AFIP Wednesday Slide Conference 1980-81

Index

af.	Case #	Date of Conference	Contrib. #	AFIP #	Contributor	Species	Organ	Diagnosis
l	I	17 Sep 80	78310	1668870	L.A. Co. Dept of Health	Dog	Skin	Hemangiopericytoma
	II	"	78-0594	1667115	Univ. of AZ	Wallaby	Muscle	Vit.E-Se myopathy
	III	"	27535	1714910	USAMRIID Ft. Detrick, MD	Dog	Heart	Parvovirus myocarditis
	IV	"	N79-114	1713314	Univ. of Florida	Horse	Sp. cord Brain	Protozoal myelitis
	I	24 Sep 80	78-1850	1667020	Faculte de Med-Vet Quebec Canada	Pig	Kidney	Actinobacillus nephritis
	II	"	79P2080	1713948	Louisiana St. Univ.	Dog	Liver Kidney	Leptospirosis
	III	"	79-3151	1713949	Ohio St. Univ.	Dog	Heart	Myocardial necrosis Canine Distemper
	IV	"	A-19735	1667122	Animal Med. Ctr.	Dog	Intestine	Mast cell tumor

	I	1 Oct 80	13696	1755478	Univ. of Md	Rhesus Monkey	Kidney	Renal tubular necrosis
	II	"	CA5-197	1755476	Lovelace Res. Albuquerque, NM	Hamster	Uterus	Endometrial adenocarcinoma
	III	"	71537	1712658	Texas A & M	Parrot	Liver	Pacheco's Disease
	IV	"	27469	1714918	USAMRIID Ft. Detrick, MD	Dog	Kidney	Amyloidosis
4	I	8 Oct 80	23197	1619627	USAMRIID Ft. Detrick, MD	Guinea Pig	Lung	Klebsiella sp. pneumonia
	II	"	7464	1712661	C.E. Kord Diag. Nashville, TN	Horse	Liver	Crotalaria poisoning
	III	"	648T	1755477	Lovelace Res. Albuquerque, NM	Dog	Kidney	Hemoglobinuric nephrosis
	IV	"	191712-13	1713082	Mich. St. Univ.	Duck	Liver	Duck plague
5	I	15 Oct 80	176750	1681107	Mich. St. Univ.	Touraco Avian	Liver	Mycobacterial hepatitis
	II	"	77P1623	1667702	Louisiana St. Univ.	Iguana	Long bone	Multiple Chrondromatosis
	III	"	X11842	1727491	Eli Lilly & Co.	Duck	Kidney	Renal coccidiosis
	IV	"	4898K	1672455	Univ. of Penn.	Snake	Kidney	Renal gout

6	I	29 Oct 80	1959-76	1618040	Oregon St. Univ.	Dog	Brain	Cryptococcosis
	II	"	79-8074	1712745	Univ. of GA	Pig	U. Bladder	Mucinous cystitis, C. renale
	III	"	7159-79	1712701	Univ. of MO	Dog	Maxilla	Epidermal bone cyst
	IV	"	MS78-430	1667745	NIH	Mouse	Lung	Sendai virus pneumonia
7	I	5 Nov.80	968-79	1716366	Oregon St. Univ.	Dog	Stomach lung	Distemper gastritis Toxoplasmosis
	II	"	82446	1714913	L.A. Co. Dept. of Health	Horse	Brain	Pinealoma
	III	"	80-1	1757055	G.A. Parker	Squirrel	Skin	Psoregates sp. infestation
	IV	"	78-4656	1667021	Faculte de Med-Vet Quebec, Canada	Pig	Liver	Eosinophilic leukemia
8	I	12 Nov 80	II Rat	1718321	Sterling-Winthrop	Rat	Cervical mass	Chemodectoma
	II	"	D-78-9390	1668449	Univ. of IL	Horse	Brain	Leucoencephalomalacia
	III	"	26903-N	1757436	Auburn Univ.	Dog	Lung	Filaroides hirthi infection
	IV	"	NA8-119	1668789	Univ. of GA	Cat	Brain	Blastomycosis

	I	19 Nov 80	CP77-1072	1640295	S.W. Med. Sch. Dallas, TX	Cat	Lung	Pulmonary thromboembolism
	II	"	080-962	1758398	Hazelton Labs. Vienna, VA	Rat	Kidney	Renal cell carcinoma
	III	"	79-0386	1712926	Univ. of AZ	Dog	Lung	Metastatic calcification - Vit. D
	IV	"	17731/80-976	1761488	WRAIR	Rabbit	Liver	Tyzzer's disease Hepatic coccidiosis
10	I	26 Nov 80	80-381	1757304	Univ. of Penn	Bovine	Spleen	Hairy vetch poisoning
	II	"	AFIP#1	1755277	Onderstepoort,So. Africa	Dog	Brain	Babesiosis
	III	"	D-78075	1666524	Exper. Path. Labs.	Dog	Mediastinal mass	Thymoma
	IV	"	AN80/254 SN80/1310	1761487	Dept. of Ag.,NSW Australia	Bovine	Heart	1080 poisoning
11	I	11 Dec 80	77324	1756983	L.A. Co. Dept. of Health	Sea lion	Liver	Viral hepatitis
	II	"	65-1970	1727493	Univ. of Wisc.	Hamster	Subcutis	Fibrosarcoma
	III	"	80-2858	1757437	Ohio St. Univ.	Dog	Testicle	Brucella sp.,Orchitis
	IV	"	4-7-81	1758838	Montana St. Univ.	Ovine	Nasal mass	Nasal adenocarcinoma

I	17 Dec.80	A-18946	1667112	Anim. Med.Cntr.	Dog	Spinal cord	Hemangio.
II	"	13747-7	1713077	Johns Hopkins Univ.	Mouse	Kidney	Chloroform nephrosis
III	"	80P465	1758396	Colo. St. Univ.	Horse	Small intestine	Aganglionosis
IV	"	80/0623	1757043	Murdoch Univ. Australia	Goat	Spinal cord	Enzootic ataxia
I	7 Jan.81	27178	1755279	NIH	Monkey	Liver	Capillaria hepatica
II	"	R27979	1720287	FDA	Rat	Kidney	Teratoid nephroma
III	"	N79-577	1711325	Brooks AFB	Monkey	Brain	Tuberculosis
IV	"	0070	1758837	Univ. of Nebr.	Cat	Liver Lung	Cytauxzoonosis
I	14 Jan.81	79-727	1716400	Kans. St. Univ.	Cat	Brain	Ependymoma
II	"	7313-80	1757255	Univ. of MO	Horse	Colon Liver	Granulomatous colitis Hepatic necrosis
III	"	N79-420	1757300	Univ of FL	Pig	Lung	Alkaloid poisoning
IV	"	80-2687 18686	1764666	WRAIR	Dog	Adrenal Lung	Adrenal cortical carcinoma
I	21 Jan.81	1/NZP	1758395	Natl. Zoo	Kangaroo	esophagus	Candiasis
II	"	80-10933	1758391	So. Dak. St.	Bovine	Lung Spleen	Anthrax
III	"	AFIP # 2	1667519	Wash. St. Univ.	Dog	U. Bladder	Rhabdomyosarcoma
IV	"	80-214	1761490	Dept. of Ag. B.C. Canada	Horse	Liver Lung	Equine Herpes V-2

	I	28 Jan.81	80-1038	1758463	Univ. of CA	Dog	Brain	Granulomatous encephalitis
	II	"	0065	1758834	Univ. of UB	Cat	Skin	Trichosporan sp. dermatitis
	III	"	A28109	1666535	Pfizer Med. Res. Labs.	Rat	Kidney	Granulomatous nephritis
	IV	"	AF1367-79	1758462	Purdue Univ.	Turkey	Trachea	Cryptosporidiosis
17	I	4 Feb.81	OL00201	1758400	Searle Labs.	Rat	Ovary	Osteosarcoma
	II	"	PA-089-13 PA-089-21	1757379	Univ. of Pitts.	Dog	Sm. Intes. Bone marrow	Parvovirus-enteritis " - hypoplasia
	III	"	#2	1757295	Syntex Res. Inc.	Rabbit	Subcutis	**Coenurosis; Multiceps sp.**
	IV	"	CP77-1159 CP78-246	1673404	S.W. Med Sch. Dallas, TX	Dog	Kidney	Gentamycin nephrosis
18	I	11 Feb.81	C80-02	1757377	Kodak	Rat	Pancreas Kidney	Alloxan toxicosis
	II	"	Cas #1	1757415	Sterling-Winthrop Labs.	Dog	Ileum	Paralytic ileus
	III	"	UT-80-902	1757303	Univ. of TN	Bovine	lung	Aspergillosis
	IV	"	77-106	1757044	Murdoch Univ. Australia	Dog	Brain	Angiostrongylus cantonensis
19	I	18 Feb.81	G-2-14	1764737	Johns Hopkins	Goat	**Synovium**	Viral leukoencephalomyelitis arthritis virus
	II	"	80-76	1771338	AFRRI	Mouse	Liver	Mouse hepatitis virus
	III	"	OARB001	1773292	FDA	Chicken	Skin	Squamous cell carcinoma
	IV	"	79N1055	1727246	Univ. of CA	Horse	Brain	Klossiella equi - Pineal

I	25 Feb 81	AFIP "1"	1716398	Wash. St. Univ.	Horse	Abdominal mass	Cystic lymphangioma
II	"	79P2240	1757311	Louisiana St. Univ.	Dog	Heart	Trypanasomiasis
III	"	80-15072	1758403	Livestock Dis. Cntr.,KY	Bovine fetus	Brain	Brucella abortus Meningitis
IV	"	AN80/254 SN80/1310	1761486	Dept. of Ag. NSW Australia	Bovine	Rumen omasum reticulum	Kikuyu grass poisoning
I	18 Mar 81	84480	1757061	L.A. Co. Dept. of Health	Dog	Bone marrow	Coccidioidomycosis
II	"	1889	1744737	EPA	Rat	Skin	Histiocytic lymphosarco
III	"	80-0356	1757048	OK St. Univ.	Sheep	Brain	Scrapie
IV	"	9-208-80	1758839	Montana St. Univ.	Goat	Placenta	Coxiella burnetti abortion
I	25 Mar 81	186-00386	1761485	Natl. Cntr. Tox. Res., AR	Rat	Mandible	Ameloblastic odontoma
II	"	63-1435	1727492	Univ. of Wisc.	Chicken	Brain	Granulomatous encepha-litis
III	"	80-2	1757054	G.A. Parker	Dog	Bone marrow	Lymphosarcoma
IV	"	H75-461a	1667029	Pig Res. Inst.,Taiwan	Pig	Kidney	Infarct, cortical necrosis Staphylococcus sp.
I	1 Apr 81	771075	1709298	NCI-NIH	Rat	Liver	Oval cell hyperplasia
II	"	11166-12	1713078	Johns Hopkins	Dog	Liver	Chronic-active hepatiti
III	"	89192	1713374	Exper. Path. Labs.	Cat	Heart	Endocardial fibro-elastosis
IV	"	78P1132	1713947	Louisiana St. Univ.	Bovine	Lung	Brucellosis pneumonia

24	I	8 Apr.81	79-2231	1714909	Faculte de Med-Vet Quebec, Canada	Goat	Ileum	Johnes' Disease
	II	"	Pigeon	1713317	MD St. Diag. Lab.	Pigeon	Liver	Myeloblastosis
	III	"	X-12280	1755475	Eli Lilly & Co.	Sea Horse	Head	Sphaeropsidales infection
	IV	"	6497-79	1777264	Wright-Patterson AFB	Baboon	Subcutis	Yaba tumor virus
25	I	15 Apr.81	8776	1771334	Univ. of AL	Rat	Lung	Mycoplasmosis, Filimentous bacterium
	II	"	28933	1758836	Letterman Army Inst. of Res.	Rhesus monkey	Uterus	Tuberculosis
	III	"	D80-5737	1756981	Univ. of IL	Turkey	Liver	Histomoniasis
	IV	"	78-R-245	1708983	Hershey Med. Ctr. Hershey, PA	Rat	Testicle	Leydig cell tumor
26	I	22 Apr.81	79-6573	1758840	Dept. of Ag. B.C.,Canada	Dog	Brain	Toxoplasmosis
	II	"	79-10197	1711324	Livestock Dis. Diag. Cntr. KY	Fox	Liver	Mycobacteriosis
	III	"	80-241	1777001	Univ of TX Hlth. Sci. Cntr.	Monkey	Blood smear	Malaria
	IV	"	80-708-12	1757060	Kans. St. Univ.	Hamster	Kidney Liver	Amyloidosis
27	I	29 Apr.81	79050 (1)	1771039	FDA	Dog	Lung	Pulmonary endarteritis
	II	"	13576-2	1761484	Univ. of MD	Rhesus monkey	Brain	Cerebral venous thrombosis
	III	"	19643-B	1783172	FDA	Rat	Kidney	Nephroblastoma
	IV	"	BG-91	1783252	Bowman-Gray Sch. of Med.	Monkey	Adrenal	Pentastomiasis

6	I	13 May 81	80-212	1757728	Natl. Zoo	Cuban Hutia	Lung	Metastatic hepatic carcinoma
	II	"	15278	1764738	Johns Hopkins	Duck	Liver	Mycobacteriosis
	III	"	80-002	1783250	Dow Chem. Co.	Mouse	Vertebra	Fibro-osseous lesions
	IV	"	8160-80	1757063	Univ. of MO	Bovine	Liver Kidney	Copper poisoning
29	I	20 May 81	035-00787	1619721	Natl. Cntr. Tox. Res.,AR	Mouse	Cervix	Malignant granular tumor
	II	"	26035	1758835	Letterman Army Res. Inst.	Dog	Brain	Cerebellar cortical abiotrophy
	III	"	AM1593-80	1758461	Purdue Univ.	Duck	Liver	Erysipelas
	IV	"	VRL-131891	1776777	Vet. Ref. Lab. Salt Lake Cty, UT	Horse	Lung	Coccidioidomycosis
30	I	27 May 81	80434	1777000	Natl. An. Diag. Lab, Ames, IA	Horse	Skin	Lymphosarcoma
	II	"	80-83	1783194	AFRRI	Dog	Liver	Copper toxicosis
	III	"	UT-80-889	1757481	Univ. of TN	Cat	Liver	Megakaryocytic myelosis
	IV	"	BG-81	1783198	Bowman-Gray Sch. of Med	Pigeon	Testicle Liver	Seminoma with metastasis to liver

Results
AFIP Wednesday Slide Conference No. 1
17 September 1980

Case I - 78310 (AFIP 1668870).

History: This specimen was a slow growing firm mass, present for one year, on the sternum of a 10-year-old spayed female spaniel mixed dog. The mass was deeply adherent to the underlying tissues. It was quite firm and white on the cut surface.

Contributor's Diagnosis & Comments: Hemangiopericytoma, skin, canine. The tumor is characterized by the presence of small capillaries which are surrounded by whorls of pleomorphic spindle-shaped cells that are considered to be pericytes. This tumor must be differentiated from neurofibromas. Reticulum stain of the hemangiopericytoma demonstrates the presence of large numbers of reticular fibers arranged in concentric layers around the small capillaries. The tumor usually infiltrated the underlying tissues, and as a result is often incompletely excised. It is not uncommon for them to recur following excision.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Hemangiopericytoma, skin, spaniel mix, canine.

Comments: All attendees concurred on the diagnosis of hemangiopericytoma. Differential diagnoses included neurofibroma/sarcoma, fibroma/sarcoma, and melanoma, whorled and dendritic type. A number of attendees suggested this tumor be placed under the broad heading of "canine cutaneous spindle cell tumors" or "canine dermatofibrosarcoma" as classified by Moulton (3). Discussion concerning the electron microscopic features of this tumor and a recent retrospective study (yet unpublished) conducted at Cornell University, reclassified 50% of the tumors diagnosed neurofibrosarcomas as hemangiopericytomas based on the presence of collagen fibers, basement membranes, microtubules, and actin filaments. Characteristics of the neurofibromas/sarcomas are presence of desmosomes, incomplete basement membranes, collagen and more rough endoplasmic reticulum and mitochondria than observed in hemangiopericytomas. Comparative studies have shown that human hemangiopericytomas bear little resemblance to canine hemangiopericytomas and that those of humans have tight junctions which are not present in the canine tumor. A number of attendees remarked that incomplete excision was a possibility in that the sections they examined contained no natural borders.

Contributor: Comparative Medical and Veterinary Services, Los Angeles County Department of Health Services, 12824 Erickson Ave., Downey, CA 90242.

References:

1. Mills, J. H. L. and Nielsen, S. W.: Canine haemangiopericytomas, a survey of 200 tumors. J. Small Animal Pract. 8: 599-604, 1967.

2. Smith, H. A., Jones, T. C. and Hunt, R. D.: Veterinary Pathology, 4th Edition, Lea & Febiger, Philadelphia, 1972, pp 205-208.

3. Stannard, A. A. and Pulley, L. T.: Tumors of the skin and soft tissue. In Tumors of Domestic Animals, edited by J. E. Moulton, University of California Press, Berkeley, 1978, pp 36-38.

Case II - 78-0594 (AFIP 1667115).

History: A year old female Bennett's wallaby (Wallabia rufogrisea), the second of three to "go down" in a zoo exhibit area, appeared to be in shock. She was unresponsive to treatment with antibiotics, fluids, B vitamins and corticosteroids and was euthanatized after 6 days. The animal was excessively fat. No impressive gross lesions were described, although pale areas were noted in the psoas and gluteal muscles.

Laboratory Results:

	May 21, 1978	May 25, 1978
WBC	10,000.	6,000.
RBC	4,810,000.	4,930,000.
Hb	15.5 gm%	16.0 gm%
Hct	45.9 %	47.1 %
Seg	93. %	60. %
Band	2. %	2. %
Lymph	4. %	38. %
Mono	1. %	0.
Alkaline Phosphatase	198. U/L	299. U/L
LDH	3,780. U/L	5,000. U/L
Glucose	27. mg/dl	88. mg/dl
Potassium	6.2 meq/L	4. meq/L
BUN	69. mg/dl	48. mg/dl
SGOT	304. U/L	5,000. U/L
SGPT	1,310. U/L	2,610. U/L
Creatinine	1.0 mg/dl	0.5 mg/dl
Bilirubin	0.1 mg/dl	0.2 mg/dl
Calcium	8.4 mg/dl	9.9 mg/dl
Phosphorus	7.3 mg/dl	4.0 mg/dl
Albumin	1.9 g/dl	2.4 g/dl
Total Protein	4.4 g/dl	5.3 g/dl

Contributor's Diagnosis & Comments: Myopathy (myodegeneration), subacute, diffuse, severe, skeletal muscle, Bennett's wallaby, possibly of Vit. E-Se dependent type.

Tongue, esophagus, and skeletal muscle were affected. A third wallaby treated for a three week period before necropsy had heavy collagenous tissue between muscle fiber bundles and lighter connective tissue surrounding surviving fibers. Lymphocytes and plasma cells were present in muscle from this animal, and both cardiac and skeletal muscle fibers had undergone mineralization. Subsequently, 2 "joeys", one out of the pouch, the other still nursing, developed sluggishness and locomotor difficulty but responded dramatically to E-Se injections. The wallabies grazed on pasture and were fed rabbit and rodent pellets, greens, and dry dog food. Bennett's variety consumed all the dog food. All three that died were overfat.

Attendees' Diagnosis & Comments: Myodegeneration and necrosis, acute, diffuse, severe, skeletal muscle, Bennett's wallaby, marsupial. Various diagnoses were given including degenerative myopathy, subacute myositis, and rhabdomyolysis.

Etiologic Diagnosis: Nutritional myopathy, Vit. E-Se deficiency.

Differential diagnosis most often given was "capture myopathy".

