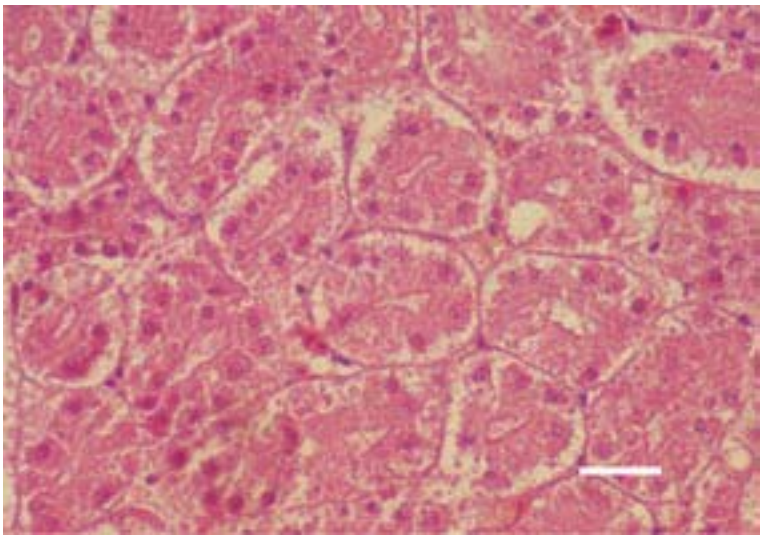


Fig. 7. Serofibrinous pericarditis and inflammatory oedema of the myocardium in ***E. coli*** septicaemia. H/E, Bar = 30 μm.



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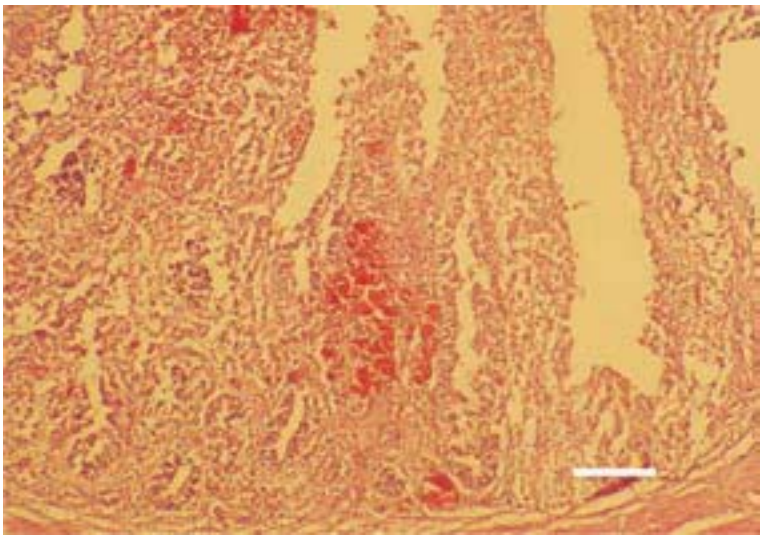

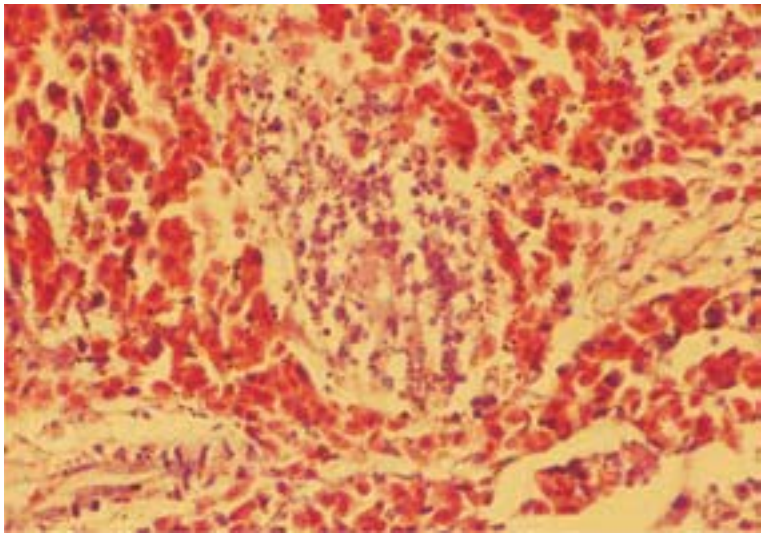

Fig. 10. Element of Fig. 9. Massive haemorrhages surrounding lymphatic clusters in the intestinal wall. H/E, Bar = 25 μm.

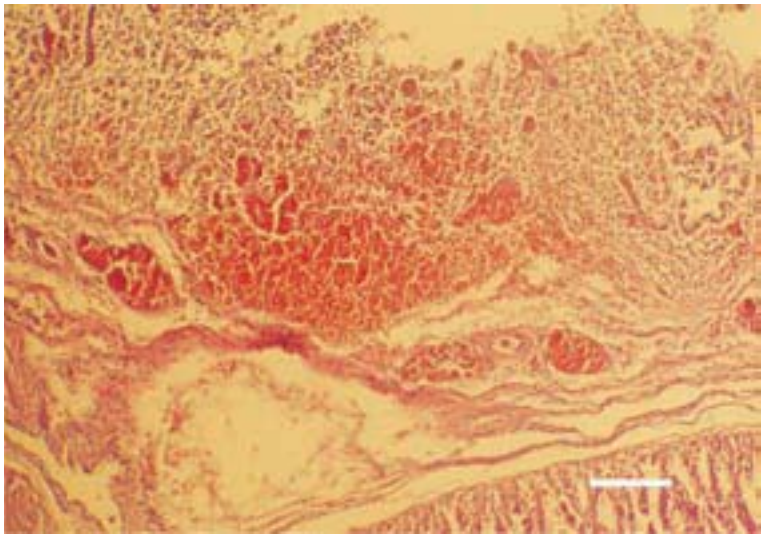

Fig. 11. Haemorrhages in the proventriculus mucous coat in colisepticaemia of enteric origin, secondary to necrotic enteritis. H/E, Bar = 100 μm.

