SYLLABUS

Veterinary Pathology Department Wednesday Slide Conference 1982-1983

Armed Forces Institute of Pathology
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Syllabus

VETERINARY PATHOLOGY DEPARTMENT, AFIP,

WEDNESDAY SLIDE CONFERENCE

1982-1983

100 microslides

26 2x2 lantern slides

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PREFACE

The Department of Veterinary Pathology, Armed Forces Institute of Pathology, has conducted the annual Wednesday Slide Conference for more than two decades. The cases presented each Wednesday throughout the academic year are also distributed to over 100 active participants, including military and civilian veterinary pathologists throughout the United States and Canada, as well as many foreign countries. The list of active contributors continues to grow. The diagnosis for each case and a synopsis of the discussion for most cases are forwarded to participants weekly.

This study set has been assembled in an effort to make the material presented at our weekly conferences available to a wider range of interested pathologists and other scientists. Discussion and comments are abbreviated in some cases in this syllabus for succinctness.

A selection of 96 cases, including 100 microslides and 26 2x2 lantern slides, has been made from the 120 cases studied during the 1982-1983 conferences.

We wish to thank each contributor for his or her participation and for the permission to use cases in this study set.

LIST OF SLIDES

Slide number	Animal	Tissue	Diagnosis
1, 11-2	Lamb	Oral cavity	Contagious ecthyma
2	Ovine	Epididymis	Epididymitis caused by Brucella ovis
3, 13	Porcine	Stomach	Dyskeratotic gastritis
4	Gerbil	Sebaceous gland pad	Sebaceous adenoma
5	Baboon	Liver	Eosinophilic granuloma caused by Hepatocystis sp merocyst
6	Rat	Submaxillary salivary gland, lymph node	Sialoadenitis and lymphoid hyperplasia caused by sialodacryoadenitis virus (coronavirus)
7	Lamb	Kidney	Malignant catarrhal fever
8	Lamb	Ileum, lymph node, liver	Johne's disease (ovine paratuberculosis)
9	Pig	Stomach	Vitamin D toxicity
10	Cat	Liver	Necrotizing hepatitis caused by Francisella tularensis (Tularemia)

11, 14	Cat	Liver and unidentified tissue	Megakaryocytic myeloproliferative disease
12	Cat	Lung	Cartilaginous hamartomas
13	Dog	Eye	Endophthalmitis and retinal detachment caused by Prototheca sp
14, 15	Pig	Eye, adrenal gland	Keratoconjunctivitis and adrenal necrosis caused by pseudorabies virus
16	Dog	Eye	Chorioretinitis caused by Blastomyces dermatitidis
17	Dog	Eye	Cataract, retinal degeneration and asteroid hyalosis
18	Dog	Lung	Pulmonary blastoma
19, 15-6	Guinea pig	Lung	Bronchitis and bronchiolitis caused by an adenovirus
20	Rat	Lung	Necrotizing pneumonia caused by Corynebacterium kutcheri
21	Dog	Kidney	Immune complex glomerulonephritis
22	Rat	Liver	LGL leukemia, oval cell hyperplasia, bile duct hyperplasia. Neoplastic nodules, hepatocellular cytomegaly, eosinophilic foci of cellular alteration

23	Dog	Kidney	Atrophy and pyelonephritis: Dioctophyma renale
24	Bovine	Liver	Cirrhosis caused by aflatoxicosi
25, 17	Avian	Skin	Feather follicular epithelial ballooning degeneration caused a papovavirus
26, 18-9	Dog	Brain stem	Neoplastic reticulosis
27	Monkey	Cerebrum	Cerebral venous thrombosis
28	Monkey	Colon	Morbilliviral colitis
29	Dog	Small intestine	Ganglioneuroma
30	Cat	Lung, liver, kidney	Cytauxzoon sp parasitemia
31	Cat	Ear	Chondrosarcoma
32	Dog	Brain	Meningitis caused by Cryptococcus neoformans
33	Bovine	Spinal cord	Rabies viral myelitis
34, 110-11	Rabbit	Liver	Hepatic coccidiosis caused by Eimeria stiedai
35, 36	Foal	Small intestine	Adenomatosis
37	Cat	Cerebrum	Vasculitis caused by ethylene glycol toxicosis
38	Equine	Spinal cord	Leptomeningitis and poliomalacia caused by Strongylus sp
39	Rat	Accessory sex gland	Scirrhous adenocarcinoma

40, L12	Equine	Spinal cord	Leptomeningitis caused by equine infectious anemia (retrovirus)
41	Lamb	Salivary gland	Vitamin A deficiency
42	Boa constrictor	Kidney	Renal trematodiasis caused by Spathlodora sp
43	Rat	Spinal cord	Kilham rat viral myelopathy
44	Rat	Clitoral gland	Cystic papillary adenocarcinoma
45, L13	Baboon	Liver	Encysted pentastome nymph
46-47	Monkey	Liver, spleen	Hepatitis and splenitis caused by Yersinia pseudotuberculosis
48	Monkey	Colon	Mucinous adenocarcinoma
49	Rabbit	Kidney	Congenital polycystic disease
50	Bovine	Lung	Bronchopneumonia, Pasteurella multocida, bovine herpesvirus-1
51	Equine	Colon	Colitis caused by Salmonella sp
52	Alligator	Lung	Granulomas caused by Metarhizium anisopliae
53	Rat	Kidney	Nephroblastoma
54	Rabbit	Skin	Shope fibroma
55	Monkey	Lung, liver	Pyogranulomas caused by Mycobacterium tuberculosis
56	Orangutan	Ovary	Granulosa cell tumor
57	Avian	Liver	Hepatitis caused by Pasteurella multocida
58	Avian	Liver, spleen	Pacheco's disease (herpesvirus)

59	Monkey	Oral cavity	Squamous cell carcinoma
60	Equine	Brain	Nigropallidal encephalomalacia
61, L14	Equine	Brain	Encephalomeningitis caused by Listeria monocytogenes
62	Dog	Cerebrum	Choroid plexus papilloma
63	Dog	Liver	Hepatic necrosis caused by idiosyncratic drug reaction (mebendazole)
64, L15-16	Dog	Liver	Copper toxicity
65	Rat	External ear canal	Zymbal's gland carcinoma
66	Cow	Kidney	Hairy vetch toxicity
67	Wallaby	Small intestine	Protozoal enteritis caused by Eimeria sp
68	Goat	Skeletal muscle	Vitamin E-selenium deficiency myopathy
69	Equine	Periadrenal fat	Vitamin E-selenium deficiency syndrome
70	Dog	Mediastinum	Pyogranulomas caused by Coccidioides immitis
71	Dog	Cerebrum	Lead toxicity
72	Dog	Eye	Retrobulbar extracranial meningoma
73	Eagle	Lung	Necrogranulomas caused by Mycobacterium avium

74	Cockatoo	Liver	Psittacosis trematode eggs
75	Calf	Esophagus	Esophagitis caused by Candida albicans
76	Fish	Skin	Lymphocystis disease
77, L17-18	Dog	Skin	Neuroendocrine cell carcinoma (Merkel cell)
78	Fish	Skin	Cutaneous saprolegniasis
79	Dog	Tooth	Canine distemper virus, enamel organ
80	Kangaroo	Skin	Molluscum contagiosum-like dermatitis
81	Mouse	Small & large intestine	Luminal Hymenolepis sp, Spironucleus sp, Giardia sp and trichomonads; adenovirus
82-83	Foal	Liver	Tyzzer's disease
84	Equine	Uterus	Contagious equine metritis
85	Bovine	Small intestine, heart	Visceral blackleg (Clostridium chauvoei)
86	Cow	Mammary gland	Mastitis caused by Nocardia sp
87	Guinea pig	Urinary bladder	Transitional cell carcinoma
88	Rat	Adrenal gland	Pheochromocytoma
89	Mouse	Kidney, lymph node	Lymphocytic proliferation (S.L.E.)
90	Dog	Colon	Granulomatous colitis caused by Prototheca sp
91, L19	Cow	Lung	Contagious bovine pleuro-pneumonia
92	Avian	Lung, air sac	Pneumonia, airsacculitis and vasculitis caused by Aspergillus fumigatus
93	Rat	Oral cavity	Adenomatous odontogenic tumor with a Gorlin cyst
94-95, L20-23	Dog	Rib, kidney, mandible	Renal 2° hyperparathyroidism, progressive renal disease of Shih Tzus
96	Rat	Oral cavity	Complex odontoma with a Gorlin cyst
97, L24	Avian	Bone	Osteosarcoma
98	Equine	Skin	Equine sarcoid
99, L25-26	Calf	Lymph node, liver	Mesothelioma
100	Cat	Skeletal muscle	Fibrodysplasia ossificans progressiva

COMMENTARY ON SLIDES

Slide 1, 11-12

History. This lamb arrived at a laboratory when it was 2 days old and found to be normal upon thorough examination. The lamb received a left thoracotomy; the endotracheal tube and instruments were sterile. The investigators always wore gowns and gloves at surgery as well as during postsurgical care. Supplemental feedings were given via a stomach tube. T lamb showed a hemorrhagic oral exudate 17 days later. (1 gross photo and 1 provided.)

Diagnosis. Stomatitis, proliferative and necrohemorrhagic, subacute to chronic-active, multifocal, severe, oral cavity, ovine, etiology, poxvirus (contagious ecthyma).

Comment. The histologic lesions, particularly the downward epidermal proliferation and ballooning degeneration, along with the electron-microscop characteristics are typical of a poxvirus—most likely contagious ecthyma. Eosinophilic cytoplasmic inclusions are not a convincing feature of the lesi and are reported by Smith, Jones, and Hunt to be transitory and without established specificity. The exophytic gingival lesions seen in the gross photo are similar to those reported in Rocky Mountain bighorn sheep (Lance et al.).

This was an interesting case in that the original diagnosis was made by a M.D. and the transmission of disease to this lamb and to another one was via

Slide 4

History. A 2- to 2-1/2-year-old male gerbil was presented with a circumscribed (1.5 cm) raised mass that was firm and irregular on the skin surface. It was located on the midline ventrally in the region of the umbilicus. It was firm and white on cut surface.

Diagnosis. Adenoma, sebaceous, solitary, sebaceous gland pad, gerbil.

Comment. This neoplasm resembles a sebaceous epithelioma because of the presence of large numbers of undifferentiated basal cells that transition to mature sebaceous cells. Moulton's text places this classification between sebaceous adenoma and carcinoma based on the level of maturation displayed by cells, while stating that it more accurately reflects the true spectrum of sebaceous gland growths as well as standardizing the nomenclature used for man and domestic animals. The use of this classification for sebaceous gland growths in the gerbil could not be documented.

Initially, these neoplasms were called midventral marking gland neoplasms, with some being malignant. The glands are present in both males and females but are less developed in females. The gland increases in size in response to gonadal steroids.

The distinctive histologic feature of the sebaceous gland pad is its composite structure of 200 to 300 or more tubuloalveolar holocrine glands, each with its own duct. The hairs arising from follicles in the deeper portions of the gland have flattened curved surfaces resembling troughs and may contribute to the movement of sebum out of the duct and onto the surface of the glandular pad.

Contributor. Department of Pathology (VCS), Kansas State University Manhattan, Kansas.

Suggested reading.

Benitz, K. F., and Kramer, A. W., Jr.: Spontaneous tumors in the Mongoli gerbil. Lab. Anim. Care 15(5): 281-294, 1965.

Glenn, M. E., and Gray, J.: Effect of various hormones on the growth and histology of the gerbil (Meriones unguiculatus) abdominal sebaceous gland pad. Endocrinology 76: 1115-1123, 1964.

Moulton, J. E.: Tumors in Domestic Animals, ed. 2. Berkeley, University of California Press, 1978, pp. 53-54.

Rowe, S. E., Simmons, J. L., Pingler, D. H., et al.: Spontaneous neoplasms in aging gerbillinae. Vet. Pathol. 11: 38-51, 1974.

Vincent, A. L., and Ash, L. R.: Further observations on spontaneous neoplasms in the Mongolian gerbil, Meriones unguiculatus. Lab. Anim. Sci. 28(3): 297-300, 1978.

Vincent, A. L., Porter, D. D., and Ash, L. R.: Spontaneous lesions and parasites of the Mongolian gerbil, Meriones unguiculatus. Lab. Anim. Sci. 25(6): 711-722, 1975.

Vincent, A. L., Rodrick, G. E., and Sodeman, W. A., Jr.: The pathology of the Mongolian gerbil Meriones unguiculatus: A review. Lab. Anim. Sci. 29(5): 645-651, 1979.

Slide 5

History. An adult female baboon (Papio sp.) was put to death during the quarantine period because of a positive intrapalpebral tuberculin reaction. At necropsy, caseous granulomas were observed in the lungs and bronchial lymph

nodes. In addition, seven white granulomatous foci measuring 3 to 4 mm were randomly scattered on the hepatic capsule. Mycobacterium gordonae, Staphylococcus aureus, and Escherichia coli were cultured from the pulmonary and lymph nodal lesions.

Diagnosis. Granuloma, eosinophilic, solitary with central protozoal merocyst, liver, baboon (Papio sp.), nonhuman primate, etiology, Hepatocystis sp.

Comment. The morphologic characteristics of the merocysts are most compatible with Hepatocystis simiae, in accordance with the criteria reported by Garnham. These criteria include a multilocular mature merocyst that attains a size of 300 u and contains polyhedral crystals similar to those seen in this case. H. simiae granulomas are common incidental lesions in baboons. Another malarial parasite, H. kochi, causes similar lesions in African green monkeys (Cercopithecus pygerythus and C. aethiops).

Contributor. Comparative Pathology Section, National Institutes of Health, Bethesda, Maryland.

Suggested reading.

Garnham, P. C.: Malarial Parasites and Other Haemosporidia. Oxford, Blackwell Scientific Publications, 1966, pp. 856-861.

Krier, J. P.: Parasitic Protozoa. New York, Academic Press, 1977, vol. III, pp. 239-264.

Leathers, C. W.: The prevalence of Hepatocystis kochi in African green monkeys. Lab. Anim. Sci. 28: 186-189, 1978.

McConnell, E. E., Basson, P. A., de Vos, V., et al.: A survey of diseases among 100 free-ranging baboons (Papio ursinus) from the Kruger National Park. Onderstepoort J. Vet. Res. 41: 126-128, 1974.

Ruch, T. C.: Diseases of Laboratory Primates. Philadelphia, W. B. Saunders & Co., 1959, pp. 325-328.

Vickers, J. H.: Hepatocystis kochi in Cercopithecus monkeys. J. Am. Vet. Med. Assoc. 149: 906-908, 1966.

Slide 6

History. Control animal from a subacute toxicity study. Except for a swelling of the ventral neck, the young adult male Sprague-Dawley rat appeared healthy. On necropsy the ventral neck was observed to be swollen, with edema surrounding the submandibular salivary glands.

Diagnoses. 1) Sialoadenitis, necrotizing, subacute, multifocal, mild with severe periglandular inflammation, submaxillary salivary gland, Sprague-Dawley rat. 2) Hyperplasia, lymphoid, diffuse, mild, lymph node. Etiology, sialodacryoadenitis virus (coronavirus).

Comment. Foci of necrosis and acute inflammation involving ducts and acini are present in the submandibular (submaxillary) salivary gland. Lobules are surrounded and separated by edema, fibrin, neutrophils, lymphocytes, plasma cells, and macrophages. The parotid and one portion of the submandibular gland are not involved in this case: the sublingual salivary gland (present in some sections) is never involved.

Sialodacryoadenitis virus is highly epitheliotropic, but replication in the respiratory tract is largely limited to mucosal epithelium (Jacoby et al.). This contrasts with the interstitial pneumonitis induced by rat coronavirus. The mode of viral spread from the respiratory tract to salivary and lacrimal glands is apparently unknown as is the reason for the sublingual gland remaining uniformly free of lesions and viral antigens (Jacoby et al.). The lesion was staged as subacute because of the presence of ductal squamous metaplasia combined with the mixed inflammatory response.

Contributor. Schering Corporation, P. O. Box 32, Lafayette, New Jersey.

Suggested reading.

Bhatt, P. N., Percy, D. H., and Jonas, A. M.: Characterization of the virus of sialodacryoadenitis of rats: a member of the coronavirus group. J. Infect. Dis. 126(2): 123-130, 1972.

Hunt, R. D.: Dacryoadenitis in the Sprague-Dawley rat. Am. J. Vet. Res. 24(100): 638-641, 1963.

Jacoby, R. O., Bhatt, P. N., and Jonas, A. M.: Pathogenesis of sialodacryoadenitis in gnotobiotic rats. Vet. Pathol. 12: 196-209, 1975.

Jonas, A. M., Croft, J., Black, L., et al.: Sialodacryoadenitis in the rat. Arch. Pathol. Lab. Med. 88: 611-622, 1969.

Slide 7

History. This 3- to 4-month-old white-faced, mixed-breed female lamb had lost weight during the last month of its life and had been dyspneic, anorexic, and pyrexic with temperatures up to 105.2°F several days prior to death. It had been treated parenterally with penicillin and tylosin. The ventral lobes

of the lungs were firm and "liverlike," and there were dark-red patchy areas in the dorsal parts of the lungs. Purulent exudate was in the trachea and bronchi. The liver had enlarged bile ducts visible on capsular and cut surfaces. The kidneys appeared large and "grossly abnormal."

Diagnosis. Arteritis, granulomatous, with intimal proliferation, diffuse, severe, muscular arterioles, kidney, mixed breed, ovine, etiology, compatible with malignant catarrhal fever (MCF).

Comment. This tissue was taken from one of two unrelated lambs from widely separated locations that had similar lesions. Both lambs had lymphoid vasculitis and arterial fibrinoid necrosis in their livers, lungs, and urinary bladders as well as in their kidneys. The livers also showed biliary hyperplasia, and acute fibrinopurulent pneumonia was present in both lambs. Epithelial degeneration and lymphocytic infiltration of the urinary bladders of both lambs was marked. The lesions in these two sheep parallel those of MCF in cattle. Portions of renal pelvis (not present on all slides) have a mononuclear inflammatory infiltrate, and there is an interstitial nephritis that is considered to be related to the primary lesion. The arterial lesions are suggestive of a cell-mediated immune pathologic process much like those seen with periarteritis nodosa. A PTAH stain demonstrated a fibrinoid material within the walls of some of the affected arteries. Wildebeests are asymptomatic carriers of MCF in Africa, and a wild animal park with wildebeests was located about 20 miles from one of the farms. Border disease is considered a primary differential diagnosis.

Sheep are suspected of serving as reservoirs of MCF of cattle but are not considered susceptible to the disease. Experimental induction of MCF in sheep has recently been reported. A report of polyarteritis in sheep in 1959 describes lesions compatible with MCF.

Contributor. School of Veterinary Medicine, Oregon State University, Corvallis, Oregon.

Suggested reading.

Castro, A. E., and Daley, G. G.: Electron microscopic study of the African strain of malignant catarrhal fever virus in bovine cell cultures. Am. J. Vet. Res. 43(4): 576-582, 1982.

Helmboldt, C. F., Jungher, E. L., and Hwang, J.: Polyarteritis in sheep. J. Am. Vet. Med. Assoc. 134: 556-561, 1959.

Kalunda, M., Ferris, D. H., Dardiri, A. H., et al.: Malignant catarrhal fever. III. Experimental infection of sheep, domestic rabbits, and laboratory animals with malignant catarrhal fever virus. Can. J. Comp. Med. 45: 310-314, 1981.

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Liggitt, H. D., and DeMartini, J. C.: The pathomorphology of malignant catarrhal fever. II. Multisystemic epithelial lesions. Vet. Pathol. 17: 73-83, 1980.

Piercy, S. E.: Studies in bovine malignant catarrh. V. The role of sheep in the transmission of the disease. Br. Vet. J. 39: 508-516, 1954.

Schmitz, J. A., and Grumbein, S. L.: Two possible cases of malignant catarrhal fever in sheep. Proceedings of the American Association of Veterinary Laboratory Diagnosticians 24: 61-66, 1981.

Slide 8

History. Tissues from a Dorper lamb. The lamb had a history of chronic weight loss but otherwise appeared normal. Other lambs in the flock were not losing weight. The lamb was put to death.

Diagnoses. Ileitis, granulomatous, diffuse, moderate to severe, ileum, Dorper, ovine. Lymphadenitis, granulomatous, diffuse, moderate to severe, lymph node. Hepatitis, granulomatous, multifocal, mild to moderate, liver. Lymphangitis, granulomatous, with lymphangiectasia, segmental, moderate to severe, lymph node and ileum. Etiology, Mycobacterium paratuberculosis (Johne's disease).

Comment. Acid-fast stained sections showed numerous organisms within macrophages in the lamina propria. A few bacilli were also present in macrophages in the mesenteric lymph node and liver. The lamb was never noted to have diarrhea. Wasting in the absence of diarrhea in sheep with paratuberculosis has been described and attributed to protein loss into the intestinal lumen. Such a mechanism is probable in this case, especially in view of the marked submucosal lymphangiectasia (apparently the result of the obliterating granulomatous lymphangitis best seen in the serosa).

Although the presence of modest numbers of acid-fast organisms along with the histologic lesions are considered typical of ovine paratuberculosis (Johne's disease), for a definitive diagnosis a culture of the organism is required. The gross intestinal lesions can be easily missed due to the diffuse nature of the mural thickening.

Contributor. Onderstepoort Veterinary Research Institute, Onderstepoort 0110, Republic of South Africa.

Suggested reading.

Allen, W. M., Bennet, S., and Patterson, D. S. P.: A biochemical study of experimental Johne's disease. I. Plasma protein leakage into the intestine of sheep. J. Comp. Pathol. 84: 381-384, 1974.

Fodstad, F. H., and Gunnarsson, E.: Postmortem examination in the diagnosis of Johne's disease in goats. Acta Vet. Scand. 20: 157-167, 1979.

Newsholme, S. J., and Fletcher, J. M.: A mycobacteriosis in a sheep resembling paratuberculosis (Johne's disease). J. S. Afr. Vet. Assoc. 52(2): 143-145, 1981.

Slide 9

History. A group of mixed breed pigs, weighing approximately 50 kg, became ill within 1 day after being fed from a new batch of feed. Many pigs vomited, but none had diarrhea. Tissues are from a pig that died after consuming the feed for 2 days.

Gross Pathology. There was hemorrhage from the fundic portion of the gastric mucosa. The lungs were diffusely congested and somewhat firm and wet. The kidneys were slightly mottled.

Diagnosis. Necrosis and mineralization, diffuse, mild to moderate, mid-zonal mucosa, fundus, stomach, porcine, etiology, compatible with vitamin D toxicity.

Comment. A small amount of mineralization is present throughout the submucosa and muscular tunics; however, special stains dramatically accentuate the predominance of the mid-zonal mucosal mineralization.

Analysis of the feed showed 26,000 units vitamin D_3/kg (NRC requirements, 125 to 200 u/kg). Mineralization was extensive in the lung, stomach, and kidney of pigs that died after consuming the feed for 4 days. Many of the pigs ate very little of the feed. These pigs survived, but reportedly "never did well."

Excess doses of vitamin D are well tolerated; it takes an extreme overdose to induce toxicity. Raschek et al. state that degeneration is the primary soft tissue lesion in vitamin D_3 toxicosis, but the mechanism by which an excess of vitamin D_3 exerts its toxic effect and initiates dystrophic calcification is not known.

The microscopic differential diagnosis is uremic gastropathy.

Contributor. Animal Disease Diagnostic Laboratory, Purdue University, West Lafayette, Indiana.

Suggested reading.

Raschek, W. M., Krook, L., Kallfelz, F. A., et al.: Vitamin D toxicity. Initial site and mode of action. Cornell Vet. 68: 324-364, 1978.

Howard, J. L., (Ed.): Current Veterinary Therapy Food Animal Practice. Philadelphia, W. B. Saunders Co., 1981, pp. 303, 332-333.

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Whitehair, C. K., and Miller, E. R.: Nutritional deficiencies. Vitamin D. In Diseases of Swine, ed. 4. Ames, Iowa State University Press 1978, pp. 1103-1104.

Slide 10

History. Three cats on a farm in southeast Georgia became sick and died within a 2-week period. One dead and one live cat were presented to a local practitioner. The live cat was anemic, debilitated, and hypothermic. Postmortem examinations were conducted by the practitioner, and frozen tissue specimens were submitted to the diagnostic laboratory. Multiple white foci, measuring 1 to 3 mm in diameter, were present on capsular and cut surfaces of the liver and spleen.