Attendees commented on the acute nature of the lesion and lack of an inflammatory response. Abnormalities in the clinical chemistries with elevated values included WBC, SGPT, LDH, initial potassium and BUN. Clinical chemistries with low values were: glucose, total protein, phosphorus, and calcium. Attendees agreed that the history was most consistent with Vit. E-Se deficiency. The history in "capture myopathy" usually indicates recent periods of restraint and/or muscular exertion. It was noted that the condition in these wallabies was responsive only to massive doses of Vit. E and not to Selenium as reported by others (5).

Contributor: Department of Veterinary Sciences, University of Arizona, Tucson, Arizona 85721.

References:

1. Strafuss, A. C. and Kennedy, G. A.: Degenerative myopathy in a giraffe. JAVMA 163, 551, 1973.
2. Herbert, D. M. and McTaggart, C.: White muscle disease in the Mountain Goat. J. Wildlife. Mgmt. 35: 752, 1971.
3. Owen, R. R., Moore, J. N., Hopkins, J. B. and Arthur, D.: Dystrophic myodegeneration in adult horses. JAVMA 171: 343, 1977.
4. Smith, H. A., Jones, T. C. and Hunt, R. D.: Veterinary Pathology, Lea & Febiger, Philadelphia, 1972, p. 1049.
5. MacKenzie, W. F. et al.: A myopathy in Goodfellow-Tree kangaroos (Dendrolagus goodfellowi) associated with confinement and responsive to massive doses of Vit. E. Abstracts - Symposium on Comparative Pathology of Zoo Animals, National Zoological Park, 1978.

Cas III - 27535 (AFIP 1714910).

History: In a litter of 5 cocker spaniel pups, four died acutely between the ages of 4 and 5 weeks. No clinical signs of illness were noted. At necropsy the heart and liver of these animals were reported to be enlarged.

Contributor's Diagnosis & Comments: Myocarditis, disseminated, subacute, severe, heart, canine, associated with intranuclear inclusions consistent with parvovirus infection.
Comments will be forwarded pending EM results.

Attendees' Diagnosis & Comments: Myocarditis, acute, diffuse, severe, heart, cocker spaniel, canine.
Etiologic Diagnosis: Viral myocarditis.
Etiology: Parvovirus.
Comments: Some attendees felt the changes in the tissue section were of a peracute nature while others believed the changes were more of an acute to subacute nature, as several described inflammatory infiltrates and reactive sarcolemmal cells in the tissue sections. Discussion of age-related lesions with parvovirus and clinical signs and forms were held. The myocardial form is usually found in dogs less than 8 weeks of age whereas the intestinal form is found in older dogs.

Discussion of available diagnostic tests included HA of swine RBC's with canine serum and electron microscopy of fecal samples for parvoviral particles. Caution was expressed in reliability of EM fecal examination as canine minute virus is similar in morphology, and is considered nonpathogenic for dogs. One attendee commented that there have been 216 confirmed cases of canine parvovirus infection in the State of Maryland.

Contributor: Pathology Division, USAMRIID, Fort Detrick, MD 21701.

References:

1. Hayes, M. A., Russell, R. G., Babuik, L. A.: Sudden death in young dogs with myocarditis caused by parvovirus. JAVMA 174: 1197-1203, 1979.
2. Jezyk, P. F., Haskins, M. E., and Jones, C. L.: Myocarditis of probably viral origin in pups of weanling age. JAVMA 174: 1204-1207, 1979.
3. Eugster, A. K., Bendele, R. A. and Jones, L. P.: Parvovirus infection in dogs. JAVMA 173: 1340-1341, 1978.
4. Thomson, G. W. and Gagnon, A. N.: Canine gastroenteritis associated with parvovirus-like agent. Can. Vet. J. 19: 346, 1978.

Case IV - N79-114 (AFIP 1718314).

History: Tissue is from an equine. For about two weeks prior to the admission, there had been a progressive, profound pelvic limb paresis. This was noticeably worse in the right rear with a tendency to knuckle. There was also urine retention but no dribbling of urine had been observed. Cutaneous muscle response was reduced posterior to L-4. When the animal was first admitted, it could stand, however, now it was paraplegic. In addition to the clinical signs in the posterior limbs, there was left-sided peripheral spastic paralysis.

Laboratory Results: Non-contributory.

Contributor's Diagnosis & Comments: Meningoencephalomyelitis, chronic, multifocal, granulomatous. Etiology: Protozoa, probably sarcosporidia.
Comments: None.

Attendees' Diagnosis & Comments: 1) Meningomyelitis, subacute, multifocal to diffuse, severe, spinal cord, equine. 2) Myelitis, nonsuppurative, multifocal to diffuse, severe, equine. 3) Encephalitis, nonsuppurative, multifocal to diffuse, severe, medulla, equine.
Etiologic Diagnosis: Protozoal.
Etiology: 1) Toxoplasma-like. 2) Sarcocystis sp. 3) Toxoplasma gondii.
Comment: Various diagnoses were given depending upon what tissue was present in the distributed case slides. Various modifiers also were used in describing the inflammatory reaction in the assorted sections. Some attendees described the presence of PMN's and microabscess formation in their sections. None reported the presence of either eosinophils or multinucleated giant cells, as previously reported (1). A few attendees reported they observed rosette formation within neurons. All attendees reported they observed banana-shaped organisms 2 u X 4 um in size throughout the affected areas. Other changes reported were axonal

degeneration, demyelination, and necrosis in the gray matter. The morphology and staining reactions of T. gondii were reviewed. Special stains that were provided were non-contributory. Considerable controversy exists over the exact etiology of Equine Protozoal Myeloencephalitis (EPM). Some authors prefer a Toxoplasma-like organism while a few others have incriminated T. gondii as the definitive agent. A number of reports have been generated which discuss this disease in the equine and negative results with special stains for T. gondii and animal inoculation studies. Most authors agree that the agent belongs to the large family Eimeridae, but the genus is still a matter of dispute. The intermediate stages of a sarcocystis species with the dog as the final host is considered a likely probability.

Contributor: Department of Pathology, University of Florida, Box J-145, JHMHC, Gainesville, FL 32610.

References:

1. Beech, J. and Dodd, D. C.: Toxoplasma-like encephalomyelitis in the horse. Vet. Path. 11: 87-96, 1974.

2. Mayhew, I. G. et al.: Equine protozoal myeloencephalitis in spinal cord disease in the horse. Cornell Vet. 68: 137-138, 1978.

3. Dubey, J. P., Davis, G. W. et al.: Equine encephalomyelitis due to a protozoan parasite resembling T. gondii. JAVMA 165: 249-255, 1974.

4. Dubey, J. P.: Toxoplasmosis in Horses. Letter to the Editor. JAVMA 165: 668, 15 Oct 1974.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Histories
AFIP Wednesday Conference - No. 1
17 September 1980

Case I - 78310 - (AFIP 1668870).

This surgical specimen was a slow growing firm mass, present for 1 year, on the sternum of a 10-year-old spayed female Spaniel mix dog. The mass was deeply adherent to the underlying tissues. It was quite firm and white on the cut section.

Case II - 78-0594 - (AFIP 1667115).

A year old female Bennett's Wallaby (Wallabia rufogrisea), the second of three to "go down" in a zoo exhibit area, appeared to be in shock. She was unresponsive to treatment with antibiotics, fluids, B complex vitamins and corticosteroids and was euthanatized after 6 days. The animal was excessively fat. No impressive gross lesions were described, although pale areas were noted in psoas and gluteal muscles.

Laboratory Results:

	May 21, 1978	May 25, 1978
WBC	10,000.	6,000.
RBC	4,810,000.	4,930,000.
Hb	15.5 gm%	16.0 gm%
Hct	45.9 %	47.1 %
Seg	93. %	60. %
Band	2. %	2. %
Lymph	4. %	38. %
Mono	1. %	0.
Alkaline Phosphatase	198. U/L	299. U/L
LDH	3,780. U/L	5,000. U/L
Glucose	27. mg/dl	88. mg/dl
Potassium	6.2 meq/L	4. meq/L
BUN	69. mg/dl	48. mg/dl
SGOT	304. U/L	5,000. U/L
SGPT	1,310. U/L	2,610. U/L
Creatinine	1.0 mg/dl	0.5 mg/dl
Bilirubin	0.1 mg/dl	0.2 mg/dl
Calcium	8.4 mg/dl	9.9 mg/dl
Phosphorus	7.3 mg/dl	4.0 mg/dl
Albumin	1.9 g/dl	2.4 g/dl
Total Protein	4.4 g/dl	5.3 g/dl

Case III - 27535 - (AFIP 1714910).

A litter of 5 cocker spaniel pups, four died acutely between the ages of 4 & 5 weeks. No clinical sign of illness were noted. At necropsy the heart and liver of these animals were reported to be enlarged.

Case IV - N79-114 (AFIP 1718314).

Tissue is from an equine. For about 2 weeks prior to the admission there had been a progressive, profound pelvic limb paresis. This was noticeably worse in the right rear with a tendency to knuckle. There was also urine retention but no dribbling of urine had been observed. Cutaneous muscle response was reduced posterior to L-4. When the animal was first admitted, it could stand, however, now it was paraplegic. In addition to the clinical signs in the posterior limbs, there was left-sided peripheral spatial paralysis.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Histories
AFIP Wednesday Slide Conference - No. 2
24 September 1980

Case I - 78-1850 - (AFIP 1667020).

Four kidneys were received from a slaughter-house. They had been taken from 7-month-old boars from the same herd. Of the 18 pigs slaughtered, 5 showed red spotted kidneys. The lesions measured 2 to 5 mm in diameter, were red with a whitish center and protruded slightly from the parenchyma.

Case II - 79N2080-1 - (AFIP 1713948).

Weimaraner, female, 4-month-old, was presented with a history of frequent vomiting and severe icterus for two days. Vaccination history unknown. The owner requested euthanasia.

Laboraory Results: WBC 23,000/UL, PCV 24%, Hb 8.2 g/dl, RBC 2.4 X 10^6/UL, Reticulocytes 0.3%, Platelet 50,000, 3+ target cells, poikilocytosis. Urinalysis: SG 1.013, 3+ Bilirubin, 10-15 RBC/HPF. BUN 198 mg/dl.

Case III - 79-3151 - (AFIP 1713949).

A beagle dog from a gnotobiotically derived and maintained litter was infected at 7 days of age with virulent CDV (R252 strain) via intraperitoneal route. At 22 days PID, acute respiratory difficulty developed without apparent neurologic dysfunction and the dog was euthanized *in extremis* by cardiac perfusion with fixative at 24 days PID.

Case IV - A-19735 - (AFIP 1667122).

A 9-year-old male German shepherd presented with diarrhea of two months duration. Became severe during the last five days prior to presentation. Physical and clinical work-up indicated mid-abdominal mass and gastrointestinal obstruction. Exploratory and necropsy revealed intraluminal pedunculated mass at ileocecal junction, enlarged mesenteric lymph nodes, ceco-colic intussusception and pinworms.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Results
AFIP Wednesday Slide Conference - No. 2
24 September 1980

Case I - 78-1850 - (AFIP 1667020).

Four kidneys were received from a slaughter-house. They had been taken from 7-month-old boars from the same herd. Of the 18 pigs slaughtered, 5 showed red spotted kidneys. The lesions measured 2 to 5 mm in diameter, were red with a whitish center and protruded slightly from the parenchyma.

Laboratory Results: Actinobacillus-like organisms were isolated from the lesions.

Contributor's Diagnosis & Comments: Septic embolic nephritis associated with Actinobacillus spp.

Actinobacillus spp. infection is not uncommon in piglets and has been reported occasionally from adult swine.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Nephritis, embolic, suppurative, multifocal, moderate, kidney, pig.

Etiologic Diagnosis: Gram negative bacteria.

Etiology: Salmonella spp. was the favored etiologic agent mentioned, while several others differentially preferred such organisms as Hemophilus and Pasteurella.

Comments: The participants discussed the distribution of the lesions and felt the multifocal nature and close association of blood vessels was consistent with a septicemic condition. Mention was made of glomerular involvement and some felt glomerulitis, and glomerulonephritis also were appropriate diagnoses. The Gram stain provided by the contributor demonstrated Gram negative bacilli, which most participants felt were compatible with Salmonella spp. as this is the most commonly isolated pathogenic Gram negative organism found in this species. Other bacterial agents associated with septic conditions in swine such as: E. coli, Erysipelothrix spp. and Streptococcus spp. also were briefly discussed.

Contributor: Animal Pathology Laboratory, Quebec Dept. of Agriculture, St. Hyacinthe, Quebec, Canada J25 7C6.

References:

1. Werdin, R. E. et al.: Porcine abortion caused by A. equuli. JAVMA 169: 704, 1976.

2. Bouley, C.: Etude d'une sounche d'Actionobacillus suis (Van Jartheld) isolee en Nornamdie. Rec. Med. Vet. 142: 25-29, 1966.

3. Edwards, P. R. and Taylor, R. L.: Shigella equirulis infection in a sow. Cornell Vet. 3: 393, 1941.

Case II - 79N2080-1 - (AFIP 1713948).

Weimaraner, female, 4-month-old, was presented with a history of frequent vomiting and severe icterus for two days. Vaccination history unknown. The owner requested euthanasia.

Laboratory Results: WBC 23,000/UL, PCV 24%, Hb 8.2 g/dl, RBC 2.4 X 10^6/UL, Reticulocytes 0.3%, Platelet 50,000, 3+ target cells, poikilocytosis. Urinalysis: SG 1.013, 3+ Bilirubin, 10-15 RBC/HPF. BUN 198 mg/dl.

Contributor's Diagnosis & Comments: Leptospirosis. At necropsy the animal was severely icteric and moderately emaciated. There were widely disseminated petechial and ecchymotic hemorrhages in the subcutis and on the serosal surfaces of the thoracic and abdominal visceral organs. Petechiation was most prominent in the renal cortex and lungs. The liver appeared normal except for being slightly more friable. Many lymph nodes were dark red.

Microscopic renal lesions consisted of proximal tubular degeneration, necrosis, multifocal mineralization, diffuse interstitial nephritis, characterized by moderate infiltration of lymphocytes, plasma cells, and macrophages; edema and hemorrhages. Liver showed diffuse severe individualization of hepatocytes. The lymph nodes were markedly hemorrhagic with severe erythrophagocytosis. Microscopic findings of the kidney and liver are considered characteristic of canine leptospirosis. Dark field examination of urine collected at the time of necropsy revealed numerous spirochetes.

Attendees' Diagnosis & Comments:

Morphologic Diagnoses: 1) Nephritis, interstitital, subacute, diffuse, moderate, kidney, canine. 2) Hepatocellular dissociation, diffuse, severe, liver.

Etiologic Diagnosis: Leptospirosis.

Etiology: Leptospiria spp.

Comments: Attendees unanimously agreed with the contributor's diagnosis, and histologic findings. In addition to the diffuse infiltration of the interstitium with lymphoplasmocytic cells and the proximal tubular degeneration, some attendees felt there was a mild early fibrosis accompanying the inflammation. The changes in the liver tissue were discussed and felt to be highly suggestive of leptospirosis, although other conditions in which there is high body temperature may mimic this change. A few attendees commented on a possible hepatocellular degeneration along with the dissociation. The role of leptospiral organisms in canine chronic interstitial nephritis was discussed briefly and viewpoints both for and against were expressed.

Contributor: Department of Veterinary Pathology, Louisiana State University, Baton Rouge, LA 70803.

References:

1. Higgins, R. and Cousineau, G.: The pathogenesis of leptospirosis. I. Hemorrhages in experimental leptospirosis in guinea pigs. Can. J. Comp. Med. 41: 174-181, 1976.

2. Higgins, R. and Cousineau, G.: The pathogenesis of leptospirosis. II. Jaundice in experimental leptospirosis in guinea pigs. Can. J. Comp. Med. 41: 182-187, 1976.

3. Morrison, W. I. & Wright, N. G.: Canine leptospirosis: An immunopathological study of interstitital nephritis due to L. canicola. J. Pathol. 120: 83-89, 1976.

Case III - 79-3151 - (AFIP 1713949).

A beagle dog from a gnotobiotically derived and maintained litter was infected at 7 days of age with virulent CDV (R252 strain) via intraperitoneal route. At 22 days PID, acute respiratory difficulty developed without apparent neurologic dysfunction and the dog was euthanized *in extremis* by cardiac perfusion with fixative at 24 days PID.

Contributor's Diagnosis & Comments: Myocardial necrosis, multifocal, disseminated with mineralization.

Multiple small whitish linear streaks were restricted to the ventricular myocardium and lungs appeared edematous. Similar gross and microscopic lesions were noted in dogs from 4 similarly treated litters with 1-4 dogs affected in each litter. No lesions were noted in age matched uninfected parallel controls or in dogs receiving non-neurovirulent tissue culture adopted CDV (R252) via the same route at the same age. In addition this dog had a viral encephalopathy with widespread glial and neuronal infection without demonstrable inflammatory cell response, generalized lymphoid tissue depletion and mild interstitial pneumonia. Ultrastructural examination of cardiac lesions demonstrated intracytoplasmic tubular filamentous aggregates, morphologcally compatible with CDV paramyxovirus, restricted to degenerating myocytes and interstitial macrophages and intact myocytes at the periphery of lesions. Previous *in vitro* studies have shown that neonatal dogs infected with virulent R252-CDV experience a depression of both cell mediated and humoral immunologic responses along with a cell and plasma associated viremia. Serum/plasma samples from involved litters were tested weekly for Ca, PO_4 and Mg levels and terminally for selenium, glutathione peroxidase and vitamin E levels. All levels were within normal limits compared to conventionally raised dogs. Slight elevations of SGOT but not CPK were observed but did not correlate with occurrence of lesions. The possible contributory role of CNS involvement, slight anemia and depressed immunologic responsiveness in the development of these lesions is not known. The lesions presented here must be differentiated from canine parvovirus myocarditis.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Degeneration and necrosis with mineralization, acute, multifocal, moderate, heart, canine.

Etiologic Diagnosis: Viral myocarditis.

Etiology: Canine Distemper Virus.

Comments: All attendees agreed with the contributor's diagnosis and etiologic agent. Discussion of viruses that cause myocarditis in other species included Foot and Mouth Disease in cattle (picornavirus), Encephalomyocarditis in swine (picornavirus), Coxsackie - B virus in neonatal mice and coronavirus in mice and rabbits. Also discussed were the pathogenesis of calcification and the early anoxic changes which may be confused with or secondary to viral myocarditis.

Contributor: Deartment of Veterinary Pathobiology, College of Veterinary Medicine, The Ohio State University, 1925 Coffey Road, Columbus, OH 43210.

References:

1. Krakowska, S. and Wallace, A. L.: Lymphocyte associated immune responses and measles virus in distemper-infected gnotobiotic dogs. Am. J. Vet. Res. 40: 669-672, 1979.

2. Higgins, R. J. et al.: Canine distempter virus-associated cardiomyopathy in the dog. (In Press).

Case IV - A-19735 (AFIP 1667122).

History: A 9-year-old male German shepherd was presented with diarrhea of two months duration that became severe during the last five days prior to presentation.

Laboratory Results: Physical and clinical work-up indicated a mid-abdominal mass and gastrointestinal obstruction. Exploratory surgery and necropsy revealed an intralumenal pedunculated mass at the ileocecal junction, enlarged mesenteric lymph nodes, ceco-colic intussuseption and whipworms, (previous history given indicating pinworms was in error). No cutaneous neoplasms were seen. Histologically neoplastic cells also were seen in the lymph nodes, bone marrow, spleen, liver and lungs. These cells were confirmed via electron microscopy as well.

Contributor's Diagnosis & Comments:

Diagnosis: Malignant mast cell tumor, intestine, ileocecal valve.

Comment: The neoplasm is considered malignant because of the presence of neoplastic cells in other organs. Primary mast cell tumors in the intestine have been described in cats, but no references were found for the dog. The mast cells seen in this dog are similar to cutaneous neoplasms, in contrast to the cells in the intestine of cats.