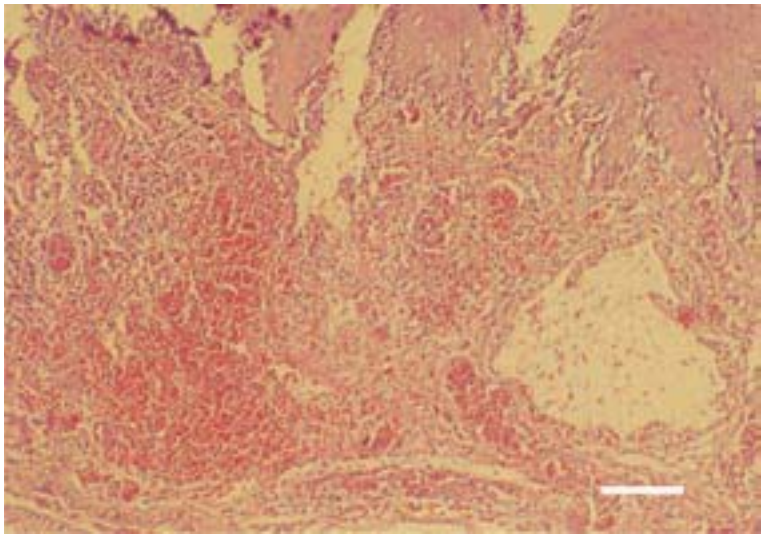

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Fig. 13. Massive haemorrhages in the parenchyma of tonsila caecalis in colisepticaemia of enteric origin, secondary to necrotic enteritis. H/E, Bar = 50 μm.

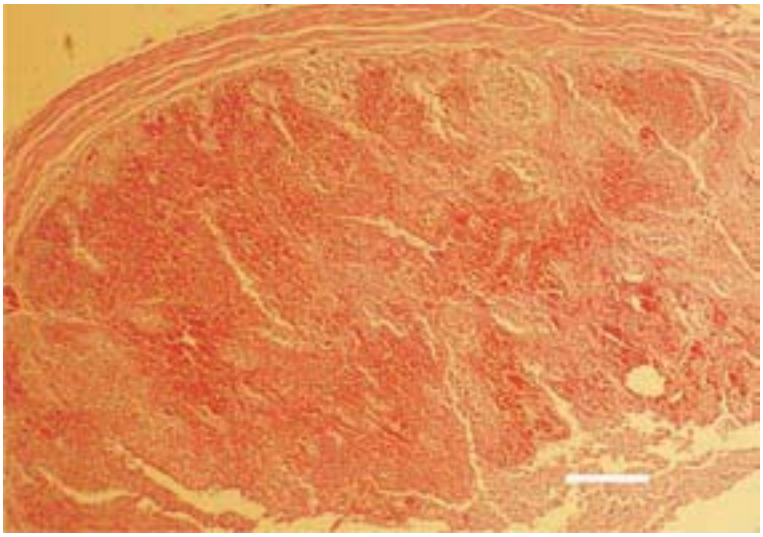
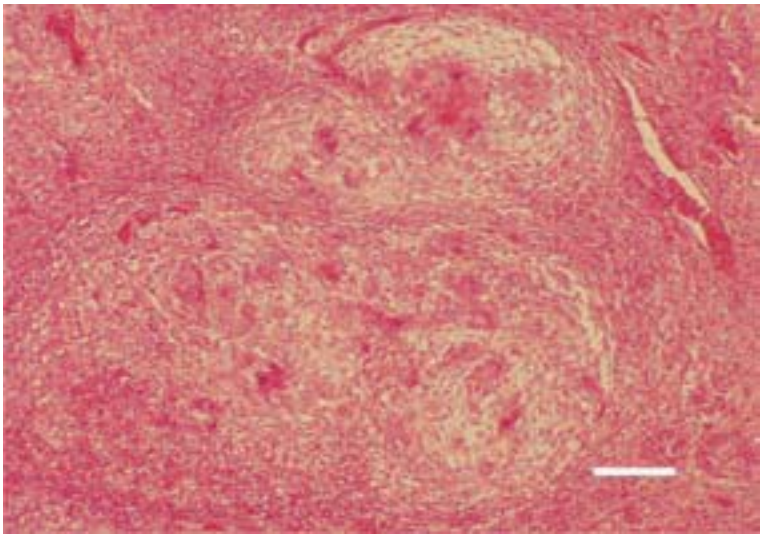


Fig 14. Coligranuloma (Hjarre's disease). Conglomerates of granulomatous nodes against the intestinal wall. Central necrotic detritus and marked acidophilia. Single foreign body-type giant cells. H/E, Bar = 30 μm.