Laboratory Results. Pasteurella multocida was cultured from the lung, while no growth was obtained from the liver, spleen, and kidney. Francisella tularensis was identified in the liver with fluorescent antibody by the Center for Disease Control, Atlanta.

Diagnosis. Hepatitis, necrotizing, acute, multifocal, moderate, liver, feline, etiology--Francisella tularensis.

Comment. Numerous gram-negative coccobacilli are present in Brown & Hopp's-stained sections. The owner of these cats developed a small ulcerative lesion on the thumb and had a fever and mildly painful axillary lymphadenopathy. Illness was severe enough to warrant hospitalization. Cat scratch fever was initially suspected, but a high antibody titer to Francisella tularensis was later demonstrated. Differential diagnoses for the cat include Tyzzer's disease, salmonellosis, listeriosis, and toxoplasmosis.

Contributor. Veterinary Diagnostic Laboratory, P. O. Box 1389, Tifton, Georgia.

Suggested reading.

Gillespie, J. H., and Timoney, J. F.: Hagan and Bruner's Infectious Diseases of Domestic Animals. Ithaca, Cornell University Press, 1981, pp. 124-125.

Jubb, K. V., and Kennedy, P. C.: Pathology of Domestic Animals. New York, Academic Press, 1970, Vol. 1, p. 179.

Tularemia associated with domestic cats - Georgia, New Mexico. CDC Morbidity and Mortality Weekly Report 34(4): 39-41 (5 Feb.) 1982.

Slide 11, 14

History. A 3-year-old spayed female cat was presented to a practicing veterinarian because of generalized lymph node enlargement. There was no

evidence of depression, weakness, inappetence, or fever. Enlarged lymph nodes, spleen, and liver were noted at necropsy. (Factor VIII photo included.)

Diagnosis. Myeloproliferative disease with megakaryocytic differentiation, liver and unidentified tissue, feline.

Comment. Histopathologic examination of the spleen and bone marrow revealed similar changes as those observed in the initial biopsy specimen taken from the lymph node. Parenchyma of both marrow and spleen were replaced by a diffuse hematopoietic neoplasm which consisted predominately of the megakaryocytic tumor cells. Periportal areas in the liver and interstitium of the kidney were infiltrated by predominately megakaryocytic-megablastic cells. Blood smears of the cat contained blast cells, some of hich had pseudopodlike processes that had the staining characteristics of platelets in the smear.

In the cat, lymphoproliferative disorders outrank myeloproliferative disease by a ratio of 20:1. Granulocytic leukemia occurs less frequently, while only three cases of megakaryocytic proliferation have been described in bone marrow neoplasia.

The use of immunoperoxidase localization of Factor VIII to identify megakaryocytes and immature megakaryocytic precursors in a case of human megakaryocytic leukemia has been recently reported. This technique was used to identify part of the blast cell population in this case using a peroxidase antiperoxidase method with an antihuman Factor VIII antibody. Factor VIII is

synthesized in large quantities by endothelial cells and megakaryocytes. The localization of Factor VIII in both cell types has been demonstrated by immunochemical techniques.

Contributor. C. E. Kord Animal Disease Diagnostic Laboratory, Division of Animal Industries, Nashville, Tennessee.

Suggested reading.

Burgdorf, W. H. C., Nakai, K., and Rasai, J.: Immunohistochemical identification of Factor VIII-related antigen in endothelial cells of cutaneous lesions of alleged vascular nature. Am. J. Clin. Pathol. 75: 167-171, 1981.

Harvey, J. W.: Myeloproliferative disorders in dogs and cats. Vet. Clin. North Am. [Small Anim. Pract.] 11(2): 349-361, 1981.

Innes, D. J., Mills, S. E., and Walker, G. K.: Megakaryocytic leukemia. Identification utilizing anti-Factor VIII immunoperoxidase. Am. J. Clin. Pathol. 77: 107-110, 1982.

Michel, R. L., O'Handley, P., and Dade, A. W.: Megakaryocytic myelosis in a cat. J. Am. Vet. Med. Assoc. 168: 1021-1025, 1976.

Nakai, K., Rasai, J., and Burgdorf, W. C.: Localization of Factor VIII related antigen in vascular endothelial cells using an immunoperoxidase method. Am. J. Surg. Pathol. 4: 273-276, 1980.

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Sutton, R. H., McKellow, A. M., Bottrill, M.: Myeloproliferative disease in the cat: a granulocytic and megakaryocytic disorder. N.Z. Vet. J. 26(11): 273-274, 1978.

History. A 5-month-old male domestic cat was presented with a history of respiratory distress for about two weeks. The cat was severely dyspneic. Radiographs revealed numerous small radiopaque nodules in the right lung.

Diagnosis. Hamartoma, cartilagenous, multiple, moderate to severe, lung, feline.

Comment. The lung lesions are composed of varying numbers of cartilagenous plaques that are separated from one another by clefts or spaces lined by a respiratory epithelium ranging in shape from low cuboidal to pseudostratified columnar. Calcification is seen in several of the cartilagenous plaques. These microscopic findings are similar to those reported for pulmonary chondromatous hamartoma in man. In man, however, the condition is usually characterized by a solitary mass in the lung.

Contributor. Department of Pathology & Microbiology, University of Montreal, St. Hyacinthe, QC J2S 7C6, Canada.

Suggested reading.

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Slide 13

History. This crossbred adult male dog had thin bloody stools of one month's duration. The dog did not respond to treatment. One week before being put to death, the dog appeared bilaterally blind.

Gross Pathology. The colon was markedly thickened and had patchy congested areas in the mucosa. The distal ileum was also thickened but to a lesser degree. Both retinas were detached due to the accumulation of a thick, white-to-pale yellow exudate between the retina and choroid.

Diagnosis. Endophthalmitis, granulomatous, severe, with diffuse retinal detachment and degeneration, eye, canine, etiology—Prototheca sp.

Comment. Protothecal organisms and granulomatous inflammation are present in the vitreous, retina, and choroid. This inflammation also involves the ciliary body. A minimal keratitis is present in some sections. A diagnosis of endophthalmitis is based on Yanoff and Fine's definition, which states that the inflammation must involve one or more coats of the eye and adjacent cavities. The wall of the organism and daughter cells are birefringent. Species identification would require immunofluorescence in the absence of cultural studies (Migaki et al.).

Prototheca organisms were also present in the colon, ileum, mesenteric lymph node, kidney, and heart. Organisms were most abundant in the submucosa of the colon and in the exudate in the eyes. These stained well with PAS and GMS stains.

Contributor. Department of Veterinary Pathology, College of Veterinary Medicine, Texas A&M University, College Station, TExas.

Suggested reading.

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Slides 14-15

History. A fatal disease was found on a closed pig farm. The disease occurred among both suckling pigs and sows. In suckling pigs, the disease followed an acute course and spread rapidly. Symptoms included anorexia, depression, diarrhea or constipation, and staggering gait. Death occurred only in suckling pigs. This case is a 14-day-old male Duroc-Jersey.

Gross Pathology. There were multiple areas of necrosis in the liver, spleen, adrenal glands, and tonsils. The eyes had a discharge.

Laboratory Results. The total leukocyte numbers in the affected pigs remained in the normal range. Pseudorabies virus was isolated from the liver, spleen, adrenal gland, and brain.

Diagnoses. Keratoconjunctivitis, necrotizing and ulcerative, chronic, diffuse, severe, cornea, Duroc-Jersey, porcine. Necrosis, acute, focal, mild to moderate, adrenal gland. Etiology, pseudorabies virus (herpesvirus).

Comment. The lesions present in the adrenal gland and eye are compatible with a diagnosis of pseudorabies. Some sections of adrenal gland contain small numbers of inflammatory cells associated with the necrosis. Typical herpetic intranuclear inclusions are also present. In piglets dying of the natural infection, there may be focal necrosis in the liver, spleen, and adrenals (Jubb and Kennedy).

According to Gelatt, experimentally induced pseudorabies keratoconjunctivitis in the pig resembles the deeper forms of herpes simplex keratitis in man and rabbit. The corneal ulcers are initially punctate but spread rapidly. Healing is indicated by vascularization, scarring, and re-epithelialization (Gelatt).

Contributor. Animal Industry Research Institute, TSC, No. 1 Tapu Chunan, Miaoli, Taiwan 350, Republic of China.

Suggested reading.

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Slide 16

History. This 2-year-old male sheep dog was presented with bilateral blepharospasms, inflamed conjunctiva, and a brief history of pawing at both eyes.

Laboratory Results. Radiographs revealed consolidation of the left middle lung lobe. Lung aspiration demonstrated blastomycosis.

Diagnosis. Chorioretinitis, with nontapetal retinal detachment, pyogranulomatous, subacute to chronic, segmental, moderate to severe, eye, sheep dog, canine, etiology, Blastomyces dermatitidis.

Comment. The pyogranulomatous inflammatory response present in this eye characteristic of ocular blastomycosis. The retinal detachment probably resulted from subretinal exudates. The multiple cysts within the ciliary bod are a nonspecific change. Although readily visible on the H&E-stained sectio a PAS stain clearly demonstrates the thick-walled PAS-positive yeast forms th are generally 6 u to 15 u in diameter. Single wide-based budding of the yeas forms and multiple nuclei are present.

Contributor. College of Veterinary Medicine, University of Illinois, Urbana, Illinois.

Suggested reading.

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Slide 17

History. This tissue is from an overweight, 10 and 1/2-year-old, castrate male German shepherd that was put to death.

Diagnoses. Cataract, cortical, diffuse, moderate, lens, eye, German shepherd, canine. Degeneration, retinal, chronic, diffuse, severe, with tapetal retinal detachment, eye. Asteroid hyalosis, moderate, vitreous body, eye.

Comment. An etiology for the retinal degeneration and cataracts could not be demonstrated. There is multifocal mineralization of lens fibers in most sections. Asteroid hyalosis is a common degenerative change that occurs in middle-aged or older dogs (Rubin). These birefringent asteroid bodies are composed of a calcium lipid complex and remain suspended in the vitreous humor (Rubin) unlike the free-floating, glittering cholesterol crystals seen with synchysis scintillans.

Contributor. Wilford Hall USAF Medical Center/SGVM, Lackland AFB, Texas.

Suggested reading.

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Slide 18

History. A 4-month-old mixed-breed male canine was presented with a sudden onset of congestive heart failure, pulmonary edema, right atrial dilation, and pleural effusion. No heart sounds were audible on the right side. A loud systolic murmur was detected high on the left side at the 5th intercostal space.

Necropsy Results. A 12-cm mass was found in the right apical lung lobe. On cut surface, the mass was solid, white, and fibrous with areas of cartilage. There was metastasis to other lung lobes and hilar lymph nodes.

Diagnosis. Blastoma, pulmonary, lung, canine.

Comment. According to Spencer, a pulmonary blastoma is growth originating from a pluripotent embryonic cell of one germ layer (mesoderm) that can be distinguished from hamartomas of single germ layer derivation because of their progressive and sometimes unlimited growth. Some investigators contend that the tumor is derived from both entodermal and mesodermal cells, while still

others consider the tumor to be derived solely from entodermal cells. Tamai et al. suggest that a metaplastic change of the neoplastic entodermal cell or trans-germ-layer differentiation (dysdifferentiation) occurs in pulmonary blastoma. Pulmonary blastoma may be an inappropriate term but remains in use out of convention.

The bronchiolelike structures are well-developed and appear to be trapped in the proliferating spindle cell component rather than being lined by neoplastic epithelium. The lung parenchyma at the margin of the tumor is compressed, and there is a mild diffuse interstitial pneumonia. The arterial wall thickening seen in this portion of the lung may be related to pulmonary hypertension.

Contributor. Laboratory of Pathology, School of Veterinary Medicine, University of Pennsylvania, Philadelphia, Pennsylvania.

Suggested reading.

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Slide 19, 15-16

History. Two adult female Hartley (Crl:HA/BR) guinea pigs being used in anthrax vaccine trails showed clinical signs of hunched posture; weight loss; rough hair coat; rapid, labored breathing; and catarrhal exudate around the external nares and on the front feet. One animal died after the fifth vaccine injection; the other, a control, was moribund and was killed.

Gross Pathology. A catarrhal exudate was found throughout the upper respiratory tract and external nares. All lung lobes remained partially distended with well-delineated reddish foci of varying sizes. No other significant gross lesions were noted. (PM included.)

Laboratory Results. Aerobic bacterial cultures of the lung, liver, and middle ears produced no growth. No pathogenic species were isolated from bacterial cultures of proximal trachea or pharyngeal swabs.

Diagnosis. Bronchiolitis and bronchitis, proliferative and necrotizing, subacute, diffuse, moderate to severe, lung, guinea pig, etiology—adenovirus.

Comment. There is a diffuse interstitial pneumonia that radiates from the bronchioles and bronchi. The epithelial proliferation indicates a reparative process in addition to the necrosis. There are some foci of early squamous metaplasia in the bronchiolar epithelium.

Ultrastructurally, viral particles were observed in intranuclear inclusion bodies of both sloughed and intact bronchiolar epithelial cells. The particles were primarily confined to nuclei with intact ruptured nuclear membranes. Virions were arranged individually and in crystalline arrays in the nuclei. In addition to the virions, small aggregates of electron-dense material of unknown composition were observed in the nuclei of all infected cells. Viral particles ranging in size between 68 and 72 nm were seen in different forms, varying only in density of the virion.

The primary differential diagnosis is a herpesvirus infection. On electron-microscopic examination, adenovirus particles have a more prominent hexagonal shape than herpesvirus and have surface projections (penton fibers) that hold them together to form paracrystalline arrays. Budding of the nuclear membrane would be expected with herpesviruses. Herpesviruses can form arrays, but the surfaces of the virions are smooth, lacking penton fibers.

Contributor. Pathology Division, US Army Medical Research Institute of Infectious Disease, Fort Detrick, Maryland.

Suggested reading.

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Slide 20

History. Several Sprague-Dawley rats in a colony exhibited rales, rough hair coat, and diarrhea. On necropsy, their lungs were observed to be mottled red and white with firm areas.

Laboratory Results. Corynebacterium kutscheri was isolated from the lungs.

Diagnosis. Pneumonia, necrotizing, acute to subacute, multifocal and coalescing, severe, lung, rat, etiology—Corynebacterium kutscheri.

Comment. This case exhibits lung lesions considered typical of Corynebacterium kutscheri in rats. Numerous gram-positive rods were demonstrated by a Brown-Brenn tissue Gram stain. The distribution of the pneumonia is considered to result from embolic foci that enlarge and coalesce. The involvement of bronchioles or bronchi occurs late in the disease process.

Contributor. Midwest Research Institute, Kansas City, Missouri.

Suggested reading.

Ford, T. M., and Jolinter, G. N.: Pneumonia in a rat associated with Corynebacterium pseudotuberculosis. A case report and literature survey. Lab. Anim. Care 18: 220-223, 1968.

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Slide 21

History. This young adult male mixed-breed canine was given 250 ml of 7% human serum albumin by rapid infusion through a catheterized cephalic vein. Twenty-one days later it was presented in a moribund condition. (EM included.)

Gross Pathology. Gross necropsy revealed a focal ulcer on the tongue and diffuse serosal reddening of the GI tract. The gastrointestinal mucosa was mottled red and white.

Laboratory Results.

WBC - 32,400 (S - 68%, L - 26%, M - 4%, B - 2%).
BUN - 188.5 mg/DL.

Diagnosis. Glomerulonephritis, proliferative, diffuse, moderate to severe, kidney, canine.

Comment. The light and electron microscopic changes are compatible with a proliferative glomerulonephritis caused by immune complex disease. The EM isolachrone demonstrates endothelial swelling and swelling of foot processes along with the deposition of material compatible with immune complexes within a slit pore and in subepithelial locations. The H&E stain and a PAMS stain 1-u

thick demonstrate a large number of mononuclear cells in interstitial capillaries and glomerular capillaries. The PAMS did not show glomerular basement membrane thickening.

Recent literature indicates that in situ immune complex formation is an important mechanism in the genesis of subepithelial deposits and that insoluble complexes play no part in their generation (Cameron et al.).

Contributor. Department of Pathology, Letterman Army Institute of Research, Presidio of San Francisco, California.

Suggested reading.

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Slide 22

History. This 15-month-old F344 male rat was given a commonly used anticonvulsant in the drinking water for 166 days and lost 100 g over several weeks.

Gross Pathology. The spleen was enlarged many times the normal size. The liver had several foci, nodules, and masses, the largest of which was 1.3 cm in diameter.

Diagnoses. Leukemia, mononuclear cell, liver, F344 rat. Hepatocellular eosinophilic foci of cellular alteration, multifocal, severe, liver. Hepatocellular cytomegaly, multifocal, severe, liver. Neoplastic nodules, hepatocellular, multiple, liver. Bile duct hyperplasia with fibrosis, multifocal, moderate, liver. Oval cell hyperplasia, multifocal, mild to moderate, liver.

Comment. Mononuclear cell leukemia is a major cause of death in aged F344 rats. Morphologically, the leukemic cells resemble large granular lymphocytes (LGL) of rats and humans, which have been demonstrated to have natural killer cell activity (Ward et al.). Aging F344 rats frequently have increased numbers of bile ducts in portal areas with or without fibrosis. The oval cell hyperplasia is thought to originate from the epithelium of the canals of Hering (bile ductules) and is a common response to hepatocarcinogens (J.N.C.I., vol. 64). Eosinophilic foci of cellular alteration are considered to be stages in the development of a hepatocarcinoma, as they are cytologically similar to the cellular elements of the neoplastic nodules and may be a part of a spectrum progressing toward the nodule (J.N.C.I., vol. 64). The eosinophilic foci and

nodules in the liver were induced by phenobarbital. Many of the nonneoplastic liver lesions are associated with the leukemia. The cytomegaly is a toxic lesion induced by phenobarbital.

Contributor. Laboratory of Comparative Carcinogenesis, National Cancer Institute, Frederick, Maryland.

Suggested reading.

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Slide 23

History. An adult male beagle appeared to be in good health and was kill by exsanguination under sodium pentobarbital anesthesia.

Gross Pathology. The right kidney was grossly enlarged. The cortex was thin, and the dilated pelvis was filled with giant kidney worms (Dioctophyma

renale). The worms ranged from approximately 20 to 100 cm in length and 0.2 to 1.0 cm in diameter. Worms were also present in the peritoneal cavity.

Diagnosis. Atrophy and pyelonephritis, chronic, diffuse, severe with Dioctophyaidae eggs, kidney, beagle, canine. Dioctophyme renale, transverse section.

Comment. The morphologic changes in this kidney are compatible with chronic hydronephrosis caused by Dioctophyme renale. Throughout the remaining renal parenchyma are varying densities of eosinophils, neutrophils, macrophages, and multinucleated giant cells. There is a chronic fibrosing capsulitis along with supracapsular granulomatous inflammation. Extracapsular granulomas contain parasite eggs with bipolar caps. Within the interstitial fibrosis are a few remaining tubules and occasional parasite eggs. The glomeruli remain, for their blood supply is greater than that of the tubules.

This aphasmid is characterized by its large diameter; coelomyarian, polyayarian musculature of uniform height; a large triradiate glandular esophagus; a large ventral nerve cord and multiple smaller accessory cords; no obvious lateral cords; no bacillary bands; a thick cuticle; long intestinal microvilli; and the presence of a mesentery (Chitwood et al.). The characteristic morphology of the eggs is also an aid in identification.

This is the largest nematode. It has a complex life cycle (eggs passed in host's urine; L1 hatches in annalid, Cambarincola chirocephala—commensal on crayfish; L1 encyst in crayfish; L3 - L4 encyst in bull head; bull head ingested by carnivore host) and has a predilection for the right kidney. This

nematode is primarily a parasite of fish-eating carnivores (dog, fox, otter), pole cat, marten, mink, and weasel), but it has also been found in cattle, pigs, horses, and man.

The exact mechanism by which D. renale causes progressive destruction of the kidneys is not clear, but hydronephrosis and/or pyelonephritis are thought to play an important role (Osborne et al.).

Contributor. Department of Safety Assessment, Merck Sharpe & Dohme Research Laboratory, West Point, Pennsylvania.

Suggested reading.

Chitwood, M., and Lichtenfels, J. R.: Identification of parasitic metazoa in tissue sections. Exp. Parasitol. 32: 511, 1972.

Fernando, S. S. E., and Jelihovsky, T.: The giant kidney worm. Pathology 12(2): 292-293, 1980.

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Osborne, C. A., Stevens, J. B., Hanlon, G. F., et al.: Dioctophyme renale in the dog. J. Am. Vet. Med. Assoc. 155(4): 605-620, 1969.

Slide 24

History. One week prior to death this 1 and 1/2-year-old Holstein heifer was noted to be straining, anxious, and isolated from the other animals. She appeared to be disoriented and staggered. She fell into a creek the day before her death; the following day she was found down and later died. Another heifer

died with similar signs about one month earlier. The animals were fed a mixture of ground oats, corn, and a dairy feed.

Gross Pathology. The abdomen contained a large volume of straw-colored fluid. The intestinal tract and mesentery were extremely edematous, particularly the colon. The liver was firm, and mesenteric lymph nodes were enlarged.

Laboratory Results. The corn was analyzed for aflatoxin and was found to contain 21 to 100 ppb. The oats and dairy feed contained none (detection level is 15 ppb).

Diagnosis. Cirrhosis, diffuse, severe, liver, Holstein, bovine, etiology, compatible with aflatoxicosis.

Comment. The most important histopathologic finding in this liver is the fibrosis that occludes sinusoids and central veins producing what is called veno-occlusive disease. Portal hypertension occurs followed by intestinal and mesenteric edema, as seen in this case.

Veno-occlusive disease is considered to be the diagnostic feature of pyrrolizidine alkaloid toxicity in cattle and horses. It is also reported in chronic aflatoxicosis of cattle. Plants containing pyrrolizidine alkaloids were not present in this heifer's feed. The presence of 21 to 100 ppb aflatoxin in the corn suggested a diagnosis of chronic aflatoxicosis. Aflatoxin levels can vary radically within a feed, as the production of the toxin has strict temperature and moisture requirements and the toxin is

inactivated by light. It is likely that the sampling in this case was not representative; this could account for a lower level of aflatoxin than was actually present in the feed consumed by the animal.

Veno-occlusive disease is a common feature of chronic pyrrolizidine alkaloid toxicity and chronic aflatoxicosis in cattle and horses. In cattle the histopathology of the liver is identical in both diseases. Hepatocytic megalocytosis is an additional feature of pyrrolizidine alkaloid toxicity not reported to occur in chronic aflatoxicosis of the horse. This feature can be used to distinguish the two diseases on histopathologic examination. Veno-occlusion may not be a specific response in either the cow or the horse but rather may represent a general response to injury, a response that may be produced by a variety of hepatotoxins.

Contributor. Department of Pathology, Virginia-Maryland Regional College of Veterinary Medicine, Virginia Polytechnical Institute, Blacksburg, Virginia

Suggested reading.

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Guthrie, L. D., and Bedell, D. M.: Effects of aflatoxin in corn on production and reproduction in dairy cattle. Proc. Annu. U.S. Anim. Health Assoc. 83: 202-204, 1979.

Heathery, M. W.: Aflatoxicosis and zinc metabolism in dairy calves. J. Dairy Sci. 64 (Suppl. 1): 145, 1980.

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Slide 25, L7

History. A group of fledgling budgerigars (Melopsittacus undulatus) about 3 to 4 weeks of age, raised commercially, had a very high mortality rate at about 2 to 4 weeks of age.

Gross Pathology. Emaciation, anemia, and feather abnormalities were the most frequent findings. Some birds had abdominal fluid and grossly evident foci of necrosis in internal organs, particularly the liver. (See gross kodachrome.)

Laboratory Results. Significant bacteria were not recovered. Initial attempts to recover a virus were not successful. Papovaviruslike particles, about 45 nm in diameter, were found in the intranuclear inclusions of feather follicle epithelium.

Diagnosis. Ballooning degeneration, multifocal, moderate, feather follicles, with hyperkeratosis, skin, budgerigar, avian, etiology—compatible with papovavirus.

Comment. Necrosis with characteristic inclusion bodies was present in a wide variety of tissues, particularly the liver, kidney, heart, and brain. In the skin, feather follicles had ballooning degeneration of epithelium with large intranuclear inclusion bodies. Asymmetrical and distorted feathers that were evident grossly were attributed to focal epithelial lesions in the developing feather. Although the mortality rate was high, in those few birds that survived normal feathering developed in about 4 months.

Suggested reading.

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Bozeman, L. H., Davis, R. B., Gaudry, D., et al.: Characterization of a papovavirus isolated from fledgling budgerigars. Avian Dis. 25: 972-980, 1981.