Attendees' Diagnosis & Comments: Mast cell tumor, malignant, large intestine, canine. Variations in designating this neoplasm included malignant mastocytoma and mastocytosis.

Comment: There was total agreement with the contributor's diagnosis. Giemsa stained sections of the tissue revealed numerous cells containing various numbers of Giemsa positive intracytoplasmic granules, consistent with mast cells. The association of eosinophils and mast cell tumors was discussed and is considered to be due to chemotactic factors for eosinophils elaborated by mast cells as well as other eosinophil attracting factors such as $C5_a$, $C3_a$, lymphokines and histamine (weakly). Vaso-active amine release with resultant edema usually associated with mast cell tumors also was discussed.

Contributor: The Animal Medical Center, 520 E. 62 St., New York, NY 10021.

Reference:

Alroy, J. et al.: Distinctive intestinal mast cell neoplasms of domestic cats. Lab. Invest. 33: 159-167, 1975.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Histories
AFIP Wednesday Slide Conference - No. 3
1 October 1980

Case I - 13696 - (AFIP 1755478).

Tissue is from a year old male rhesus monkey born and reared in a primate colony that suddenly became anorectic, weak, hypothermic and dehydrated. Despite symptomatic treatment, the animal died 6 days after the onset of symptoms and was necropsied.

Laboratory Results: The hematological values were within normal limits except for a slight monocytosis (15%). SV40 was isolated from the spleen postmortem.

Case II - CA5-197 - (AFIP 1755476).

A female nulliparous Chinese hamster died at 992 days of age. A yellowish white nodular mass measuring about 1 cm diameter at its greatest dimensions surrounded the uterus and colon.

Case III - 71537 - (AFIP 1712658).

Adult cockatoo, one of several psittacine birds in a pet shop which died following a brief illness marked by profuse green watery diarrhea. These birds became sick one week after the arrival of a shipment of domestically-raised cockatiels and parakeets.

Case IV - 27469 - (AFIP 1714918).

A 9-year-old female mixed breed canine was presented on 25 Jan 79 because of vomiting over the previous 3 days. It responded to symptomatic treatment. On 30 Apr 79 it was returned after constant vomiting during the previous week. It was emaciated, very depressed, and was markedly dehydrated. Blood urea nitrogen levels remained elevated despite intravenous fluid administration and attempts at peritoneal dialysis. The owner requested euthanasia. Necropsy done by the clinician revealed pale firm kidneys and no other abnormalities.

Laboratory Results:

Date	PCV	TP	WBC	BUN	Na+	K+	SGPT	GLU
27 Jan 79	45	7	15,000 (84% Seg)	19	143	3.5	35	93
30 Apr 79	48	9	5,700 (83% Seg)	126	143	3.7	20	75
1 May 79				110				

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Results
AFIP Wednesday Slide Conference - No. 3
1 October 1980

Case I - 13696 - (AFIP 1755478).

History: Tissue is from a year old male rhesus monkey born and reared in a primate colony that suddenly became anorectic, weak, hypothermic and dehydrated. Despite symptomatic treatment, the animal died 6 days after the onset of symptoms and was necropsied.

Laboratory Results: The hematological values were within normal limits except for a slight monocytosis (15%). SV40 was isolated from the spleen postmortem.

Contributor Diagnosis & Comments:
1) Kidney, renal tubular necrosis, multifocal, marked.
2) Kidney, renal tubular cytomegaly, with inclusions, multifocal.
3) Kidney, chronic pyelitis, mild.

Comments: Besides the renal tubular necrosis, the animal also had a severe patchy interstitial pneumonia with similar associated basophilic intranuclear inclusions. These inclusions were positive for SV40 antigen by immunoperioxidase staining of paraffin sections of affected organs. Electron microscopy revealed characteristic papovavirus particles in these tissues. This is the first report indicating that Simian Virus 40 may produce pulmonary and renal disease in its natural host.

Attendees' Diagnosis and Comments:
Morphologic Diagnosis: Necrosis, tubular, diffuse, severe with intranuclear inclusion bodies and multifocal tubular regeneration, kidney, rhesus monkey.
Etiologic Diagnosis: Viral renal tubular necrosis.
Etiology: SV40 virus.

Comments: The above morphologic diagnosis was the consensus following considerable discussion. Attendees' diagnoses turned in before the conference are listed in order of frequency: 1) Nephritis, interstitial, subacute, diffuse, severe, kidney. 2) Tubulo-interstitial nephritis, subacute, diffuse, marked, kidney. 3) Glomerulonephritis, subacute, diffuse, moderate to severe, kidney.

Attendees also listed the chronic pyelitis as either a separate morphologic diagnosis or included it in their primary diagnosis. Other etiologic diagnoses given were toxic and immune mediated. A substantial number of conference participants listed cytomegalovirus or adenovirus as the probable etiology. Herpes B virus was listed by a few as a possible etiologic agent. Intranuclear inclusion bodies were observed by all in the nuclei of the convoluted tubular epithelium both in intact and necrotic cells. Discussion of the SV40 virus included its previously stated nonpathogenicity in natural hosts (1), such as rhesus and African Green Monkeys; the contamination of early polio vaccines prepared from kidney tissue cultures of these animals; and evidence of early vertical transmission (3). Tumors induced by the virus include fibrosarcomas in suckling hamsters inoculated subcutaneously; ependymomas in suckling hamsters inoculated intracerebrally and in newborn mastomys inoculated subcutaneously; and other malignancies such as anaplastic sarcomas, lymphomas and osteogenic sarcomas in 3-week-old hamsters inoculated intravenously (1).

Contributor: Department of Pathology, School of Medicine, University of Maryland, Baltimore, MD 21201.

References:
1. Hunt, R. D. et al.: Viral diseases (SV40). In The Pathology of Laboratory Animals, Ed. by K. Benirschke, F. M. Garnner & T. C. Jones, Springer-Verlag, 1978, pp 1325-1326.
2. Sheffield, W. D. et al.: Simian virus 40 associated fatal interstitital pneumonia and renal tubular necrosis in a rhesus monkey. J. Infect. Dis. (to be published in October issue).
3. Kaschula, V. R., Van Dellen, A. F. and DeVos, D.: Some infectious diseases of wild Vervet monkeys (Cercopithecus aethips pyserythrus) in South Africa. J. South Africa Vet. Assoc. 49: (3) 223-227, 1978.

Case II - CA5-197 - (AFIP 1755476).

History: A female nulliparous Chinese hamster died at 992 days of age. A yellowish white nodular mass measuring about 1 cm diameter at its greatest dimensions surrounded the uterus and colon.

Contributor's Diagnosis & Comments: Endometrial adenocarcinoma.

Comments: Endometrial neoplasms occur with a relatively high incidence in aged Chinese hamsters. This high incidence, their low chromosome number (2N=22) and small size make them suitable subjects for experimental studies of endometrial cancer.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Adenocarcinoma, uterus, hamster.

Comments: Attendees felt "poorly differentiated" was an appropriate additional modifier for this neoplasm. An adenosquamous variant of this neoplasm is seen in significantly older hamsters. Other tumors of the uterus in this species include carcinosarcomas and malignant mixed mesodermal tumors of Mullerian origin and leiomyomas and leiomyosarcomas (2). Spontaneous endometrial adenocarcinoma also occurs frequently in older rabbits of various breeds (3).

Contributor: Lovelace Inhalation Toxicology Research Institute, P. O. Box 5890, Albuquerque, NM 87115.

References:
1. Benjamin, S. A. and Brooks, A. L.: Spontaneous lesions in Chinese hamsters. Vet. Pathol. 14: 449-462, 1977.
2. Brownstein, D. G. and Brooks, A. L.: Spontaneous endometrial neoplasms in aging Chinese hamsters. JNCI 64: 1209-1214, 1980.
3. Baba, N. and von Haam, E.: Spontaneous endometrial adenocarcinoma in aged rabbits. In Handbook: Animal Models of Human Disease, Fasc. 1, Model No. 21, Ed. by T. C. Jones, D. B. Hackel and G. Migaki, Registry of Comparative Pathology, AFIP, 1972.
4. Ward, B. C. and Moore, W.: Spontaneous lesions in a colony of Chinese hamsters. Lab. Anim. Care 19: 516-521, 1969.

Case III - 71537 - (AFIP 1712658).

History: Adult cockatoo, one of several psittacine birds in a pet shop which died following a brief illness marked by profuse green watery diarrhea. These birds became sick one week after the arrival of a shipment of domestically-raised cockatiels and parakeets.

Contributor's Diagnosis & Comments:

Acute multifocal hepatic necrosis with associated herpesviral inclusion bodies (Pacheco's Disease).

Attendees' Diagnosis & Comments:

Necrosis, acute, diffuse, severe, liver, Cockatoo.

Etiologic Diagnosis: Viral hepatitis.

Etiology: Herpes virus.

Comments: Although some attendees preferred a morphologic diagnosis of necrotizing hepatitis, the consensus was that the primary lesion was necrosis and not inflammation. Other etiologic agents listed differentially were adenovirus and paramyxovirus. The intranuclear eosinophilic inclusions were considered to be characteristic of herpesviruses, however, the diffuse distribution of the lesion compared to the multifocal nature of most herpetic lesions in the liver of other species was considered unusual by some. A minimal inflammatory component was reported by several attendees. Fixatives which may be used to enhance the intranuclear inclusions such as Bouin's and Zenker's were mentioned.

Contributor: Department of Veterinary Pathology, College of Veterinary Medicine, Texas A&M University, College Station, TX 77843.

References:

1. Simpson, C. F., Hanley, J. E. & Gaskin, J. M.: Psittacine herpesvirus infection resembling Pacheco's Parrot Disease. J. Infect. Dis. 131: 390-396, 1975.

2. Simpson, C. F. and Manley, J. E.: Pacheco's Parrot Disease of psittacine birds. Avian Dis. 21: 209-219, 1977.

Case IV - 27469 - (AFIP 1714918).

A 9-year-old female mixed breed canine was presented on 25 Jan 79 because of vomiting over the previous 3 days. It responded to symptomatic treatment. On 30 Apr 79 it was returned after constant vomiting during the previous week. It was emaciated, very depressed, and was markedly dehydrated. Blood urea nitrogen levels remained elevated despite intravenous fluid administration and attempts at peritoneal dialysis. The owner requested euthanasia. Necropsy done by the clinician revealed pale firm kidneys and no other abnormalities.

Laboratory Results:

Date	PCV	TP	WBC	BUN	Na+	K+	SGPT	GLU
27 Jan 79	45	7	15,000 (84% Seg)	19	143	3.5	35	93
30 Apr 79	48	9	5,700 (83% Seg)	126	143	3.7	20	75
1 May 79				110				

Contributor's Diagnosis & Comments: Renal amyloidosis.

All renal glomeruli had moderate to severe segmental to panglomerular deposits of hyaline homogeneously acidophilic material. Examination of Congo red stained sections using crossed Nicol prisms revealed green birefringence of the material within glomeruli consistent with amyloid.

Additional renal changes included degeneration of tubules, interstitital plasma cell infiltrates, and intratubular proteinaceous casts and aggregates of mineralized debris.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Amyloidosis, glomerular, diffuse, severe, kidney, mixed breed canine.

Etiologic Diagnosis: Amyloidosis.

Etiology: Spontaneous amyloidosis or secondary amyloidosis.

Comment: Attendees unanimously agreed with the contributor's diagnosis and comments. Laboratory results commented on were relative anemia and hypoproteinemia masked by the reported marked dehydration. A leucopenia was felt to be present in the latter stage of the disease along with the anemia. Changes in serum chemistries commented on were initial low normal Na+ and K+ levels, and later marked elevations in BUN. Attendees discussed the association of complicating thrombosis in renal amyloidosis, which is postulated to be due to hemoconcentration, hyperfibrinogenemia, hyperglobulinemia, and clot promoting factors producing a hypercoagulable state (2). The apparent lack of immunogenicity of amyloid depositions in various tissues was discussed. One attendee reported several cases of amyloidosis in nasal masses of horses seen at New Bolton Center, University of Pennsylvania.

The pathogenesis of this disease remains uncertain. Primary amyloidosis has been reported in humans, dogs and mice. It is associated with multiple myeloma in man. Secondary amyloidosis is considered more common in animals and is thought to be associated with chronic disease states including inflammatory and neoplastic conditions. Serum proteins associated with primary amyloidosis are AL or light chains, and those associated with secondary amyloidosis are SAA proteins, both related to immunoglobulins. Daily injections of casein in mice causes chronic antigenic stimulation and results in renal amyloidosis after 22-26 weeks. Recently it was observed that amyloid was redistributed to the kidney from the primary sites in the spleen and liver in animals receiving daily injections of casein for 21 days and then discontinued for periods of 2 to 6 months. A similar condition also has been reported in rabbits (3).

Contributor: USAMRIID, Pathology Division, Fort Detrick, MD 21701.

References:

1. Osborne, C. A. et al.: Renal amyloidosis in the dog. JAVMA 153: 668-669, 1968.

2. Slauson, D. O. & Gribble, D. H.: Thrombosis complicating renal amyloidosis in dogs. Vet. Pathol. 8: 352-363, 1971.

3. Shirahama, T. & Cohen, A. S.: Redistribution of amyloid deposits. Am. J. Path. 99: 539-550, 1980.

4. DiBartola, S. P. et al.: Urinary protein excretion and immunopathologic findings in dogs with glomerular disease. JAVMA 177: 73-77, 1980.

5. Glenner, G. G.: Amyloid deposits and amyloidosis (2 parts). N. Eng. J. Med. 302: 1283-1292, and 302: 1333-1343, 1980.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Histories
AFIP Wednesday Slide Conference - No. 4
8 October 1980

Case I - 23197 - (AFIP 1619627).

Tissue from a female guinea pig. Clinically, the animal was emaciated, reluctant to move, and had a scant amount of dried, yellow nasal discharge.

Case II - 7464 - (AFIP 1712661).

Horse had signs of colic. Initially noted staggering in pasture. The animal had a "dummy attitude" in the terminal stage.

Case III - 648T - (AFIP 1755477).

Female beagle, 4.25 years old, presented to clinic weak and depressed. Physical examination revealed icterus, pale mucous membranes, dehydration, temperature = 104°F, pulse 156/min. respiration 60/min., bloody urine. No evidence of external blood loss other than hematuria. No known exposure to toxic materials. Treated with corticosteroids, electrolytes and antibiotics, but died 3 days later.

Laboratory Results:

Hematology	RBC X 106/cmm	Hct	Hb	Platelets X 103/cmm	NRBC	WBC/cmm	Bands
48 days before	7.6	5.5	17.2	305	-	11,000	3%
Clinic admiss.	0.76	5.0	3.5	15	22/100	15,300	30%

Spherocytosis - Anisocytosis +3

Clin. Chem. Total Protein 7.6 mg%, BUN 50 mg/dl; SGPT 700Iu/mL

Case IV - 191712-713 - (AFIP 1713082).

The history reported was that 10 from a group of 20 Muscovy ducks had died within the past 3 days. It was noted that other ducks were on the premise, but there had not been a problem in the other ducks. It was reported that the red faces of the birds got very dark, almost black. The birds stopped eating but did drink water and then died within 2 to 3 days. It was noted that some birds had died in 24 hours. The birds had not been medicated or vaccinated. The diet consisted of corn, bread, lettuce and chicken mash for the baby birds. Housing was in the woods with a shelter with 1 side of the area boarded by a creek. It was noted that there were cement and plastic pools in the area as well. Additional history indicated that by 5 days after submission of these particular specimens, a majority of the Muscovy ducks on the premise had died.

Microbiologic examination: Pasteurella multocida was recovered from the lung, liver, kidney and intestine submitted for culture.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Results
AFIP Wednesday Slide Conference - No. 4
8 October 1980

Case I - 23197 (AFIP 1619827).

History: Tissue from a female guinea pig. Clinically, the animal was emaciated, reluctant to move, and had a scant amount of dried, yellow nasal discharge.

Contributor's Diagnosis & Comments: Bronchopneumonia, multifocal, subacute, severe, guinea pig.

Comments: A Brown-Hopps stain revealed numerous encapsulated Gram negative bacterial rods. Klebsiella pneumoniae was cultured from the lung.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Bronchopneumonia, suppurative, subacute, multifocal to diffuse, severe, lung, guinea pig.

Etiologic Diagnosis: Bacterial pneumonia.

Etiology: Most compatible with a Gram negative bacterial rod. A differential included Bordetella sp., Klebsiella sp., Pasteurella sp., and Pseudomonas sp.

Comments: The histological features of the lesion discussed were the predominant neutrophilic response, the clear zones noted surrounding many of the bacteria, and the numerous and diffuse vacuoles or small clear spaces both within foci of pneumonia and thickened septal structures surrounding clear alveoli. The moderator commented he had observed similar vacuolization in tissue sections of other guinea pigs dying from this and other causes and felt this change was due to fat-droplet emboli. The origin of the fat correlates with a corresponding fatty liver often seen in sick and dying guinea pigs, and is apparently related to anorexia and emaciation. The contents of these vacuoles would have been removed during processing. The pronounced infolding of the bronchiolar epithelium was postulated to be due to contraction of the prominent bronchiolar musculature upon contact with the fixative. Bacterial agents responsible for pneumonias in guinea pigs were discussed.

Contributor: USAMRIID, Division of Pathology, Fort Detrick, MD.

References:

1. Berendt, R. F., Long, G. G., Abeles, P. G., Canonico, F. B., Elwell, M. R., and Powanda, M. D.: Pathogenesis of respiratory Klebsiella pneumoniae infection in rats: Bacteriological and histological findings and metabolic alterations. Infect. & Immunol. 15: 586-593, 1977.

2. Kunz, L. L. and Hulton, G. M.: Diseases of the laboratory guinea pig. Veterinary Scope, pp 12-20, 1971, The Upjohn Co., Kalamazoo, Michigan.

Case II - 7464 - (AFIP 1712661).

History: Horse had signs of colic. Initially noted staggering in pasture. The animal had a "dummy attitude" in the terminal stage.

Laboratory Results: No laboratory work was performed. The practicing veterinarian submitted fresh and formalin fixed tissues to the laboratory. There were negative virus isolation results and no significant findings in bacteriology.

Contributor's Diagnosis & Comments: Crotalaria Poisoning.

Comments: The practitioner also submitted plants the animal had been eating, which were identified as Crotalaria spectablis.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Degeneration and necrosis, centrilobular, subacute to chronic, multifocal to diffuse, severe, liver.

Comments: The morphologic diagnosis was agreed upon after some discussion. Some attendees felt there was a mild inflammatory component and a diagnosis of hepatitis was more appropriate. Most attendees commented on a mild variation in size of the hepatocytes, primarily those in periportal areas of the hepatic lobules. A few attendees commented on an occasional cell which appeared to be undergoing regeneration. Centrilobular degeneration and necrosis and mild vacuolization of the hepatocytes was reported by all. There was considerable controversy over the reported etiology. Participants which have had some experience reviewing lesions due to pyrrolizidine alkaloids felt that this was an unusual presentation of a naturally occurring case and considered the lesions more consistent with those observed in serum hepatitis of horses. A number of features which are usually present in liver tissue from animals with pyrrolizidine alkaloid toxicosis were absent. These features include: portal fibrosis, extreme megalocytosis, and bile duct proliferation. It also was pointed out that the usual case is that of chronic intoxication rather than acute toxicity as the plant is not considered palatable in large quantities consumed over a short period of time. Serum enzyme tests for pyrrolizidine alkaloid toxicosis include gamma glutamyl transpeptidase (GGT) and alkaline phosphatase (AP). Although nonspecific, enzyme levels exceeding 35 IU/L of GGT and 300 IU/L of AP are considered indicative of hepatic damage of the type caused by pyrrolizidine alkaloids of Senecio jacobaea in horses and cattle in the Pacific Northwest.