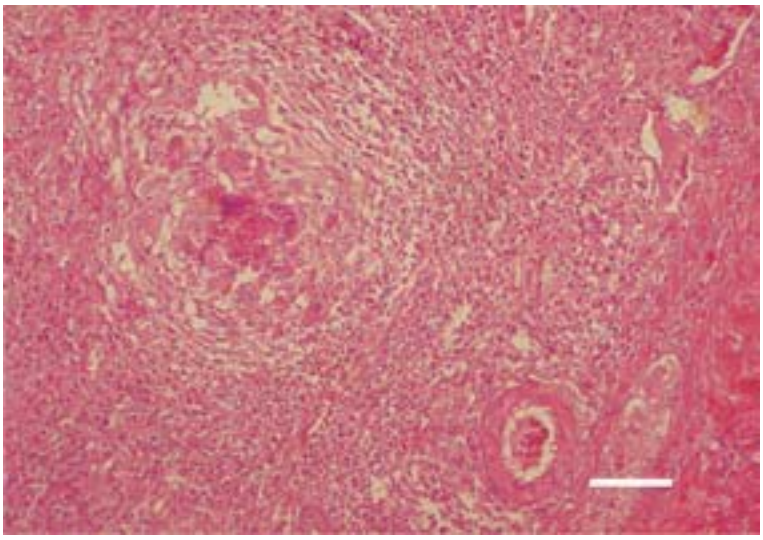


Fig 15. Coligranuloma (Hjarre's disease). Coligranuloma, liver. H/E, Bar = 35 μm.

Fig. 16. Coligranuloma (Hjarre's disease). Coligranuloma, liver. The typical heterophilic debris among the central necrotic detritus. H/E, Bar = 25 μm.

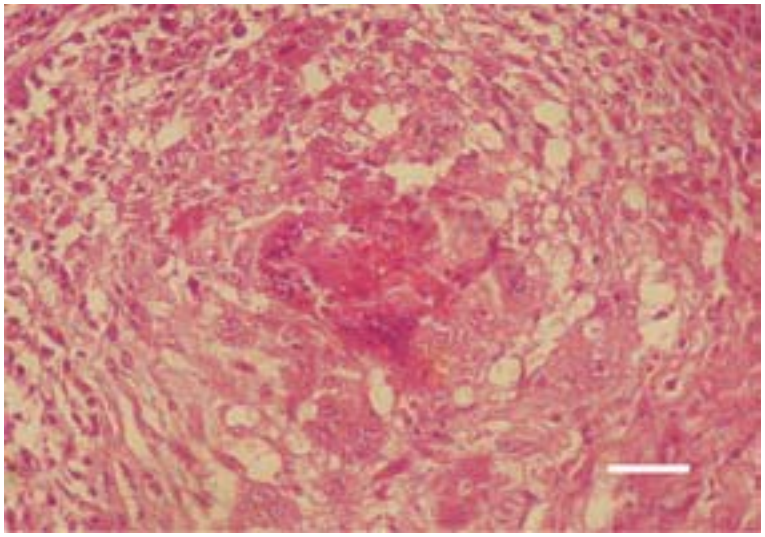
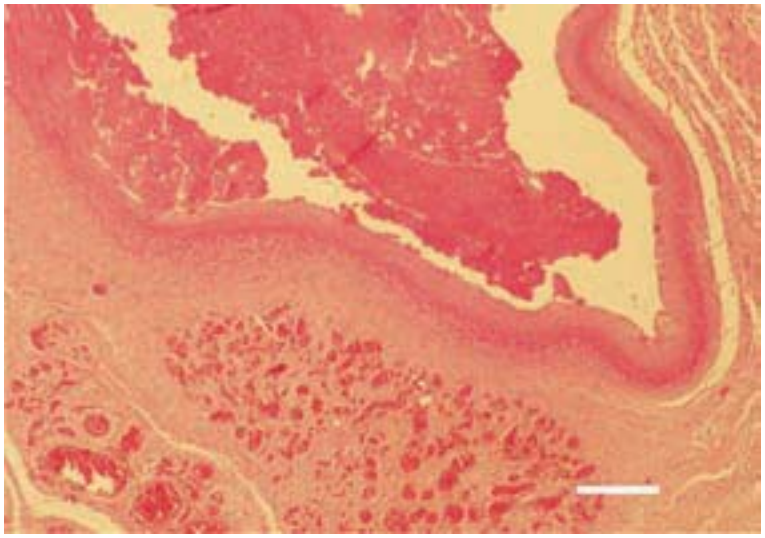


Fig. 17. Sternal bursitis. Filling of the sternal bursa with fibrinous caseous exudate. Peripheral outgrowth of fibrous tissue and inflammatory hyperaemia. H/E, Bar = 100 μm.



SALMONELLOSES

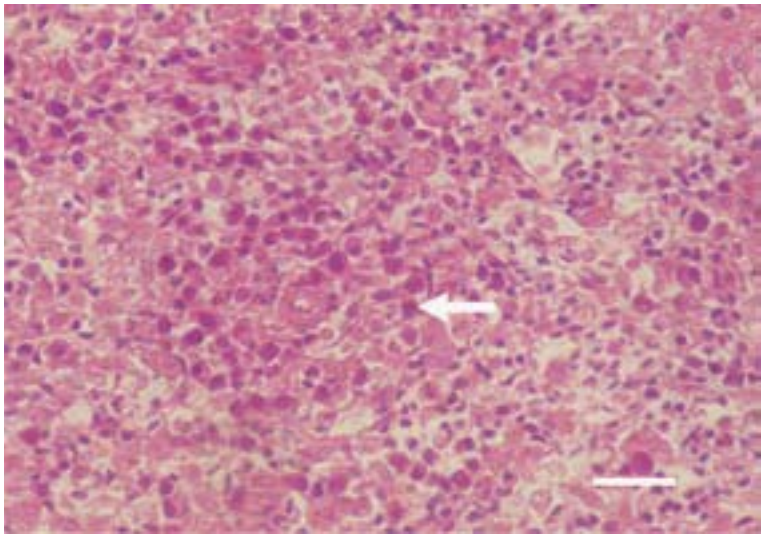


Fig. 1. Pullorum disease. Perivascular proliferative inflammatory focus in the liver, consisting mainly of histiocytes and single epitheloid cells (arrow). H/E, Bar = 25 μm.