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Slide 26: L8-L9

History. The second digit of the right forepaw of a female Scottish terrier was amputated when she was 9 years old to remove a malignant melanoma. There was no recurrence at the operative site. At 12 years of age, she developed a head tilt and circling movements to the right. The dog had difficulty holding food in its mouth. Head bobbing occurred when the dog stood still. Treatment with high doses of corticosteroids and antibiotics resulted in limited temporary improvement. Two months from the onset of these symptoms, the dog was put to death because of her worsening condition. Until the time

of her death, the head tilt and circling remained and ataxia, stumbling, loss of palpebral reflexes, bilateral corneal ulceration, and weakness of the tongue and oral musculature were evident. (EM included.)

Gross Pathology. The surface of the ventral hypothalamus was discolored red, and a 4-mm red mass protruded through the right cerebellar pontine angle. On sectioning, a firm red mass extended from the ventral thalamus caudally through the pons and emerged through the cerebellar-pontine angle. The greatest diameter of the mass in the pons was 11 mm. The urinary bladder contained a 2-cm mass (transitional cell carcinoma) projecting into its lumen.

Diagnosis. Reticulosis, neoplastic, brain stem, canine.

Comment. The predominant cell type of the mass belongs to the perithelial reticulohistiocytic system of the CNS. Reticulin stains of this tumor showed a very delicate reticular network in the tumor that appeared to center about and was heaviest near blood vessels which course through the tumor. Ultrastructurally, the tumor cells have prominent microbodies or peroxisomes and filopodia.

Primary CNS reticulosis may be either of the granulomatous/inflammatory or neoplastic types. Primary CNS neoplastic reticuloses are a relatively common type of canine CNS neoplasm.

Neoplastic reticuloses have been classified as adventitial sarcomas, perithelomas, perithelial sarcomas, reticulosarcomas, reticulum cell sarcomas, and fibrosarcomas when the neoplasm was thought to arise from the perivascular

reticulohistiocytic system or primitive reticulum cells. They have also been classified as microgliomatosis or microgliomas when the neoplasm appeared to originate from resident CNS phagocytes or microglial cells.

Similarities between the reticuloses and chronic encephalitides have generated speculation about the role of infectious agents in primary CNS reticuloses. Both rabieslike and canine distemper inclusion bodies and canine distemper antigen have been found in cases of canine reticulosis, but there ha been no direct evidence causally linking viruses to reticulosis.

The spectrum of lesions classified as reticulosis remains controversial a covers a wide range. The diffuseness of the lesion in this case is considere unusual.

Contributor. Toxicology Section, Health, Safety, & Human Factors Laboratory, Eastman Kodak Company, B-320, Kodak Park, Rochester, New York.

Suggested reading.

Pankhauser, R., Luginbuhl, H., and McGrath, J. T.: V. Tumors of the nervous system. Bull. W.H.O. 50: 68, 1974.

Koestner, A., and Zaman, W.: Primary reticuloses of the central nervo system in dogs. Am. J. Vet. Res. 23: 381-393, 1962.

Vandevelde, M., Kristensen, B., and Greene, C. E.: Primary reticulos the central nervous system in the dog. Vet. Pathol. 15: 673-675, 1978.

Wuia, O., and Mehrsein, P.: Primary reticulosis of the central nervo system. J. Neurol. Sci. 14: 469-482, 1971.

Slide 27

History. This tissue was taken from a 4-year-old rhesus monkey (Macaca mulatta). No clinical problems were noted until one month before its death, when the animal escaped from its cage. It was breathing very heavily after a short pursuit, but no abnormalities were found on physical examination or blood work. For three days, a week before its death, water intake was severely restricted as part of the experimental protocol. During the subsequent week the monkey ate poorly, but when offered water, he drank heavily. He became increasingly depressed and died despite supportive care.

Gross Pathology. The animal was in a good state of nutrition with no lesions noted outside the central nervous system. Sectioning the fixed brain revealed symmetrical hemorrhagic foci in the white matter of the frontal and occipital cortex.

Diagnosis. Thrombosis and ectasia, venous, acute to chronic, multifocal, moderate, with perivascular hemorrhage, hemosiderosis and gemistocytosis, cerebral white matter, brain, rhesus monkey, (Macaca mulatta), nonhuman primate.

Comment. Eight cases of cerebral venous thrombosis were seen in a colony of 250 monkeys within a one-year period. The disease has been most commonly associated with dehydrated monkeys, secondary to a variety of causes, but has also presented in otherwise normal animals. A relationship to hypertension was considered: however, this lesion has not been seen in monkeys used in a hypertension study at the contributing institution. There is variation in the

stage of the lesions between affected veins. This is considered to be a multistage condition that remains confined to the white matter. The pathogenesis and etiology of this lesion remain unclear.

The condition resembles a syndrome described in people: this is also termed cerebral venous thrombosis. The condition in monkeys has also been termed "leukoencephalosis and perivascular myelosis." The age of the lesions in this case probably antedates the clinical history of one week.

Contributor. Division of Comparative Medicine, Johns Hopkins University School of Medicine, Baltimore, Maryland.

Suggested reading.

Averback, P.: Primary cerebral venous thrombosis in young adults: The diverse manifestations of an underrecognized disease. Ann. Neurol. 3: 81-86, 1978.

Sheffield, W. D., Squire, R. A., and Strandberg, J. D.: Cerebral venous thrombosis in the rhesus monkey. Vet. Pathol. 18: 11-76, 1978.

Slide 28

History. The tissue submitted is from a lethargic adult male owl monkey (Aotus trivirgatus) that had a bloody diarrhea. Despite treatment with fluids, antibiotics, and steroids, he died on the sixth day of illness. This monkey was one of approximately 30 Aotus monkeys out of approximately 300 in this colony that died over a 2-week period.

Diagnosis. Colitis, subacute, diffuse, moderate, with crypt cell necrosis, syncytial cell formation and intranuclear/intracytoplasmic inclusion bodies, colon, owl monkey, Aotus trivirgatus, nonhuman primate, etiology, compatible with morbillivirus.

Comment. Pathologic, serologic, and culture results indicated that the outbreak of disease was due to infection with paramyxovirus, most likely measles. Clinical signs were variable and included sudden death, mucopurulent nasal discharge, respiratory rales, and diarrhea with or without blood. The latter clinical sign was observed most frequently. Major lesions involved the gastrointestinal and respiratory systems. There was distention of the stomach and segments of the small intestine and colon, with blood-tinged or soft brown ingesta. The liver was swollen and pale. Many monkeys had bronchopneumonia or interstitial pneumonia. Histologically there was necrosis of gastric or intestinal epithelium with occasional epithelial syncytia and intranuclear and intracytoplasmic inclusions. There were syncytia and inclusions in the lung, bile ducts, and occasionally in spleen and lymph nodes. This outbreak of disease in the Aotus colony was associated with evidence of measles infection in baboons and cynomolgus and rhesus monkeys housed in adjacent areas. Many of these Old World monkeys had a rise in hemagglutination inhibition titer to measles. One cynomolgus and two rhesus monkeys developed a maculopapular skin rash. Of the Aotus monkeys that died, 10 out of 10 were seropositive and 6 out of 10 had a rise in titer to measles. Of the surviving Aotus monkeys, over 75% were seropositive for measles.

With morbilliviruses, a transient primary viremia as a result of a local respiratory tract lesion is followed by an asymptomatic phase in which viral

replication occurs in the lymph nodes draining the respiratory tract. Syncytia formation results from the virus-induced cell membrane modification that facilitates fusion and virus spread. Lysis of infected cells occurs with the developing immune response, producing a secondary viremia and dissemination throughout the body. Viral replication occurs in all epithelia and the reticuloendothelial system.

The high fatality rate in the affected colony may be related to the virulence of the virus as well as to the host immune response capability.

Contributor. Division of Pathology, Walter Reed Army Institute of Research, Washington, D.C.

Suggested reading.

Albrecht, P.: Fatal measles infection in marmosets: Pathogenesis and prophylaxis. Infect. Immun. 27(1): 969-978, 1980.

Fraser, C. E. O.: A paramyxovirus causing fatal gastroenterocolitis in marmoset monkeys. Primates Med. 10: 261-270, 1978.

Hall, W. C.: Pathology of measles in rhesus monkeys. Vet. Pathol. 8: 307-319, 1971.

O'Brien, A. W.: Measles virus antibodies in a laboratory colony of monkeys (Aotus trivirgatus). Lab. Anim. 15: 343-345, 1981.

Slide 29

History. A 1 and 1/2-year-old male Doberman pinscher presented clinically with a 4-month history of persistent vomiting, intermittent diarrhea and weight loss.

Gross Pathology. A greatly enlarged and firm segment of distal jejunum was removed surgically following exploratory laparotomy. Grossly, the distal jejunum was irregularly thickened up to 4 cm with scattered ulcerative and hemorrhagic zones along the mucosal surface. The tumor tissue was white and firm.

Diagnosis. Ganglioneuroma, tunica muscularis, small intestine, Doberman pinscher, canine.

Comment. The tumor consists of an admixture of ganglion cells, nerve fibers, and associated Schwann cells that infiltrate and replace the muscular tunics. Ganglion cells occur singly or in small groups: the nerve fiber component usually predominates. Secondary inflammatory changes are present.

Ganglioneuromas of the intestinal tract are quite rare. These slow-growing benign tumors are considered to arise from immature neuroblastic tissue.

Contributor. Department of Pathology & Parasitology, School of Veterinary Medicine, Auburn University, Alabama.

Suggested reading.

Coordy, D. R.: Tumors of the nervous system and eye. In Tumors in Domestic Animals, ed. 2, edited by Moulton, J. E. Berkeley, University of California Press, 1978, pp. 437-439.

Dahl, E. V.: Gastrointestinal ganglioneuromas. Am. J. Pathol. 33: 953-965, 1957.

Ganer, M., and Feuchtwanger, M. M.: Ganglioneuroma of the duodenum. Gastroenterology 51: 689-693, 1966.

Harkin, J. C., and Reed, R. J.: Tumors of the Peripheral Nervous System, Ser. 2, Fascicle 3, of Atlas of Tumor Pathology. Washington, D.C., Armed Forces Institute of Pathology, 1969, pp. 145-149.

Patnaik, A. K.: Intestinal ganglioneuroma in a kitten. A case report and review of literature. J. Small Anim. Pract. 19: 735-742, 1978.

Slide 30

History. An 18-month-old domestic short-hair cat vaccinated at a free rabies clinic on 5/5/82 became lethargic, anorexic, and developed seizures by 5/10/82. The animal died 5/11/82 and was submitted for rabies examination, though no human exposure was reported.

Gross Pathology. There was marked icterus of all tissues with moderate hydrothorax and ascites: petechiation of heart, lung, kidney, urinary bladder, mesenteric lymph nodes, and cerebral meninges: splenomegaly (2x): and hepatomegaly (1/2x).

Laboratory Results. Rabies fluorescent antibody (impression smears) studies of the hippocampus, cerebellum, cerebellar peduncles, and medullary vellum were negative.

Diagnoses. Parasitemia, protozoal, erythrocytes and mononuclear cells, diffuse, severe, lung, liver, kidney, domestic short-hair cat, feline, etiology—Cytauxzoon sp. Hepatitis, portal, subacute, diffuse, minimal to mild, liver.

Comment. Veterinarians commonly associate lethargy, anorexia, anemia, and/or icterus with feline infectious anemia, feline infectious peritonitis, and sometimes ethylene glycol (antifreeze) toxicity. Cytauxzoonosis was recognized in domestic cats in Missouri in 1976 (Wagner); this was one of the first reports of this disease in domestic animals within the USA. The disease is apparently being recognized with increasing frequency in cats in the United States and consequently should be included among the tentative diagnoses considered by veterinary clinicians and veterinary pathologists when lethargic, anemic, icteric cats are encountered. This case was unusual clinically because CNS seizures are apparently uncommon with cytauxzoonosis in the domestic cat.

Some confusion exists as to whether the cells parasitized in the vessels are monocytes and/or endothelial cells. The differential diagnosis includes Hepatozoon sp.: however, this does not infect RBC's and gametogony occurs in circulating neutrophils.

The domestic cat is an unlikely natural host for the organism in that the disease is invariably fatal. Some investigators have suggested that the bobcat may be the natural reservoir host for the parasite (Glenn et al.). The bobcat is susceptible to both the fatal and nonfatal form of the disease, and the disease has been experimentally transmitted from bobcats to domestic cats (Kier et al.).

Contributor. Department of Microbiology, Pathology, & Parasitology, School of Veterinary Medicine, North Carolina State University, Raleigh, North Carolina.

Suggested reading.

Glenn, B. L., Bolley, R. E., and Kocan, A. A.: Cytauxzoon-like piroplasms in erythrocytes of wild-trapped bobcats in Oklahoma. J. Am. Vet. Med. Assoc. 181(11): 1251-1253, 1982.

Kier, A. B., Wagner, J. E., and Morehouse, L. G.: Experimental transmission of Cytauxzoon felis from bobcats (Lynx rufus) to domestic cats (Felis domesticus). Am. J. Vet. Res. 43: 97-101, 1982.

Levine, N. D.: Protozoan Parasites of Domestic Animals and of Man, ed. 2. Minneapolis, Burgess Publishing Co., 1973, pp. 317-346.

McCully, R. M., Keep, M. E., and Basson, P. A.: Cytauxzoonosis in a giraffe [Giraffa camelopardalis (Linnaeus, 1758)] in Zululand. Onderstepoort J. Vet. Res. 37: 7-9, 1970.

Neitz, W. O.: Theileriosis, gonderiosis, and cytauxzoonosis. A review. Onderstepoort J. Vet. Res. 27: 275-430, 1957.

Wagner, J. E.: A fatal cytauxzoonosis-like disease in cats. J. Am. Vet. Med. Assoc. 168: 585-588, 1976.

Wagner, J. E., Ferris, D. H., and Kier, A. B., et al.: Experimentally induced Cytauxzoonosis-like disease in domestic cats. Vet. Parasitol. 6: 305-311, 1980.

Slide 31

History. The submitting veterinarian performed a biopsy on a necrotic ulcerated mass, approximately 2 cm in diameter, that was located on the right ear of a 16-year-old domestic short-hair male cat that had been previously castrated.

Diagnosis. Chondrosarcoma, right ear, breed unspecified, feline.

Comment. Chondrosarcomas are considered rare in cats, with the usual sites being the scapulae, vertebrae, and limbs (Pool). Neoplasms of the feline pinna are uncommon and are usually squamous cell carcinomas (Scott). Various types of neoplasms have been reported infrequently on the ears of cats, including basal cell carcinoma, fibroma, hemangioendothelioma, mastocytoma, malignant melanoma, and papilloma (Legendre).

The well-differentiated cartilagenous portions appear to be arising from the undifferentiated spindle cells and in this respect somewhat resemble the mesenchymal chondrosarcoma of man. In man, however, the spindle cells are even less differentiated and definitely mesenchymal in nature.

Contributor. School of Veterinary Medicine, Oregon State University, Corvallis, Oregon.

Suggested reading.

Legendre, A. M., and Krahwinkel, D. J.: Feline ear tumors. J. Am. Anim. Hosp. Assoc. 17: 1035-1037, 1981.

Pepe, A. J., Kuhlmann, R. F., and Miller, M. D.: Mesenchymal chondrosarcoma. J. Bone Joint Surg. [Am.] 59(2): 256-258, 1977.

Pool, R. R.: Tumors of bone and cartilage. In Tumors in Domestic Animals, ed. 2., edited by Moulton, J. E., Berkeley, Univ. of California Press, 1978, pp. 125-126.

Salvador, A. H., Beabout, J. W., and Dahlin, D. C.: Mesenchymal chondrosarcoma - observations on 30 new cases. Cancer 28(3): 605-615, 1971.

Slide 32

History. The tissue on this slide is from a 10-year-old male Irish Setter weighing 45 lbs. The animal had a history of weight loss and the recent appearance of multifocal ulcerative lesions measuring 1 to 3 cm in diameter involving the skin, oral mucous membranes, and tongue.

Diagnosis. Meningitis, pyogranulomatous, multifocal, mild to moderate, brain, Irish setter, canine, etiology, Cryptococcus neoformans.

Comment. Pyogranulomatous lesions containing typical narrow-based-budd cryptococcal organisms with wide mucoid capsules were found in the skin, or mucosa, kidneys, optic nerve, urethra, lymph node, and lung in addition to lesions presented. The pyogranulomatous response is considered to be more typical of blastomycosis or histoplasmosis. Cryptococcus neoformans was cultured from the lung, lymph node, CNS, and skin ulcer.

The eyes, oral cavity, and upper respiratory tract are the most probabl portals of entry followed by direct extension and hematogenous dissemination other organs.

Contributor. Department of Pathology, Division of Comparative Medicine University of Maryland School of Medicine, Baltimore, Maryland.

Suggested reading.

Barron, C. N.: Cryptococcosis in animals. J. Am. Vet. Med. Assoc. 127 125-132, 1955.

Binford, C. H., and Connor, D. H. (Eds.): Pathology of Tropical and Extraordinary Diseases. Washington, D.C., Armed Forces Institute of Pathology, 1976, pp. 572-573.

Gelatt, K. N., McGill, L. D., and Peiman, V.: Ocular and systemic cryptococcosis in a dog. J. Am. Vet. Med. Assoc. 163: 1370-1375, 1973.

Perfect, J. R., Lang, S. D. R., and Durack, D. T.: Chronic cryptococcal meningitis. A new experimental model in rabbits. Am. J. Pathol. 101: 177-194, 1980.

Slide 33

History. The initial clinical signs seen in this 4-year-old Polled Hereford male were incoordination and anorexia. Supportive therapy was administered by the local veterinarian. Upon presentation at the hospital, the bull was incoordinated and weak on the left front leg. He was aggressive, which was not his usual temperament, and had a temperature of 103.5°F. Supportive therapy was given. The bull continued to deteriorate and was put to death that afternoon (3 days after initial signs were observed by the owner).

Diagnosis. Myelitis, lymphocytic, multifocal, mild, with hemorrhage, gliosis, and intracytoplasmic neuronal inclusions consistent with Negri bodies, spinal cord, bovine, etiology, lyssavirus (Rabies).

Comment. The perivascular cuffing by lymphocytes along with a small amount of hemorrhage and gliosis are considered typical of the spinal cord lesions found in ruminants with rabies. The Negri bodies are very pleomorphic and often multiple. Negri bodies consist of a compact mass of viral nucleocapsid surrounded by numerous bullet-shaped viral particles budding from

intracytoplasmic membranes. Their shape is determined by molding to their environment, and they are often surrounded by a thin halo.

Fluorescent antibody examination was positive for rabies.

Contributor. Department of Veterinary Pathology, College of Veterinary Medicine, Iowa State University, Ames, Iowa.

Suggested reading.

Gillespie, J. H., and Timoney, J. F.: Hagan and Bruner's Infectious Diseases of Domestic Animals. Ithaca, Cornell University Press, 1981, pp. 758-772.

Jubb, K. V. F., and Kennedy, P. C.: Pathology of Domestic Animals, ed. 2. New York, Academic Press, 1970, vol. 2, p. 414.

Mohanty, S. B., and Dutta, S. K.: Veterinary Virology. Philadelphia, Lea & Febiger, 1981, pp. 217-223.

Slide 34, L10-L11

History. This male New Zealand white rabbit that weighed 2 kg was found dead in its cage a day after it was received at a laboratory. A large quantit of watery fecal material was found in the cage bottom.

Gross Pathology. Multiple yellow nodules of various sizes were seen in th liver. The cecum and colon contained yellow watery fecal material. (See gros Kodachromes.)

Diagnosis. Ectasia and papillary hyperplasia, bile ducts, with intraductal and intraepithelial coccidia, multifocal, severe, with mild chronic pericholangitis, liver, New Zealand white rabbit, etiology, Eimeria stiedai.

Comment. The intrahepatic bile duct epithelium is proliferative, forming fronds with numerous coccidial organisms (compatible with Eimeria stiedai) in various stages of development in the epithelial cells and lumen of the ducts. The basis for the hyperplasia is not fully understood. A portal hepatitis was present on most slides.

Following ingestion of sporulated oocysts, sporozoites are released and penetrate the intestinal mucosa. The method of sporozoite transport to the liver is not clear, but the hematogenous route seems most likely. Once in the liver, the sporozoites invade the bile duct epithelium and occasionally hepatocytes, where they become schizonts. The number of asexual generations is not known, but following gametogony unsporulated oocysts break out of host cells and are passed in the feces. The ectatic ducts can rupture resulting in a granulomatous response.

Escherichia coli was considered the most likely cause of the diarrhea reported in the history.

Contributor. Mobil Oil Corporation, Toxicology Division, Princeton, New Jersey.

Suggested reading.

Pakes, S. P.: Protozoal diseases. In Biology of the Laboratory Rabbit, edited by Weisbroth, S. H., Flatt, R. E., and Kraus, A. L. New York, Academic Press, 1974, pp. 264-268.

Shadduck, J. A., and Pakes, S. P.: Protozoal and metazoal diseases. In Pathology of Laboratory Animals, edited by Benirschke, K., Garner, F. M., and Jones, T. C. New York, Springer-Verlag, 1978, vol. II, pp. 1605-1606.

Slide 35-36

History. A 6-month-old Arabian foal had a history of anorexia, depression and copious black diarrhea for 4 days prior to being hospitalized in a moribund state.

Gross Pathology. The wall of the distal duodenum, jejunum, and ileum was thickened irregularly up to 1.2 cm. The mucosa was prominently corrugated. Mesenteric lymph nodes were swollen and slightly congested.

Diagnosis. Crypt proliferation and hyperplasia with villous atrophy, diffuse, severe, small intestine, Arabian, equine, etiology, compatible with Campylobacter sputorum subsp. Mucosalis.

Comment. There was no evidence of an immunodeficiency. Electron microscopy revealed rod-shaped bacteria in the apical cytoplasm of intestinal crypt epithelial cells. These bacteria were also demonstrated in histologic sections from Levaditi-stained tissue. The ultrastructure and staining properties of the bacteria were similar to those of Campylobacter sputorum subsp. Mucosalis associated with intestinal adenomatosis in swine.

The diarrhea may be related to the loss of the villar epithelium and its absorptive function. There is an associated mild enteritis in those sections of small intestine examined. The intestinal crypts were elongated and branching, with the epithelium occasionally being pseudostratified.

The pathogenesis of "intestinal adenomatosis" is not completely known. In swine, Campylobacter sputorum subsp. Mucosalis (CSM) has been associated with or incriminated as the cause of the proliferation or hyperplasia, but consistent production of the disease experimentally has been difficult. CSM organisms have a predilection for crypt epithelium, are capable of entering epithelial cells without phagocytosis, and are usually seen free within the cytoplasm. A similar proliferative enteritis has been reported in hamsters, sheep, swine, and a blue fox. A CSM-like organism has been observed in the crypt epithelium of each of the affected species.

Contributor. Department of Veterinary Pathology, School of Veterinary Medicine, University of California, Davis, California.

Suggested reading.

Duhamel, G. F., and Wheeldon, E. B.: Intestinal adenomatosis in a foal. Vet. Pathol. 19: 447-450, 1982.

Jacoby, R. O.: Transmissible ileal hyperplasia of hamsters. I. Histogenesis and immunocytochemistry. Am. J. Pathol. 91: 433-450, 1978.

Lomax, L. G., and Glock, R. D.: Naturally occurring porcine proliferative enteritis: Pathologic and bacteriologic findings. Am. J. Vet. Res. 43: 1608-1614, 1982.

Lomax, L. G., Glock, R. D., Harris, D. L., et al.: Porcine proliferative enteritis: Experimentally induced disease in cesarean-derived colostrum deprived pigs. Am. J. Vet. Res. 43: 1622-1630, 1982.

Lomax, L. G., Glock, R. D., and Hogan, J. E.: Experimentally induced porcine proliferative enteritis in specific-pathogen-free pigs. Am. J. Vet. Res. 43: 1615-1621, 1982.

Love, D. N., and Love, R. J.: Pathology of proliferative hemorrhagic enteropathy in pigs. Vet. Pathol. 16: 41-48, 1979.

Slide 37

History. A 3-year-old domestic short-hair cat had a normal litter one week prior to being found in lateral recumbency with a temperature of 104°F. Her eyes were reportedly alternating between myosis and mydriasis. Central nervous signs progressed to clonic convulsions before she was put to death one day after the first signs were noted.

Laboratory Results. Virus isolation attempts were negative, FAT for feline infectious peritonitis was negative.