Other toxicities discussed were locoweed poisoning in horses in which there is a mononuclear response in addition to portal fibrosis, and moldy corn poisoning in cattle and sheep which have some features in common with pyrrolizidine alkaloid poisoning such as centrilobular degeneration and necrosis in acute cases and extensive fibrosis and bile duct proliferation in chronic cases.

Etiologic Diagnosis: Toxic hepatitis.

Etiology: Pyrrolizidine alkaloid poisoning or serum hepatitis (Theiler's Disease of horses).

Contributor: C. E. Kord Animal Disease Laboratory, P. O. Box 40627, Melrose Station, Nashville, TN 37204.

References:
1. Jubb & Kennedy: Pathology of Domestic Animals, Vol. 2, Academic Press, 1970, pp 216-219 and pp 221-224.
2. Craig, A. M. et al.: Serum enzyme tests for pyrrolizidine alkaloid toxicosis. Amer. Assoc. Veterinary Laboratory Diagnosticians, 21st Annual Proceedings, 1978, pp 161-178.

Case III - 648T - (AFIP 1755477).

History: Female beagle, 4.25 years old, presented to clinic weak and depressed. Physical examination revealed icterus, pale mucous membranes, dehydration, temperature = 104°F, pulse 156/min. respiration 60/min., bloody urine. No evidence of external blood loss other than hematuria. No known exposure to toxic materials. Treated with corticosteroids, electrolytes and antibiotics, but died 3 days later.

Laboratory Results:

Hematology	RBC X 106/cmm	Hct	Hb	Platelets X 103/cmm	NRBC	WBC/cmm	Bands
48 days before	7.6	55.	17.2	305	-	11,000	3%
Clinic admiss.	0.76	5.0	3.5	15	22/100	15,300	30%

Spherocytosis - Anisocytosis +3

Clin. Chem. Total Protein 7.6 mg%, BUN 50 mg/dl; SGPT 700Iu/mL

Contributor's Diagnosis & Comments: A diagnosis of autoimmune hemolytic anemia was made based on the clinical history and hematologic findings. In addition to the tubular necrosis and hemaglobinuria the dog also had centrilobular necrosis of the liver and mild marrow erythroid hyperplasia.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Degeneration and necrosis, tubular, acute, multifocal to severe, with multifocal hemoglobin casts, severe, kidney.

Etiologic Diagnosis: Biliary and hemoglobinuric nephrosis.

Etiology: Autoimmune Hemolytic Disease.

Comments: There was general agreement with the contributors diagnosis and comments. Other morphologic diagnoses given by a few participants were glomerulonephritis and glomerulonephrosis; however, after some discussion these were viewed as unsubstantiated without appropriate thin sections and special stains. Glomerular lesions are not a reported feature of this condition in dogs, but are seen in systemic lupus erythematosus hemolytic anemia of dogs. The pathogenesis of the tubular lesions in this case was discussed. Hypoxia was favored over a direct toxic effect of the hemoglobin deposition, as a plausable explanation. The left shift in the neutrophilic series was commented on and is reported in this condition. The underlying cause was not ascertained, but may be related to the bone marrow hyperplasia and release of blood cells.

Contributor: Lovelace Inhalation Toxicology Research Institute, P. O. Box 5890, Albuquerque, NM 87115.

References:

1. Dodds, W. Jean: Autoimmune hemolytic disease and other causes of immune-mediated anemia: An overview. JAAHA 13: 437-441, 1977.
2. Harvey, J. W.: Canine hemolytic anemias. JAVMA 176: 970-974, 1980.

Case IV - 191712-713 - (AFIP 1713082).

The history reported was that 10 from a group of 20 Muscovy ducks had died within the past 3 days. It was noted that other ducks were on the premise, but there had not been a problem in the other ducks. It was reported that the red faces of the birds got very dark, almost black. The birds stopped eating but did drink water and then died within 2 to 3 days. It was noted that some birds had died in 24 hours. The birds had not been medicated or vaccinated. The diet consisted of corn, bread, lettuce and chicken mash for the baby birds. Housing was in the woods with a shelter with 1 side of the area boarded by a creek. It was noted that there were cement and plastic pools in the area as well. Additional history indicated that by 5 days after submission of these particular specimens, a majority of the Muscovy ducks on the premise had died.

Microbiologic examination: Pasteurella multocida was recovered from the lung, liver, kidney and intestine submitted for culture.

Contributor's Diagnosis & Comments:

Gross Lesions: The ducks were in generally good body condition and the skin on the head was very cyanotic. Internal lesions included many petechial hemorrhages on the heart, mesentery of the gut and liver. There was a marked pericarditis with apparent focal necrosis in the heart. The spleens were very soft and hemorrhagic and blood exuded freely from the cut surface. The livers had a very mottled appearance with apparent areas of necrosis. There were discrete areas of necrosis in the intestine with a fibrinonecrotic membrane attached to the surface. There was free blood in the lumen of the gut in the bird which had been dead the longest. There was also hemorrhage at the junction of the proventriculus and esophagus in the bird which had died before the owner had left home. It was reported that 1 duck had died since the owner had left home and this particular bird had very similar lesions with the exception of free blood in the lumen of the gut and the hemorrhage at the junction of the proventriculus and esophagus.

Laboratory Findings:

Virologic Examination: Virus isolation attempts are in progress both in the virologic section at MSU and at the National Animal Disease Center in Ames, Iowa. When these results are obtained a supplemental report will be sent. Virus has not been isolated at this point in time from the tissues of the ducks.

Microbiologic Examination: Pasteurella multocida was recovered from the lung, liver, kidney and intestine submitted for culture.

Histopathologic Examination: On microscopic examination there were fairly severe lesions in the intestinal tract of both birds. There was desquamation of epithelium as well as hemorrhage into the submucosa and tunica muscularis in some areas. There was necrosis of lymphoid tissue within the submucosa of the intestine in some locations. In addition, there were many bacterial colonies generally on the mucosal surfaces or the ulcerated mucosa of the intestine as well as in a number of the visceral organs. There was desquamation and necrosis on the

epithelium of the trachea in the bird which had been dead the longest. There were numerous bacterial colonies on the mucosal surface of the trachea in this particular bird. There was severe hyperemia and focal hemorrhage in the lung without much indication of inflammatory infiltrate into the lung. There was fairly marked hemorrhage in the myocardium extending in some areas throughout the total thickness of the myocardium from the pericardium to the endocardium. In several sections of posterior intestine from the birds there was massive hemorrhage into the submucosa with necrosis of lymphoid nodules within the submucosa in these same sections of intestine as well as some of those anterior to this location. There was necrosis of much of the epithelium even down into the crypts while the interstitium surrounding the crypt epithelium was relatively normal. There were often large bacterial colonies within the areas of hemorrage and necrotic tissue. On examination of the liver there were areas of necrosis with intranuclear inclusion bodies in varying stages of development in numerous hepatic cells within and at the edges of the areas of necrosis. In addition, there were areas of eosinophilic globules surrounded by halos in the cytoplasm of some of the degenerating hepatocytes. Similar cytoplasmic changes had been noted in the sections of intestine examined. There appeared to be disruption of the endothelial lining of the sinusoids in the liver at some locations with hemorrhage into the surrounding hepatic parenchyma. There was no indication of inflammatory change in the liver and bacterial colonies were not noted in the liver of the duck which had recently died. There was complete disappearance of lymphoid tissue within the spleen. There were areas of hemorrhage and variable amounts of congestion in nearly all the other tissues examined.

Conclusions: The history, gross and microscopic lesions are consistent with a provisional diagnosis of duck plague. In addition, it was felt that the 2 birds submitted to the laboratory had a complicating terminal Pasteurell multocida infection or fowl cholera. (Virus isolation attempts as well as other laboratory methods of demonstration of viral agents are in progress and will be reported at a later date as the results become available.)

Supplemental Report:

Virologic examination: An oral report has been received from the National Animal Disease Center, Ames, Iowa, indicating that the virus of duck plague, or Duck Virus Enteritis, was isolated from the tissues submitted for examination.

Conclusions: This finding confirms our previous diagnosis of duck plague.

Attendees' Diagnoses & Comments:

Morphologic Diagnoses: 1) Degeneration and necrosis, acute, multifocal to diffuse, severe, with eosinophilic intranuclear inclusion bodies, liver, Muscovy duck, avian. 2) Enteritis, necrotizing, acute to subacute, diffuse, moderate to severe, small intestine. 3) Congestion, acute, diffuse, severe, spleen. 4) Pneumonia, hemorrhagic, peracute to acute, multifocal, moderate, lung.

Etiologic Diagnosis: Duck Viral Enteritis (DVE), with secondary Pasteurella pneumonia.

Etiology: Herpesvirus.

Comments: The above diagnoses were reached after some discussion of the lesions. The tissues present in the submitted slides varied as to which organs were present. Most slides contained liver and small intestine. The distribution and severity of lesions in the different sections varied from multifocal to diffuse and from moderate to severe. A number of attendees commented on a moderate

postmortem change present in their slides and felt this masked some of the lesions and accounted for an apparent bacterial overgrowth within the intestinal and pulmonary tissues. Some participants did not feel a true pneumonia was present in the sections of lung, but agreed there was congestion and hemorrhage present. The eosinophilic intranuclear inclusion bodies were observed by all and considered characteristic of herpes viral inclusions. A few attendees reported seeing similar inclusions in some of the sloughed and necrotic intestinal lining cells. Comment was made on the severe congestion of the spleen and a lymphoid depletion. A loose reticular appearance around Schweigger-Seidel sheaths was reported but considered normal in view of the congestion and postmortem change. Discussion included species susceptability among Anseriformes and vaccination (immunization) effectiveness among birds.

Contributor: Animal Health & Disease Laboratory, Michigan State University, P. O. Box 30076, Lansing, MI 48909.

References:

1. Leibovitz, L.: Gross and histopathologic changes of duck plague (Duck Virus Enteritis). Am. J. Vet. Res. 32 (2): 275-290, 1971.

2. Proctor, S. J.: Pathogenesis of duck plague in the Bursa of Fabricius, thymus, and spleen. Am. J. Vet. Res. 37 (4): 427-431, 1976.

3. Toth, T. E.: Active immunization of White Pekin ducks against Duck Virus Enteritis (Duck Plague) with modified live virus vaccine: Serologic and immunologic response of breeder ducks. Am. J. Vet. Res. 32 (1): 75-81, 1971.

4. Leibovitz, L.: Diseases of Poultry, 7th Ed., 1978, pp 621-632.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Histories
AFIP Wednesday Slide Conference - No. 5
15 October 1980

Case I - 176750 (AFIP 1681107).
This Ross Leg Touraco (band #459) was found dead in the exhibit. A second bird was found down on Aug 22, 1978, removed from the exhibit, and seemed to recover after being isolated from the group.

Case II - 77PN1623D - (AFIP 1667702).
Tissues from a Black iguana, female, about three years old, presented to necropsy because multiple protuberances in most lung bones of the body. On necropsy the protuberances were observed to be a white, firm tissue, surrounding the bones.

Case III - X-11842 - (AFIP 1727491).
Tissue from a common loon (Gavia immer). Two days prior to death the bird was weak, unable to fly, and had blood-tinged milky white droppings.

Case IV - 4898-K - (AFIP 1672455).
Mated 2 springs ago, laid 7 eggs. This spring mated but no eggs produced. Several lumps felt in the posterior abdomen adjacent to cloaca. Thought to be impacted eggs, verified on radiologic examination. Removed forcibly by speezing through cloaca. Snake died 12 hours later.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Results
AFIP Wednesday Slide Conference - No. 5
15 October 1980

Case I - 176750 (AFIP 1681107).

History: This Ross Leg Touraco (band #459) was found dead in the exhibit. A second bird was found down on Aug 22, 1978, removed from the exhibit, and seemed to recover after being isolated from the group.

Contributor Diagnosis & Comments:

Gross Lesions: This bird was dead on arrival and in fair condition. External lesions - The right leg had broken bones and skin over the leg was slightly hemorrhagic and discolored (purplish-black). Internal lesions - The liver was somewhat enlarged with multiple foci that varied from 0.5 to 1 mm in diameter which were yellowish white in color. The spleen was markedly enlarged, being 3 to 5 times normal size, and also contained similar whitish foci to those described in the liver. Many nodules were seen along the intestine which were whitish in color and quite hard. There were some areas of caseous necrosis present in the mouth.

Laboratory Findings: Microbiologic Examination: The lung, liver, and kidney contained heavy growth of beta-hemolytic Streptococcus sp. and Klebsiella pneumoniae and a moderate growth of E. coli. The beta-hemolytic Streptococcus sp. was sensitive to ampicillin, cephalothin, chloramphenicol, gentamicin, lincomycin, neomycin, nitrofurantoin, penicillin, polymyxin B, and tetracycline and resistant to sulfadimethoxine and tribrissen. The Klebsiella was sensitvie to cephalothin, chloramphenicol, gentamycin, neomycin, nitrofurantoin, polymyxin B, tribrissen, and tetracycline and resistant to ampicillin, lincomycin, penicillin, and sulfadimethoxine.

Histopathologic Examination: The liver had multifocal areas of caseation necrosis that were frequently surrounded by large foamy macrophages with occasional multinucleated giant cells being present. This had a relatively random distribution. The spleen had similar-type lesions but with more necrosis being present. The similar-type lesions were also seen in the oral mucosa that was described grossly. Similar-type lesions were also seen in the ventriculus. On special stain (acid-fast) there were prodigious numbers of acid-fast bacilli seen throughout virtually all tissues examined. This was particularly remarkable change due to the overwhelming numbers of these organisms.

Conclusion: Avian tuberculosis. (This has to be one of the most severe cases of avian tuberculosis I have ever seen. It is a strong possibility that there will be other losses due to this disease with birds that were in the same housing.)

Attendees' Diagnoses & Comments: 1) Hepatitis, granulomatous, multifocal, severe, liver. 2) Splenitis, granulomatous, diffuse, severe, spleen. 3) Proventriculitis, granulomatous, multifocal, moderate, proventriculus. 4) Myositis, subacute to chronic, diffuse, moderate, skeletal muscle.

Etiologic Diagnosis: Gastrointestinal Tuberculosis.

Etiology: Mycobacterium sp. probably M. avium.

Comments: Attendees commented on the apparent granulomatous response observed in many tissues. Differential diagnoses given included entities such as E. coli infection (coligranulomas) and Xanthomatosis or other storage diseases. An acid fast stain provided demonstrated large numbers of acid fast bacilli (AFB) in all the tissues. The AFB primarily were within multinucleated giant cells surrounding the granulomas. Other bacterial elements noted in the submitted sections were aggregates of basophilic cocci within the center of several granulomas, but not in cells. The moderator (Dr. Montali, National Zoological Park) pointed out that this was an atypical presentation of tuberculosis in avian species in that the cellular response was primarily histiocytic and typical tubercles were not present. Avian tubercles characteristically consist of caseous centers surrounded by spindle-shaped epithelioid cells. Defenses of the host, serotype of the organism, and adaptibility of the organism play important roles in the disease. The necrotic centers within the granulomas were considered to be degenerating and necrotic histiocytes. The absence of cavitation and lack of abscess formation in birds is attributed to the absence of proteolytic enzymes in the avian heterophil. Most cases of avian tuberculosis are caused by M. avium intercellulariae serotypes 1-3. Serotypes 4-20 usually are not involved. Tuberculosis due to M. avium occasionally occurs in immunosuppressed people and has been observed in a number of captive marsupials at the National Zoo. A large number of these marsupials had caseous lesions which also are common in avian T.B.. In addition to avian strains, parrots are susceptible to M. tuberculosis (hominis) and M. bovis. A few participants had observed lesions in chickens that grossly and microscopically resembled tuberculosis but yielded Corynebacterium equi upon culture. Impression smears for acid fast stains should be prepared at necropsy from all granulomatous lesions of birds over 6 months of age.

Contributor: Animal Health Diagnostic Laboratory, College of Veterinary Medicine, Michigan State University, Lansing, MI, 48909.

Reference:

Montali, R. J., et al.: Tuberculosis in captive exotic birds. JAVMA 169 (9): 920-927, 1976.

Case II - 77PN1623D - (AFIP 1667702).

History: Tissues from a black iguana, female, about three years old, presented to necropsy because multiple protuberances in most long bones of the body. On necropsy the protuberances were observed to be a white, firm tissue, surrounding the bones.

Contributor Diagnosis & Comments: Multiple Chondromatosis.

Comments: The decalcified cross sections of this skeletal bone show marked periosteal cartilagenous proliferation with some ossification. The cortical bone shows no evidence of involvement with any pathological process and seems normally calcified. The presence of this change in the majority of the bones would be consistent with a nutritional deficiency with this particular reaction in this species or better, a virally induced reaction similar to that of osteopetrosis in chickens. No evidence of either was present in this case.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Osteochondromatous hyperplasia, diffuse, severe, long bone, iguana.

Etiologic Diagnosis: Nutritional osteodystrophy.

Etiology: Calcium-phosphorus imbalance.

Comments: Histologic features of the lesion which were discussed included the lack of mineralization of the osteoid, the chondroid hyperplasia, and the scattered endochondral ossification. The differential diagnosis given was osteochondroma. The lesions present paralleled a similar condition that has been reported in green iguanas (1). The lesions in this species have been attributed to calcium-phosphorus imbalance in conjunction with stress. The disease has been seen in active animals especially. The lesions described were fibrocartilagenous proliferation, osteodystrophy, parathyroid hyperplasia, and hypocalcemic tetany. The condition appears to be peculiar to lizzards. Dietary manipulation with low calcium or high phosphorus diets or combinations there of, result in the production of this disease in green iguanas. In natural conditions fibrous osteodystrophies of undetermined cause have been observed in hatching iguanas, mature iguanas, and Geckos.

Contributor: Department of Veterinary Pathology, School of Veterinary Medicine, Louisiana State University, Baton Rouge, LA 70803.

Reference:

Anderson, M. P. and Capen, C. C.: Nutritional osteodystrophy in captive green iguanas (Iguana iguana). Virchows Arch B Cell Patn 21: 229-247, 1976.

Case III - X-11842 - (AFIP 1727491).

History: Tissue from a common loon (Gavia immer). Two days prior to death the bird was weak, unable to fly, and had blood-tinged milky white droppings.

Laboratory Results:
Aerobic bacterial cultures of liver and kidney were negative. Direct FA for Newcastle Disease was negative. Lead analysis of kidney also was negative.

Contributor Diagnosis & Comments: Renal Coccidiosis due to Eimeria gaviae.
Comment: This is the first reported case of renal coccidiosis in a member of the Order Gaviformes. Morphologically, the coccidian was considered to be an Eimeria sp. distinct from Eimeria truncata. It was named Eimeria gaviae, n. sp.

Attendees' Diagnoses & Comments:
Morphologic Diagnosis: Pyelonephritis, subacute, multifocal, mild, kidney, Common Loon, Gavia immer.
Etiologic Diagnosis: Renal coccidiosis.
Etiology: Eimeria gaviae.
Comments: Discussion of the lesions included the mild subacute inflammatory reaction and the presence of coccidian parasites within the renal pelvis and distal medulla. Some participants commented on the apparent necrosis of the epithelium lining the collecting tubules and the renal pelvis. Various stages of the parasite were present in large numbers, but there was a lack of host response to their presence. This stimulated discussion as to the pathogenecity of the organism, and it was felt these were generally nonpathogenic and considered an incidental finding. A few attendees commented on a mild hyperplasia of the ureter epithelium. Eimeria truncata is the most commonly found occidian parasite in birds and is considered innocuous in most species.

Contributor: Eli Lilly & Company, Greenfield Laboratories, P. O. Box 708, Greenfield, IN 46140.