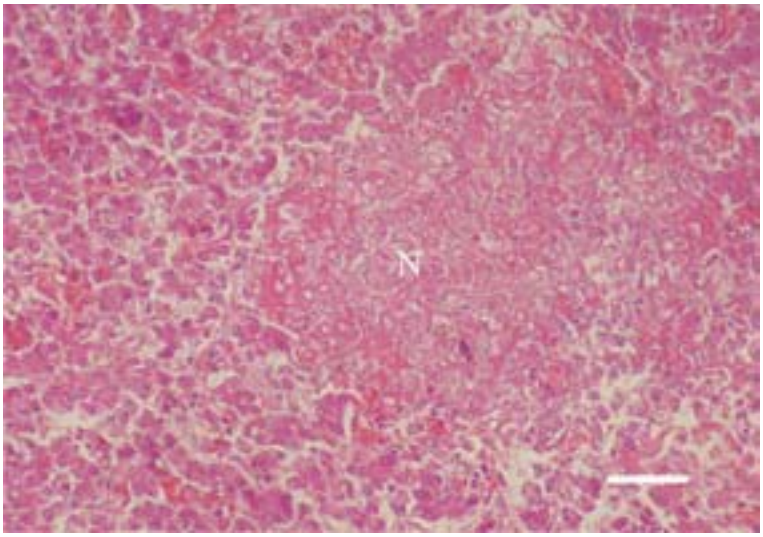
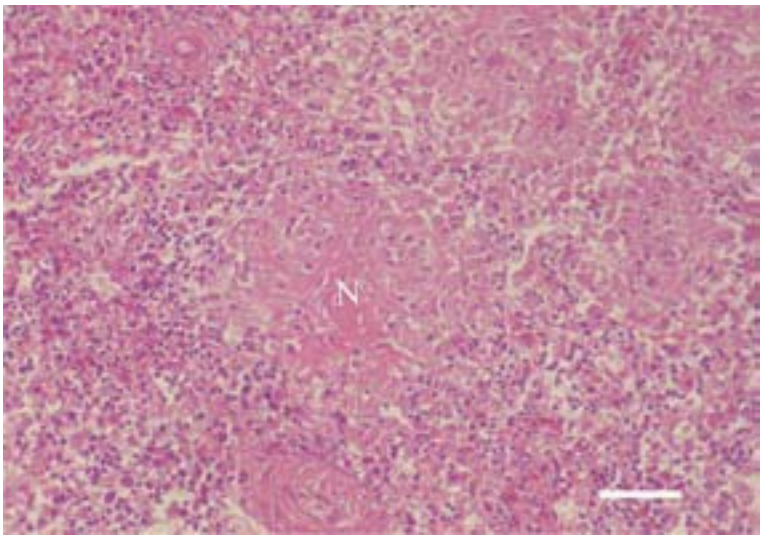


Fig. 2. Fowl typhoid. Areactive fibrinoid necrosis (N) in the liver. H/E, Bar = 40 μm.

Fig. 3. Fowl typhoid. Periarteriolar fibrinoid necrosis (N) in the spleen. Marked cell reaction in the periphery of the necrotic foci involving lymphocytes, histiocytes and single granulocytes. H/E, Bar = 50 μm.



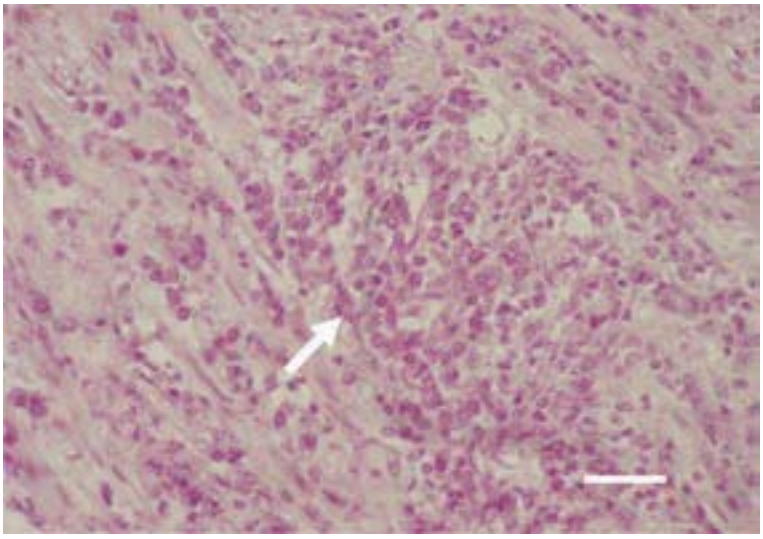


Fig. 4. Fowl typhoid, hen. Mononuclear inflammatory cell proliferate in the myocardium (arrow). H/E, Bar = 30 μm.