Diagnoses. Vasculitis, acute, segental, moderate, cerebrum and meninges, feline. Crystals, anisotropic, multifocal, meningeal and cerebral vessels, brain, compatible with ethylene glycol crystals.

Comment. The morphologic features of the numerous anisotropic, multicolored crystals in the lumen and walls of meningeal and cerebral vessels are compatible with oxalates. The primary lesion is considered to be a

vasculitis, but the apparent lack of association between the presence of crystals and inflammation is confusing. There is necrosis of vessels in some sections.

Histologic alterations were also prominent in the kidneys of this cat, as there were large numbers of refractile crystals in cortical tubules with attenuation or degeneration of adjacent tubular epithelial cells. Scattered tubules were moderately dilated and occasional hyaline casts were present.

Contributor. Animal Disease Diagnostic Laboratory, School of Veterinary Medicine, Purdue University, West Lafayette, Indiana.

Suggested reading.

Bove, K. E.: Ethylene glycol toxicity. Am. J. Clin. Pathol. 45: 46-50, 1966.

Buck, W. B., and Osweiler, G. D.: Clinical and Diagnostic Veterinary Toxicology. Dubuque, Iowa, Kendall/Hunt Publishing Co., 1976, pp. 125-127.

Hadlow, W. J.: Acute ethylene glycol poisoning in a cat. J. Am. Vet. Med. Assoc. 130: 296, 1957.

Nersting, E. J., and Nielsen, S. W.: Experimental ethylene glycol poisoning in the dog. Am. J. Vet. Res. 27: 574-582, 1966.

Slide 38

History. A 5-month-old quarter horse colt had a sudden onset of hind limb ataxia leading to posterior paraplegia.

Gross Pathology. There were bilateral hemorrhages in the ventral grey matter of the caudal lumbar and sacral spinal cord. A Verminous arteritis was present at the root of the cranial mesenteric artery.

Laboratory Results. Fluorescent antibody studies for rabies and EHV-1 were negative.

Diagnoses. Leptomeningitis, granulomatous, focal, moderate with a nematode parasite, ventral sulcus, spinal cord, quarter horse, equine. Poliomalacia, multifocal, moderate, with vacuolation, axonal swelling and chromatolysis in adjacent neuropil, spinal cord. Etiology, compatible with Strongylus sp.

Comment. The nematode is degenerating and could not be positively identified. Striations are demonstrable in portions of the cuticle, and a microvillar border is seen lining the lumen of the intestine in some sections. These characteristics, along with the size of the parasite, are compatible with a diagnosis of Strongylus sp. The bilateral poliomyelomalacia and Wallerian degeneration in the spinal tracts suggest that the parasite was occluding the central branch of the ventral spinal artery with consequent infarction of the cord.

Cerebrospinal nematodosis is well-documented in the horse (Swanstrom et al. and Little), and S. vulgaris is considered to be the species most commonly involved. Experimental reproduction of the brain lesions has been achieved by intracarotid inoculation of larvae (Little et al.).

Contributor. Department of Pathology, College of Veterinary Medicine, Kansas State University, Manhattan, Kansas.

Suggested reading.

Chitwood, M., and Lichtenfels, J. R.: Identification of parasitic metazoa in tissue sections. Exp. Parasitol. 32: 407-519, 1972.

Little, P. B.: Cerebrospinal nematodiasis of equidae. J. Am. Vet. Med. Assoc. 160: 1407-1411, 1972.

Little, P. B., Sein, L. V., and Pretz, P.: Verminous encephalitis of horses: Experimental induction with Strongylus vulgaris larvae. Am. J. Vet. Res. 35: 1501-1510, 1974.

Swanstrom, O. G., Rising, J. L., and Carlton, W. W.: Spinal nematodiosis in a horse. J. Am. Vet. Med. Assoc. 155: 748-753, 1969.

Slide 39

History. This tissue represents an incidental finding in a 2-year-old Sprague-Dawley rat.

Diagnosis. Adenocarcinoma, scirrhous, accessory sex gland, Sprague-Dawley rat.

Comment. The neoplastic epithelial element appeared to provoke an extensive desmoplasia that caused the separation and distortion of the carcinomatous acini; lack of orientation was evident. Most of the tubules contain purulent exudate. Some sections show large areas of suppurative inflammation with extensive and diffuse permeation of the fibrous stroma with neutrophils. A mantle of mononuclear cells, interrupted in places, encloses

the mass. Peripheral to the mass are a few large nonmalignant glands. The mass infiltrates the wall of the vas deferens, and there is occasional thrombus formation. The prostate gland may be the origin of the tumor.

Contributor. Bio/dynamics, Inc., East Millstone, New Jersey.

Suggested reading.

Franks, L. M., and Waldague, P.: Tumors of the accessory male sex glands. In Pathology of Tumors in Laboratory Animals, edited by Turusov, V. S. Lyon, IARC, 1976, vol. 1, part 2, pp. 151-154.

Hall, W. C., Nielsen, S. W., and McEntee, K.: XIX. Tumors of the prostate and penis. Bull W.H.O. 53: 1976, pp. 247-250.

Ward, J. M., Rennik, G., Stinson, S. F., et al.: Histogenesis and morphology of naturally occurring prostatic carcinoma in the ACI/segHapBR rat. Lab. Invest. 43: 517-522, 1980.

Slide 40, L12

History. A 16-year-old thoroughbred gelding had an episode of incoordination and high fever that was treated successfully with antibiotics and steroids by a local practitioner. Three months later, there was another episode of ataxia of two weeks' duration that did not resolve. The incoordination involved all four limbs with a slight dragging of the left hind toe and stumbling over uneven ground. Radiographs of the cervical spine revealed no abnormality. (See koilachrone.)

Gross Pathology. Significant gross lesions were confined to the cerebral meninges. The external surface of the arachnoid was finely granular over both

marginal gyri from the occipital poles to an area just [illegible] ectosylvian sulci. This area was approximately 1 cm in width on both cerebral hemispheres, lateral to the medial longitudinal sulcus.

Laboratory Results. A CBC and panel at the time the gelding was put to death were noncontributory. CSF cytologic findings—18 RBC's per mm^3 and 267 nucleated cells per mm^3 (92% lymphocytes, 3% neutrophils, 5% macrophages). CSF protein—338 mg/dl. EHV, VEE, and EEE titers were low. The blood sample taken just prior to death was positive by the Coggin's test for retrovirus (EIA).

Diagnosis. Leptomeningitis, lymphocytic and plasmacytic (nonsuppurative), diffuse, moderate, spinal cord, thoroughbred, equine, etiology, retrovirus (EIA).

Comment. The inflammatory response is characterized by the infiltration of lymphocytes and plasma cells with occasional macrophages. As a result of persistent antigenic stimulation, Russell body cells are also present. The histologic lesions are compatible with a diagnosis of EIA. A spinal meningitis similar to that seen in the brain extended throughout the length of the cord but was not uniform in distribution. It chiefly involved the pia over the dorsal funiculus and occasionally the ventral median fissure. Cells surrounded the dorsal rootlets and occasionally the ventral rootlets but did not infiltrate the nerves or cause Wallerian degeneration.

Contributor. Department of Pathobiology, University of Tennessee, Knoxville, Tennessee.

Gillespie, [illegible] Diseases of Domestic Animals. Ithaca, Cornell University [illegible] 797-803.

Issel, C. J., and Coggins, L.: Equine infectious anemia: Current knowledge. J. Am. Vet. Med. Assoc. 174: 727-732, 1979.

McClure, J. J., Lindsay, W. A., Taylor, W., et al.: Ataxia in four horses with equine infectious anemia. J. Am. Vet. Med. Assoc. 180(3): 279-283, 1982.

McIlwraith, C. W., and Kitchen, D. N.: Neurological signs and neuropathology associated with a case of equine infectious anemia. Cornell Vet. 68: 238-249, 1978.

Slide 41

History. This 8-month-old mixed-breed female lamb was the third out of a group of 17 experimental lambs to die over a 3-week period. Enterotoxemia was suspected as a possible cause of death.

Diagnosis. Squamous metaplasia, diffuse, severe, interlobular ducts, with diffuse lobular atrophy and lymphocytic infiltration, salivary gland, ovine, etiology, compatible with vitamin A deficiency.

Comment. This lamb was part of a nutritional study on vitamin A and vitamin E deficiency. There were renal changes consistent with a diagnosis enterotoxemia as the cause of death. Lesions compatible with vitamin E deficiency were white muscle disease. Lesions compatible with vitamin A deficiency include squamous metaplasia of the salivary glands and the glan the nasal submucosa.

In the salivary gland there is diffuse atrophy of lobules with interstitial lymphocytic infiltration. This is thought to be a result of back pressure in the ducts and leakage of secretory material into the lobule. The amount of fibrous tissue within this gland is considered by most attendees to be normal. Some slides contain a portion of thymus that is atrophied. This lamb is considered to be too young for thymic involution; the deficient diet of the lamb may have been responsible for the thymic atrophy observed.

Suggested reading.

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Jubb, K. V., and Kennedy, P. C.: Pathology of Domestic Animals. New York, Academic Press, 1970, vol. 2, pp. 539-542.

Wills, J. H. L.: Experimental pathology of dairy calves ingesting one-third the daily requirement of carotene. Acta Vet. Scand. 8: 324-346, 1967.

Robbins, S. L.: Pathologic Basis of Disease. Philadelphia, W. B. Saunders & Co., 1979, pp. 488-491.

Slide 42

History. Five days prior to death, this 10-year-old female boa constrictor (Constrictor constrictor) was severely bitten by a rat that was offered to it as food. The snake had not eaten for one year. Physical exam revealed extensive lacerations, mild "mouthrot," and extreme emaciation.

Diagnosis. Tubular ectasia with epithelial hyperplasia and squamous metaplasia, multifocal, moderate, with intraluminal trematodes, kidney. Granulomas, chronic, multifocal, mild, containing trematode eggs, kidney.

Comment. The morphologic characteristics of this intratubular metazoan parasite including the absence of a body cavity along with the presence of oral and ventral suckers, paired ceca, intrauterine operculated eggs, and testes and vitellaria are typical of trematodes. Although this parasite is probably Styphlodora sp., the genus of the trematode cannot be determined from the sections examined.

Death in this snake was attributed to emaciation and trauma. Since renal trematodiasis can be asymptomatic (Myers), the clinical significance of the renal styphlodoriasis in the present case is uncertain. The renal lesions are similar to those previously described (Myers and Kazacos et al.).

Contributor. Department of Veterinary Pathology, Ohio State University, Columbus, Ohio.

Suggested reading.

Chiodini, R., and Sundberg, J. P.: Styphlodora horrida in the kidneys and ureters of a boa constrictor (Constrictor constrictor). VM/SAC: 877-878 (May) 1980.

Kazacos, K. R., and Fisher, L. F.: Renal styphlodoriasis in a boa constrictor. J. Am. Vet. Med. Assoc. 171: 876-878, 1977.

Myers, T. S.: Neral Trematodiasis in a boa constrictor (Constrictor constrictor). Lab. Anim. Sci. 21: 423-425, 1971.

Slide 43

History. A group of young adult male Lewis rats were irradiated (total body) with 950 rads 3 weeks prior to death. One day later they were given a bone marrow transplant that had been treated with cyclophosphamide. Paralysis was noted one day before death: rear leg reflexes could be elicited.

Gross Pathology. The spleen and lymphoid organs were notably reduced in size. The only other gross lesions were found in the spinal cord: red streaks measuring 1 to 4 mm in length and about 1 to 2 mm in diameter were found most numerous in the thoracic cord.

Laboratory Results. Bacterial cultures of the spleen and lung were negative. A fecal exam was also negative.

Diagnosis. Hemorrhage and malacia, acute, multifocal, moderate to severe, with necrotizing vasculitis, spinal cord, rat.

Comment. Within different sections there are varying degrees of vascular necrosis, with some affected vessels having a prominent inflammatory response. Vascular endothelium is highly susceptible to parvovirus-induced damage. Hepatocytes, especially in young animals, are also susceptible owing to the virus' affinity for mitotically active cells (Coleman et al.).

Other lesions commonly seen with Kilham rat virus (KRV) infections include a necrotizing and hemorrhagic orchitis (considered virtually pathognomonic) and hepatitis. Hemorrhages in the medulla, cerebellum, and cerebral cortex are often present as are widespread subserosal and pulmonary hemorrhages. Carthew et al. report that KRV can cause inflammation and necrosis in the lungs of rats. Fetal resorption and cerebellar hypoplasia can result from infection by KRV. KRV commonly induces a persistent, asymptomatic infection in adult rats (Coleman et al.).

This outbreak of spinal cord disease occurred in an isolated colony. Surviving animals in the colony showed marked elevations in titer to Kilham rat virus. Lesions were most apparent in the spinal cord of all animals examined.

Contributor. Division of Comparative Medicine, Johns Hopkins University School of Medicine, Baltimore, Maryland.

Suggested reading.

Coleman, G. L., Jacoby, R. O., Bhatt, P. N., et al.: Naturally occurring lethal parvovirus infection of juvenile and young adult rats. Vet. Pathol. 20: 49-56, 1983.

Jacoby, R. O.: Viral diseases. A. parvoviruses. In Biology of the Laboratory Rat. New York, Academic Press, 1979, vol. I, pp. 273-283.

Jortner, B. S., and Percy, D. H.: The nervous system. In Pathology of Laboratory Animals, edited by Benirschke, K., Garner, F. M., and Jones, T. C. New York, Springer-Verlag, 1978, pp. 129-131.

Slide 44

History. This tissue is from a Sprague-Dawley female rat, approximately 670 days of age, with a round, firm 20-mm red-gray mass beneath its tail.

Diagnosis. Adenocarcinoma, cystic, papillary, clitoral gland, rat.

Comment. This large neoplasm is invading through its compression capsule. Papillary cystadenocarcinoma is a common tumor of the clitoral gland of rats, but metastasis is rare. Hyperplastic and neoplastic lesions of this gland usually develop from the acinar cells, basal cells, or the squamous epithelium of the ducts and only rarely from the mesenchymal tissue (Resnik et al.). The diagnosis of clitoral gland neoplasia is occasionally difficult to distinguish from infections of the gland, which are common, or from invading neoplasms from the adjacent mammary glands or skin.

Contributor. Pathology Services Division, National Center for Toxicological Research, Jefferson, Arkansas.

Suggested reading.

Resnik, G., and Resnik-Schuller, H.: Pathology of the clitoral and preputial glands in aging F344 rats. Lab. Anim. Sci. 30: 845-850, 1980.

Resnik, G., and Ward, J. M.: Morphology of hyperplastic and neoplastic lesions in the clitoral and preputial gland of the F344 rat. Vet. Pathol. 18: 228-238, 1981.

Slide 45, 1113

History. An incidental observation at the necropsy of an experimental mature male baboon (Papio sp.) is represented on this slide.

Gross Pathology. A coiled, cylindrical, annulated, fawn-colored, parasite approximately 10 mm in length and 2 mm in diameter was present subjacent to Glisson's capsule. (See kodachrome.)

Diagnosis. Encysted pentastome nymph, subcapsular, focal, liver, baboon (Papio sp.).

Comment. The gross appearance and several microscopic features of the parasite present in this case including chitinous cuticle with sclerotized openings, villous digestive tract, acidophilic digestive glands, and metamerically arranged striated musculature are characteristic of pentastomids. Noted with most encysted nymphs is the shed cuticle from the previous molt. These parasites have a wide distribution including both Eastern and Western hemispheres. There are at least three genera that have been described in nonhuman primates: Linguatula, Porocephalus, and Armillifer. Armillifer nymphs lack cuticular spines and have prominent projecting annuli that were consistent with the gross appearance of the parasite present in the liver of this baboon. The life cycle of pentastomes is heteroxenous. The definitive host is usually a carnivore, often a snake, and the intermediate host can be any number of animals, usually mammals. The adult parasite is generally present in the respiratory system of the definitive host. The parasitic larvae develops in various tissues of the intermediate host. The intermediate host ingests the

eggs, which hatch in the host's gut. The larval from then migrates in body tissues often to become encysted in lungs, liver, serosal surfaces, or omentum. The carnivorous definitive host becomes infected when the intermediate host is eaten. Pentastomiasis is usually asymptomatic. Commonly, as in this case, there is little host response to the viable encysted parasite; but should the nymph die, a foreign-body type inflammatory response usually occurs. Marked, sometimes fatal, peritonitis can occur in severe infections. This is invariably associated with penetration of the gut wall by massive numbers of larvae.

Contributor. Pathology Branch, AFAMRL/THP, Wright Patterson AFB, Ohio.

Suggested reading.

Chitwood, M., and Lichtenfels, J. R.: Identification of parasitic metazoa in tissue sections. Exp. Parasitol. 32: 415-419, 1972.

Cosgrove, G. E., Nelson, B. M., and Self, J. T.: The pathology of pentastomid infection in primates. Lab. Anim. Care 20(2): 354-360, 1970.

Flynn, R. J.: Pentastomids. In Parasites of Laboratory Animals. Ames, Iowa State University Press, 1973, chap. 16, pp. 493-503.

Toft, J. D. II: The pathoparasitology of the alimentary tract and pancreas of nonhuman primates: A review. Vet. Pathol. 19(Supp 7): 44-92, 1982.

Slide 46-47

History. The tissue is from an adult male Capuchin monkey. The animal was part of a collection maintained in a safari park. It sickened quite suddenly

and expired in a matter of a few days. At necropsy the liver was observed to be slightly enlarged and marked by large numbers of small yellow-gray, sharply demarcated areas, measuring 0.1 to 0.5 cm, throughout the parenchyma. The spleen was enlarged.

Diagnosis. Hepatitis and splenitis, necrotizing and pyogranulomatous, subacute, multifocal, moderate to severe, with associated bacterial colonies, liver and spleen, Capuchin monkey, etiology - Yersinia pseudotuberculosis.

Comment. The morphologic characteristics of the splenic and hepatic lesions in this case are typical of Yersinia pseudotuberculosis infections. Occasionally, these gram-negative pleomorphic bacilli display bipolar staining. In the spleen, there is diffuse reticuloendothelial cell hyperplasia in addition to the microgranulomas.

A pure culture of Yersinia pseudotuberculosis was taken from the liver and spleen. When this pathogen is suspected, culture material may be held at 4° to 7°C (cold enrichment) in isotonic saline solution to reduce competitive organism growth before culturing.

Contributor. Laboratory and Pathology Service, Veterans Administration Medical Center, Roseburg, Oregon.

Suggested reading.

Benirschke, K., Garner, F. M., and Jones, T. C.: Pathology of Laboratory Animals. New York, Springer-Verlag, 1978, vol. II, pp. 1463-1466.

Gillespie, J. H., and Timoney, J. F.: Hagan and Bruner's Infectious Diseases of Domestic Animals. Ithaca, Cornell University Press, 1981, pp. 96-97.

Paff, J. R., Triplett, D. A., and Saari, T. N.: Clinical and laboratory aspects of Yersinia pseudotuberculosis infections, with a report of two cases. Am. J. Clin. Pathol. 66: 101-110, 1976.

Rosenberg, D. P., Lerche, N. W., and Henrickson, R. V.: Yersinia pseudotuberculosis infection in a group of Macaca fascicularis. J. Am. Vet. Med. Assoc. 177: 818-819, 1980.

Slide 48

History. An aged female rhesus monkey experienced weight loss, anorexia, and a bloated abdomen. Radiography revealed a gas-distended bowel. A clinical diagnosis of lower bowel obstruction was rendered.

Gross Pathology. There was a firm, gray annular constriction (napkin ring) of the colon, approximately 15 cm proximal to the rectum.

Diagnosis. Adenocarcinoma, mucinous, colon, rhesus monkey.

Comment. These tumors are common in man but rare in animals. Although this tumor has invaded to the serosa, extensions or metastases outside of the colon were not observed in this case. The neoplasm has lakes of mucus as well as numerous glands distended by mucus. There is a moderate amount of desmoplasia associated with some areas of the tumor.

DePaoli et al. consider the increase in the number of reported tumors in nonhuman primates to be related to the increased average age of these animals as a result of the establishment of breeding colonies and regional primate centers. They further report that the association of cancer with advancing age in the nonhuman primate is evident, particularly with malignant neoplasms of the gastrointestinal tract.

Contributor. Sterling-Winthrop Research Institute, Department of Toxicology, Rensselaer, New York.

Suggested reading.

DePaoli, A., and McClure, H. M.: Gastrointestinal neoplasms in nonhuman primates: A review and report of eleven new cases. Vet. Pathol. 19(Supp 7): 104-125, 1982.

Lushbaugh, C. C., Hasson, G. L., Swartzendruber, D. C., et al.: Spontaneous colonic adenocarcinoma in marmosets. Primate Med. 10: 119-124, 1978.

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Slide 49

History. On 4-25-82 an 8-month-old female domestic New Zealand white rabbit (Oryctolagus cuniculus) was noticed to be much thinner than her cage mate. On physical examination two large masses were palpated in the lumbar regions of the kidneys.

Gross Pathology. Gross examination revealed both the right and left kidney to be enlarged, weighing 30 and 36 g respectively. The kidneys had a spongy appearance. A stone was present in the pelvis of the right kidney. Multiple calcified cystic nodules were associated with the common bile duct and pancreas. The parathyroids were normal in size.

Diagnosis. Cysts, multiple, cortex, kidney, New Zealand white rabbit, etiology compatible with congenital polycystic disease.

Comment. The polycystic kidney meets the criteria of Potters Type III classification, which requires normal and cystic nephrons admixed in the involved kidney. With this classification, remnants of glomeruli may be found in some cysts. The cysts are confined to the renal cortex.

In addition to hereditary cortical renal cysts in rabbits, the production of polycystic renal disease in newborn rabbits can be produced by the injection of corticosteroids such as methylprednisolone acetate.

Contributor. Southwestern Medical School, Dallas, Texas.

Suggested reading.

Fox, R. R., Krinsky, W. L., and Crary, D. D.: Hereditary cortical cysts in the rabbit. J. Hered. 62: 105-109, 1971.

Ojeda, J. L., and Garcia-Porrero, J. A.: Structure and development of parietal podocytes in renal glomerular cysts induced in rabbits with methylprednisolone acetate. Lab. Invest. 47: 167-176, 1982.

Osathanondh, V., and Potter, E. L.: Pathogenesis of polycystic kidneys. Arch. Pathol. Lab. Med. 77: 459-465, 1964.

Robbins, S. L.: Pathology Basis of Disease. Philadelphia, W. B. Saunders & Co., 1979, pp. 1123-1127.

Vlachos, J. D.: A new experimental model of polycystic kidneys. Am. J. Dis. Child. 123: 118-121, 1972.

Wiesbroth, S. H.: The Biology of the Laboratory Rabbit, ed. 1. New York and London, Academic Press, 1974, pp. 391-392.

Slide 50

History. This tissue is from a 16-month old female Hereford that was one of a group of animals that died during an outbreak of respiratory disease in a feedlot of 2,000 animals.

Gross Pathology. The tracheal mucosa is overlaid with yellowish exudate. Major bronchi contain mucopurulent exudate. The anteroventral portions of both lungs are firm and dark red in color.

Diagnosis. Bronchopneumonia, fibrinopurulent, acute to subacute, multifocal to diffuse, severe, lung, Hereford, bovine, etiology, compatible with Pasteurella multocida.

Comment. Infectious bovine rhinotracheitis virus and Pasteurella multocida were isolated from the lungs. Tracheitis and necrotizing bronchitis/bronchiolitis attributable to the infectious bovine rhinotracheitis virus is difficult to identify. The bronchopneumonia in this case is very suggestive but not pathognomonic of a Pasteurella infection. There are some fibrin thrombi within alveolar capillaries, prompting consideration of possible disseminated intravascularization.

Forman et al. report that BHV-1 infected macrophages may have suppressed phagocytic activity and this in turn may explain the increased susceptibility of calves to bacterial infection. They go on to suggest that the alterations in immunologic activity of the alveolar macrophages occurs long before morphological changes in the cells become apparent. Allen et al. report that following intranasal infection with BHV-1 there is extensive loss of cilia on tracheal epithelium, thus reducing the efficiency of the mucociliary defense mechanism.

Contributor. Western College of Veterinary Medicine, University of Saskatchewan, Saskatoon, Saskatchewan, Canada S7N 0W0.

Suggested reading.

Allan, E. M., and Mscolla, P. M.: Scanning electron microscopy of the tracheal epithelium of calves inoculated with bovine herpesvirus I. Res. Vet. Sci. 29: 325-327, 1980.