Reference:
Montgomery, R. D., Novilla, M. N. and Shillinger, R. B.: Renal coccidiosis caused by Eimeria gaviae n. sp. in a Common Loon (Gavia immer). Avian Diseases 22: 809-814, 1978.

Case IV - 4898-K - (AFIP 1672455).

History: Mated 2 springs ago, laid 7 eggs. This spring mated but no eggs produced. Several lumps felt in the posterior abdomen adjacent to cloaca. Thougnt to be impacted eggs, verified on radiologic examination. Removed forcibly by speezing through cloaca. Snake died 12 hours later.

Contributor Diagnosis & Comments: Kidney, snake, gouty tophi, chronic inflammation, and glomerulosclerosis.

Comment: The lesions are similar to those seen in snakes given gentacin therapy, but are more extensive than in those cases.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Nephritis, necrotizing, chronic, disseminated, kidney, snake.

Etiologic Diagnosis: Renal gouty tophi.

Etiology: Dehydration, gentamycin therapy, or high protein diet.

Comments: The morphologic diagnosis given above was reached after some discussion. Other diagnoses given were tubular nephrosis, and glomerulonephritis. Changes in the tissue which were commented on included the segmental nature of the lesion, a moderate interstitital fibrosis, the presence of multinucleated giant cells surrounding the urate deposits, and the abundance of birefringent urate crystals present. The segmental nature of the lesion and the apparently unaffected peripheral nephrons were considered unique in this case. The usual presentation in gentamycin and dehydration nephrosis is a diffuse lesion wherein interstitial edema precedes tubular necrosis and urate deposition in tubules which in turn result in interstitial fibrosis and chronic inflammation. A few participants speculated on a local ischemia due to a vascular injury resulting in the lesion present. Sequential biopsies also have produced similar lesions in kidneys of snakes undergoing experimental procedures according to the moderator. Occasional sclerotic glomeruli were noted in the peripheral regions of the tissue but the majority appeared unaffected and were considered normal. The enlarged amorphous appearance of some glomeruli is considered a normal finding in reptilian kidneys, and without special stains on thin sections, the diagnosis of glomerulonephritis would be unsubstantiated. Gouty tophi occur in a number of different organs in addition to the kidneys of reptiles with renal disease. Tophi are observed in the lung, liver, heart, pericardium, and other visceral organs; this reflects the hyperuricemia which results from the inability to excrete the breakdown products of protein catabolism.

Contributor: Laboratory of Pathology, School of Veterinary Medicine, University of Pennsylvania, Philadelphia, PA 19104.

References:

Wallach, J. R. and Hoessle, D.: Visceral gout in captive reptiles. JAVMA 151: 897-899, 1967.

Jacobsen, E. R.: Gentamyacin related visceral gout in two bold snakes. WM/SAC 71: 361-363, 1967.

Montali, R. J., Bush, M. and Smeller, J. M.: The pathology of nephrotoxicity of gentamycin in snakes. Vet. Path. 16: 108-115, 1979.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Histories
AFIP Wednesday Slide Conference - No. 6
29 October 1980

Case I - 1959-76 (AFIP 1618040).
History: A 4-year-old domestic shorthair cat had dilated pupils for 4 weeks and respiratory problems 2 weeks prior to examination. The cat was anorexic, debilitated and had diarrhea. Ocular exam revealed discrete areas of bilateral retinitis with mild papilledema.

Case II - 79-8074 (AFIP 1712745).
History: A recently bred 6-month-old gilt exhibited hematuria for 3 or 4 days before death. Necropsy findings included pyelonephritis, hemorrhagic ureteritis and mucohemorrhagic cystitis.

Case III - 7153-79D or 7153-79E (AFIP 1712701.
History: A 5-year-old, spayed female Irish Setter was presented to the veterinarian because of a hard 1.5 cm swelling of the gum at the base of the right upper canine tooth.

Radiographic Findings:
Expansile lytic lesion of the maxilla at the base of the right canine tooth. The lytic lesion is surrounded by a uniform thin rim of sclerotic bone.

Case IV - Ms 78-430 (AFIP 1667745).
History: Numerous DBA/1 mice of both sexes appeared ill 14 days after arrival from a commercial breeder. Morbidity was over 50% with moderate mortality. Clinically, the affected mice exhibited extreme lassitude, ruffled hair, and dyspnea.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Patnology
Department of Veterinary Pathology

Results
AFIP Wednesday Slide Conference - No. 6
29 October 1980

Case I - 1959-76 (AFIP 1618040).

History: A 4-year-old domestic shorthair cat had dilated pupils for 4 weeks and respiratory problems 2 weeks prior to examination. The cat was anorexic, debilitated and had diarrhea. Ocular exam revealed discrete areas of bilateral retinitis with mild papilledema.

Laboratory Results: Toxoplasma titer-negative.

Contributor's Diagnosis & Comments: Mycotic meningoencephalitis; Cryptococcus sp.

Comments: Cryptococcus organisms were found in lesions in the eye.

Attendees' Diagnoses & Comments: Cryptococcal meningoencephalitis, multifocal, moderate to severe, brain, domestic shorthair, feline.

Comments: The above diagnosis was reached after some discussion of the lesions and host response to the organisms. An etiomorphologic diagnosis was preferred on the basis of the large numbers of organisms involved, the destruction of considerable neuropil, and the complete lack to minimal inflammatory response of this animal. Various morphologic diagnoses were submitted which included descriptors such as: nonsuppurative, chronic, granulomatous, subacute, and fungal meningoencephalitis. Comments on the lesions included distribution, and tissue destruction. The distribution was felt to be closely associated with the meningeal extensions into the neurophil via the pial lined blood vessels. A number of the lesions in the brain substance contained central blood vessels with organisms in the distended Virchow-Robbins space, or in areas formerly occupied by brain tissue. Discussion of other lesions commonly associated with this organism in various animals included sinal, ocular, pulmonary, mammary and cutaneous lesions. Extension of cryptococcal infection through the cribiform plate to the brain was discussed. Invasion by the organisms via the meninges covering the olfactory nerves seems more plausable than direct extension. Extension to the eyes from the CNS also was considered meningeal in origin. Hematogenous spread was considered unlikely in this case. Participants generally agreed with the proposal that this animal was not responding to the organism due either to immunosuppression, lack of antigenic stimulus of the organism or both. Concurrent infection with feline leukemia virus and its clinical manifestation of generalized immunosuppression could account for this. Also the antigenic determinants of the cryptococcal capsule play a role in the host response. Lesions also occur in immunologically privileged sites with little host response such as meninges, iris, and joints. Lesions in other animals discussed were cryptococcal mastitis in cows, pulmonary granulomas in donkeys and horses, and CNS and pulmonary lesions in dogs. Clinical differential diagnoses for ocular cryptococcal infection in cats include: FeLV, toxoplasmosis, and FIP. Diagnostic aids in detection of the CNS form should include examination of CSF preparations stained with India ink. Staining characteristics of the organism in tissue sections in most cases are as follows: The cell wall and capsule stain deeply with mucicarmine and less intensely with PAS. The capsule also is Alcian blue positive. GMS also stains the cell wall.

Contributor: Veterinary Diagnostic Laboratory, Oregon State University, Corvallis, OR 97331.

References:
1. Fisher, C. A.: Intraocular cryptococcosis in two cats. JAVMA 158: 191-198, 1971.
2. Gelatt, K. N. et al.: Ocular and systemic cryptococcosis in a dog. JAVMA 162: 370-375, 1973.
3. Perfect, J. R. et al.: Chronic cryptococcal meningitis: A new experimental model in rabbits. Am. J. Pathol. 101: 177-194, 1980.

Case II - 79-8074 (AFIP 1712745).

History: A recently bred 6-month-old gilt exhibited hematuria for 3 or 4 days before death. Necropsy findings included pyelonephritis, hemorrhagic ureteritis and mucohemorrhagic cystitis.

Laboratory Results:
Corynebacterium suis was cultured from the urinary bladder and ureter.

Contributor's Diagnosis & Comments: Mucinous cystitis associated with Corynebacterium suis infection.

Comments: The anaerobic bacterium Corynebacterium suis has been associated with cystitis and pyelonephritis in sows and is suspected of being transmitted by the boar. A high percentage of healthy adult males carry the organism in the prepuce. The lesions in the bladder may not be limited to infection with C. suis but metaplasia of the transitional epithelium to a mucus-secreting, glandular type seems to be common with the infection.

Attendees' Diagnoses & Comments:
Morphologic Diagnosis: Cystitis, mucinous, and hyperplastic, subacute to chronic, urinary bladder, pig.
Etiologic Diagnosis: Bacterial cystitis.
Etiology: Corynebacterium suis.
Comments: Other diagnoses considered were polypoid and hyperplastic cystitis. Other bacterial agents considered differentially were C. renale, Stapn. sp., Proteus, and E. coli. Discussion of the lesions centered on the metaplastic response of the urothelium to mucus secreting cells rather than the normal transitional cell epithelium. This same mucosal change has been reported in other infections in swine and has been associated with exudative epidermititis, Coliform enteritis, and hog cholera (1). A similar metaplastic change is reported to occur in guinea pigs, bovines and humans with urinary tract infections. Infection in sows with Corynebacterium suis is most commonly encountered in pregnant and postparturent animals in which death may occur in 2-4 days. Gross lesions include pyelonephritis, dilation of the ureters with exudate in the lumina, and suppurative and hemorrhagic cystitis, in addition to the mucinous cystitis. Discussion also included the mild inflammatory infiltrate in the lamina propria and edema of the bladder wall. Characteristics of the organism discussed were Gram positive diptheroids, anaerobic, and restricted media requirements.

Contributor: Veterinary Diagnostic Laboratory, University of Georgia, P. O. Box 1389, Tifton, GA 31794.

References:
1. Brobst, D. F., Cottrell, R. and Delez, A.: Mucinous degeneration of the epithelium of the urinary tract of swine. Vet. Path. 8: 485-489, 1971.
2. Soltys, M. A. and Spratling, F. R.: Infectious cystitis and pyelonephritis of pigs: A preliminary communication. Vet. Rec. 69: 500-504, 1957.
3. Soltys, M. A.: Corynebacterium suis associated with a specific cystitis and pyelonephritis in pigs. J. Path. Bact. 81: 441-446, 1961.

Case III - 7153-79D or 7153-79E (AFIP 1712701.

History: A 5-year-old, spayed female Irish Setter was presented to the veterinarian because of a hard 1.5 cm swelling of the gum at the base of the right upper canine tooth.

Radiographic Findings:
Expansile lytic lesion of the maxilla at the base of the right canine tooth. The lytic lesion is surrounded by a uniform thin rim of sclerotic bone.

Contributor's Diagnosis & Comments: Epidermal inclusion cyst of bone.
Comments: The lesion was curetted and 2-3 cc of keratinized debris was removed. The tissue removed from the cyst was submitted for histopath examination. At the six month follow-up the lesion had not recurred.

Attendees' Diagnoses and Comments:
Morphologic Diagnosis: 1) Keratinizing squamous epithelial cyst, alveolar bone, maxilla, dog. 2) Microfilariasis, blood.
Comments: There was considerable variation in the preconference morphologic diagnoses listed by the participants. The diagnosis given above was reached only after considerable discussion and consultation. Attendees considered other primary and differential entities such as dentigerous cyst, keratinizing and calcifying odontogenic tumor, ameloblastic variant, and gingival hyperplasia. Possible origin of the tissue was discussed but there was no concensus of opinion. These included embryological rests of odontogenic epithelium, downgrowth of gingival epithelium, and sequelae of traumatic insult with implantation of surface epithelium. The bone lesions described and viewed on the submitted radiographs were discussed and felt to be secondary to the expansion of the cyst with keratin. Bone resorption can occur with any expansile lesion over bone without being involved in the lesion directly. This lesion was considered to have some but not all the characteristics of the human keratinizing and calcifying odontogenic cyst, as large quantities of keratin are present and areas of mineral deposition are present. The tumor lacks the myxomatous tissue surrounding the tumor, and the conspicuous ghost cells found in man. At the end of the discussion, it was felt that the lesion probably was of odontogenic origin but could not be definitively determined. Microfilaria were described in the tissue sections by several attendees.

Contributor: Department of Veterinary Pathology, College of Veterinary Medicine, University of Missouri, Columbia, MO 65211.

References:
1. Dahlin, D. C.: Bone Tumors: General Aspects and Data on 6,221 Cases. 3rd Edition, Charles Thomas Publishing, Springfield, IL, 1978, p. 424.
2. Shafer, W. G.: Textbook of Oral Pathology, 4th Edition, W. B. Saunders Co., Philadelphia, 1974, p. 249.

Case IV - Ms 78-430 (AFIP 1667745).

History: Numerous DBA/1 mice of both sexes appeared ill 14 days after arrival from a commercial breeder. Morbidity was over 50% with moderate mortality. Clinically, the affected mice exhibited extreme lassitude, ruffled hair, and dyspnea.

Laboratory Results: 1:630 titre to Sendai virus.

Contributor's Diagnosis & Comments: Severe, subacute, disseminated hyperplastic bronchiolitis with squamous metaplasia and peribronchiolar parenchymal consolidation.

Etiology: Sendai virus.

Comment: The columnar epithelium of the bronchioles is markedly hyperplastic with early focal areas of squamous metaplasia. The lumina of the bronchioles contain a moderate to large amount of cellular detritus, and the peribronchiolar parenchyma has a variable degree of consolidation with a mixed but predominately mononuclear infiltrate.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Pneumonia, interstitial, with bronchiolar hyperplasia and metaplasia and bronchiolitis, subacute, disseminated, moderate to severe, lung, mouse.

Etiologic Diagnosis: Viral pneumonia.

Etiology: Sendai virus (Myxovirus parainfluenza type I)

Comments: The preconference morphologic diagnosis listed by most conference participants was bronchopneumonia with modifiers such as hyperplastic and metaplastic, chronic, and subacute. However, based on the viral etiology and the diffuse interstitial changes, this diagnosis was amended. Pneumonias caused by bacterial agents and those secondary to aspiration are traditionally classified as bronchopneumonias, whereas viral, chlamydial, and irritant inhalation are classified as interstitial pneumonias. The lesions in this case were considered quite characteristic of mouse Sendai virus pneumonia. There was considerable discussion of the hyperplastic and metaplastic changes in the bronchioles, coupled with the mild mixed inflammatory response, with particular reference to squamous metaplasia. The primary morphologic change in Sendai virus infection involves necrosis followed by proliferation of the bronchiolar epithelium which may progress to complete occlusion of the lumen. Subsequently this proliferative change

extends into the peribronchiolar alveolar spaces. Squamous metaplasia follows the proliferative lesions in both areas. The cell of origin of the hyperplastic epithelium is believed to be the clara cell. This cell is also the precursor for the type II pneumocyte. Type II pneumocytes in turn replace desquamated type I pneumocytes. A number of attendees reported seeing intracytoplasmic eosinophilic inclusion-like bodies within the hyperplastic epithelium along with a few mitotic figures involving similar cells. Intracytoplasmic inclusion bodies have been reported in conventional mouse strains; intranuclear inclusion bodies have been reported in nude mice; and both intranuclear and intracytoplasmic viral particles have been observed in conventional and nude mouse strains infected with Sendai virus. One attendee commented on syncychial cell formation in their section, however, this is not the usual finding in this condition. Syncytial cell formation, however, is known to occur in tissue cultures infected with Sendai virus. Mice, rats, hamsters and guinea pigs are susceptable hosts for Sendai virus infection. Morbidity and mortality are highest in mice, especially young animals. Prevalence of mouse colonies infected ranges from 40-50% in the U. S. The complement fixation test is considered the test of choice for detection of infected animals. The CF test is more sensitive and persists longer than the routinely employed hemagglutination inhibition test. Differentiation of the lesions of Sendai virus and pulmonary neoplasia is based on the distribution of the lesion rather than cellular morphology which can be confusing.

Contributor: Comparative Pathology Section, Veterinary Resources Branch, NIH, Bethesda, MD 20014.

References:

1. Appell, L. H., Kovatch, R. M. et al.: Pathogenesis of Sendai virus infection in mice. Am. J. Vet. Res. 32: 1835-1841, 1971.

2. Anderson, M. S. et al.: Cell-mediated immunity to Sendai virus infection in mice. Infect. Immunol. 15: 239-244, 1977.

3. Parker, J. C. et al.: Susceptibility of inbred and outbred mouse strains to Sendai virus and prevalence of infection in laboratory rodents. Infect. Immunol. 19: 123-130, 1978.

4. Ward, J. M.: Naturally occurring Sendai virus disease of mice. Lab. Anim. Sci. 24: 938-942, 1974.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Histories
AFIP Wednesday Slide Conference - No. 7
5 November 1980

Case I - 968-79 (AFIP 1716366).
History: A 4-month-old Australian shepherd dog reportedly began salivating and went into convulsions. At necropsy, the lungs were somewhat congested and contained multiple greyish foci, 2 to 3 mm in diameter. There were no other gross lesions.

Case II - 82446 (AFIP 1714913).
History: This tissue is from an adult male horse which had a history of neurologic signs for 5 months, which included circling, ataxia, and incoordination. The clinical signs had been getting progressively more severe, and the horse was euthanatized.

A mass was found in the brain, appearing to be midline in origin, with invasion of both lateral ventricles. The mass extended from the occipital lobes forward to about 4 cm short of the anterior aspect of the cerebrum. The cerebellum was not involved. There was some necrosis in the tumor.

Case III - 80-1 (AFIP 1757055).
History: Cutaneous mass located below the ear of a flying squirrel.

Case IV - 78-4656 (AFIP 1667021).
History: Formol fixed tissues were received from a cross-bred 4-year-old sow which had been sick for 48 days. The main symptoms were anorexia, fever and progressive weakness. She aborted at 89 days of pregnancy.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Results
AFIP Wednesday Slide Conference - No. 7
5 November 1980

Case I - 968-79 (AFIP 1716366).

History: A 4-month-old Australian shepherd dog reportedly began salivating and went into convulsions. At necropsy, the lungs were somewhat congested and contained multiple greyish foci, 2 to 3 mm in diameter. There were no other gross lesions.

Contributor's Diagnosis & Comments: Canine distemper and pulmonary toxoplasmosis.

Comments: Numerous toxoplasma organisms are present in foci throughout the lung. In the stomach there are many intranuclear and intracytoplasmic eosinophilic inclusion bodies in the gastric glands.

Attendees' Diagnoses & Comments: Morphologic diagnoses: 1) Pneumonia, necrotizing, protozoal, multifocal, moderate to severe, lung, canine. 2) Pneumonia, interstitial, diffuse, mild, lung. 3) Inclusion bodies, intranuclear and intracytoplasmic, gastric glands, stomach.

Etiologic Diagnosis: Toxoplasmosis and Canine Distemper.

Etiology: Toxoplasma sp. and Canine Distemper Virus (Morbillivirus).

Comments: Tissue sections containing the Toxoplasma lesions were not present on all the slides submitted, therefore the necrotizing pneumonia was not diagnosed by all. The diffuse interstitial pneumonia was observed by all the participants and several noted intranuclear eosinophilic inclusion bodies in the epithelial cells of the bronchioles. All the participants observed the intranuclear and intracytoplasmic eosinophilic inclusions in the gastric glands. A few participants mentioned degeneration and necrosis of gastric gland cells and preferred a diagnosis of a mild acute gastritis. The interstitial pneumonia and inclusions in the stomach were attributed to the canine distemper virus. Those that had sections containing the Toxoplasma lesions commented on the large number of organisms both within cells and free in alveolar spaces. The necrotic lesions correspond to the multiple greyish foci observed grossly. A brief discussion followed concerning the simultaneous infection of these agents. These conditions frequently are observed together in young animals. Infection by the virus depresses the the immunological status of the animal and renders it susceptable to infection and proliferation of toxoplasma organisms. CNS signs observed clinically in these animals could be attributed to either infection. Examination of the brain microscopically could reveal lesions of one or both infections.