FOWL CHOLERA

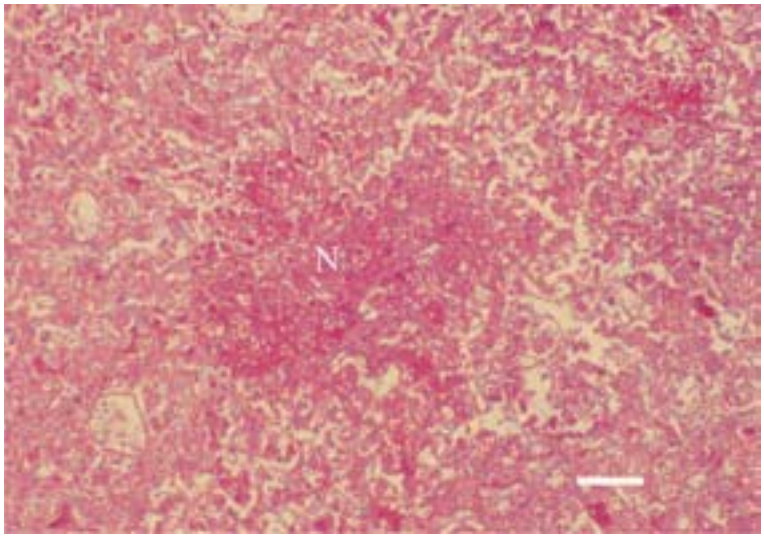
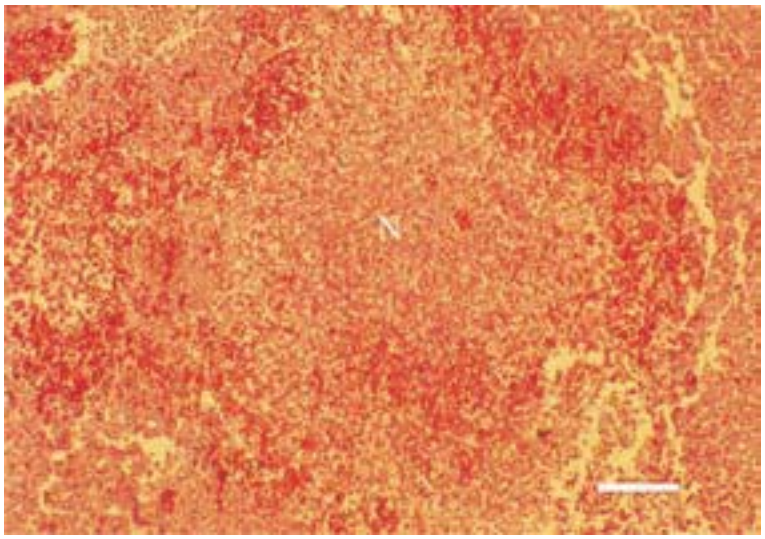


Fig. 1. Acute fowl cholera. Coagulative areactive necrosis (N) in the liver of a hen. Multiple nuclear debris among the necrotic tissue. H/E, Bar = 35 μm.

STAPHILOCOCCUS AUREUS INFECTIONS

Fig. 1. Widespread coagulative necrosis (N) in the liver, surrounded by a haemorrhagic zone as a manifestation of staphylococcal septicaemia in a hen. H/E, Bar = 35 μm.



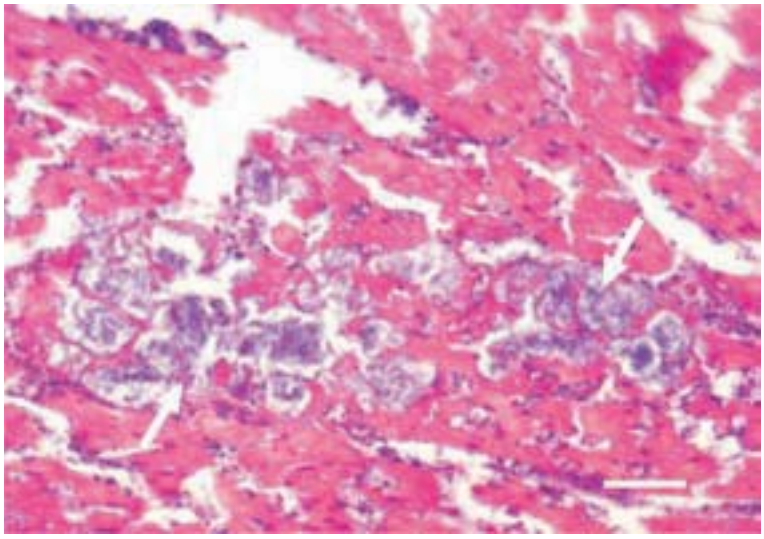


Fig. 2. Interstitial and parenchymatous myocarditis in a broiler chicken. Bacterial colonization and bacterial thrombi in the myocardium (arrows) in staphylococcal septicaemia. H/E, Bar = 25 μm.

Fig. 3. Myocardial infarction (I) in a bird that has survived a staphylococcal septicaemia. H/E, Bar = 30 μm.