Forman, A. J., and Babiuk, L. A.: Effect of infectious bovine rhinotracheitis virus infection on bovine alveolar macrophage function. Infect. Immun. 35: 1041-1047, 1982.

Jericho, K. W. F., and Darcel, G. C. Q.: Response of the respiratory tract of calves kept at controlled climatic conditions to bovine herpesvirus I in aerosols. Can. J. Comp. Med. 42: 156-167, 1978.

Rehmtulla, A. J., and Thomson, R. G.: A review of the lesions in shipping fever of cattle. Can. Vet. J. 22: 1-8, 1981.

Yates, W. D. G.: A review of infectious bovine rhinotracheitis, shipping fever pneumonia and viral-bacterial synergism in respiratory disease in cattle. Can. J. Comp. Med. 46: 225-263, 1982.

Slide 51

History. This 5-month-old Arabian male foal was admitted to the University of Georgia Veterinary Hospital with diarrhea two weeks before being put to death. The fecal sample taken on the day of admission was negative for Salmonella sp.

Laboratory Results. Salmonella typhimurium type 8 was cultured from the feces one week following the first culture.

Diagnosis. Colitis, necrotizing and suppurative, subacute, diffuse, with multifocal ulceration, severe, colon, Arabian, equine, etiology, Salmonella typhimurium.

Comment. The colonic lesions are considered characteristic of salmonellosis; however, the patchy distribution is somewhat atypical.

Several authors suggest that as many as five negative fecal cultures are needed to rule out salmonellosis and that there is a likelihood that some cases diagnosed clinically as colitis X may be salmonellosis. A neutropenia early in the clinical course of disease followed by a neutrophilia would be suggestive of salmonellosis. Smith et al. suggest that many clinically normal horses harbor salmonella in the intestinal tract and that stress (surgical, nutritional, etc.) is the key factor in the initiation of severe enteric disease.

Contributor. College of Veterinary Medicine, University of Georgia, Athens, Georgia.

Suggested reading.

Gillespie, J. H., and Timoney, J. F.: Hagan and Bruner's Infectious Diseases of Domestic Animals, ed. 7. Ithaca, New York, Cornell University Press, 1981, pp. 84-93.

Owen, R. R., Fullerton, J. N., Tizard, I. R., et al.: Studies on experimental enteric salmonellosis in ponies. Can. J. Comp. Med. 43: 247-254, 1979.

Smith, B. P., Reina-Guerra, M., and Hardy, A. J.: Prevalence and epizootiology of equine salmonellosis. J. Am. Vet. Med. Assoc. 172: 353-356, 1978.

Smith, B. P., Reina-Guerra, M., Hardy, A. J., et al.: Equine salmonellosis: Experimental production of four syndromes. Am. J. Vet. Res. 40: 1072-1077, 1979.

Slide 52

History. This tissue is from an alligator (Alligator mississippiensis).

Diagnosis. Granuloma, with fungal hyphae, multifocal and coalescing, severe, lung, alligator (Alligator mississippiensis).

Comment. While the morphologic characteristics of the fungus in the lung lesions are morphologically compatible with Metarhizium anisopliae, a culture is considered necessary for a definitive diagnosis. Differential diagnoses in addition to Aspergillus sp. include Beauveria bassiana and Paecilomyces sp. The latter has only been reported once in an alligator but may prove to be a potent pathogen of reptiles (Austwick et al.). Beauveria bassiana is known to be fatal in alligators, but Frostling et al. consider this to be an accidental pathogen in vertebrates.

Metarhizium anisopliae is a green muscardine fungus that is used as a pesticide. It occurs in soil and on plant remains, and its pathogenicity to reptiles has been confirmed by the successful inoculation of a lizard. The appearance and color of the hyphal mats in the airways of the affected

alligator closely resemble those of Aspergillus fumigatus as seen in the airsacs in avian aspergillosis. In lesions, the hyphae can reach 7 um in diameter and are generally thin-walled and contorted. Within this host, gross lung lesions include: emphysema with miliary nodules in the parenchyma: soft, hollow bullae lined with dark green fungal mats: mycetomata up to 50 mm in diameter within airways: trachea and bronchi with raised yellow, firm lesions up to 20 mm in diameter: and lingual ulcers.

Contributor. Cardiothoracic Institute, University of London, Brompton Hospital, Frimley, Surrey, GU16 5QE, United Kingdom.

Suggested reading.

Austwick, P. K. C., and Keymer, I. F.: Fungi and actinomycetes. In Diseases of the Reptilea, edited by Cooper, J. E., and Jackson, O. F., New York, Academic Press, 1981, vol. I, pp. 193-231.

Frostling, R. A., Jensen, J. M., Robinson, B. E., et al.: Fatal mycotic pulmonary disease of captive alligators. Vet. Pathol. 16: 428-431, 1979.

Slide 53

History. This mass from a 4-month-old male Fischer rat was detected at necropsy.

Diagnosis. Nephroblastoma, kidney, Fischer rat.

Comment. This neoplasm contains some well-differentiated tubules that blend into undifferentiated blastema-type stromal tissue. The neoplasm has a distinct lobulated appearance, and glomeruloid structures may be visualized. Nephroblastomas are generally considered to be the most common renal tumor found in rats.

Hard et al. state that the nephroblastoma in the rat can be exclusively an epithelial neoplasm with the connective tissue component being a benign connective tissue reaction of host origin.

In man, hypertension is an important finding associated with phroblastomas that is mediated by the renin-angiotensin system as a result of renal arterial obstruction, perirenal compression, or production of a humoral pressor agent by the tumor itself (Bresnahan et al.). Bresnahan et al. report a nephroblastoma in a rat that died of a ruptured aortic aneurysm as a result of suspected hypertension.

Contributor. Department of Pathology, TheraGenics, Inc., 1800 East Pershing Road, Decatur, Illinois.

Suggested reading.

Bresnahan, J. F., and Wagner, J. E.: Nephroblastoma with associated aortic rupture in a rat. Lab. Anim. Sci. 32: 169-170, 1982.

Hard, G. C., and Noble, R. L.: Spontaneous rat nephroblastoma. Arch. Pathol. Lab. Med. 106: 418-422, 1982.

Migaki, G., and Casey, H. W.: Comparative pathology of nephroblastoma in humans and animals. In Tumours of Early Life in Man and Animals, edited by Severi, L. Perugia, Perugia Quadrennial International Conferences on Cancer, 1978, pp. 1053-1069.

Slide 54

History. This 8-week-old male New Zealand white rabbit (Oryctolagus cuniculus) developed a single, large nodular cutaneous mass on the top of its head that appeared to grow rapidly over a 3-week period. This was the only animal affected out of fourteen rabbits maintained in outdoor hutches.

Gross Pathology. On surgical removal, the tumor appeared grossly as a superficially ulcerated spherical nodule involving both the intercutaneous and subcutaneous tissue. The tumor measured 4 cm in diameter with no evidence of invasion into the subjacent bone. The lesion had a firm consistency and was pale and glistening on the cut surface.

Diagnosis. Fibroma, dermis, with epidermal intracytoplasmic inclusion bodies, skin, rabbit, etiology compatible with Shope fibroma virus (SFV).

Comment. The Shope or rabbit fibroma is a viral-induced neoplasm caused by a leporipoxvirus and is believed to be spread by arthropods from the wild rabbit population (where the condition is enzootic) to domestic rabbits (Pulley et al. and Raflo et al.). These tumors are characterized by (1) intracytoplasmic inclusion bodies that are obvious in the prickle cell layer

and less obvious in the neoplastic proliferation, (2) a diffuse proliferation of pleomorphic fibroblastic-to-histiocytic cells in the dermis, and (3) a prominent necroinflammatory component composed of lymphocytes, plasma cells, and macrophages (Pulley et al. and Prose et al.).

According to Sell et al., tumor regression in adult rabbits with the Shope fibroma is associated with a decline in infectious virus in the tumor, the appearance of circulating antibody, the development of delayed cutaneous reactivity, the inhibition of macrophage migration in vitro in the presence of fibroma antigen, and the acquisition of killer T-cells specific for fibroma-infected target cells in vitro. They report that no correlation between interferon production and fibroma rejection has been found. They further indicate that SFV has a systemic effect on the reticuloendothelial system of the neonatal rabbit that may be responsible for progressive tumor formation.

In a recent report by Pogo et al., it was concluded that the Shope fibroma virus-1 (SFV-1) genome contains information for both cytolysis and tumorigenesis. The authors go on to state that this unusual virus may be a recombinant between an orthopoxvirus and a leporipoxvirus. The SFV-W strain did not produce cytopathic effects in the study by Pogo et al. but was tumorigenic. It is not known if the cytocidal genes are present but repressed in the SFV-W strain.

Contributor. Department of Pathology, Michigan State University, East Lansing, Michigan.

Suggested reading.

Pogo, B. G. T., Preimuth, P., and Stein, A.: Shope fibroma virus. I. Biological and molecular properties of a cytocidal and a noncytocidal strain. J. Virol. 41: 97-103, 1982.

Prose, P. H., Preidman-Klein, A. E., and Vilcek, J.: Morphogenesis of rabbit fibroma virus. Am. J. Pathol. 64: 457-481, 1971.

Pulley, L. T., and Shively, J. N.: Naturally occurring infectious fibroma in the domestic rabbit. Vet. Pathol. 10: 509-519, 1973.

Raflo, C. P., Olsen, R. G.; Pakes, S. P., et al.: Characterization of a fibroma virus from naturally occurring skin tumors in domestic rabbits. Lab. Anim. Sci. 23: 525-532, 1973.

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Tompkins, W. A. F., and RamaRao, G. V. S. V.: Defective macrophage immunity in newborn rabbits with fibroma virus-induced tumors. J. Reticuloendothel. Soc. 23: 161-166, 1978.

Slide 55

History. This male Cynomolgus monkey (Macaca fascicularis) was obtained from a primate importer. This monkey was one of 65 primates in a colony that died or was put to death.

Laboratory Results. Mycobacterium tuberculosis was cultured from the trachea, spleen, liver, and lungs.

Diagnoses. Pyogranulomas, multifocal and coalescing, moderate to severe, lung and liver, Cynomolgus monkey (Macaca fascicularis), etiology Mycobacterium tuberculosis. Pleuritis, chronic, diffuse, moderate, lung. Hepatitis, portal, subacute, diffuse, mild.

Comment. In the center of some of the granulomas there is caseous necrosis, one of the hallmarks of this disease. The relationship of the pleuritis to the mycobacteria could not be established, although pleuritis is commonly seen with tuberculosis in a variety of species. Hepatic portal inflammatory infiltrates, similar to those present in this case, are a common finding in research primates. Hepatocytes, in the sections examined, frequently contain variable amounts of glycogen. As is typical with this organism, acid-fast stains of the lung and liver rarely demonstrate organisms. This disease is infrequently seen in other than newly imported primates.

Contributor. Pathology Section, National Institute Occupational Safety & Health, DHHS, Cincinnati, Ohio.

Suggested reading.

Benirschke, K., Garner, F. M., and Jones, T. C.: Pathology of Laboratory Animals. New York, Springer-Verlag, 1978, vol. II, pp. 1418-1427.

Gillespie, J. H., and Timoney, J. F.: Hagan and Bruner's Infectious Diseases of Domestic Animals. Ithaca, Cornell University Press, 1981, pp. 247-265.

Montali, R. J.: Mycobacterial Infections of Zoo Animals. Washington, D.C., Smithsonian Institution Press, 1978.

Robbins, S. L.: Pathologic Basis of Disease. Philadelphia, W. B. Saunders & Co., 1979, pp. 396-404.

Wolinsky, E.: Nontuberculous mycobacteria and associated diseases. Am. Rev. Respir. Dis. 119: 107-159, 1979.

Slide 56

History. This 30-year-old female orangutan (Pongo pygmaeus) had repeated episodes of diarrhea and upper respiratory disease over a 6-month period. Strongyloides sp. organisms were occasionally found in the feces. Two months before her death, an episode of vaginal bleeding was reported. An exploratory laparotomy done one week before death revealed a severe chronic peritonitis.

Gross Pathology. At necropsy, a ruptured gallbladder and a severe chronic peritonitis were found. Three cholioliths were obstructing the cystic duct of the gallbladder. In the pelvic region, numerous fibrous adhesions were observed between the uterus, colon, and urinary bladder. A large poorly defined yellow polycystic mass was adherent to the body of the uterus.

Diagnosis. Granulosa cell tumor, ovary, orangutan (Pongo pygmaeus).

Comment. This tumor is composed of small rounded and elongated cells that have high nuclear-to-cytoplasmic ratios and poorly defined cell margins. The cells are arranged in solid lobules of various sizes, a few of which have small cystic centers. In many lobules, rosette formation is evident around small focal proteinaceous deposits or capillaries. Dilated ductular structures lined by a single layer of ciliated columnar epithelium course through the tumor. These ductlike structures are thought to be segments of the fallopian tube.

The nuclei in many of the tumor cells were slightly pale with a prominent dark-staining groove that is considered by Scully to be a common finding with this tumor. Granulosa cell tumors may have many different patterns and include areas showing luteinization.

The gross and histologic appearances of the tumor are considered characteristic of a granulosa cell tumor. The focal deposits of proteinaceous material within lobules resemble the Call-Exner bodies observed in human granulosa cell tumors. No evidence of tumor metastasis was found. No ovarian tumors have been previously reported in orangutans.

In addition to the granulosa cell tumor, this animal also had endometriosis and adenomyosis. Many of the adhesions between the pelvic viscera were caused by the endometriosis.

Contributor. New Mexico Veterinary Diagnostic Services, Albuquerque, New Mexico.

Suggested reading.

Graham, C. E., and McClure, H. M.: Ovarian tumors and related lesions in aged chimpanzees. Vet. Pathol. 14: 380-386, 1977.

Moulton, J. E.: Tumors in Domestic Animals, ed. 2. Berkeley, University of California Press, 1978, pp. 111-136.

Nielsen, S. W., Misdorp, W., and McEntee, K.: XV. Tumors of the Ovary. Bull. W.H.O. 53: 213-214, 1976.

Scully, R. E.: Tumors of the Ovary and Maldeveloped Gonads, Ser. 2, Fascicle 16, of Atlas of Tumor Pathology. Washington, D.C., Armed Forces Institute of Pathology, 1979 [1980], pp. 153-173.

Slide 57

History. This tissue is from one of several wild fowl with a history of a high rate of mortality that were submitted for necropsy. Many types of birds were involved, but the majority were diving and old squaw ducks.

Gross Pathology & Laboratory Results. Gross lesions consisted of petechial and ecchymotic hemorrhages on the epicardia. Livers were enlarged and revealed multiple small whitish foci. Pasteurella multocida organisms were isolated from the liver and spleen samples. Cultures for the presence of other organisms like Erysipelas and Salmonella were negative.

Diagnosis. Hepatitis, necrotizing, acute, multifocal, moderate, with numerous phagocytosed and extracellular bacteria, liver, avian, etiology, Pasteurella multocida.

Comment. The hepatic histopathologic changes are considered to be characteristic of avian Pasteurella multocida infections. Hepatocytes and Kupffer cells occasionally contain bacteria, but some of the extracellular bacterial proliferation is considered to be postmortem growth. The cause of death was probably related to septicemia. Hunter et al. report that death in the case of avian cholera is likely to be due to terminal endotoxemia: however, the microscopic lesions in the liver sections examined in this case do not reflect this.

Contributor. Animal Health Laboratory, Centreville, Maryland.

Suggested reading.

Heddlestone, K. L.: Avian Pasteurellosis. In Diseases of Poultry, ed. 7, edited by Hofstad, M. S., et al. Ames, Iowa State University Press, 1978, pp. 188-191.

Hunter, B., and Wobeser, G.: Pathology of experimental avian cholera in mallard ducks. Avian Dis. 24: 403-414, 1980.

Montgomery, R. D., and Stein, G.: The 1978 epornitic of avian cholera on Chesapeake Bay. Avian Dis. 23(4): 965-978, 1979.

Whiteman, C. E., and Bickford, A. A.: Avian Disease Manual. Fort Collins, Colorado State University Press, 1979, pp. 82-85.

Slide 58

History. This tissue is from a cockatoo from a group of Moluccan cockatoos that died acutely with diarrhea and terminal convulsions. At necropsy, 2 ml of a thin, straw-colored ascitic fluid were found. The liver was mottled with numerous petechiae on the serosal surface, and there was moderate splenomegaly.

Laboratory Results. A herpesvirus was isolated in tissue culture and by chick embryo inoculation.

Diagnosis. Necrosis, multifocal, moderate to severe, with intranuclear inclusion bodies, liver and spleen, Moluccan cockatoo, avian, etiology, herpesvirus (Pacheco's parrot disease).

Comment. A morphologic diagnosis of necrotizing hepatitis may be preferred by some; however, the primary lesion is necrosis and not inflammation. While most prominent in periarteriolar sheaths, there are also foci of splenic necrosis in the red pulp. Characteristic herpetic intranuclear inclusion bodies are abundant in the liver but rare in the spleen.

The birds were kept for a time in a warehouse that also contained Nanday conures. This species of conure can be an asymptomatic carrier of the virus.

Contributor. Experimental Pathology Laboratories, Inc., Herndon, Virginia.

Suggested reading.

Hitchner, S. B., et al. (Eds.): Isolation and Identification of Avian pathogens, ed. 2. New York, Creative Printing Company, Inc., 1980, pp. 98-101.

Miller, T. D., Millar, D. L., and Naqi, S. A.: Isolation of Pacheco's disease herpesvirus in Texas. Avian Dis. 23: 753-756, 1979.

Petrak, M. L.: Diseases of Cage and Aviary Birds. Philadelphia, Lea & Febiger, 1982, pp. 524-525.

Simpson, C. F., Hanley, J. E., and Gaskin, J. M.: psittacine herpesvirus infection resembling Pacheco's parrot disease. J. Infect. Dis. 131: 390-396, 1975.

Simpson, C. F., and Hanley, J. E.: Pacheco's parrot disease of psittacine birds. Avian Dis. 21: 209-219, 1977.

Slide 59

History. This tissue is from an adult female rhesus monkey (Macaca mulatta) in a large free-ranging breeding colony. There was a growth protruding from the mandibular gum area.

Diagnosis. Squamous cell carcinoma, oral cavity, rhesus monkey (Macaca mulatta).

Comment. There is a proliferation of neoplastic squamous epithelium with extensive invasion of the underlying fibrous connective tissue. Features include increased mitoses, pleomorphism of nuclei, giant nuclei, keratin pearl formation, individual cell keratinization, and hyperchromatism.

A differential diagnosis of a mucoepidermoid carcinoma was considered; however, a mucicarmine stain was negative. Oral tumors in nonhuman primates are considered rare, with the increased reporting of such tumors possibly being related to the prolonged lifespan of colony-maintained monkeys.

Contributor. Pathobiology & Primatology Branch, Division of Product Quality Control, Bureau of Biologics, FDA, Bethesda, Maryland.

Suggested reading.

Head, K. W.: Tumours of the upper alimentary tract. Bull. W.H.O. 53: 149, 1976.

Moulton, J. E. (Ed.): Tumors of Domestic Animals, ed. 2. Berkeley, University of California Press, 1978, pp. 45-46.

Slide 60

History. This 16-month-old mustang that was caught in the wild was admitted to a teaching hospital with a primary complaint of difficulty in prehension. When attempting to drink water, it bit at the water. Its lips were retracted, and the horse was emaciated.

Diagnosis. Malacia, symetrical, diffuse, severe, substantia nigra, midbrain, equine, etiology, compatible with yellow star thistle toxicity.

Comment. The histopathologic lesions in the brain of this horse are characteristic of nigropallidal encephalomalacia. Malacia is usually bilateral in the globus pallidus and substantia nigra. While the pathogenesis of this disease remains unclear, there may be a block of oxidative metabolism or a neurotransmitter-substance defect that isolates the lesion to the classic locations. The clinical signs associated with this disease suggest there is a more widespread effect on the brain than the microscopic lesions indicate. The yellow star thistle plant (Centaurea solsitialis is known to cause this disease, and Russian knapweed (Centaurea repens) produces essentially the same lesions.

Contributor. Department of Veterinary Microbiology & Pathology and Washington Animal Disease Diagnostic Lab, School of Veterinary Medicine, Washington State University, Pullman, Washington.

Suggested reading.

Farrel, R. K., Sande, R. D., and Lincoln, S. D.: Nigropallidal encephalomalacia in a horse. J. Am. Vet. Med. Assoc. 158(7): 1201-1204, 1971.

Fowler, M. E.: Nigropallidal encephalomalacia in the horse. J. Am. Vet. Med. Assoc. 147(6): 607-616, 1965.

Larson, K. A., and Young, S.: Nigropallidal encephalomalacia in horses in Colorado, J. Am. Vet. Med. Assoc. 156(5): 626-628, 1970.

Young, S., Brown, W. W., and Klinger B.: Nigropallidal encephalomalacia in horses caused by ingestion of weeds on the genus Centaurea. J. Am. Vet. Med. Assoc. 157(11): 1602-1605, 1970.

Slide 61: L14

History. A 16-year-old castrated male Welsh pony was anorectic for 1 week. Signs of facial paralysis and circling to the right developed. There was evidence of deficits of cranial nerves V, VII, VIII, and XII. Clinical signs progressed, and the animal became recumbent. It was put to death.

Gross Pathology. Multiple yellow, soft areas, measuring 0.5 cm in diameter, were present in the brain stem caudal to the olive. Similar lesions were observed in the left confluence of the cerebellar peduncles, in the right midbrain obliterating the red nucleus, and in the lateral caudal thalamus. The cranial cervical spinal cord was involved. (See kodachrome.)

Laboratory Results. Listeria monocytogenes organisms were cultured from sections of medulla oblongata. Small numbers of small gram-positive rods were detected on frozen sections of medulla oblongata.

Diagnosis. Encephalomeningitis, nonsuppurative, subacute, multifocal, mild to moderate, medulla oblongata, Welsh pony, equine, etiology - Listeria monocytogenes.

Comment. No immunologic deficiency was detected in this pony, and there was no history of contact with ruminants or access to silage. The lesions of listeriosis were limited to the CNS. Listeria infections in horses can cause abortion, septicemia, and encephalitis, but reported cases are few. The route of infection by L. monocytogenes is thought to be umbilical or oral. It also has been reported that the organisms gain entrance to the calvarium via the trigeminal nerves. The organism has been isolated from a variety of environments including silage, mud, feces, sewage, and garden soil.

Jones and Hunt state that the primary brain stem lesion is a circumscribed collection of mononuclear cells, with or without neutrophils, in close proximity to blood vessels but that diffuse cellular infiltration and frank microabscesses may occur with relatively little tissue necrosis.

Many consider the movement of organisms this large within nerves to be unlikely, yet the general restriction of the lesions to the spinal cord and brain stem is confusing if they are of hematogenous origin.

Contributor. Department of Experimental and Comparative Pathology, College of Veterinary Medicine, University of Florida, Gainesville, Florida.

Suggested reading.

Clark, E. G., Turner, A. S., Boysen, B. G., et al.: Listeriosis in an Arabian foal with combined immunodeficiency. J. Am. Vet. Med. Assoc.a 172: 363-366, 1978.

Emerson, F. G., and Jarvis, A. A.: Listeriosis in ponies. J. Am. Vet. Med. Assoc. 152: 1645-1646, 1968.

Gillespie, J. H., and Timoney, J. F.: Hagan and Bruner's Infectious Diseases of Domestic Animals. Ithaca, Cornell University Press, 1981, pp. 221-225.

Jones, T. C., and Hunt, R. D.: Veterinary Pathology, ed. 5. Philadelphia, Lea & Febiger, 1983, pp. 631-634.

Slide 63

History. A 9-year-old spayed German short-hair pointer was in apparent good health. A stool sample was positive for hookworms and whipworms. Telmintic dewormer (Pitman-Moore) was dispensed. The second deworming was started two weeks after completion of the first regimen. On the third day of treatment the dog became incoordinated and would not eat. When seen by the veterinarian the animal was depressed and icteric. The dog died 4 hours later.

Diagnosis. Necrosis, hemorrhagic, massive, diffuse, severe, liver, German short-hair pointer, canine.

Comment. The massive hepatic necrosis is considered to be related to the administration of mebendazole. The modest number of lymphocytes in portal areas are considered to be pre-existing. There is some bile duct obstruction and reticuloendothelial cells frequently contain iron pigment.

Drugs that cause hepatic disease are classified as either intrinsic (dose dependent, short latency period, and reproducible in laboratory animals) or idiosyncratic (nonpredictable). Idiosyncratic hepatotoxicity may reflect an allergic reaction to a drug or a metabolic aberration in the host that results in hepatotoxic metabolite production. This reaction is considered to be of

the idiosyncratic type. The role of aflatoxins or other hepatotoxins should also be considered in the differential diagnosis.