Contributor: Veterinary Diagnostic Laboratory, School of Veterinary Medicine, Oregon State University, Corvallis, OR 97331.

References:

1. Benirschke, K., et al.: Pathology of Laboratory Animals. Vol. I. Springer-Verlag, New York, 1978, pp 95 and 370-371.

2. Capen, C. C. and Cole, C. B.: Pulmonary lesions in dogs with experimental and naturally occurring toxoplasmosis. Path. Vet. 3: 40-63, 1966.

3. McCullough, B. et al.: Experimental canine distemper virus induced lymphoid depletion. Am. J. Pathol. 74: 155, 1974.

Case II - 82446 (AFIP 1714913).

History: This tissue is from an adult male horse which had a history of neurologic signs for 5 months, which included circling, ataxia, and incoordination. The clinical signs had been getting progressively more severe, and the horse was euthanatized.

A mass was found in the brain, appearing to be midline in origin, with invasion of both lateral ventricles. The mass extended from the occipital lobes forward to about 4 cm short of the anterior aspect of the cerebrum. The cerebellum was not involved. There was some necrosis in the tumor.

Contributor's Diagnosis & Comments: Pineoblastoma.

Comments: The tumor was found to be densely cellular and was composed of small round to slightly elongated cells which had hyperchromic nuclei and very sparce cytoplasmic material. There was no apparent differentiation. The tumor was very vascular. There was minimal mitotic activity. Tumor cells had seeded out the ventricular walls in several foci. The similarity to medulloblastoma is apparent. The origin of the tumor was consistent with the diagnosis of pineoblastoma. Some favor the term primative neuroectodermal tumor for this group of neoplasms. Luginbuhl found 30.5% of a series of 400 tumors of the nervous system in animals to be neuroectodermal in origin, but none appeared to be pineoblastomas. While no reference has been found of pineoblastoma in animals, pinealoma has been reported in a horse, a rat, and silver fox.

Attendees' Disgnoses & Comments:

Morphologic Diagnosis: Primative neuroectodermal tumor, brain, horse.

Comments: The above diagnosis was reached after some discussion of the lesion. Other diagnoses given included; astrocytoma, ependymoma, glioblastoma, ogliodendroma, and undifferentiated mixed tumor of nervous tissue. Numerous differential diagnoses also were included such as medulloblastoma and mixed glioma in addition to those listed above. Cellular constituents and morphology commented on were the predominant small hyperchromic round cells which resembled lymphocytes, larger less abundant pale round cells associated with trabecular structures, and small hyperchromic spindle-shaped cells with carrot-shaped nuclei arranged in bundles and whorls. This latter cell type was not present in all sections. The location of the lesion and destruction of the neuropil was commented on and felt to be attributable to several of the listed neoplasms. Special stains including Bielschowsky's stain would be helpful in demonstrating argyrophilic cellular processes associated with pineal gland tumors. Both pinealomas and pineoblastomas have been described as having malignant behavior with invasion of adjacent structures.

Contributor: Comparative Medical & Veterinary Services, Los Angeles County Department of Health Services, 12824 Erickson Ave., Downey, CA 90242.

References:

1. Russell, D. S. & Rubinstein, L. J.: Pathology of Tumors of the Nervous System. 4th Ed., Williams & Wilkins Co., Baltimore, 1977, pp 283-298.

2. Luginbuhl, H.: Comparative aspects of tumors of the nervous system. In Epizootiology of Cancer in Animals. Ann. N. Y. Acad. Sci. 108: 702-721, 1963.

Case III - 80-1 (AFIP 1757055).

History: Cutaneous mass located below the ear of a flying squirrel.

Contributor's Diagnosis: *Psorergates* spp. infestation.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Dermatitis, proliferative and pyogranulomatous, focally extensive, skin, flying squirrel.

Etiologic Diagnosis: Acariasis.

Etiology: *Psorergates* spp.

Comments: Descriptive terms for the dermatitis turned in before the conference by various attendees included chronic-active, granulomatous, suppurative, chronic-suppurative, and papillary-cystic. The above diagnosis was agreed upon after some discussion. Differential etiologic agents listed were *Sarcoptes* spp., *Notoedres* spp. and *Cheyletiella* spp. Characteristics of the lesion commented on were the mixed inflammatory response to the presence of the mites, the close association of the parasites to hair follicles, and the tunnels formed by the mites in the epidermis. Photographs of the gross specimen from this animal were viewed along with photomicrographs of the parasites. Grossly the specimen appears as a honeycomb or multiple microcystic mass within the skin. Features of the parasites included a round body with 4 pairs of legs each with 2 terminal hooks and a medially directed spine at the base of each leg. Histologic features include a chitinous body wall, appendages or sites of appendage attachment to the body and striated skeletal muscle within the body cavity. The small basophilic spherical structures noted in many of the parasites were interpreted to be elements of the reproductive organs of the mites.

Contributor: Dr. George A. Parker, Veterinary Pathologist, 11101 Streamview Court, Great Falls, VA 22066.

Reference:

1. Flynn, R. J.: Parasites of Laboratory Animals. University of Iowa Press, 1973, pp 460 and 478.

2. Soulsby, E.J.L.: Hilminths, Arthropods, and Protozoa of Domestic Animals. (Monnig) 6th Ed., Williams & Wilkins Co., Baltimore, 1968, pp 499-51.

Case IV - 78-4656 (AFIP 1667021).

History: Formol fixed tissues were received from a cross-bred 4-year-old sow which had been sick for 48 days. The main symptoms were anorexia, fever and progressive weakness. She aborted at 89 days of pregnancy.

Laboratory Results: On postmortem there was fibrinous pleuritis and pericarditis and peritoneal effusion. Liver was enlarged, very firm and yellowish on section. Spleen was enlarged, thick and brownish. Kidneys and mesenteric lymph nodes were larger than normal and white spots were scattered throughout their parenchyma.

Contributor's Diagnosis & Comments: Eosinophilic myeloid leukemia.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Myeloid leukemia, eosinophilic, liver, kidney, and spleen, pig.

Comments: There was general agreement with the contributor's diagnosis. Modifications of the same diagnosis listed by the participants were eosinophilic myeloid sarcoma, granulocytic sarcoma, and well differentiated eosinophilic myeloproliferative disease. All the attendees noted the large eosinophilic granules within the cytoplasm of the neoplastic cells and occasional indented and lobulated nuclei. The majority of the cells, however, possessed round nonlobulated nuclei. The presence of these neoplastic cells within blood vessels reinforced the leukemic nature of this condition in this animal. Grossly these lesions have been described as green discolored areas affecting various tissues in cattle and pigs. Microscopically these lesions would have to be differentiated from eosinophilic lymphadenitis and eosinophilic myositis when present in muscle. Eosinophils present in the other conditions would appear as mature well lobulated cells compared to these immature appearing neoplastic cells.

Contributor: Animal Pathology Laboratory, Quebec Department of Agriculture, St. Hyacinthe, Quebec, Canada J 25 7C6.

References:

1. Moulton, J. E. and Dungworth, D. E.: Tumors of lymphoid and hematopoietic tissues. In Tumors in Domestic Animals, 2nd Edition, J. E. Moulton, Editor, University of California Press, 1978, pp 150-204.

2. Migaki, G.: Hematopoietic Neoplasms of Slaughter Animals. National Cancer Institute Monograph 32: 121-139, 1969.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Histories
AFIP Wednesday Slide Conference - No. 8
12 November 1980

Case I - SWRI Rat Case II (AFIP 1718321).
History: Male, stock, Charles River, approximately 10 week old rat presented with a large (6 cm. diameter) mass in the right ventral side of neck.

Case II - D78-9390 (AFIP 1668449).
History: This was a 3-year-old female cross-bred POA admitted to the clinic with a history of an acute onset of anorexia, depression and head pressing. The animal appeared severely dehydrated. Death occurred within 24 hours of admission. The owners reported that another mare died recently with CNS symptoms.

Case III - 26903-N (AFIP 1757436).
History: Progressive dyspnea in a dog began one month after it had been hit by a car. Radiographs revealed diffuse interstitial and alveolar pneumonia.

Case IV - NA8-119 (AFIP 1668789).
History: An 8-year-old, DSH spayed female cat with a history of posterior ataxia for 7 weeks was presented for examination and treatment. Euthanasia was performed 2 days after posterior paralysis developed.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Results
AFIP Wednesday Slide Conference - No. 8
12 November 1980

Case I - SWRI Rat Case II (AFIP 1718321).

History: Male, stock, Charles River, approximately 10 week old rat presented with a large (6 cm. diameter) mass in the right ventral side of neck.

Contributor's Diagnosis & Comments: Carotid body tumor (chemodectoma; nonchromaffin paraganglioma).

Comment: The tumor consists of round to oval epithelioid cells with indistinct, eosinophilic cytoplasm. Cells are arranged in cords, nests, and clusters. In many areas fibrous trabeculae divide the tumor into an alveolar arrangement. The tumor is highly vascular, and a peritheliomatous pattern is evident. Mitotic figures are rare. Many areas of necrosis are present.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Chemodectoma, malignant, ventral neck region, rat.

Comments: Other diagnoses submitted before the conference included: Primative neuroectodermal neoplasm, undifferentiated tumor of primative cell origin, poorly differentiated carcinoma, and reticulum cell sarcoma. Comments on the morphology of the tumor included the prominent endocrine pattern, the small round to spindle-shaped cells with scant cytoplasm, the variable mitotic rate, and numerous foci of necrosis. The tumor was considered to be malignant, based on the high mitotic rate observed in some areas of the tumor and invasion of the capsule with extension into adjacent tissues present in some sections. This tumor is considered quite rare in the rat, as only one has been reported previously (1). These tumors are more often encountered in the canine, where the majority of them are of aortic body origin. Aortic body tumors in the dog are 5 to 8 times more frequent than are carotid body tumors. Histologically the two are morphologically similar. In man carotid body tumors are encountered more frequently than aortic body tumors. The functions of the carotid body and aortic body were reviewed. These chemoreceptor organs are sensitive to changes in blood carbon dioxide, pH, and oxygen tension, and aid in the regulation of respiration and circulation. These organs can initiate an increase in the depth, rate and minute volume of respiration by way of parasympathetic innervation and increase heart rate and blood pressure by way of sympathetic innervation. These tumors in dogs are considered to be nonfunctional. The exact cell of origin has not been precisely determined although it is thought to arise from perivascular mesodermal cells that are invaded by cells of neuroectodermal origin (2). This tissue also is found in the internal jugular vein along the innominate artery, below the middle ear, along the recurrent branch of the glossopharyngeal nerve, nodose ganglion, pancreas, and the ciliary ganglion in the orbit (2).

Contributor: Sterling-Winthrop Research Institute, Rensselaer, NY 12144.

References:

1. Squire, R. A. et al.: Tumors. In Pathology of Laboratory Animals, Vol. II, Benirschke, K. et al. (Eds), Springer-Verlag, New York, 1978, pp 1085-1088.

2. Capen, C. C.: Tumors of the endocrine glands. In Tumors in Domestic Animals, J. E. Moulton, (Ed), Univ. of California Press, Berkeley, 1978, pp 414-420.

3. Benirschke, K. et al.: Pathology of Laboratory Animals, Vol. II. 1978, p. 1088.

4. Ashley, D.J.B.: Evans' Histological Appearances of Tumors, Vol. I, 3rd Edition, 1978, pp 92-95.

Case II - D78-9390 (AFIP 1668449).

History: This was a 3-year-old female cross-bred POA admitted to the clinic with a history of an acute onset of anorexia, depression and head pressing. The animal appeared severely dehydrated. Death occurred within 24 hours of admission. The owners reported that another mare died recently with CNS symptoms.

Contributor's Diagnosis & Comments: Leucoencephalomalacia (moldy corn poisoning).

Comments: On gross examination two areas of malacia, each 2-3 cm. in diameter were found in the cerebral white matter. Lesions were not found in other organs. The toxin responsible for "moldy corn poisoning" in horses has not been characterized. Fusarium moniliforme has been isolated from feed toxic to horses and is thought to be responsible for production of the toxin which causes the disease.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Leucoencephalomalacia, acute, multifocal to focally extensive, severe, cortical white matter, brain, horse.

Etiologic Diagnosis: Leucoencephalitic mycotoxicosis.

Etiology: Moldy corn poisoning, Fusarium moniliforme.

Comments: An equally acceptable morphologic diagnosis listed by many participants was necrosis, and hemorrhage, acute, multifocal, severe, white matter, brain. Characteristics of the lesions commented on were the large areas of necrosis present in the tissue, the lack of a significant inflammatory response, and multifocal hemorrhages, most common around vessels. Considerable discussion ensued concerning the changes in and around vessels most notably the hypertrophy and possible hyperplasia of endothelial cells in small vessels, the perivascular hemorrhage and edema, the influx of some eosinophils, and the presence of small variably sized hyaline bodies around some of the vessels. The presence of these hyaline bodies in other toxic CNS conditions such as lead poisoning in monkeys strongly suggests a correlation with or a relationship to toxic entities. These bodies are PAS positive. They also have been observed in monkeys poisoned with arsenic. Other lesions associated with "moldy corn poisoning" in horses are centrilobular hepatic necrosis and fibrosis and bile duct proliferation which may occur independent of the CNS lesion or in conjuction with it. CNS lesions also may occur without hepatic lesions (2). Differental diagnosis of malacic lesions in the equine brain should include Equine Herpes Virus I. Lesions in this herpetic disease include malacia which is not restricted to the white matter, hemorrhage around vessels, a necrotizing arteritis and lymphocytic perivascular cuffs. Other diseases of the equine which may produce CNS signs and lesions include WEE, VEE, EEE, equine viral arteritis, and purpura hemorrhagica.

Contributor: Veterinary Diagnostic Laboratory, University of Illinois, Urbana, Illinois, 61801.

References:

1. Badiali, L. et al.: Moldy corn poisoning as the major cause of an encephalomalacia syndrome in Egyptian equidae. Am. J. Vet. Res. 29: 202-209, 1968.

2. Marasas, W.F.O. et al.: Leucoencephalomalacia: A mycotoxicosis of equidae caused by Fusarium moniliforme Sheldon. Onderstepoort J. Vet. Res. 43 (3): 113-122, 1976.

3. Wilson, B. J. et al.: Causative fungus agent of leucoencephalomalacia in equine animals. Vet. Rec. 88: 484-486, 1971.

Case III - 26903-N (AFIP 1757436).

History: Progressive dyspnea in a dog began one month after it had been hit by a car. Radiographs revealed diffuse interstitial and alveolar pneumonia.

Contributor's Diagnosis: Pulmonary nematodiasis due to Filaroides hirtni.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Pneumonia, granulomatous, diffuse, severe, lung, poodle, canine.

Etiologic Diagnosis: Verminous pneumonia.

Etiology: Filaroides sp.

Comments: Other diagnoses submitted by the participants prior to the conference were interstitial granulomatous pneumonia, interstitial verminous pneumonia, and chronic interstitial pneumonia. Differential parasites listed were Filaroides milksi and F. osleri.

The parasite was identified as a nematode. Furthermore, the worm was classified as a member of the family Metastrongylidae. Characteristics that enabled this diagnosis were: 1) small size of worms; 2) low, (poorly developed) musculature, 3) larvae in utero; and 4) the presence of a large intestine composed of few cells. The intestinal cells also contained large amounts of hemosiderin. The morphology of the worm was undistinguishable from known cases of infection with Filaroides spp. and thus the generic diagnosis was Filaroides. Specific identification was not possible on histological section but since a granulomatous reaction was surrounding both adults and larvae the most probable specific diagnosis was F. hirthi.

A similar granulomatous reaction may be observed around larvae and degenerating worms in F. milksi infection but is not commonly observed around adult F. milksi nematodes. Filaroides osleri would also have to be considered as a possible species in this case but these nematodes are usually associated with nodule formation at the bifurcation of the trachea and in the larger bronchi. Positive identification of the worm would require examination of the entire parasite including body length, characteristics of the buccal cavity, and reproductive features. The life cycle of F. hirthi has not been completely worked out but a mollusc is suspected as the intermediate host . Considerable evidence exists indicating infection also may occur by ingestion of the L_1 stage of the parasite (2). Other nematode parasites found in the lung of canine include Capillaria aerophilia, Crenosoma vulpis, and Angiostrongyles vasorum. One also must be alert for Aleurostrongyles abstrusina, a lungworm of cats. Dogs may be an unnatural host.

Contributor: Department of Pathology & Parasitology, School of Veterinary Medicine, Auburn University, AL 36849.

References:

1. August, J. R. et al.: Filaroides hirthi in a dog. Fatal hyperinfection suggestive of autoinfection. JAVMA 176: 331-334, 1980.

2. Georgi, J. R. et al.: Preliminary investigation of the life history of Filaroides hirthi. Cornell Vet. 66: 309-323, 1976.

3. Dorrington, J. E.: Studies on Filaroides osleri infestation in dogs. Onderstepoort J. Vet. Res. 35: 225-286, 1968.

Case IV - NAB-119 (AFIP 1668789).

History: An 8-year-old, DSH spayed female cat with a history of posterior ataxia for 7 weeks was presented for examination and treatment. Euthanasia was performed 2 days after posterior paralysis developed.

Laboratory Results: CSF-162mg% protein
39 WBC/mm^3 (90% lymphocytes)

Contributor's Diagnosis & Comments: Blastomycosis, granulomatous meningomyelitis.

Comments: The spinal cord and meninges from C1 through the cauda equina contained a submeningeal pyogranulomatous process with Blastomyces dermatitidis seen in the lesions of each section. There were focal submeningeal pyogranulomatous lesions in the area of the left hippocampus and occipital regions.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Meningomyelitis, pyogranulomatous, diffuse, severe, spinal cord, DSH, feline.

Etiologic Diagnosis: Mycotic meningomyelitis.

Etiology: Blastomyces dermatitidis.

Comments: There was general agreement with the contributor's diagnosis and comments. A few participants felt the spinal cord itself was not signifcantly involved in their sections and preferred the diagnosis of meningitis alone. All participants agreed that the primary lesion was in the meninges with secondary extension into the spinal cord. This extension was characterized by inflammation penetrating into the substance of the cord at the periphery and destruction of the involved neuropil. Numerous organisms were observed by all. Features characteristic of blastomyces organisms were size (5-15mu), presence of a thick cell wall, and broad base budding. Special stains which accentuated these features included GMS, PAS, and Gridley's fungus stain. The route of infection in this case was conjectured to be via meningeal vessels or CSF. Discussion included the geographic distribution of the organism and the natural soil life cycle. Pulmonary lesions are most commonly seen in animals, but skin, bone and visceral lesions also occur. Differential diagnoses include paracoccidioidomycosis, histoplasmosis, cryptococcosis, and coccidioidomycosis.

Contributor: Department of Veterinary Pathology, University of Georgia, Athens, GA 30602.

References:

1. Migaki, G. et al.: Fungal diseases. Chapt. 16, in Pathology Laboratory Animals, Vol. II, Benirschke, K. et al. (Ed.), Springer-Verlag, New York, 1978, pp 1560-1562.

2. Emmons, C. W. et al.: Blastomycosis. Chapter 21, in Medical Mycology, 3rd Edition, Lea & Febiger, Philadelphia, 1977, pp 342-364.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Histories
AFIP Wednesday Slide Conference - No. 9
19 November 1980

Case I - 2 & 3 or Skin & Lung (AFIP 1640295).