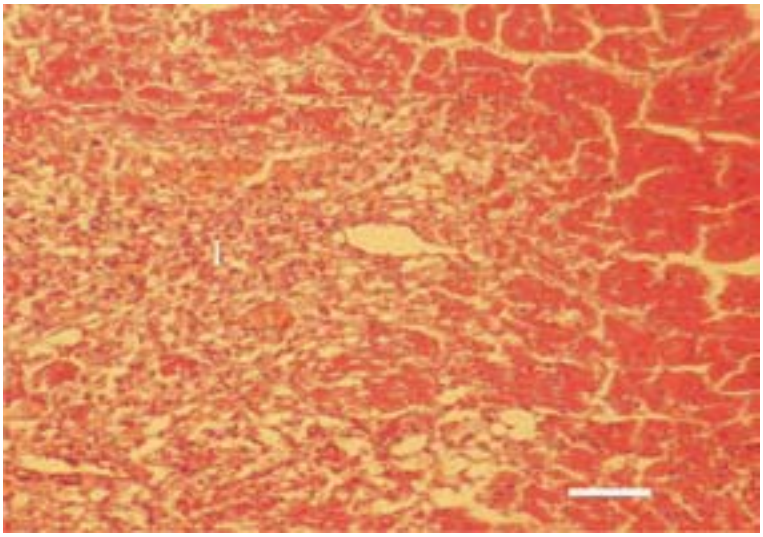
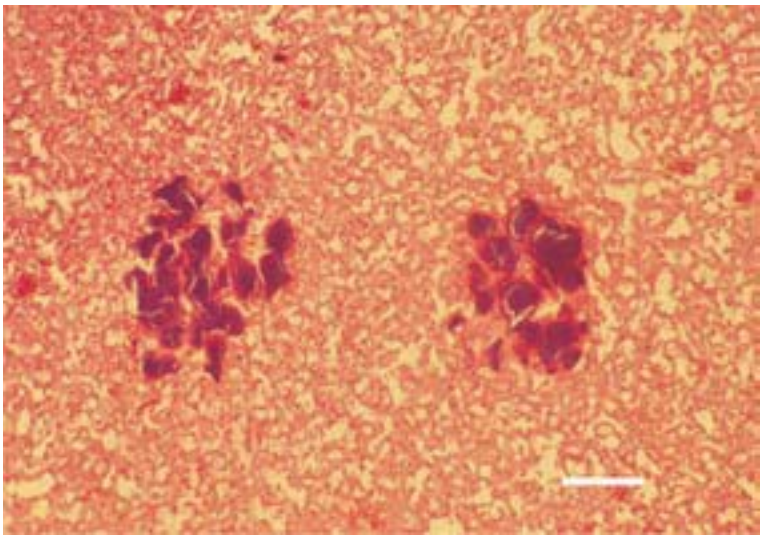


Fig. 4. Bacterial emboli obturating capillary sinusoids and intensive degenerative necrobiotic lesions of the liver, resulting from metastasis of a local focus of staphylococcal infection. H/E, Bar = 35 μm.



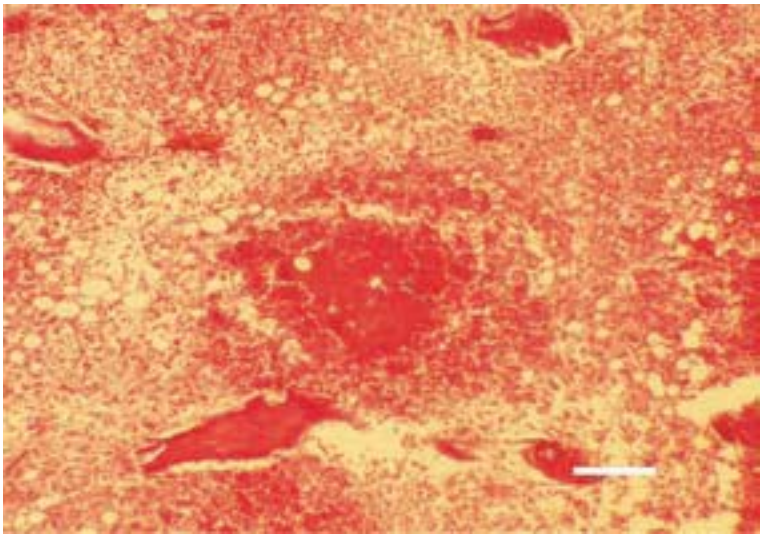


Fig. 5. Staphylococcal osteomyelitis, secondary to septicaemia. An inflammatory necrotic focus, bacterial colonization and bone marrow congestion. H/E, Bar = 30 μm.

Fig. 6. Staphylococcal osteomyelitis. Bacterial thrombus (arrow) obturating a bone marrow sinusoid. H/E, Bar = 25 μm.

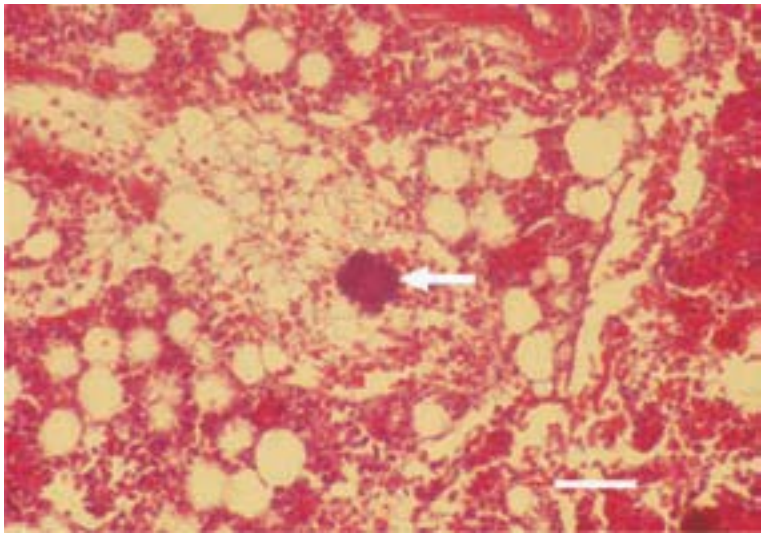


Fig. 7. Staphylococcal osteomyelitis. Focal inflammatory necrotic lesions in the growth plate of the proximal femur in a broiler chicken. H/E, Bar = 40 μm..