It has not been established if mebendazole has a direct hepatotoxic effe or if drug and environmental interactions increase its toxicity. The drug i used extensively in both human and veterinary medicine, with only a few "idiosyncratic" hepatotoxicities reported in dogs.

Contributor. Department of Veterinary Pathology, College of Veterinary Medicine, University of Missouri, Columbia, Missouri.

Suggested reading.

Polzin, D. J., Stowe, C. M., O'Leary, T. P., et al.: Acute hepatic necrosis associated with the administration of mebendazole to dogs. J. Am. Vet. Med. Assoc. 179: 1013-1016, 1981.

Swanson, J. F., and Breider, M. A.: Hepatic failure following mebendazol administration to a dog. J. Am. Vet. Med. Assoc. 181: 72-73, 1982.

Slide 64, L15-L16

History. An 8-year-old male Bedlington terrier was presented with a history of recurrent vomiting. The dog was slightly dehydrated. (See gross and EM kodachromes.)

Laboratory Results.

Glucose	120 mg/dl (105)*	
Cholesterol	670 mg/dl (250)*	
Total Bilirubin	1.3 mg/dl (.6)*	
Alkaline Phosphatase		1034 U/l (90)*
SGPT	552 U/l (25)*	

Serum Copper - elevated markedly

*high normal value

Histochemistry. Copper granules confirmed using Dimen's stain.
Granules do not contain iron (Perl's).
Fat globules are abundant (ORO).

Diagnosis. Cirrhosis, with hepatocellular granular material, diffuse, moderate, liver, Bedlington terrier, canine, etiology - copper toxicity.

Comment. The first of the kodachromes shows the gross appearance of the liver at necropsy. The second slide demonstrates the copper granules associated with lysosomes in an electron micrograph.

The histopathologic findings, special stains, and laboratory results support a diagnosis of chronic progressive hepatitis caused by copper toxicosis. A morphologic diagnosis of cirrhosis is preferred because of the presence of necrosis, biliary hyperplasia, fibrosis, and regenerative nodules: however, some slides do not demonstrate regenerative nodules and are diagnosed as chronic active hepatitis. Hepatocytes in regenerative nodules have less prominent cytoplasmic granules than those in the remainder of the liver.

This disease is considered to have a hereditary basis and in some ways is similar to Wilson's disease in humans; however, humans with this disease often show extrahepatic signs of the copper toxicosis, such as CNS symptoms, and have low serum copper levels as well as low ceruloplasmin levels. The low total serum copper results from an increase in free copper but a greater decrease in ceruloplasmin-bound copper. Affected dogs show only hepatic signs and have normal serum copper and ceruloplasmin levels. Hemolysis seen in both dogs and man is thought to be caused by a rapid rise in serum copper concentrations as a result of massive hepatocellular necrosis, release of intracellular copper into the peripheral circulation, and a subsequent alteration of red cell membrane stability (Hardy et al.).

Contributor. Roechst-Roussel Pharmaceuticals, Inc., Somerville, New Jersey.

Suggested reading.

Hardy, R. M., and Stevens, J. B.: Chronic progressive hepatitis in Bedlington terriers (Bedlington liver disease). In Current Veterinary Therapy I, Small Animal Practice, edited by Kirk, R. W. Philadelphia, W. B. Saunders Co., 1977, pp. 996-998.

Hardy, R. M., and Stevens, J. B.: Chronic progressive hepatitis in Bedlington terriers. Proceedings of the American Animal Hospital Association, 1978, pp. 187-190.

Hardy, R. M., Stevens, J. B., and Stowe, C. M.: Chronic progressive hepatitis in Bedlington terriers associated with elevated liver copper concentrations. Minn. Vet. 15: 13-24, 1975.

Ludwig, J., Owen, C. A., Benham, S. S., et al.: The liver in the inherited copper disease of Bedlington terriers. Lab. Invest. 43: 82-87, 1980.

Robbins, S. L., and Cotran, R. S.: Pathologic Basis of Disease, ed. 2. Philadelphia, W. B. Saunders Co., 1979, pp. 241-243.

Twedt, D. C., Sternlieb, I., and Gilbertson, S. R.: Clinical, morphologic, and chemical studies on copper toxicosis of Bedlington terriers. J. Am. Vet. Med. Assoc. 175(3): 269-275, 1979.

Slide 65

History. This 14-month-old male Sprague-Dawley rat died spontaneously with a large (3 X 4 cm) ulcerated mass on the left side of the head beneath the ear. The large mass displaced the jaw with resultant malocclusion and emaciation.

Diagnosis. Carcinoma, Zymbal's gland, external ear canal, rat.

Comment. Zymbal's gland carcinomas are a recognized neoplastic entity that can have squamous and sebaceous differentiation at both the primary and metastatic sites. These tumors are generally malignant, but metastasis is rare. While metastasis is generally to a regional lymph node, the sections of lymph node examined in this case were free of the tumor. Invasion of the brain and lung metastasis did occur in this case.

Contributor. Toxicology Research Laboratory, The Dow Chemical Company, Midland, Michigan.

Suggested reading. Pliss, G. B.: Tumors of the auditory sebaceous glands. In Pathology of Tumours in Laboratory Animals, edited by Turusov, V. S. Lyon, IARC Scientific Publications, 1973, vol. 1, pp. 29-30.

Slide 66

History. This tissue is from a 7-year-old Angus cow. Three of 4 ill animals in a group of 40 died after an illness of about 2 weeks. Clinical signs in this animal included bloody mucoid diarrhea. One or more of the other ill animals were constipated. Several had a papular or scaly dermatitis.

Diagnosis. Nephritis, interstitial, granulomatous, multifocal to coalescing, kidney, Angus, bovine, etiology compatible with hairy vetch toxicity.

Comment. The most interesting feature of this case is the presence of numerous multinucleated giant cells in association with a toxic condition. Special stains failed to demonstrate an infectious agent. The lesions in this case are considered characteristic of hairy vetch toxicity, but their pathogenesis remains unclear. A similar syndrome in Wales occurred in cattle without access to vetch. The disease in Wales has been observed in cattle consuming grass silage to which a preservative (sulfuric acid, formalin, methanol urea) was added.

Contributor. Department of Pathology, College of Veterinary Medicine, Oklahoma State University, Stillwater, Oklahoma.

Suggested reading.

Jones, T. C., and Hunt, R. D.: Veterinary Pathology, ed. 5. Philadelphia, Lea & Febiger, 1983, pp. 942-944.

Panciera, R. J.: Hairy vetch (Vicia villosa Roth) poisoning in cattle. In Effects of Poisonous Plants on Livestock, edited by Keeler, VanKampen, and James. New York, Academic Press, 1978, pp. 555-563.

Thomas, G. W.: Pyrexia with dermatitis in dairy cows. In Practice 1: 16-18, 1979. (Supplement to Veterinary Record (London).)

Slide 67

History. A baby male Dama wallaby (Wallabia parma), weighing 1 kg, would not drink milk, was given normosol-a the following day, and died on the third day.

Diagnosis. Enteritis, subacute, diffuse, moderate, with multiple mucosal and submucosal intracellular protozoal schizonts, small intestine, Dama wallaby, etiology, Eimeria sp.

Comment. The presence of this Eimeria sp. in the intestine of the wallaby has created confusion in the past, having been mistaken for Besnoitia sp. According to Hammond, certain stages of some Eimeria spp. invade and develop in mesodermal cells. A number of species of Eimeria including E. bovis, E. gilruthi, and E. leuckarti have unusually large schizonts that are accompanied by considerable hypertrophy and modification of the host cell, with this host-parasite complex behaving as an autonomous organism. The merozoites in the schizonts of Eimeria sp. often form rosettes, as seen in this case.

This protozoa closely resembles Besnoitia spp., but schizonts of Besnoitia spp. are not found in intestinal mucosa and submucosa. Also, the merozoites of Besnoitia spp. are not known to form the rosettes seen in schizonts of many Eimeria spp.

Contributor. USAF School of Aerospace Medicine (VNSP), Brooks AFB, Texas.

Suggested reading.

Hammond, D. M., and Long, P. L.: The Coccidia. Baltimore, University Park Press, 1973, pp. 50-54.

Soulsby, E. J. L.: Helminths, Arthropods and Protozoa of Domesticated Animals, ed. 7. Philadelphia, Lea & Febiger, 1982, pp. 594-619.

Todd, K. S., Jr., and Ernst, J. V.: Coccidia of mammals except man. In Parasitic Protozoa, edited by Kreier, J. P. New York, Academic Press, 1977, vol. III, pp. 71-89.

Slide 68

History. This 3-month-old female Nubian goat did not grow well, and the day prior to submission she acted slightly weak, lying by herself away from the herd until her death that evening. She was one of a set of triplets. She was given sulfaquinoxaline (1 oz per gallon) in a pail for 2 to 3 days prior to her death. Her diet consisted of sheep milk replacer and a grain mixture containing 500 lb of corn, 600 lb of oats, 100 lb of soybean meal, 50 lb of linseed meal, 50 lb of calf manna, 2 lb of vitamins A, D, & E mixture, 10 lb of mineral supplement, and 100 lb of molasses. The goat had been dehorned at approximately 3 to 4 weeks of age.

Diagnosis. Degeneration and necrosis, subacute, focally extensive, severe, skeletal muscle, Nubian, caprine.

Comment. It is not possible to make a definitive diagnosis from this slide. Differential diagnoses included myopathies with a nutritional, neurogenic, metabolic, toxic, or infectious basis, but a nutritional vitamin E-selenium deficiency is the most likely etiology. On some sections there is evidence of early myofiber regeneration. With the possible exception of a lack of mineralization, the myodegenerative changes are considered consistent with those described by Jubb and Kennedy for nutritional myopathies.

Vitamin E and selenium are thought to control peroxidative reactions by independent but related mechanisms, with Vitamin E sequestering free radicals that initiate the peroxidative process and selenium being a component of the enzyme glutathione peroxidase (GSH-Px) that reduces hydrogen peroxide and lipid hydroperoxidases to less reactive metabolities. Recent studies have shown there may be a relationship between the tissue distribution of selenium-dependent and nonselenium-dependent GSH-Px and the lesions associated with Vitamin-E and/or selenium deficiencies in various species.

Contributor. Michigan State University, AHDL, Lansing, Michigan.

Suggested reading.

Jubb, K. V., and Kennedy, P. C.: Pathology of Domestic Animals. New York, Academic Press, 1970, vol. 2, pp. 482-488.

Scholz, R. W., Cook, L. S., and Todhunter, D. A.: Distribution of selenium-dependent and nonselenium-dependent glutathione peroxidase activity in tissues of young cattle. Am. J. Vet. Res. 42: 1724-1729, 1981.

Underwood, E. J.: Trace Elements in Human & Animal Nutrition, ed. 4. 1977, pp. 302-340.

Van Vleet, J. F.: Current knowledge of selenium-vitamin E deficiency in domestic animals. J. Am. Vet. Med. Assoc.a 176: 321-325, 1980.

Slide 69

History. This 7-month-old male quarter horse was presented for progressive weakness and emaciation over a 2-month period. He had prepucial edema and hard swellings over the supraorbital fossae and ligamentum nuchae. Radiography showed mineralized densities within the nuchal ligament. The foal continued to weaken over a 9-day hospital course despite a voracious appetite and was found dead on the ninth day.

Gross Pathology. All fat depots in the animal's body were similarly affected. The adipose tissue was firm and yellow-brown and fractured easily. On cutting, it was gritty and had chalky white foci throughout. The cut surface of the nuchal ligament had similar chalky foci. Supraorbital swellings were composed of similarly affected adipose tissue. Skeletal and cardiac muscle were grossly normal.

Laboratory Results. Serum levels of arsenic, cadmium, mercury, lead, and thallium were insignificant. The serum selenium leovel of 0.030 ug/ml was considered unusually low. Insufficient serum was collected for alpha tocopherol assay.

Diagnosis. Steatitis and necrosis with mineralization, diffuse, moderate, periadrenal adipose tissue, quarter horse, equine, etiology, compatible with vitamin E-selenium deficiency.

Comment. The histopathologic changes are compatible with a diagnosis of a vitamin E-selenium deficiency syndrome. The yellow-brown pigment (ceroid) within macrophages is PAS-positive in addition to being acid-fast and autofluorescent. Mineralized foci are positive with a von Kossa stain for phosphates and carbonates, and an alizarin red stain is positive for calcium salts.

There is a spectrum of lesions associated with vitamin E-selenium deficiency syndromes. The role of Vitamin-E as a free radical scavenger, the role of selenium and glutathione peroxidase in inactivating free radicals, and the role of excess dietary polyunsaturated fatty acids must be considered in the pathogenesis of the lesions.

A similar syndrome has been reported in New Zealand and Europe either with or without accompanying muscular degeneration. A vitamin E-selenium deficiency etiology was postulated but not proven. The serum selenium levels in this foal were considered extremely low. The soil of the Pacific Northwest is endemically low in selenium, and typical Vitamin E-selenium deficiency syndromes in all species are common. Although several other foals on the same farm were unaffected, the low dietary selenium is a likely etiology in this case.

Contributor. Department of Veterinary Microbiology and Pathology, Washington State University, Pullman, Washington.

Suggested reading.

Dodd, D. C.: Muscle degeneration and yellow fat disease in foals. N.Z. Vet. J. 8: 45-50, 1960.

Hartley, W. J., and Dodd, D. C.: Muscular dystrophy in New Zealand livestock. N.Z. Vet. J. 5: 61-66, 1957.

Kroneman, J., and Wenswoort, P.: Muscular dystrophy and yellow fat disease in Shetland pony foals. Neth. J. Vet. Sci. 1(1): 42-49, 1968.

Platt, H., and Whitwell, K. E.: Clinical and pathological observations on generalised steatitis in foals. J. Comp. Pathol. 81: 449-506, 1971.

Slide 70

History. An 8-year-old female German shepherd was presented for chronic regurgitation immediately after eating and for weight loss. At necropsy, a large fibrotic mass was found enveloping the base of the heart, hilus, and esophagus.

Diagnosis. Pyogranulomatous, multifocal and coalescing, severe, with central yeast forms, mediastinum, thorax, German shepherd, canine, etiology, compatible with Coccidioides immitis.

Comment. The yeast forms are characteristic Coccidioides immitis spherules, some containing endospores. In the dog, C. immitis lesions are

often found in the lungs and thoracic lymph nodes; confinement to the mediastinum is considered unusual in this species. No pulmonary involvement was noted other than the mass at the hilus, nor was there any evidence of disseminated disease.

Contributor. Pathology Division, US Army Medical Research Institute of Infectious Disease, Fort Detrick, Maryland.

Suggested reading.

Ajello, L. (Ed.): Coccidioidomycosis. Proceedings of the 2nd Coccidioidomycosis Symposium. Tucson, University of Arizona Press, 1967.

Chandler, F. W., Kaplan, W., and Ajello, L.: Color Atlas and Text of the Histopathology of Mycotic Diseases. Chicago, Year Book Medical Publishers Inc, 1980, pp. 50-53, 182-189.

Emmons, C. W., Binford, C. H., Utz, J. P., et al.: Medical Mycology, ed. 1. Philadelphia, Lea & Febiger, 1977, pp. 230-253.

Jungerman, P. F., and Schwartzman, R. M.: Coccidioides immitis. In Veterinary Medical Mycology. Philadelphia, Lea & Febiger, 1972.

Slide 71

History. This 8-month-old male kelpie cross was dosed orally with a mixture of lead chloride, lead bromide, and lead sulphate in 1:1:2 proportions respectively at the rate of 5mg/kg/day for 50 days, to give a total dosage of 1.2 g. The dog was maintained on a high-fat, low-calcium diet. Clinical changes were noted on day 24 and on day 50. On day 24 the dog was noted to be

unusually aggressive. On day 50 the dog had 3 convulsive episodes approximately 30 minutes in duration. Between fits the dog circled and appeared to be blind. The animal died during the third fit.

Laboratory Results.

Tissue lead levels (ppm). Radius - 111, Calvarium - 144, Lumbar vertebra - 227, Liver - 45, Kidney - 126, Brain (frontal lobe) - 3.3. Blood - 112 ug/l (pretreatment - 18 ug/dl).

Diagnosis. Spongiosis and foci of endothelial proliferation, focally extensive, mild to moderate, cerebrum, etiology compatible with lead toxicity

Comment. This section demonstrates the essentially spongiform character the cerebrocortical lesions, their tendency for laminar involvement, and the proliferative vascular reaction.

The capillary proliferation in the cerebrum is very suggestive of lead toxicity with radiation injury as the major differential diagnosis. Neuronal death is not a prominent feature of lead poisoning, as illustrated by this case.

According to Stowe et al., a high-fat, low-calcium diet increases the absorption of lead by as much as ten times. Lead-induced endothelial damage the brain leads to lead accumulation that results in convulsions when brain lead levels reach 3 ppm.

Contributor. University of Melbourne, Veterinary Clinical Centre, Werribee, Victoria, Australia 3030.

Suggested reading.

Robbins, S. L.: Pathologic Basis of Disease. Philadelphia, W. B. Saunders & Co., 1979, pp. 537-539.

Stowe, H. D., and Vandevelde, M.: Lead-induced encephalopathy in dogs fed high fat, low calcium diets. J. Neuropathol. Exp. Neurol. 38: 463-474, 1979.

Zook, B. C.: The pathologic anatomy of lead poisoning in dogs. Vet. Pathol. 9: 310-327, 1972.

Slide 72

History. This tissue is from a 12 and 1/2-year-old neutered female miniature poodle with presenting signs of right-sided exophthalmia, blindness, and corneal ulceration. The eye was surgically removed.

Gross Pathology. A large mass was attached to the posterior globe and enveloped the optic nerve.

Diagnosis. Meningioma, extracranial, retrobulbar, canine.

Comment. A large cellular mass was attached to the posterior border of the sclera and completely surrounded the optic nerve, which was undergoing early compression atrophy. The neoplastic cells are round to ovoid in shape and are arranged as compact sheets and islands in a fine fibrous stroma. Some of the

neoplastic cells exhibit a whorling pattern. The tumor contains numerous foc of metaplastic cartilage, some of which is calcified. The mass did not appea to be encapsulated, and it was not possible to determine whether the meningio originated from the optic nerve or intracranial meninges (via extension).

Examination of the cornea revealed a large necrotic ulcerated area that contained a fibrous exudate and foci of active fibroplasia. The iris was adhered to the affected portion of the cornea. The retina showed marked swelling and degenerative changes.

Contributor. New Hampshire Veterinary Diagnostic Laboratory, University of New Hampshire, Durham, New Hampshire.

Suggested reading.

Biryukovihci, N.: Orbital meningioma with intraocular invasion in a dog. Histology and ultrastructure. Vet. Pathol. 14: 521-529, 1977.

Moulton, J. E.: Tumors in Domestic Animals. Berkeley, University of California Press, 1978, pp. 439-441.

Slide 73

History. This tissue is from a wild eagle that was found dead. Lesions were confined to the lungs.

Laboratory Results. Mycobacterium avium-serotype I was cultured from the pulmonary lesions.

Diagnosis. Necrogranulomas, parabronchi, multifocal, severe, lung, eagle, etiology Mycobacterium avium.

Comment. The large number of acid-fast organisms present in this case is considered typical for Mycobacterium avium infections; however, it is very unusual for the lesions to be confined to the lung. M. avium lesions in birds are usually found in the digestive organs and spleen, with osteomyelitis and bone marrow involvement being common. M. avium is rarely isolated from free-living birds.

Contributor. Syntex Research Laboratories, Palo Alto, California.

Suggested reading.

Gillespie, J. H., and Timoney, J. F.: Hagan and Bruner's Infectious Diseases of Domestic Animals. Ithaca, Cornell University Press, 1981, pp. 247-266.

Montali, R. J., Bush, M., and Smith, E. E.: Pathology of tuberculosis in captive exotic birds. IN Mycobacterial Infections of Zoo Animals, edited by Montali, R. J. Washington, D.C., Smithsonian Institution Press, 1978, pp. 209-215.

Wolinsky, E.: Nontuberculous mycobacteria and associated diseases. Am. Rev. Respir. Dis. 119: 107-159, 1979.

Slide 74

History. This 9-month-old female cockatoo had diarrhea for several weeks. The bird was treated with Amoicillin, Keflex, and eventually Gentocin. No response was observed. Fecal examination was negative for parasites.

Diagnosis. Granuloma, chronic, multifocal, liver, cockatoo, avian, etiology trematode eggs. Intracytoplasmic organisms, disseminated, liver. Necrosis, multifocal, liver, etiology, compatible with Chlamydia psittaci.

Comment. The numerous intracellular, round, Price-Giemsa stain-positive organisms of various sizes (less than 1.0 u) are compatible with Chlamydia. Culture results are considered necessary for a definitive diagnosis of Chlamydia psittaci infection. The size variability may represent elementary and initial bodies. The granulomas appear to be intraductal.

The fluke is most likely a dicrocoelid, possibly Platynosum sp. A negative fecal examination may be due to the eggs not being shed at the time of the test; however, a routine fecal flotation will not usually demonstrate trematode eggs.

Contributor. Veterinary Reference Laboratory, Salt Lake City, Utah.

Suggested reading.

Arnstein, P., and Meyer, K. F.: Psittacosis and ornithosis. In Diseases of Cage and Aviary Birds, edited by Petrak, M. L., Philadelphia, Lea & Febiger, 1982, pp. 528-534.

Gillespie, J. H., and Timoney, J. F.: Hagan and Bruner's Infectious Diseases of Domestic Animals. Ithaca, Cornell University Press, 1981, pp. 334-344.

Page, L. A.: Avian chlamydiosis (ornithosis). In Disease of Poultry, ed. 7, edited by Hofstad et al. Ames, Iowa State University Press, 1978, pp. 337-365.

Strano, A. J., and Binford, C. H.: Ornithosis (psittacosis). In Pathology of Tropical and Extraordinary Diseases, edited by Binford, C. H., and Connor, D. H. Washington, D.C., Armed Forces Institute of Pathology, 1976, pp. 76-78.

Slide 75

History. This 9-day-old male giraffe calf was born with difficulty at a zoo in the southeastern United States and did not receive colostrum. It remained recumbent unless forced to stand. Evidence of cerebellar deficiency was noted. It was treated with ampicillin, gentamicin, and fluid therapy from birth until death.

Gross Pathology. Starting with the distal third of the esophagus and extending into the rumen and reticulum, there was a diffuse, white, raised granular layer that left an eroded area when scraped away. Multiple areas of ulceration measuring up to 2 cm in diameter were seen in the rumen, especially over the pillars.

Diagnosis. Esophagitis, acute, with intraepithelial microabscesses and fungal elements, diffuse, severe, esophagus, giraffe, Giraffidae, etiology - compatible with Candida albicans.

Comment. The histomorphologic features of the fungal hyphae including alignment perpendicular to the mucosal surface, a width of 2 to 3 u with bulbous thickenings, septations, and nondichotomous branching are considered characteristic of Candida. The large infiltrate of neutrophils is considered to be somewhat unusual as is the presence of minimal hyperkeratosis in the sections examined. Binford et al. state that a purulent reaction is present with acute candidiasis and a lymphocytic component increases with time. Culture results are needed for species identification of the fungus. The candidiasis is probably secondary to prolonged antibiotic therapy. There was neither gross nor microscopic evidence of cerebellar lesions.

In their study on C. albicans-induced immunosuppression, Rivas et al. report that B-lymphocyte responses are often normal and occasionally hyperactive. They suggest that a component of the Candida cell may induce suppressor B-cell activity and that these cells may contribute to the depressed T-cell responsiveness.

Contributor. Department of Veterinary Pathology, College of Veterinary Medicine, University of Georgia, Athens, Georgia.

Suggested reading.

Binford, C. H., and Connor, D. H. (Eds.): Pathology of Tropical and Extraordinary Diseases. Washington, D.C., Armed Forces Institute of Pathology, 1976, vol. 2, pp. 568-569.

Jones, T. C., and Hunt, R. D.: Veterinary Pathology, ed. 5. Philadelphia, Lea & Febiger, pp. 695-696.

Myerowitz, R. L.: Gastrointestinal and disseminated candidiasis. Arch. Pathol. Lab. Med. 105: 138-143, 1981.

Rivas, V., and Rogers, T. J.: Studies on the cellular nature of Candida albicans induced suppression. J. Immunol. 130: 376-379, 1983.