History: When this 5-year-old male Siamese cat was presented on 8/15/77 to the attending veterinarian, he was depressed, had a rectal temp. of 104°F and a wound puncture in the left thigh. The animal was treated with Penicillin IM and hospitalized. The next day, the patient's temperature dropped to 102°F. X-rays of the left rear leg revealed no broken bones or any additional lesions. On the 2nd day of hospitalization the animal's temp. dropped to 98°F. and had pale mucus membranes. Laboratory tests revealed a PCV of 10% and a WBC of 12,000. Two weeks following the first day of hospitalization the patient was still very depressed and icteric. The rectal temp. at the time was 96°F. Laboratory tests revealed a PCV 10%, a WBC of 11,000 and a blood film with polychromosia and numerous nucleated RBC. Two slides, one each skin & lung.

Case II - 080-962 (AFIP 1758398).

History: Incidental finding in a 2-year-old control female rat.

Gross: Right kidney has a large firm yellow nodule 1.5 x 1.2 cm in cortex, extending into medulla.

Case III - 79-0386 (AFIP 1712926).

History: Unvaccinated female toy poodle, 9 weeks old, was presented to a veterinary emergency service at 3:00 a.m. after a second convulsion. Treatment consisted of sedation at first and later by anesthetization. The pup weakened progressively and died at 4:20 p.m. It was presented for necropsy after 3 days of refrigeration. Gross necropsy observations were noncollapsing, congested, edematous lungs with foam in trachea.

Case IV - 17731/80-976 (AFIP 1761488).

History: A severe diarrhea was observed 3 Mar 80 in this rabbit. It died eleven days later.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Results
AFIP Wednesday Slide Conference - No. 9
19 November 1980

Case I - 2 & 3 or Skin & Lung (AFIP 1640295).

History: When this 5-year-old male Siamese cat was presented on 8/15/77 to the attending veterinarian, he was depressed, had a rectal temp. of 104°F and a wound puncture in the left thigh. The animal was treated with Penicillin IM and hospitalized. The next day, the patient's temperature dropped to 102°F. X-rays of the left rear leg revealed no broken bones or any additional lesions. On the 2nd day of hospitalization the animal's temp. dropped to 98°F. and had pale mucus membranes. Laboratory tests revealed a PCV of 10% and a WBC of 12,000. Two weeks following the first day of hospitalization the patient was still very depressed and icteric. The rectal temp. at the time was 96°F. Laboratory tests revealed a PCV 10%, a WBC of 11,000 and a blood film with polychromosia and numerous nucleated RBC. Two slides, one each skin & lung.

Laboratory Results:
2nd day: PCV 10%, WBC 12,000, rectal temp. 98°F.
3rd day: PCV 10%, WBC 11,000, polychromasia, nucleated RBC's, rectal temp. 96°F.

Contributor's Diagnosis & Comments
Morphologic Diagnosis: Pulmonary thrombosis. Acute and chronic fasciitis with vasculitis and thrombosis formation.
Etiologic Diagnosis: Bacterial thromboembolic septicemia.
Comments: It is felt that the thigh wound was the site of the initial infection which led to a septicemia resulting in a regenerative hemolytic anemia and pulmonary thrombosis.

Attendees' Diagnoses & Comments:
Morphologic Diagnoses: 1) Cellulitis, subacute, focally extensive to diffuse, moderate to severe, subcutis, skin, Siamese cat, feline. 2) Thrombosis, venular and arteriolar, multifocal to disseminated, subcuticular, dermal, and pulmonary vessels, moderate to severe, skin and lung. 3) Hyperplasia, medial, and intimal, moderate to severe, pulmonary arteries, lung. 4) Vasculitis, subacute, multifocal, mild, venules, subcutis, skin. 5) Pneumonia, interstitial and granulomatous, multifocal moderate, lung.
Etiologic Diagnosis: Unresolved.
Comments: The presence of the pneumonia and thrombosis in the lung varied from one section to the next. The etiologic diagnosis was deferred, as a general concensus could not be reached. Differential etiologic diagnoses included disseminated intravascular coagulation, bacterial septicemia, feline infectious anemia, and myeloproliferative disease. A proposed possible etiologic diagnosis for the medial hyperplasia listed by many was pulmonary nematodiasis caused by Aleurostrongyles sp. Many participants felt the skin lesion and the pulmonary thrombosis and associated lesions were related. Additionally, a large number of participants wondered whether a concurrent infection with Hemobartonellosis or erythemic myelosis was present in this case. Some attendees reported observing

suspicious granular densities on RBC's and could not rule out Hemobartonella organisms or Howell-Jolly bodies. Binucleated red blood cells and numerous immature cells were reported by a few attendees and felt to be representative of a myeloproliferative disease. Medial hyperplasia was preferred over the usual diagnosis of medial hypertrophy because of increased number of smooth muscle cells apparent in these arteries. The intimal hyperplasia was discussed and proposed to be due to direct bacterial endotoxic effects or to hypoxic change due to the thrombosis. At the close of the discussion of this case, it was suggested that additional laboratory tests and other organ tissue sections such as liver, kidney and spleen may have been helpful in determining the specific cause(s) of the lesions present.

Contributor: University of Texas Southwestern Medical School, Dallas, TX 75235.

Case II - 080-962 (AFIP 1758398).

History: Incidental finding in a 2-year-old control female rat.

Gross: Right kidney has a large firm yellow nodule 1.5 x 1.2 cm in cortex, extending into medulla.

Contributor's Diagnosis & Comments: Renal tubular carcinoma.

Comments: The considerable variability of the cellular morphology with areas that are forming tubular areas, papillary areas, sheets of cells, and clear cells or "hypernephroma" have been previously reported in the literature. No metastatic lesions were seen.

Attendees' Diagnoses & Comments: 1) Renal carcinoma. 2) Nephrosis, multifocal, mild, kidney.

Comments: There was general agreement with the contributor's diagnosis and comments. While various names are acceptable for this neoplasm including renal carcinoma, renal cell carcinoma, renal tubular carcinoma and adenocarcinoma, the first two names are used most widely. The mitotic rate was low but some of the figures were very prominent. Vascular invasion was not observed. Differentially some participants felt this neoplasm was an adenoma. Distinction between renal carcinomas and adenomas often is difficult. In the absence of metastasis or distinct invasion, there are no exact gross, histologic or ultrastructural features that consistently differentiate renal carcinomas and adenomas. Even tiny tumors less than 2mm may exhibit features of malignancy including necrosis, hemorrhage, prominent mitoses and cellular pleomorphism. These tumors do not metastasize commonly except in humans and dogs. It was pointed out that several chemical compounds have been shown to produce lesions in the kidney ranging from tubular hyperplasia to carcinoma. Lesions in the ureters and urinary bladder may accompany these tubular changes. Incidence of primary carcinomas of the kidney in rats is considered quite low. Spontaneous mixed sarcomas have been reported in the Osborne-Mendel and Brown Norway rat (1,3). A short discussion followed concerning the other lesions present in this case. The term nephrosis was preferred based on the glomerular, tubular, and interstitial changes and the presence of proteinaceous

casts in some tubules. A number of equally acceptable synonyms for this condition also were mentioned. This lesion is present in many strains of rats. The incidence, and age of occurrence vary with the strain of rat. Nephrosis often interferes with the interpretation of experimental procedures.

Contributor: Pathology Department, Hazleton Laboratories, Inc., 9200 Leesburg Turnpike, Vienna, VA.

References:
1. Turusov, V. S.: Pathology of Tumors in Laboratory Animals. Vol. I, Part 2, pp 73-75.
2. Bulletin of the World Health Organization. Vol. 53: No. 2-3, p. 238.
3. Benirschke, K. F., et al.: Pathology of Laboratory Animals. Vol. I, pp 142-145, and Vol. II, p. 1161, Springer-Verlag, New York, 1978.

Case III - 79-0386 (AFIP 1712926).

History: Unvaccinated female toy poodle, 9 weeks old, was presented to a veterinary emergency service at 3:00 a.m. after a second convulsion. Treatment consisted of sedation at first and later by anesthetization. The pup weakened progressively and died at 4:20 p.m. It was presented for necropsy after 3 days of refrigeration. Gross necropsy observations were noncollapsing, congested, edematous lungs with foam in trachea.

Contributor's Diagnosis & Comments: Calcification, metastatic, lung.

Comments: There was no mineralization of the heart or its blood vessels, but some fiber degeneration was indicated by HBFP stain reaction. Kidney contained lesions morphologically similar but less extensive than those in the lung. Kidney and lung deposits were Alizarin Red and PAS positive, iron and fat negative. EM photomicrographs demonstrated laminated structures associated with degenerating cell organelles, with collagen fibers or with interstitial space. Intensely electron dense needle-shaped crystals noted in and at the edge of laminated structures and in the lining of vessels resembled hydroxyapatite. There were no significant lesions in other tissues. There was tension between owner and breeder which made it unjudicious to inquire about the feeding routine to which this young dog had been exposed.

Attendees' Diagnoses & Comments: 1) Calcification/mineralization, metastatic, diffuse, severe, lung, toy poodle, canone. 2) Pneumonia, interstitial, granulomatous, chronic, focal, lung.

Etiologic Diagnosis: Unresolved but suspect hypervitaminosis D or uremia.

Comments: There was unanimous agreement with the contributor's diagnosis. The diffuse mineralization in this case was considered compatible with vitamin D toxicosis or uremia. Possible causes listed prior to the conference were secondary hyperparathyroidism, congenital renal hypoplasia, and functional pituitary adenoma. A focus of pneumonia with interstitial fibrosis, macrophages and giant cells was present in some sections. If this focus of inflammation was a response to the mineral, we are unable to explain the focal nature of the response when the mineral was diffuse in septal walls, vessel walls, and bronchiolar walls. Mineral also was present in many alveolar spaces. There was some discussion of the

vaccination status of this animal and the possibility of the CNS signs being attributed to the metastatic calcification or some infectious process, i e. canine distemper, or toxoplasmosis.

Contributor: Department of Veterinary Science, University of Arizona, Tucson, AZ 85721.

References:
1. Hunt, R. D., et al.: Hypervitaminosis in New World Monkeys. Am. J. Clin. Nutr. 22: 358-366, 1969.
2. Capen, C. C. et al.: The pathology of hypervitaminosis D in cattle. Path. Vet. 3: 350-378, 1968.
3. Mulligan, R. M. et al.: Metastatic calcification produced in dogs by hypervitaminosis D and haliphagia. Am. J. Path. 26: 451-473, 1948.

Case IV - 17731/80-976 (AFIP 1761488).

History: A severe diarrhea was observed 3 Mar 80 in this rabbit. It died eleven days later.

Contributor's Diagnosis & Comments: 1) Hepatitis, necrotizing, acute, multifocal, severe, liver, etiology: Bacillus piliformis. 2) Coccidiosis, hepatic, multifocal, severe, liver, etiology: Eimeria stiedae. 3) Hyperplasia, biliary, multifocal, severe, bile duct, liver.

Comments: The intrahepatic bile duct epithelium is proliferative forming fronds with numerous coccidial organisms in varying stages of development in the epithelial cells and lumen of the ducts. Also focal areas of necrosis are scattered throughout the hepatic parenchyma. Degenerating hepatocytes at the margin of the areas of necrosis contain filamentous bacteria consistent with Bacillus piliformis. This case probably represents a truly coincidental, simultaneous occurrence of two infectious processes in one tissue. Both Eimeria steidae and Bacillus piliformis are primary pathogens, neither requiring the action of a predisposing agent. Bacillus piliformis was, however, considered to be the cause of diarrhea and death. Lesions caused by B. piliformis were not demonstrated in other tissues of this rabbit due to extreme postmortem autolysis. However, other rabbits received at the same time from the same supplier and colony had lesions caused by B. piliformis in the caecum and heart as well as the liver.

Attendees' Diagnoses & Comments:

Morphologic Diagnoses: 1) Hyperplasia, biliary, multifocal, moderate to severe, with ductal ectasia, liver, rabbit. 2) Necrosis, hepatocellular, multifocal, moderate, liver.

Etiologic Diagnoses: 1) Hepatic coccidiosis. 2) Bacillary hepatitis (Tyzzer's disease).

Etiology: 1) Eimeria stedae. 2) Bacillus piliformis.

Comments: An equally acceptable morphologic diagnosis of proliferative cholangitis was listed by several attendees as there was a mild inflammatory component surrounding the affected bile ducts in many sections. This inflammatory component was composed primarily of lymphocytes and a few heterophils. Large numbers of coccidian parasites were observed in the hyperplastic epithelium and free within the lumen of the bile ducts by all the participants. Some preferred an etiomorphologic diagnosis of protozoal cholangitis. All of the participants reported observing the multiple foci of hepatic necrosis and several reported seeing intracellular organisms in the hepatocytes at the periphery of these lesions. These bacterial organisms were characterized as long bacilli arranged in a cross stick fashion in the cytoplasm of hepatocytes. Subsequently a GMS stain was provided which accentuated this morphology. A brief discussion followed concerning the life cycle of the coccidian parasite. Points discussed were mode of transmission, migration to the liver, bile ducts, and stages of replication. The simultaneous occurrence of these diseases in this rabbit suggests a decrease immune status due to stress and hepatic coccidiosis which in turn led to the establishment of B. piliformis as an opportunistic infection.

Contributor: Division of Pathology, Walter Reed Army Institute of Research, Washington, DC 20012.

References:

1. McClure, H. M. et al.: The Digestive System. In Pathology of Laboratory Animals, ed. by K. Benerscke, et al., New York, Springer-Verlag, 1978. Vol. I, pp 20.

2. Squire, R. A. et al.: Tumors in Pathology of Laboratory Animals, ed. by K. Benerscke, et al., New York, Springer-Verlag, 1978. Vol. II, pp 1160.

3. Carlton, W. W. and Hunt, R. D.: Bacterial Diseases. In Pathology of Laboratory Animals, ed. by K. Benerschke, et al., New York, Springer-Verlag, 1978. Vol. II, pp 1380-1384.

4. Shadduck, J. A. and Pakes, S. P.: Protozoal and Metozoal Diseases. In Pathology of Laboratory Animals, ed. by K. Benerschke, et al., New York, Springer-Verlag, 1978, Vol. II, pp 1603-1607.

5. Smetana, H.: Coccidiosis of the liver in rabbits III: Experimental study of the histogenesis of coccidiosis of the liver. Arch. Pathol. 15:516-536, 1933.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Histories
AFIP Wednesday Slide Conference - No. 10
26 November 1980

Case I - 80-331 (AFIP 1757304).

History: Of 20 heifers at pasture, four went off feed and developed dermatitis, swollen lymph nodes and had blood in their feces. An enlarged kidney was palpated per rectum in one heifer. All four cattle died within two days of each other.

Case II - Unmarked (AFIP 1755277).

History: CNS tissue from a 2-week-old Airdale puppy from South Africa. Four pups died from a litter of eight. The pups died rapidly showing vague CNS signs.

Case III - EPL D78075 (AFIP 1666524).

History: This tissue is from an 8-year-old spayed female poodle. She was euthanized following the onset of dyspnea and cranial nerve deficits. Radiographs show a mass in the anterior mediastinum. The only significant finding at necropsy was a 4.0 cm diameter mass extending from the base of the heart to the attachment of the anterior sternum that was multinodular and mottled yellow and dark red.

Case IV - WN 80/729-2 (AFIP 1761487).

History: Pellets containing 1080 were distributed to bait rabbits in a paddock containing 30 head of cattle, including cows and calves. Two calves were found dead the next day, and a cow and calf (80/729-2,3) died 3 days after baiting. The owner observed the animals "drop dead and then rapidly bloat". At autopsy there was generalized congestion of visceral organs and subepicardial hemorrhage.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Results
AFIP Wednesday Slide Conference - No. 10
26 November 1980

Case I - 80-331 (AFIP 1757304).

History: Of 20 heifers at pasture, four went off feed and developed dermatitis, swollen lymph nodes and had blood in their feces. An enlarged kidney was palpated per rectum in one heifer. All four cattle died within two days of each other.

Contributor's Diagnosis & Comments:

Diagnosis: Hairy Vetch (Vicia villosa) poisoning.

Comments: Abundant quantities of hairy vetch were found growing in the pasture. Apparently it had been introduced to the farm the previous year in a load of wheat seed bought from another farm. The wheat crop grown from the contaminated seed was so badly infested with vetch that it was not harvestable, consequently the affected field was converted to pasture; the pasture grazed by the affected cattle this spring. Gross and microscopic lesions typical of hairy vetch poisoning were seen in skin, kidney, heart, lymphoid tissues and other internal organs.

Attendees' Diagnoses & Comments:

Morphologic Diagnoses: 1) Splenitis, granulomatous, multifocal, mild to moderate, spleen, heifer, bovine. 2) Hyperplasia, lymphoid, diffuse, moderate, spleen.

Etiologic Diagnosis: Vicia villosa toxicosis (Plant toxicosis).

Etiology: Vicia villosa.

Comments: The morphologic diagnoses were reached after considerable discussion. The etiologic diagnosis was not disclosed until after presentation of the histologic findings. The majority of the attendees proposed an infectious etiologic diagnosis such as mycobacteriosis or bovine malignant catarrhal fever as their pre-conference choice. A few others listed plant toxicosis with resultant photosensitization and skin lesions, but could not relate these clinical signs with the histologic lesions in the spleen. All attendees reported observing giant cells with multiple nuclei scattered throughout the section of splenic tissue in variable numbers depending on their particular section. A few attendees felt there was an increase in the number of lymphocytic cells both in the white and red pulp, but this was considered dependent on the age of the animal. Participants who were familiar with this condition in bovines felt the lesions were quite characteristic for hair vetch poisoning and not T.B. or M.C.F. It was pointed out that the lesions lacked the typical appearance of the tuberculus granuloma with caseation of the central portion surrounded by giant cells and proliferating fibrous connective tissue. Malignant catarrhal fever was considered an unlikely cause with the absence of vasculitis and the presence of the multinucleated giant cells. Giant cells are reported only in tissue culture and not in the natural disease. Other lesions observed grossly with this plant toxicity are greyish colored nodules in the kidneys and adrenal glands. The lesions in the kidney must be differentiated from M.C.F., lymphoma and East Coast Fever (in Africa). Histologically there is a mixed cellular infiltrate consisting of lymphocytes, macrophages and giant cells in these tissues which may in some cases be confused with malignant lymphoma. A short discussion followed concerning the clinical signs observed with this toxicosis.

Photosensitization and dermatitis are reported with this and other members of the genus Vicia. Hemolytic disease also is observed with some members of this genus. The blood in the feces of these heifers was attributed to a local toxic effect in the G.I. tract.

Contributor: New Bolton Center, Pennsylvania State University, 382 W. Street Road, Kennett Square, PA 19348.

References:

1. Panciera, R. J. et al.: A disease of cattle grazing hairy vetch pasture. JAVMA 148: 804-808, 1966.
2. Smith, H. A. et al.: In Veterinary Pathology, 4th Edition, 1972, pp 885-886.

Case II - Unmarked (AFIP 1755277).

History: CNS tissue from a 2-week-old Airdale puppy from South Africa. Four pups died from a litter of eight. The pups died rapidly showing vague CNS signs.

Contributor's Diagnosis & Comments: Canine cerebral babesiosis.

Comments: This case is unusual due to the young age of the pups. Although congenital infection has not been ruled out, the general opinion is that the pups acquired the infection via tick bites just as older dogs. These pups must have been infected soon after birth.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Erythrocytic parasitemia, capillaries, cerebral cortex, brain, Airdale, canine.

Etiologic Diagnosis: Canine cerebral babesiosis.

Etiology: Babesia sp. probably B. canis.