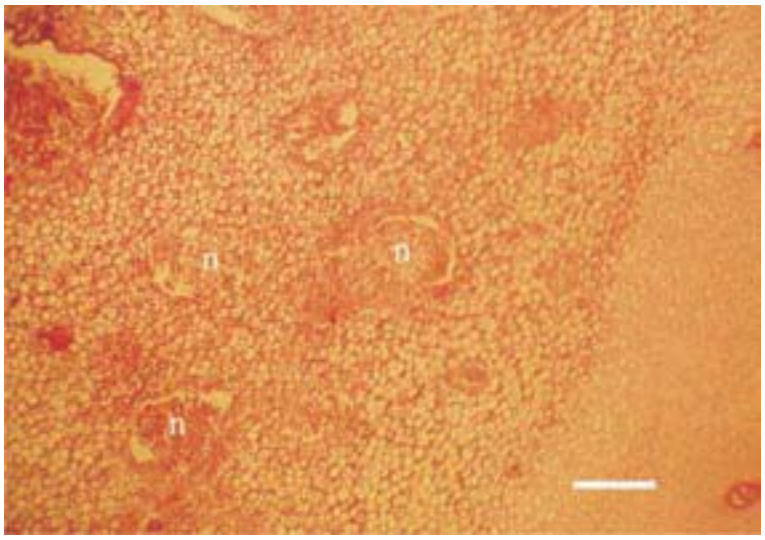
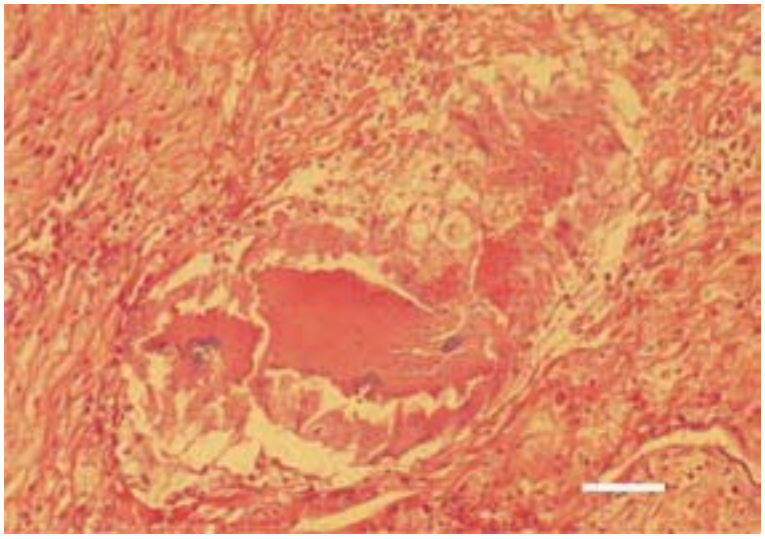


Fig. 8. Staphylococcal tenosynovitis. Serofibrinous exudate filling the synovial space, central necroses, with clusters of bacterial colonies among them. H/E, Bar = 25 μm.



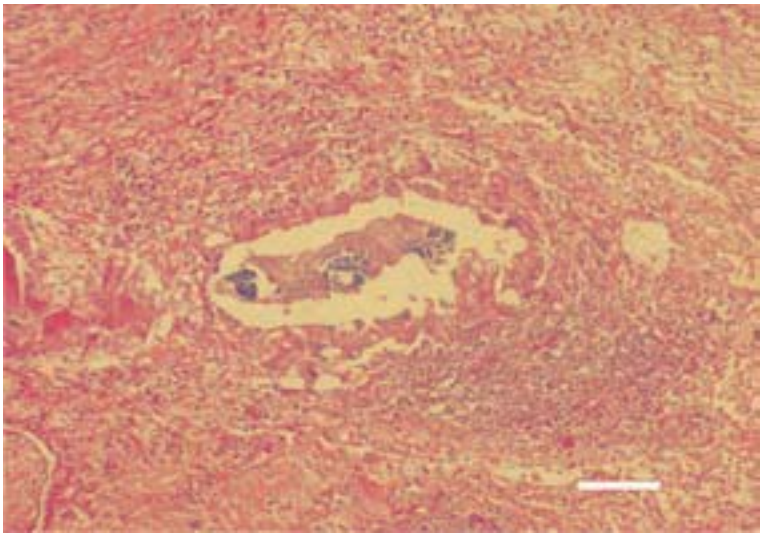


Fig. 9. Staphylococcal tenosynovitis. Central caseous necrotic lesions and clusters of bacterial colonies. Intensive inflammatory cell reaction (lymphocytes, granulocytes and macrophages) affecting the tendon sheath layers. H/E, Bar = 40 μm.

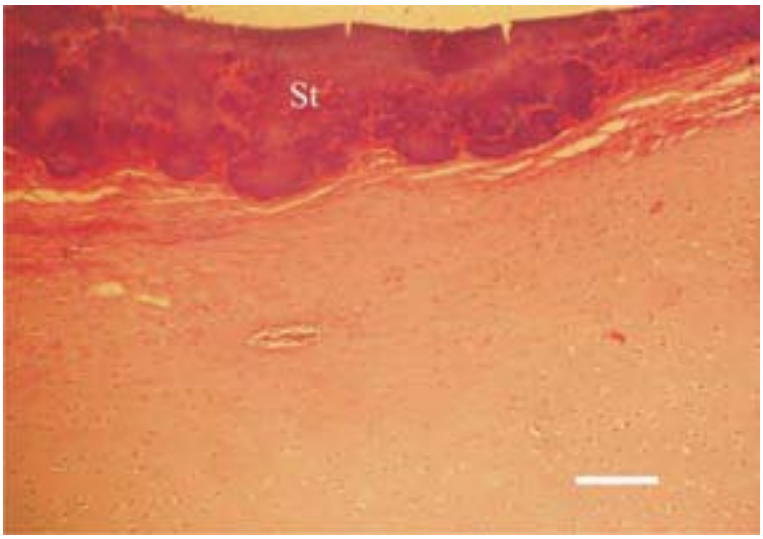


Fig. 10. Staphylococcal arthritis. Degenerative necrobiotic lesions and massive bacterial colonization (St) of the distal femoral articular cartilage in a growing male broiler breeder. H/E, Bar = 40 μm.