Slide 76

History. This tissue is from the skin of two fish from a major aquarium that have nodular lesions on the body and dorsal fins.

Gross Pathology. External examination revealed numerous white raised lesions measuring about 1 to 2 mm in diameter.

Diagnosis. Cytomegaly with cytoplasmic inclusions, multifocal, dermis, fish, pisces, etiology - iridovirus (lymphocystis disease virus).

Comments. The lesions are typical of lymphocystis disease virus. The marked fibroblast hypertrophy, hyaline capsule, granular cytoplasm, and intracytoplasmic basophilic inclusion bodies are pathognomonic for this disease. Lymphocystis disease virus is thought to be transmitted by ingestion, via the gills, or through skin wounds. Lesions usually develop 2 to 4 weeks after exposure. Infected fish remain carriers after the skin nodules slough, which usually occurs after several months.

Many sections contained intracellular protozoal spores in the skeletal muscle. There is no inflammatory response to the spores. These spores are considered to be of the order Myxosporidia and are an incidental finding.

Contributor. Department of Pathology, University of Maryland School of Medicine, Baltimore, Maryland.

Suggested reading.

Nigrelli, R. F., and Ruggieri, G. D.: Studies on virus diseases of fishes. Spontaneous and experimentally induced cellular hypertrophy (lymphocystis disease) in fishes of the new York Aquarium with a report of new cases and an annotated bibliography (1874-1965). Zoologica 50: 83, 1965.

Ribelin, W. E., and Migaki, G.: The Pathology of Fishes. Madison, University of Wisconsin, 1975, pp. 248-249.

Roberts, R. J.: Fish Pathology. London, Baillience Tindall, 1978, p. 116.

Spitzer, R. E., Koch, E. A., Reid, R. B., et al.: Metabolic-morphologic characteristics of the integument of teleost fish with mature lymphocystis nodules. Cell Tissue Res. 222: 339-357, 1982.

Slide 77, L17-L18

History. A 14-year-old female mixed-breed Boston terrier had a skin tumor on the ventral thorax and two separate tumors on the neck. All lesions were similar in gross appearance. They were composed of subepidermal skin nodules with pale, white, moist, firm, homogenous cut surfaces. The margins were sharply demarcated except for occasional infiltration into the underlying connective tissue and adjacent stroma. There was no encapsulation. The overlying skin was unremarkable. (See EM 2 X 2's.)

Diagnosis. Carcinoma, neuroendocrine cell origin (Merkel cell), dermis and subcutis, skin, Boston terrier-mix, canine.

Comment. The tumor cells are compactly grouped into irregular solid sheets, trabeculae, strands, and cords with a scanty supporting stroma. In places, the tumor cells form pseudorosettes around small blood vessels. Strands and cords of tumor cells infiltrated among bundles of collagen. Permeation of the underlying subcutaneous fat is also noted. Sprinkled among the tumor cells are a few polymorphonuclear cells and lymphocytes. The overlying epidermis is free of tumor.

By electron microscopy the tumor cells are found to be tightly packed and form acinar structures. Cells are joined infrequently by desmosomes. Golgi zones are active, and there are considerable numbers of dilated rough endoplasmic reticulum profiles. Several cells contain scattered dense secretory-type granules, and rare cells contain well-developed stage III premelanosomes, some with a paracrystalline appearance.

The electron-microscopic features of this tumor suggest a Merkel cell origin (trabecular carcinoma). The light microscopic features are not considered typical of a trabecular carcinoma as described in man, but the sectioning-induced artefacts that were present on the slides examined made thorough evaluation difficult. It was assumed from the history that the multiple skin tumors were all the same, bringing up the question of whether the tumors were primary or metastatic. Metastatic tumors to the skin are very rare. Trabecular carcinomas have been reported in multiple cutaneous sites in humans.

Trabecular carcinoma of the skin is a rare but distinct neoplastic disease. It initially and primarily involves the skin but later often involves lymph nodes and becomes widely disseminated. The patients survive for 2.25 to 10 years after the disease is first diagnosed (Toker et al.). The disease occurs in elderly patients of both sexes. The cell of origin of trabecular carcinoma of the skin was suggested by Tang and Toker as being one of the neural crest derivatives, most probably the Merkel cell. Hashimoto suggested that Merkel cells are migratory neural crest cells, which enter the epidermis or settle in the dermis. Ultrastructurally, the Merkel cell contains membrane-bound granules and occasional melanosomes. In view of the multipotential nature of these neural crest cells, it is not surprising to encounter a case in which not only neurosecretory granules but also premelanosomes were found. This finding reinforces the impression that this tumor originates from the neural crest.

Previous reports on trabecular carcinomas in humans suggest that the tumor cells may contain melanosomes, but the recent report by Zak et al. states that basal lamina material, tonofilaments, and melanosomes are not present within these tumor cells. According to Kroll et al., the presence of neurosecretory granules is not proof of derivation and it may merely denote a line of differentiation.

Contributor. C.E. Nord Animal Disease Diagnostic Laboratory, Division of Animal Industries, Nashville, Tennessee.

Suggested reading.

Hashimoto, O. O.: The ultrastructure of the skin of human embryo. X. Merkel tactile cells in the finger and nail. J. Anat. 11: 99-120, 1972.

Kroll, M. H., and Toker, C.: Trabecular carcinoma of the skin. Arch. Pathol. Lab. Med. 106: 404-408, 1982.

Tang, C. K., and Toker, C.: Trabecular carcinoma of the skin. An ultrastructural study. Cancer 42: 2311-2321, 1978.

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Slide 78

History. Tissue from a common blue gill (Lepomis macrochirus macrochirus).

Gross Pathology. The lesion was an oval, well-circumscribed, superficial excoriation, measuring approximately .5 X 1.0 cm, located on the right lateral surface of the peduncle. Adhered to the surface of the ulcer was a delicate white cottonlike material.

Diagnosis. Erosion, epidermal, with superficial fungal elements, focally extensive, severe, skin, common blue gill, pisces, etiology—compatible with Saprolegnia sp.

Comment. Only fragments of necrotic epidermis remained at the margins of the eroded skin. While the skin appears ulcerated, the basal lamina remains intact, accounting for our classification of the lesion as an erosion. The only morphological manifestations of inflammation are the few lymphocytes and

macrophages infiltrating the dermis around the scales near the base of the eroded area and a few congested blood vessels near the same location. Overlying the erosion are many nonseptate hyphae sectioned in several planes admixed with debris. Although there is some variation in the width of the hyphae, many approximate 20 microns. These hyphae are compatible with those described for a number of Saprolegnia sp. and other genera of so-called "water molds" that are commonly lumped together with the saprolegnids.

The minimal inflammatory response seen in this case is characteristic unless there is an accompanying bacterial infection. Early investigators considered these organisms to be secondary invaders or opportunists; however, it is fairly well-documented now that there are several species of Saprolegnia that are primary pathogens. Relatively mild infections with one or more ulcers can cause a high mortality rate in fish. This is apparently related to the loss of integrity of the integument. The skin is an important tissue in maintaining hemostasis of electrolytes and fluids in fish. If large enough areas are ulcerated, teleosts will die from shock.

Although many authors refer to Saprolegnia sp. as fungi, according to recent classification, they are not true fungi. They actually belong to the kingdom, Protista; class, Oomycetes.

Contributor. Pathology Branch, Aerospace Medical Research Laboratory, Wright Patterson AFB, Ohio.

Suggested reading.

Leibovitz, L.: Mycotic infections. J. Am. Vet. Med. Assoc. 177(11): 1110-1112, 1980.

Richards, R. H.: Pathology of bacterial and fungal diseases affecting fish. In The Pathology of Fishes, edited by Ribelin, W. E. Madison, University of Wisconsin Press, 1975, pp. 205-209.

Roberts, R. J.: Fish Pathology. London, Bailliere Tindall, 1978, pp. 205-215.

Slide 79

History. This tissue is from a one-day-old dog that was experimentally inoculated with a naturally occurring infectious agent. A necropsy was performed at 18 days postinoculation.

Diagnosis. Degeneration and necrosis, with syncytia and intracytoplasmic inclusions, stratum intermedium and ameloblastic layer, enamel organ, tooth, breed unspecified, canine, etiology - canine distemper virus (morbillivirus).

Comment. The section shows disruption of the normal architecture of the enamel organ. There is necrosis of ameloblasts, syncytial giant cell formation, intracytoplasmic eosinophilic inclusion formation, and infiltration by small numbers of macrophages and lymphocytes.

Contributor. Laboratory of Pathology, School of Veterinary Medicine, University of Pennsylvania, Philadelphia, Pennsylvania.

Suggested reading.

Dubielzig, R. R.: The effect of CDV on the ameloblastic layer of the developing tooth. Vet. Pathol. 16: 268-270, 1979.

Dubielzig, R. R., Higgins, and Krakowka, G. S.: Lesions of the enamel organ of developing dog teeth following experimental inoculation of gnotobiotic puppies with canine distemper virus. Vet. Pathol. 18: 684-689, 1981.

Slide 80

History. A two-centimetre] cutaneous mass on the right distal tibia and a one-centimetre] mass on a left forelimb digit were excised from a 2 and 1/2-year-old female grey kangaroo (Macropus fuliginosus).

Diagnosis. Hyperplasia, epidermal, with intracytoplasmic eosinophilic inclusions, and chronic dermatitis, multifocal, moderate, skin, grey kangaroo (Macropus fuliginosus) - etiology compatible with poxvirus.

Comment. This skin lesion in some ways resembles human molluscum contagiosum. The lesion in humans is generally associated with a minimal inflammatory reaction, and the epidermis grows down into the dermis as multiple closely packed, pear-shaped lobules. The intracytoplasmic inclusion bodies (molluscum bodies) form in the stratum spinosum. Ultrastructurally, the inclusions consist of many vacuoles filled with mature viruses. The exophytic nature of the lesion in this case is unlike that in man. The infiltrate of plasma cells, lymphocytes, and histiocytes in the dermis is considered secondary. Poxvirus infections in association with cutaneous lesions have been

observed in four species of macropod marsupials—the quokka (Setonix brachyurus), the red kangaroo (Megaleia rufa), the western grey kangaroo (Macropus fuliginosus), and the eastern grey kangaroo (Macropus giganteus). In addition, a probable molluscum contagiosum-like pox infection has also been described in sea lions, horses, and chimpanzees.

Contributor. School of Veterinary Studies, Murdoch University, Murdoch, Western Australia.

Suggested reading.

Douglas, J. D., Tanner, K. N., and Prime, J. P., et al.: Molluscum contagiosum in chimpanzee. J. Am. Vet. Med. Assoc. 151: 901-904, 1967.

Lever, W. F.: Histopathology of the Skin, ed. 5. Philadelphia, J. B. Lippincott Co., 1976, pp. 346-348.

Lutzner, M. A.: Molluscum contagiosum, verruca and zoster viruses. Arch. Dermatol. 87: 436-444, 1963.

McKenzie, R. A., Fay, P. R., and Prior, H. C.: Poxvirus infection in the skin of an eastern grey kangaroo. Aust. Vet. J. 55: 188-190, 1979.

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Wilson, T. M., and Poglayen-Neuwall, I.: Pox in South American sea lions (Otaria byronia). Can. J. Comp. Med. 35: 174-177, 1971.

Slide 81

History. A group of normal-appearing CD2F1 mice was submitted for health surveillance evaluations. This tissue is from a 4-week-old female from a commercial "barrier" facility.

Diagnosis. Luminal Hymenolepis sp., Spironucleus sp., Giardia sp. and trichomonads with epithelial intranuclear inclusions and mucosal cysticercoids intestine, mouse.

Comment. This case demonstrates (1) the value of systematic histopathologic examination of the small intestine in routine health surveillance of mice and (2) the quality of sections that may be prepared routinely after fixing the unopened mouse intestine in alcoholic formalin (10% formalin made with 70% ethanol instead of water). Similarly prepared sections of large intestine also should be examined. Done properly, histopathologic examination of the gut provides important diagnostic information on approximately one-third of the infections (parasitic, bacterial, and viral) of importance in health surveillance of mice. A small piece of large intestine in some sections has Trichomonas sp. in the lumen.

Some slides have a granulomatous lesion in a section of lymph node that contains Hymenolepis sp. cysticercoids as well as the larvae within the lamina propria of the intestine. The presence of hooks on the scolex identifies the cestode as Hymenolepis nana, a parasite that can be transmitted to man. This is a rare finding in most mouse strains but is reportedly common in RFM mice infected with Hymenolepis sp. (Van Zwieten and Durcher, 1976).

Contributor. Department of Comparative Medicine, University of Alabama, Birmingham, Alabama.

Suggested reading.

Cohen, B. J., and de Groot, F. G.: Adenovirus infection in athymic (nude) mice. Lab. Anim. Sci. 26: 955-956, 1976.

Hashimoto, K., Sugiyama, T., and Sasaki, S.: An adenovirus isolated from the feces of mice. I. Isolation and identification. Jpn. J. Microbiol. 10: 115-125, 1976.

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Takeuchi, A., and Hashimoto, K.: Electron microscope study of experimental enteric adenovirus infection in mice. Infect. Immun. 13: 569-580, 1976.

Van Zwieten, M. J., and Boorsma, C.: Selected parasitologic, bacteriologic and virologic diseases of the mouse intestinal tract. In EDLP Pathology Atlas, European Late Effects Project Group, Committee on Pathology Standardization, Gesellschaft fur Strahlen-und Umweltforschung MBH (Neuherberg), W. Germany, 1976, p. 45.

Ward, J. M., and Young, D. M.: Latent adenoviral infection of rats: Intranuclear inclusions induced by treatment with a cancer chemotherapeutic agent. J. Am. Vet. Med. Assoc. 169: 952-953, 1976.

Slides 82-83

History. A 4-week-old female thoroughbred foal, weighing 85 kg, developed seizurelike behavior. The foal had a temperature of 102.2°F, a pulse of 80, and pink mucous membranes. Two days prior to death, the animal became sluggish and lethargic.

Diagnosis. Hepatitis, necrotizing, subacute, multifocal-coalescing, severe, liver, equine, etiology - compatible with Bacillus piliformis.

Comment. The presence of silver-positive (Warthin-Starry stain) filamentous bacterial rods growing in colonies within hepatocytes adjacent to the necrotic areas is diagnostic of Bacillus piliformis infection (Tyzzer's disease).

Bacillus piliformis is an obligate intracellular parasite that has been cultivated in embryonated eggs but not in artificial media. The pathogenesis of this disease in foals remains unclear, but Turk et al. suggest that B. piliformis is a significant cause of necrotizing hepatitis in 1- to 6-week-old foals. There is a carrier state (latent infection) that can result in intestinal lesions following stress. Serum titers are being taken in rabbits to diagnose Tyzzer's disease.

Contributor. Division of Comparative Pathology, University of Florida, Gainesville, Florida.

Suggested reading.

Pully, L. T., and Shively, J. N.: Tyzzer's disease in a foal (light and electron-microscopic observations). Vet. Pathol. 11: 203-211, 1974.

Thomson, G. W., Wilson, R. W., and Physick-Sheard, P.: Tyzzer's disease in the foal: Case reports and review. Can. Vet. J. 18: 41-43, 1977.

Turk, A. M., Gallina, A. M., and Perryman, L. E.: Bacillus piliformis infection (Tyzzer's disease) in foals in northwestern United Stated: A retrospective study of 21 cases. J. Am. Vet. Med. Assoc. 178(3): 279-281, 1981.

Slide 84

History. This mare was inoculated with a bacterial suspension into the posterior uterine body 14 days previously. She developed a mucopurulent vaginal discharge.

Laboratory Results. Haemophilus equigenitalis was present in the inoculum and was recovered at necropsy 14 days later from the oviduct, uterus, and cervix. The organism stained by an indirect immunofluorescent technique and was seen in these areas as well as in the vagina and vestibule. Endometrial folds were swollen, and the uterine lumen contained a small amount of flocculent viscid fluid. The mare had seroconverted in the plate agglutination test for antibodies to the contagious equine metritis organism.

Diagnosis. Endometritis, subacute, diffuse, moderate, uterus, breed unspecified, equine, etiology, Haemophilus equigenitalis.

Comment. After natural or experimental infection with contagious equine metritis organisms, there is a mild diffuse acute endometritis and cervicitis. Neutrophils are present in small numbers in the stratum compactum of the uterus and migrate singularly or in small groups through the luminal epithelium. The stratum spongiosum of the endometrium is edematous. The severity of the endometritis and cervicitis increases, and lymphocytes, macrophages, and plasma cells join the neutrophils so that by 14 days the inflammation is most severe and predominantly plasmacytic. Thereafter, the severity of the inflammation declines, but mild diffuse or multifocal lymphocytic endometritis and cervicitis persist. Vaginitis occurs too, but is less severe and resolves by

10 weeks after inoculation. Severe subacute salpingitis has been seen, and there is evidence that the infection causes residual mild multifocal chronic salpingitis.

Contributor. New Bolton Center, University of Pennsylvania, Kennett Square, Pennsylvania.

Suggested reading.

Gillespie, J. H., and Timoney, J. F.: Hagan and Bruner's Infectious Diseases of Domestic Animals. Ithaca, Cornell University Press, 1981, pp. 98-100.

Platt, H., and Atherton, J. G.: The experimental infection of ponies with contagious equine metritis. Equine Vet. J. 10: 153-159, 1978.

Powell, D. G.: Contagious equine metritis. Adv. Vet. Sci. Comp. Med. 25: 161-184, 1981.

Ricketts, S. W., and Rossdale, P. D.: Endometrial biopsy studies of mares with contagious equine metritis 1977. Equine Vet. J. 10: 160-166, 1978.

Ricketts, S. W., and Rossdale, P. D., Wingfield-Digby, N. J., et al.: Genital infection in mares. Vet. Rec. 101: 65, 1977.

Slide 85

History. After a storm, a group of 240 yearling Charolais-cross cattle was moved to a new location to facilitate feeding. Three were found dead within 12 hours.

Gross Pathology. Fibrinous peritonitis and hemorrhage were found throughout the musculature of both right and left ventricles.

Laboratory Results. Fluorescent antibody testing on the heart was pos for Clostridium chauvoei. Blood agar plates inoculated with heart and mesentery were incubated anaerobically, yielding a growth of Clostridium chauvoei.

Diagnosis. Enteritis, myocarditis and serositis, hemorrhagic, acute, diffuse, moderate to severe, small intestine and heart, Charolais-cross, bovine, etiology - Clostridium chauvoei.

Comment. This case is considered unusual in that the intestinal lesions have not been reported, causing consideration of a possible mixed infection. It is very difficult to differentiate autolytic changes from necrosis in this intestine. Autolysis is rapid in injured tissue. Gas bubbles within the affected tissue are expected with Clostridium chauvoei infection but are not present in this case. The pathogenesis of C. chauvoei infections remains unclear.

Contributor. Veterinary Science Department, South Dakota State University, Brookings, South Dakota.

Suggested reading.

Gillespie, J. H., and Timoney, J. F.: Hagan and Bruner's Infectious Diseases of Domestic Animals. Ithaca, Cornell University Press, 1981, pp. 216-218.

Jones, T. C., and Hunt, R. D.: Veterinary Pathology, ed. 5. Phil... Lea & Febiger, 1983, pp. 578-580.

Jubb, K. V., and Kennedy, P. C.: Pathology of Domestic Animals. New York, Academic Press, 1970, vol. 2, pp. 468-470.

Sterne, M.: Clostridial infections. Br. Vet. J. 137: 443-454, 1981.

Slide 86

History. A 9-year-old Holstein cow lost 300 lb after calving, and her milk production was down. The cow was being treated for mastitis and a gastrointestinal disorder. A significant finding at necropsy was an enlarged, firm right udder.

Diagnosis. Mastitis, necrotizing and pyogranulomatous, diffuse, severe, mammary gland, Holstein, bovine, etiology - Nocardia sp.

Comment. Spontaneous infection of the bovine udder with Nocardia asteroides is an ascending infection from the teat. It produces a progressive pyogranulomatous lesion. Diagnosis was made by demonstration of numerous branching, filamentous gram-positive, acid-fast organisms in the mammary gland. These characteristics are compatible with Nocardia asteroides. The Nocardia in this case stains unusually well with H&E. The presence of large numbers of multinucleated giant cells is considered atypical. The buildup of mineral on individual bacteria may cause some confusion in that they resembled fungal hyphae. Special stains do not demonstrate fungal elements.

Contributor. Livestock Disease Diagnostic Center, University of Kentucky, Lexington, Kentucky.

Suggested reading.

Bushnell, R. B.: Clinical and diagnostic aspects of herd problems with nocardial and mycobacterial mastitis. In The Proceedings of the 22nd Annual Meeting of the American Assoc. of Vet. Lab. Diag., St. Louis, 1979, pp. 1-12.

Fuchs, H. W.: Pathology, diagnosis and pathogenesis of bovine mastitis caused by Nocardia asteroides. Arch. Exp. Veterinarmed. 26(4): 683-700, 1972.

Gillespie, J. G., and Timoney, J. F.: Hagan and Bruner's Infectious Diseases of Domestic Animals. Ithaca, Cornell University Press, 1981, pp. 244-246.

Peir, A. C.: Nocardia asteroides as a mammary pathogen of cattle. Am. J. Vet. Res. 22: 502-517, 698-703, 1961.

Slide 87

History. A 3-year-old guinea pig died after a short history of depression, lethargy, anorexia, and constipation. Necropsy examination revealed a firm, solid, polypoid mass within the bladder lumen, approximating the size of the contracted lumen.

Diagnosis. Transitional cell carcinoma, urinary bladder, guinea pig.

Comment. There is a transition of the downward projecting cords of transitional epithelium (Brunn's nests) into the infiltrating small, dark cells in the lamina propria. In some sections there is a large, fairly well-defined cellular mass in the lamina propria that has a sarcomatous appearance with areas of transition into the small cell component. A small cell component is a relatively common finding with this tumor in humans and seems to occur

neoplastic transitional epithelial cells infiltrate the lamina propria. These small cells often resemble lymphocytes. Cystitis cystica and squamous metaplasia were found within portions of the tumor. According to Koss, Brunn's nests, cystitis cystica, and cystitis glandularis are epithelial variants that can be associated with bladder cancer.

Other diagnoses considered include undifferentiated sarcoma, spindle cell carcinoma, carcinosarcoma, and carcinoma with a leiomyosarcoma. Tumors of the urinary system in guinea pigs are extremely uncommon.

Contributor. University of Pittsburgh, Central Animal Facility, Pittsburgh, Pennsylvania.

Suggested reading.

Benirschke, K., Garner, F. M., and Jones, T. C.: Pathology of Laboratory Animals. New York, Springer-Verlag, 1978, pp. 1170-1171.

Koss, L. G.: Tumors of the Urinary Bladder, Ser. 2, Fascicle 11, of Atlas of Tumor Pathology. Washington, D.C., American Registry of Pathology, Armed Forces Institute of Pathology, 1975, pp. 39-43, 101.

Pawlowski, A. M.: IV. Tumors of the Urinary Bladder. International Histological Classification of Tumors of Domestic Animals. Bull. W.H.O. 50(1-2): 45-52, 1974.

Slide 88

History. A 30-month-old M-520 male rat, at age 2 months, received .04 cc 3-methyl cholanthrene injected into the wall of the sigmoid colon.

Gross Pathology. The left adrenal gland was replaced by a soft, friable hemorrhagic tumor mass measuring 1-1/2 x 1 x 1 cm. The tissues were preserved in Kelly's fixative.

Diagnosis. Pheochromocytoma, adrenal gland, M-520 rat.

Comment. The adrenal gland contains a massive neoplastic growth that has replaced all of the normal architecture. Parts of the tumor show arrangements as nests of small, dark cells (with little cytoplasm) around vascular spaces. Other areas show lobular masses of paler cells with abundant cytoplasm and well-defined cell borders. These cells often contain large bizarre nuclei. A possible third cell type is intermediate between the two aforementioned cell types. These are arranged as large nests. The individual cells have finely granular, lightly stained cytoplasm. There are small, round-to-oval hematoxyphilic bodies in the cytoplasm of some of these cells, indicating they are of medullary origin.

In addition to the tumor, a granulomatous arteritis and a mild glomerulopathy were found in the kidney, both of which are common age-associated lesions of rats. Considering the rat's age, the kidney lesions are mild.

According to the NIH Rodent Catalogue, the Marshall 520 (M520) rat has naturally occurring adrenal medullary tumors 68% to 80% of the time after 18 months of age. Hollander et al. state that adrenal medullary tumors other than pheochromocytomas are very rare in all strains of rats and that these tumors are not prone to metastasize.

Contributor. National Cancer Institute, Registry of Experimental Cancer, Bethesda, Maryland.

Suggested reading.

Burek, J.: Adrenal gland tumors. In Pathology of Aging Rats. West Palm Beach, Florida, CRC Press, 1978, pp. 43-49.

Hollander, C. F., and Shell, K.: Tumors of the adrenal gland. In Pathology of Tumors in Laboratory Animals. Lyon, France, World Health Organization, IARC, 1976, vol. 1, pt. 2, pp. 273-281.

NIH Rodents 1980 Catalogue. National Institutes of Health Publication No. 81-606, 1980, pp. 113-114.

Slide 89

History. These tissues are from a control 5-month-old MRL/l mouse.

Gross Pathology. The animal had generalized lymph node enlargement.

Diagnoses. Lymphocytic proliferation, diffuse, severe, lymph node, MRL/l mouse. Periarteritis, granulomatous, diffuse, severe, kidney. Glomerulonephritis, membranoproliferative, subacute, diffuse, moderate, kidney. Arteritis, necrotizing, acute, segmental, moderate, kidney.

Comment. The MRL/l mouse strain, developed by Murphy and Roths, is a useful animal model for the study of spontaneous systemic lupus erythematosus (SLE) and rheumatoid arthritis. Lesions in MRL/l mice include polyarthritis similar to human rheumatoid arthritis, polyarteritis, a lymphadenopathy

characterized by massive proliferation of T-lymphocytes, and a rapidly fata immune complex glomerulonephritis. Greater than 50% of both male and femal mice die by 7 months of age because of renal lesions. The usual renal lesic is a subacute proliferative glomerulonephritis that involves proliferation o both mesangial and endothelial cells and basement membrane thickening. Immunofluorescent studies have shown granular deposits of immunoglobulins in capillary walls. In addition to the T-cell proliferation in the lymph node, there are large numbers of plasma cells and Mott (Russell body) cells.

All MRL/l mice develop massive peripheral lymph node enlargement that progresses to over 100 times control lymph node weights by 16 weeks of age. Perivascular infiltrates of lymphocytes, plasma cells, and histiocytes are routinely seen in the lung, kidney, salivary gland, and liver. There is no leukemic blood picture and no evidence that the lymphoproliferative process is malignant. It cannot be ruled out, however, that the lymphoid hyperplasia is not a preneoplastic lesion. The lymphocyte proliferation is determined by an autosomal recessive lpr (lymphoproliferation) gene. These lymphocytes are presently believed to consist of Ly null or weakly Ly 1 inducer/helper T-cells.

The subcapsular spindle cell hyperplasia present in the adrenal glands that were present in some of the sections is a spontaneous age-related lesion frequently seen in several mouse strains. The origin of the cells and the significance of the lesion are unknown.

Contributor. Wyeth Laboratories, Inc., Paoli, Pennsylvania.

Suggested reading.

Altman, A., Theofilopoulos, A. N., Weiner, R., et al: Analysis of T-cell function in autoimmune murine strains defects in production of and responsiveness to interleukin 2. J. Exp. Med. 154(3): 791-808, 1981.

Hang, L., Theofilopoulos, A. N., and Dixon, F. J.: A spontaneous rheumatoid arthritis-like disease in MRL/l mice. J. Exp. Med. 155(6): 1690-1701, 1982.

Kelly, V. E., Winkelstein, A., Izui, S., et al.: Prostaglandin E_1 inhibits T-cell proliferation and renal disease in MRL/l mice. Clin. Immunol. Immunopathol. 21(2): 190-203, 1981.

Murphy, E. D., and Roths, J. B.: Autoimmunity and lymphoproliferation: Induction by mutant gene lpr, and acceleration by a male associated factor in strain EKSB mice. In Genetic Control of Autoimmune Disease, edited by Rose/Bigazzi/Warner. Elsevier North Holland, Inc., 1978, pp 207-221.

Prudhomme, G. J., Park, C. L., Fieser, T. M., et al.: Identification of a B-cell differentiation factor(s) spontaneously produced by proliferating T-cells in murine lupus strains of the lpr/lpr genotype. J. Exp. Med. 157: 730-742, 1983.

Slide 90

History. An 8-year-old male German shepherd with a long history of allergic dermatitis developed hemorrhagic diarrhea with tenesmus. The condition did not respond to treatment, and the dog became cachectic, disoriented, and blind. The dog was put to death after a clinical course of 5 weeks.

Diagnosis. Colitis, granulomatous, focally extensive, transmural, severe, colon, German shepherd, canine, etiology - Prototheca sp.

Comment. Necropsy findings consisted of a thickened ulcerated colon, cecum, and ileum. The mesenteric lymph nodes and liver were enlarged. Eye lesions consisted of cloudy corneas and hyphema. Prototheca organisms were found in the large and small intestine, mesenteric lymph nodes, liver, kidney, heart, and eyes. The brain was not available for examination. The histopathologic lesions are typical for protothecal colitis, with the organisms staining unusually well with H&E. The organisms are prominent around vessels, which they frequently invade. Species identification would require immunofluorescence in the absence of cultural studies (Migaki et al.).

Contributor. Syntex Research, Palo Alto, California.

Suggested reading.

Carlton, W. W., and Austin, L.: Ocular protothecosis in a dog. Vet. Pathol. 10: 274-280, 1973.

Ihes, G. D., Lloyd, J., and Brightman, M. P.: Disseminated protothecosis in a dog. Onderstepoort J. Vet. Res. 44(1): 1-6, 1977.

Migaki, G., Font, R. L., Sauer, R. M., et al.: Canine protothecosis: Review of the literature and report of an additional case. J. Am. Vet. Med. Assoc. 181(8): 794-797, 1982.

Suhan, M. S., Majka, J. A., and Kaplan, W.: Primary mucocutaneous Protothecosis in a dog. J. Am. Vet. Med. Assoc. 163: 1372-1374, 1973.

Van Kruiningen, H. J.: Protothecal enterocolitis in a dog. J. Am. Vet. Med. Assoc. 157: 56-63, 1970.

Slide 91, I19

History. This tissue is from a mature cow that was one of many similarly affected in a large herd. (See gross photo of fixed tissue.)

Diagnosis. Pneumonia, interlobular, serofibrinous, subacute, diffuse, severe, lung, bovine, etiology, Mycoplasma mycoides mycoides.

Comment. The gross and microscopic lung lesions in this case are considered characteristic for contagious bovine pleuropneumonia (CBPP) caused by Mycoplasma mycoides mycoides. The organisms were demonstrated with a Giemsa stain.

This tissue is from a herd outbreak in Southwest Africa. The disease remains a problem throughout much of the world but was eradicated from the United States in 1892. Control is made difficult because of symptomless carrier animals. The disease primarily affects cattle, but buffalo, goats and sheep are susceptible to artificial infection. A different strain of the organism reportedly causes natural disease in goats in the U.S.A.

It is first necessary for the organism to penetrate the mucous layer of the respiratory tract before contacting the epithelium. The organism then attaches to ciliated epithelium after nestling in between the cilia. There is intimate contact with the host cell membrane, but membrane fusion has not been proven (Gourlay et al.).

Following attachment, the organism produces a toxic effect that with M. mycoides mycoides in cattle seems to be confined to the vascular system. The effect is variable between animal species and may have some relationship to their susceptibility to the disease. The capsule of M. mycoides mycoides may play a role in inhibition of host defenses and strain virulence. This organism has been shown to share common antigens with the bovine host that may impede recognition by the host or stimulate autoantibody.

Contributor. Onderstepoort Veterinary Research Institute, Onderstepoort, Republic of South Africa 0110.

Suggested reading.

Foreign Animal Diseases. Their Prevention Diagnosis and Control. Committee on Foreign Animal Diseases of the United States Animal Health Association, 1975, pp 76-84.

Gillespie, J. H., and Timoney, J. F.: Hagan and Bruner's Infectious Diseases of Domestic Animals. Ithaca, Cornell University Press, 1981, pp 289-293.

Gourlay, R. N., and Howard, C. J.: Respiratory mycoplasmosis. Adv. Vet. Sci. Comp. Med. 26: 289-332, 1982.

Slide 92

History. This tissue is from a 2-year-old Stellars jay that had been on exhibit for about one year in a small zoo. The bird had been treated with chloramphenicol I.M., then P.O. for leg lesions. The lesions improved and treatment was stopped after 14 days. Two days later the bird became dyspneic and died.

Laboratory Results. Aspergillus fumigatus was isolated from the lung and abdominal air sacs.

Diagnoses. Pneumonia and airsacculitis, necrotizing, acute, focally extensive, severe, with fungal elements, lung, Stellars jay, avian, etiology - Aspergillus fumigatus. Vasculitis, necrotizing, with thrombosis, acute, focally extensive, severe, with fungal elements, lung, etiology - Aspergillus fumigatus.

Comment. Congestion and multiple foci of necrosis were found in the lung. Airways contain fibrin, inflammatory cells, and branching septate fungal hyphae. Necrosis and thrombosis of lung and blood vessels with inflammatory cells and fungal hyphae were found in the vessel wall. Fungal organisms and inflammation extend into adjacent vesicles; sterigmata and chains of conidia are visible. The right lung was most severely involved. The histomorphologic features of the fungus are considered characteristic for Aspergillus fumigatus. Some of the more severely affected portions of the lung may be infarcted.

Contributor. Department of Comparative Medicine, The Milton S. Hershey Medical Center, Pennsylvania State University, Hershey, Pennsylvania.

Suggested reading.

Benirschke, K., et al.: Pathology of Laboratory Animals. New York, Springer-Verlag, 1978, pp 92, 1574.

Jones, T. C., and Hunt, R. D.: Veterinary Pathology. Philadelphia, Lea & Febiger, 1983, pp 667-669.

Montali, R. J., and Migaki, G. (Eds.): Proceedings of a Symposium on the Comparative Pathology of Zoo Animals, held at the National Zoological Park, Smithsonian Institution, Washington, D.C., October 2-4, 1978; Washington, D.C Smithsonian Institute Press, 1980, pp 271-272.

Slide 93

History. This tissue is from an aged Long-Evans rat.

Diagnosis. Adenomatous odontogenic tumor in association with a keratinizing and calcifying odontogenic (Gorlin) cyst, oral cavity, Long-Evans rat.

Comment. There are many inconsistencies in the veterinary and human literature pertaining to the classification of dental tumors. The use of the term "adamantinoma" is synonymous with the generally preferred term "ameloblastoma." The origin of the ameloblastoma is most likely the dental lamina. There is no evidence suggesting an origin from ameloblasts, and there are no hard tissue elements in these tumors. An odontoma contains hard tissue elements, and there may be nonneoplastic ameloblasts producing enamel. The use of the term odontogenic epithelium is preferred to describe cells of tooth origin in which a distinction between ameloblasts and odontoblasts can't be made. Odontogenic tumors are often found in association with keratinizing and calcifying odontogenic cysts.

Contributor. Ethicon Research Foundation, Somerville, New Jersey.

Suggested reading.

Dubielzig, R. R., and Thrall, D. E.: Ameloblastoma and keratinizing ameloblastoma in dogs. Vet. Pathol. 19: 596-607, 1982.

Gorlin, R. J.: Odontogenic tumors. In Thomas' Oral Pathology, edited by Gorlin, R. J., and Goldman, H. M. St. Louis, C. V. Mosby Co., 1970, vol. 1, pp. 481-503.

Lewis, D. J., Cherry, C. P., and Gibson, W. A.: Ameloblastoma (adamantinoma) of the mandible in the rat. J. Comp. Pathol. 90: 379-384, 1980.

Shafer, W. G.: Textbook of Oral Pathology, ed. 3. Philadelphia, W. B. Saunders Co., 1974, pp 249-267.

Slide 94-95, L20-L23

History. This 4-year-old Shih Tzu was clinically normal until she had her first litter of pups at 3 years of age. At that time, the dog began to lose weight and became progressively emaciated. Before presentation the dog was anorectic with P/U and P/D. While blood was being drawn for a heartworm test, the dog collapsed and had a cardiac arrest. She was resuscitated and subsequently developed vomiting and diarrhea. She was put to death 2 days following the cardiac arrest.
(See 202 slides).

Gross Pathology. Both kidneys were severely shrunken, pale, and firm. The left adrenal gland was somewhat enlarged. All bones and teeth were soft and costochondral junctions were enlarged.

Diagnoses. Osteopenia and fibroplasia, diffuse, severe, mandible, Shih Tzu, canine. Osteopenia, osteomalacia and fibroplasia, diffuse, severe, bony rib (progressive renal disease of Shih Tzus and renal rickets).

Comment. The osteopenia and fibroplasia present in the mandible and rib are the components of fibrous osteodystrophy characteristically associated with hyperparathyroidism. These lesions are the result of intense osteoclastic activity. The osteomalacia results from the failure of osteoid to mineralize. This follows the impaired 1 hydroxylation of the $25D_3$ metabolite in the kidney, yielding a reduced intestinal calcium absorption. Furthermore, the excessive parathyroid hormone (PTH) levels result in abnormal (unmineralizable) matrix formation. The inability of the kidney to respond to PTH causes a progressive hyperphosphatemia.

Diagnoses made from other tissues include a diffuse chronic interstitial nephritis (suggested by the gross photo) and midoral mucosal mineralization and necrosis in the stomach. The histomorphologic features of the kidney lesion are compatible with familial renal disease of Shih Tzus. The stomach lesions are caused by uremia.

The basophilic material apparently within the dentine layer of the large molar is probably dentin or a variant of dentin and either residual of a resorptive process or a primary congenital malformation of the tooth.

Contributor. Bowie Animal Hospital, Bowie, Maryland.

Suggested reading.

Capen, C. C., and Martin, S. L.: Calcium-regulating hormones and diseases of the parathyroid glands. In Textbook of Veterinary Internal Medicine, ed. 2, edited by Ettinger, S. J. Philadelphia, W. B. Saunders Co., 1983, pp. 1550-1581.

O'Brien, T. D., Osborne, C. A., Yano, B. L., et al.: Clinicopathologic manifestations of progressive renal disease in Lhasa Apso and Shih Tzu dogs. J. Am. Vet. Med. Assoc. 180: 658-664, 1982.

Robbins, S. L., and Cotran, R. S.: Pathologic Basis of Disease. Philadelphia, W. B. Saunders & Co., 1979, pp 1379-1387.

Slide 96

History. A-39-week-old CR-CD (Charles River) albino rat in a toxicology study was put to death because of mechanical interference with eating as a result of a mass on the mandible. The mass was located in the oral cavity, appeared tan and red in color with numerous small hard white projections, and measured 1.5 X 1.3 cm. The consistency on cutting was hard.

Diagnosis. Complex odontoma in association with a keratinizing and calcifying odontogenic (Gorlin) cyst, mandible, CR-CD rat.

Comment. Complex odontomas contain poorly differentiated hard tissue (dentin, enamel, cementum, etc.), and there is an essential absence of neoplastic ameloblastic tissue. Neoplastic ameloblastic tissue does not produce hard tissue(s). According to Gorlin, in normal tooth development, degeneration of the dental lamina occurs soon after hard tissue formation, and the complex odontoma corresponds to this more complete stage of induction.

A differential diagnosis might include ameloblastic odontoma; however, ameloblastic odontomas (odontoameloblastoma) are considered to be a collision tumor consisting of an obvious ameloblastoma as well as an odontoma. The occurrence of such a tumor in humans is uncommon.

Contributor. Hazleton Laboratories America, Inc., Vienna, Virginia.

Suggested reading.

Haeperl, H., and Ackerman, L. V. (Eds.): International Union Against Cancer-Illustrated Tumor Nomenclature, ed. 2. New York, Springer-Verlag, 19

Moulton, J. E.: Tumors in Domestic Animals, ed. 2 (revised). Berkeley, University of California Press, 1978, pp. 244-246.

World health Organization Bulletin, International Histological Classification of Tumors of domestic Animals, Part 2. Bull. W.H.O. 53(2-3): 1976.

Refer to references for slide 93 for this case also.

Slide 97, I24

History. A 2 and 1/2-year-old female Panama boat-billed heron (Cochlearius P. Panamensus was lame for weeks. There was a firm swelling in the left and right proximal tibiotarsus, and some small firm nodules were attached to the axial bones.

Gross Pathology. There was swelling, hemorrhage, and osteolysis in the proximal tibiotarsus as well as hard nodules in the bone marrow, liver, lung lobes, and ovaries (see 2x2 slide).

Diagnosis. Osteosarcoma, bone, heron, avian.

Comment. Multiple organs being affected by this tumor in the heron is unusual since osteosarcoma, particularly with metastases, is uncommon in cage birds. The site from which this tumor arose in the heron is unclear; however, it is most likely that the tumor originated from the left tibiotarsus, invading the vessel and metastasizing hematogenously to the other bones and soft tissues.

The lung metastases and long bone lesions are demonstrated on the radiograph. This case was reported by Liu et al.

Contributor. The Animal Medical Center, New York, New York.

Suggested reading.

Beach, J. E.: Diseases of budgerigars and other cage birds: A survey of postmortem findings. Part IV. Vet. Rec. 74: 134-140, 1962.

Liu, S. K., Dolensek, E. P., and Toppe, J. P.: Osteosarcoma with multiple metastases in a Panama boat-billed heron. J. Am. Vet. Med. Assoc. 181: 1396-1398, 1982.

Petrak, M. L., and Gilmore, C. E.: Diseases of Cage and Aviary Birds, edited by Petrak, M. L. Philadelphia, Lea & Febiger, 1969, pp 459-489.

Slide 98

History. A mass was removed from the abdominal region of a 4-year-old female quarter horse. A puncture wound occurred in the abdominal region in early fall. The wound healed, and a growth developed on the skin.

Gross Pathology. The mass was greyish white in color, pedunculated, and measured approximately 4 X 3 X 2 cm.

Diagnosis. Equine sarcoid, skin, quarter horse, equine.

Comment. The histomorphologic features of this tumor are considered to be characteristic of an equine sarcoid. In some sections, a portion of the tumor is covered by epithelium showing acanthosis, hyperkeratosis, and pseudoepitheliomatous hyperplasia. The tumor cells at the epidermal-dermal junction are frequently oriented perpendicularly to the basement membrane. Moulton and others report that almost all equine sarcoids have an epidermal and a dermal component. There is likely a viral etiology, the identity of which has yet to be determined.

Contributor. Department of Pathology, School of Veterinary Medicine, Tuskegee Institute, Alabama.

Suggested reading.

Baker, J. R., and Leyland, A.: Histological survey of tumors of the horse, with particular reference to those of the skin. Vet. Rec. 96: 419-422, 1975.

Cheevers, W. P., Roberson, S. M., Brassfield, A. L., et al.: Isolation of a retrovirus from cultured equine sarcoid tumor cells. Am. J. Vet. Res. 43: 804-806, 1982.

Moulton, J. E.: Tumors in Domestic Animals, ed. 2. Berkeley, University of California Press, 1978, pp 18-22.

Weiss, E.: Tumors of the soft (mesenchymal) tissues. Bull. W.H.O. 50: 101-110, 19754.

Slide 99, L25-L26

History. This tissue is from a veal calf 12 to 13 weeks old. The calf was being fed a commercial veal production ration. For the last 3 weeks the calf began to lose weight and became emaciated. The calf was also thought to be bloated the final 3 weeks before slaughter.

Gross Pathology. Extensive tumor growth covered the abdominal viscera. Tumor on the liver capsule did not invade. Iliac lymph nodes were enlarged. There was evidence of tumor on the serosa of the pleural cavity (see 2x2 gross photos).

Diagnosis. Mesothelioma, Glisson's capsule and abdominal lymph node, bovine.

Comment. The histomorphologic features of this tumor are consistent with the papilliferous mesotheliomas described by Schamber et al. in their recent report of neoplasms in calves. The projections are lined by large pleomorphic cells with occasional syncytia formation. The nuclei are generally round to oval, vary in size, and contain a prominent nucleoli. Fibrous connective tissue strands in the cores of the papillae and in the more solid basal areas of the neoplasm were demonstrated with a Masson's trichrome stain. Cells with clear cytoplasmic vacuoles are present in the stroma as well as lining the papillae. The parenchyma of the lymph node is largely replaced by the tumor and neoplastic cells are present in the subcapsular sinus and surrounding lymphatics.

With the transcoelomic spread of a tumor, differential diagnoses should include epithelial tumors originating in the ovary, uterus, and intestine.

Contributor. Department of Veterinary Science, University of Wisconsin, Madison, Wisconsin.

Suggested reading.

Baskerville, A.: Mesothelioma in the calf. Pathol. Vet. (Basel) 4: 149-156, 1967.

Head, K. W.: Tumors of the lower alimentary tract. Bull. W.H.O. 53: 175-182, 1976.

Moulton, J. E.: Tumors in Domestic Animals, ed. 2. Berkeley, University of California Press, 1978, pp 280-285.

Schamber, G. J., Olson, C., and Witt, L. E.: Neoplasm in calves (Bos taurus). Vet. Pathol. 19: 629-637, 1982.

Slide 100

History. A 6-year-old female domestic short-hair cat was presented with a 4-week history of a progressing inability to jump and walk. Physical examination revealed an alert animal with rigid limbs and stiff muscles. The femoral pulses were difficult to palpate. The cat appeared to be sore and could walk only with assistance. No response was noted to steroid and antibiotic therapy, and the animal was put to death.

Gross Pathology. The gross lesions are restricted to the proximal muscles of all four limbs, which are variably affected. Both subscapularis muscles are white and firm with gritty foci palpable within the fascial sheaths and the

muscle bellies. The left quadricep muscle is white, soft, and interwoven with firm white connective tissue in which are seen gritty nodules on cut section. The epaxial, hypaxial, and distal limb musculature appear normal.

Diagnosis. Fibroplasia, with chondroid metaplasia and ossification, focally extensive, severe, with associated myodegeneration, skeletal muscle, feline (compatible with fibrodysplasia ossificans progressiva.

Comment. The histomorphologic features of this section are compatible with fibrodysplasia ossificans progressiva (FOP); however, examination of additional sections and particularly whole body radiographs are considered essential for a definitive diagnosis. The primary lesion is a proliferation of epimysial connective tissue. chondroid metaplasia and membranous ossification are seen within the areas of mesenchymal proliferation. Rare foci of inflammatory cells are present in muscle as well as connective tissue. The muscle lesions are considered to result from disuse secondary to immobilization as well as entrapment of the muscle by fibrous tissue. Some islands of bone were being formed by enchondral as well as by intramembranous ossification. A striking feature in this case is the distribution of the lesion in the proximal appendicular skeleton. An interview with the owner of this cat to obtain any familial history of the disorder was unproductive. A similar disorder has been described in pigs and horses.

The condition in man is found as a rare, disastrous disorder resulting in progressive immobility of the skeleton. In FOP, hyperplasia of epimysial connective tissue results in membranous ossification. Trauma is not a required

prologue for either the proliferation or ossification. The disorder is often noted in young patients. concomitant microdactyly of the thumb and big toe as often seen in FOP. (No such abnormality was noted in the present feline case.

The etiology of the human disease is obscure. FOP appears to be inherited as an autosomal dominant trait. The incidence of the condition is so low as to implicate a point mutation as the cause. FOP has been associated with increased paternal age. Effective therapy is nonexistent.

In the 19th century and early part of the 20th century the disease was termed myositis ossificans progressiva. More recent workers tend to use FOP with the justification that inflammation of the muscles per se is an insignificant component of the disorder.

Contributor. Department of Pathology, Angell Memorial Animal Hospital, Boston, Massachusetts.

Suggested reading.

Connor, T. M., and Evans, D. A. P.: Fibrodysplasia ossificans progressiva. The clinical features and natural history of 34 patients. J. Bone Joint Surg. [Br.] (1): 24-26, 1982.

Connor, T. M., and Evans, D. A. P.: Genetic aspects of fibrodysplasia ossificans progressiva. J. Med. Genet. 19: 35-39, 1982.

McKusick, V. A.: Heritable disorders of connective tissue, ed. 4. St. Louis, C. V. Mosby, 1972.

Rogers, J. C., and Chase, G. A.: Paternal age effect in fibrodysplasia ossificans progressiva. J. Med. Genet. 16: 147-148, 1979.

Rosenstirn, J.: A contribution to the study of myositis ossificans progressiva. Ann. Surg. 1918: 450-520, 591-637, 1966.

Siebold, H. R., and Davis, C. L.: Generalized myositis ossificans (familial) in pigs. Pathol. Vet. 4: 79, 1967.

Smith, R.: Myositis ossificans progressiva: A review of current problems. Semin. Arthritis Rheum. 4(4): 369-380, 1975.