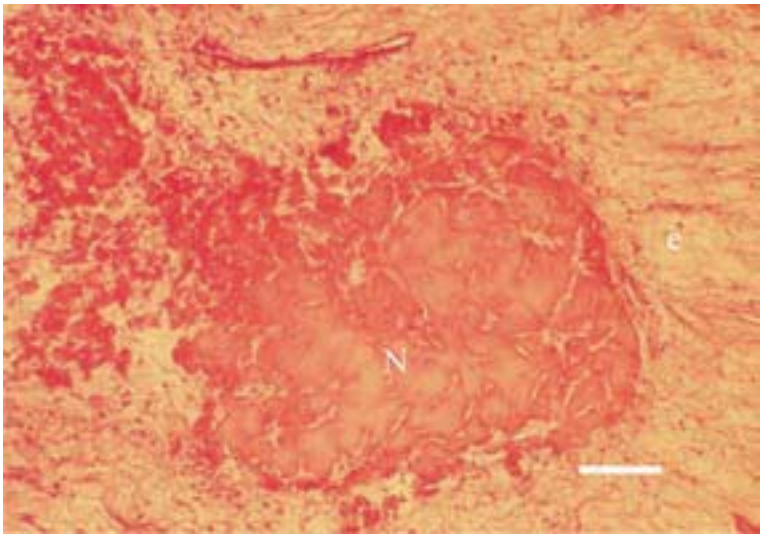


Fig. 11. Staphylococcal tenosynovitis. Axial necrosis (N) of a tendon and perifocal inflammatory oedema (e). H/E, Bar = 30 μm.

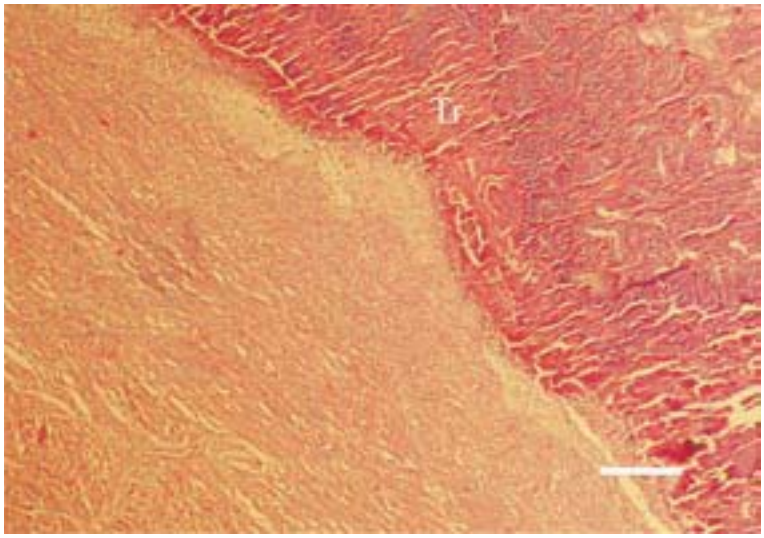


Fig. 1. Valve thromboendocarditis. Massive thrombotic masses (Tr) coating the mitral valve in the left heart side of a duck. It is generally associated with Streptococcus zooepidemicus. H/E, Bar = 40 μm.

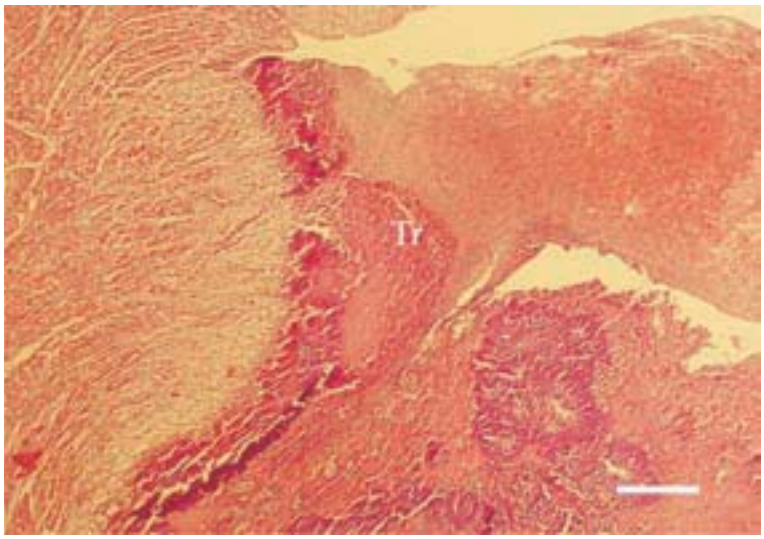


Fig. 2. Streptococcosis, duck. A mixed wall thrombus (Tr) partially occluding the left atrioventricular opening. H/E, Bar = 50 μm.

Fig. 3. Myocardial infarction (I) as a result of thromboemboly. Focal degenerative necrobiotic lesions and beginning of organization, appearance of macrophages. H/E, Bar = 25 μm.