Comments: There was unanimous agreement with the contributor's diagnosis and comments. All the participants reported observing numerous intraerythrocytic protozoal parasites within the capillaries in the brain sections. Differential etiologic agents listed included Hemobartonella canis, Hepatocystis sp. and Trypanosoma sp. Other histologic features reported by some attendees were neuronal degeneration which was not considered a prominent feature and perivascular hemorrhages which were present in a few sections. One of the contributors was present at the conference and discussed the clinical and histopathological findings of this disease in dogs in his experience. Clinically the signs are a marked fever, dry cough, pronounced anemia (15-25% PCV), icterus, hemoglobinuria, splenomegaly and hepatomegaly. Blood smears may reveal a 60-70% parasitemia of the red blood cells. Grossly the spleen and liver are enlarged and dark and the kidney also appears dark. There may be generalized icterus and subdural hemorrhages. The lungs may be heavy and wet and the urinary bladder usually contains bloody urine (red water). Histologically there is pulmonary edema, congestion in numerous organs, and hemosiderosis in the spleen. Interstitial nephritis and centrilobular hepatic necrosis may be present. Histologic lesions in the brain include congestion in the cerebral vasculature, widening of the Virchow-Robin spaces, and hemorrhage in the midbrain and base of the sulci. Diagnosis of the cerebral form is made at necropsy by observing parasitized RBC's in cerebral capillaries in Giemsa stained brain smears, particularly from the hippocampal region. Participants agreed with the contributor's comments concerning the route

of infection in this animal. The prepatent period for this disease is reported to be between 5 and 24 days. Congenital cerebral babesiosis has been reported in newborn calves (2). In Africa treatment of this disease in dogs is directed at the acute stage of the disease but not at elimination of the parasite. Complete elimination of the parasite renders the animal susceptible to subsequent infection as the immunity wanes. A low level of parasitemia is preferred with resultant premunity. Splenectomy results in loss of premunity.

Contributor: Onderstepoort Veterinary Research Institute, Onderstepoort 0110, Republic of South Africa, and the Department of Zoonotic Diseases, Armed Forces Institute of Pathology, Washington, DC 20306.

References:

1. Jubb, K.V.F. and Kennedy, P. C.: In Pathology of Domestic Animals, 2nd Ed., 1970, p. 322.

2. De Vos, A. J. et al.: Cerebral babesiosis in a newborn calf. Onderstepoort J. Vet. Res. 43(2): 75-78, 1976.

Case III - EPL D78075 (AFIP 1666524).

History: This tissue is from an 8-year-old spayed female poodle. She was euthanized following the onset of dyspnea and cranial nerve deficits. Radiographs show a mass in the anterior mediastinum. The only significant finding at necropsy was a 4.0 cm diameter mass extending from the base of the heart to the attachment of the anterior sternum that was multinodular and mottled yellow and dark red.

Contributor's Diagnosis: Anterior Mediastinum-Lymphoepithelioma.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Thymoma, anterior mediastinum, poodle, canine.

Comments: There was general agreement with the contributor's diagnosis, although the preferred morphologic diagnosis was thymoma as classified by the WHO (1). Differentially the participants listed thymic lymphosarcoma and undifferentiated carcinoma. A few others listed possible inflammatory mediastinitis. Histologic features discussed were the mixed cell population of lymphocytic and epithelial cells which generated such descriptive terms as mixed epithelial and lymphocytic and predominately epithelial or clear cell thymoma. A few participants reported they observed Hassel's corpuscles in the tissue sections, while others observed multifocal necrosis in the tumor. In a recent publication it was proposed that the Hassel's corpuscles originate from the circulating mononuclear phagocytic cells (3). The combined lymphocytic and epithelial components are considered necessary for the diagnosis of thymoma which helps to distinguish this neoplasm from pure lymphocytic sarcoma and histiocytic lymphosarcoma. Discussion followed concerning the association of thymoma and Myasthenia gravis and the suppression of acetylcholine uptake at the neuromuscular junction and the occurrence of these two conditions in the canine (4).

Contributor: Experimental Pathology Laboratories, Inc., P. O. Box 474, Herndon, VA 22070.

References:
1. Jarrett, W.F.H. & Mackey, L. J.: Neoplastic diseases of the haematopoietic and lymphoid tissue. Bull. Wld. Hlth. Org. 50: 21-34, 1974.
2. Moulton, J. E. et al.: Tumors of the lymphoid and hemopoietic tissues. In Tumors of Domestic Animals., 2nd edition, University of Calif. Press, Berkeley, CA, 1978, p. 177.
3. Blau, J. N.: Letter to the Editor. The Lancet, Jan. 20, 1979.
4. Parker, G. A., Casey, H. W.: Thymomas in Domestic Animals. Vet. Pathol. 13: 353-364, 1976.

Case IV - WN 80/729-2 (AFIP 1761487).

History: Pellets containing 1080 were distributed to bait rabbits in a paddock containing 30 head of cattle, including cows and calves. Two calves were found dead the next day, and a cow and calf (80/729-2,3) died 3 days after baiting. The owner observed the animals "drop dead and then rapidly bloat". At autopsy there was generalized congestion of visceral organs and subepicardial hemorrhage.

Contributor's Diagnosis & Comments: Multiple small foci of myocardial necrosis, often involving individual fibers, with histiocytic infiltration. 1080 toxicosis.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Degeneration and necrosis, multifocal, mild, heart, bovine.

Etiologic Diagnosis: Fluoromonoacetate toxicosis.

Etiology: Sodium monofluoracetate/fluoroacetamide (1080).

Comments: The attendees agreed with the contributor's morphologic description and etiologic diagnosis as based on the histologic lesions and case history. A few preferred a morphologic diagnosis of acute multifocal necrotizing myocarditis as supported by the obvious infiltration of histiocytic/macrophagic cells in response to the myocardial necrosis. Each diagnosis was considered equally acceptable. Differential etiologic diagnoses included water hemlock and Japanese yew toxicosis. The lesions present are considered nonspecific and diagnosis is strongly based on the clinical history, gross and microscopic findings. Other lesions associated with this rodenticide are subepicardial hemorrhage and visceral hemorrhage in cattle and pulmonary edema and congestion of the liver and kidney. Dogs, cats and pigs may become indirectly poisoned through consumption of 1080 intoxicated rodents. Clinical signs in dogs and cats must be differentiated from those of strychnine and thallium poisoning.

Contributor: Regional Veterinary Laboratory, Wollongbar, N.S.W. 2480 Australia.

References:
1. Buck, W. B. et al.: Clinical and Diagnostic Veterinary Toxicology, 2nd Ed., 1976, p. 233.
2. Smith, H. A. et al.: In Veterinary Pathology, 4th Edition, 1972, p. 823.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Histories
AFIP Wednesday Slide Conference - No. 11
10 December 1980

Case I - 77324 - (AFIP 1756983).

History: This is from one of a series of feral California Sea Lions which was found stranded on a Los Angeles County beach in the early summer of 1978. It was an animal about 9 months of age, and was quite thin. It was febrile and had signs of pneumonia. It died after a few days in captivity.

Case II - 65-1970 - (AFIP 1727493).

History: Hamster inoculated with tissue suspension, ten months prior to necropsy with mass in flank area.

Case III - 80-2858 - (AFIP 1757437).

History: A 40-pound 3-year-old mixed breed male dog was presented for lethargy, anorexia and polydipsia. The dog was febrile with enlarged warm testicles and an enlarged left mandibular lymph node. Antibiotic therpay was instituted for 2 months and castration recommended. The owner delayed the castration for 5 months at which time the dog was clinically normal. The dog had never been sexually active.

Case IV - 4-7-81 - (AFIP 1758838).

History: A 5-year-old ewe was presented with a history of dyspnea and a mucopurulent nasal discharge which had been present for a period of one week. The animal was euthanized and a necropsy performed.

Laboratory Results: Significant gross lesions were limited to the left nasal cavity. There was an irregular shaped, firm, white mass that invaded adjacent tissue and filled the entire cavity.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Results
AFIP Wednesday Slide Conference - No. 11
10 December 1980

Case I - 77324 - (AFIP 1756983).

History: This is from one of a series of feral California Sea Lions which was found stranded on a Los Angeles County beach in the early summer of 1978. It was an animal about 9 months of age, and was quite thin. It was febrile and had signs of pneumonia. It died after a few days in captivity.

Contributor Diagnosis & Comments: Acute Viral Hepatitis.

This appears to be an entity which had not been seen previously in this area, nor reported in the literature. There is acute necrosis which is multifocal throughout the liver section, and affecting large areas of some lobules. There is a sparce infiltration of neutrophils and mononuclear inflammatory cells at the periphery of the necrotic foci. Numerous intranuclear inclusion bodies are present in the hepatocytes in the affected areas. Attempts to isolate the virus were unsuccessful. Ultrastructural examination demonstrated virus which had the morphology and size of adenovirus.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Hepatitis, necrotizing, acute, diffuse, severe, liver, California sea lion.

Etiologic Diagnosis: Viral hepatitis.

Etiology: Suspected adenovirus.

Comments: There was general agreement with the contributor's diagnosis and comments. The distribution of the lesions was considered centrilobular and midzonal overall but several coalescing foci obscured the histologic picture. Generally few inflammatory cells were observed and these consisted of PMN's and macrophages as reported by the contributor. Several attendees reported basophilic intranuclear inclusions surrounded by a clear nuclear zone but these were rare. The majority of the inclusions were a smudgy eosinophilia and occupied the entire nucleus. A differential etiologic agent considered by several was herpes virus infection based on the intranuclear inclusions. It was pointed out that inclusions are not specific for viruses. The histological appearance of the tissue was reminiscent of infectious canine hepatitis. The lesions were not suggestive of leptospirosis which is endemic in sea lions. The pneumonic lesions observed grossly may be related to lungworm infestation, probably Parafilaroides decorus which is found commonly in wild sea lions. The intermediate host is a marine teleost fish, the Opaleye. The worms found in the stomach were probably Contracecum sp. and/or Anasakis sp.

Contributor: Los Angeles County Department of Health Services, Comparative Medical and Veterinary Services, 12824 Erickson Ave., Downey, CA 90242.

Reference: Britt, J. O. et al.: Acute viral hepatitis in California sea lions. JAVMA 175: 921-923, 1979.

Case II - 65-1970 - (AFIP 1727493).

History: Hamster inoculated with tissue suspension, ten months prior to necropsy with mass in flank area.

Contributor Diagnosis & Comments: Fibrosarcoma induced with bovine papilloma virus suspension.

Comments: Hamsters respond with fibrosarcoma in the subcutis or brain from local inoculation of bovine papilloma or deer fibroma virus. About 10% have late pulmonary metastases. When a flank tumor is induced as in this case, there is a marked stimulation of pigment laden cells normally concentrated in this area. The pigmented cells are principally melanocytes though mast cells also are found in these fibrosaromas.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Fibrosarcoma, skin, flank area, hamster.

Etiologic Diagnosis: Viral induced fibrosarcoma.

Etiology: Bovine papilloma virus (Papovavirus).

Comments: The morphologic diagnosis, etiologic diagnosis and etiology were not reached until after discussion and disclosure of the agent. The majority of the participants favored a malignant melanoma, spindle cell type as their preconference choice. This was based on the presence of the melanin pigment in large areas of the tumor. The amount of pigment laden cells varied from one section to the next; some had large areas of mature fibrous connective tissue with little pigment while others had considerable pigmentation in their sections. Junctional activity was absent. The two principal cell populations present were fibrocytic cells and melanocytes. Several attendees also reported observing numerous mast-like cells with cytoplasmic granules in less differentiated areas of the tumor. The concensus of opinion was that the mass appeared malignant although mitotic activity was minimal. Other viral agents which have been reported to cause fibrosarcomas in hamsters include SV-40 virus and avian adenovirus (CELO).

Contributor: Department of Pathology, University of Wisconsin, 1655 Linden Drive, Madison, WI 53706.

References:

1. Hoffman, R. A. et al.: The Golden Hamster. Iowa State University Press, Ames, Iowa, pp 100-101.

2. Robl, M. G. and Olson, Carl: Oncogenic action of bovine papilloma virus in hamsters. Cancer Res. 28: 1596-1604, 1968.

Case III - 80-2858 - (AFIP 1757437).

History: A 40-pound 3-year-old mixed breed male dog was presented for lethargy, anorexia and polydipsia. The dog was febrile with enlarged warm testicles and an enlarged left mandibular lymph node. Antibiotic therpay was instituted for 2 months and castration recommended. The owner delayed the castration for 5 months at which time the dog was clinically normal. The dog had never been sexually active.

Laboratory Results: PCV 38%. Total protein 10.6 g/dl. 824 monocytes/ul. Serum antibody titer to Brucella canis was 1:400.

Contributor Diagnosis & Comments: a) Epididymitis and orchitis, lymphoplasmocytic, chronic, diffuse, severe. b) Seminiferous tubule degeneration, diffuse, severe. c) Interstitial cell hyperplasia, multinodular, mild to marked.

Comments: An etiologic diagnosis of Brucella canis orchitis was based on the positive brucellosis titer and the degenerative and inflammatory changes in the testicle and epididymis.[1,2] Organisms were not seen with Gram stain. The cause of the interstitial cell hyperplasia (which was more diffuse and marked in the contralateral testicle) is unknown. It has been observed in man that interstitial cell populations vary inversely with the spermatogenesis.[3] Widespread nodular hyperplasia and diffuse hyperplasia of interstitial cells are not uncommon in atrophic testicles in animals.

Atendees' Diagnoses & Comments:

Morphologic Diagnoses: 1) Orchitis, chronic, diffuse, mild, testicle, canine. 2) Epididymitis, chronic, multifocal, mild to moderate, testicle. 3) Hyperplasia, interstitial cell, diffuse, moderate to severe, testicle.

Etiologic Diagnosis: Orchi-epididymal brucellosis.

Etiology: Brucella canis.

Comments: Participants generally agreed with the contributor's diagnoses and comments. A variety of morphologic diagnoses were submitted prior to the conference which either incorporated the three separate entities or listed them separately. The history was considered quite suggestive of and the lesions consistent with brucellosis in the dog. Discussion of the lesions included the severe tubular degeneration, the lymphoplasmacytic infiltration of the interstitium and the diffuse interstitial cell hyperplasia. Several attendees reported observing an increase in fibrous connective tissue characterized as a thickened eosinophilic fibrillar stroma surrounding some degenerating tubules. Whether this was absolute or relative was not resolved. The direct cause of the seminiferous tubular degeneration was postulated to be either directly related to the bacterial infection or possibly due to an increase in local tissue temperature due to inflammation. The interstitial cell hyperplasia was considered real and related to the tubular atrophy/degeneration via lack of negative feedback inhibition.[3] The inflammatory cell infiltrate was minimal in this tissue; apparently the majority of the inflammation had subsided as based on the history and histological findings. A short discussion followed concerning the immunity to Brucella organisms. Cell mediated immunity is the principal host reaction and form of elimination of the organisms. Humoral antibody production also is important in the elimination of brucella organisms. Stains employed in the demonstration of Brucella sp. include Gram's, especially the Brown & Hopps modification, and occasionally GMS silver stain. In humans the organisms sequester in the bone marrow and if attempts

to isolate the organism from the blood fail, culture of bone marrow aspirates should be attempted. In dogs bacteremia may persist for several months. Blood, vaginal discharges from bitches with recent history of abortion, testicle and prostate of the male may yield organisms on culture with prescribed culture media.[4]

Contributor: Department of Veterinary Pathobiology, The Ohio State University, Columbus, OH 43210.

References:

1. Moore, J. A. et al.: Male dogs naturally infected with Brucella canis. JAVMA 155: 1352, 1969.
2. Gleiser, C. A. et al.: Pathologic changes in dogs infected with a Brucella organism. Lab. Anim. Sci. 21: 540, 1971.
3. Warren, S. et al.: Interstitial cell growth of the testicle. Am. J. Path. 19: 307, 1943.
4. Moore, J. A. et al.: Epizootiology, diagnosis and control of Brucella canis. JAVMA 156: 1737-1740, 1970.

Case IV - 4-7-81 - (AFIP 1758838).

History: A 5-year-old ewe was presented with a history of dyspnea and a mucopurulent nasal discharge which had been present for a period of one week. The animal was euthanized and a necropsy performed.

Laboratory Results: Significant gross lesions were limited to the left nasal cavity. There was an irregular shaped, firm, white mass that invaded adjacent tissue and filled the entire cavity.

Contributor Diagnosis & Comments: Nasal adenocarcinoma.
Comment: This was a single animal from a flock of unknown size. Endemic ethmoturbinate tumors have been reported from this area.

Attendees' Diagnosis & Comments:
Morphologic Diagnosis: Adenocarcinoma, nasal cavity, sheep, ovine.
Comments: There was total agreement with the contributor's diagnosis. A few participants added such descriptive terms to their diagnosis as papillary, or papillo-tubular adenocarcinoma. Differentially several attendees listed adenoma based on the very uniform well differentiated cells composing the mass. Etiologic diagnoses listed by several attendees included endemic nasal/ethmoturbinate adenocarcinoma of sheep or simply viral induced nasal adenocarcinoma of sheep. The proposed etiologic agent was retrovirus. Participants favoring a viral etiology cited a recent report in which viral particles morphologically similar to a visna-maedi virus were detected in tumor tissues.[2] In some sections a small amount of bone was present and felt to be reactive bone versus pre-existing. Several participants reported the presence of multifocal squamous metaplasia within the tumor. Considerable variability was reported in the mitotic rate of the tumor cells. The cell of origin of this tumor is not precisely known; it could arise

from either mucosal epithelium or Bowman's glands in the lamina propria of the mucosa. A mild inflammatory infiltrate consisting of neutrophils, lymphocytes and some macrophages was observed by several attendees. These neoplasms are not reported to metastasize but are locally invasive and may progress into the cranial vault with resultant neurological lesions. Differentially based on the history Oestrus ovis parasites must be considered with greenish nasal discharges in sheep.

Contributor: Marsh Veterinary Laboratory, P. O. Box 997, Bozeman, MT 59715.

References:

1. Young, S. et al.: Neoplasms of the olfactory mucous membrane of sheep. Cornell Vet. 51: 97-112, 1961.
2. Yonemichi, H. et al.: Intranasal tumor of the ethmoid olfactory mucosa in sheep. Am. J. Vet. Res. 39: 1599-1606, 1978.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

PLEASE NOTE THE FOLLOWING CHANGES FOR CONFERENCE #10

**Addition:
Conf. #10
Case II --- Add to end of Attendees' Comments:
If treatment of the acute disease results in sterilization of infection the animal is susceptible to re-infection.

**Correction:
Conf. #10
Case III -- Attendees' Comments:
Sentence beginning - In a recent publication.....
Should read - Hassel's corpuscles function as mononuclear phagocytic cells of the R.E. system.

Histories
AFIP Wednesday Slide Conference - No. 12
17 December 1980

Case I - A-18946 - (AFIP 1667112).

History: A 6-month-old female Siberian Husky presented with a history of right forelimb lameness for a few days. The clinical diagnosis of panosteitis was made. The dog was treated with steroids, analgesics and Vitamin C. A few days later, the dog was presented non-ambulatory, with tetraparetic cervical spine pain, and upper motor neuron signs of the hind limbs. Cervical cord neoplasm (glioma) was the clinical diagnosis.

Laboratory Results: CSF analysis, Hematology and SMA were unremarkable.

Case II - 13747 - (AFIP 1713077).

History: Tissue is from one of a male C57BL mice out of a group of 10 that died suddenly. The survivor was dyspneic. Sixteen of 20 male ICR mice from the same room died at the same time.

Laboratory Results: Bacteriologic and parasitologic exams as well as virologic titers of the surviving ICR mice were negative.

Case III - 80P465 - (AFIP 1758396).

History: An all-white male foal of paint parentage, with clinical signs of dullness and colic, was presented to the CSU Clinic for euthanasia. It was one day old.

Case IV - 80/0623, H80/775 - (AFIP 1757043).

History: The tissue is from a 4-month-old Angora goat kid. The animal was found in sternal recumbancy. During the two weeks of observation it was unable to walk or rise but remained alert and continued to eat and drink. The limb withdrawal reflexes were normal. The country of origin is Australia.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Results
AFIP Wednesday Slide Conference - No. 12
17 December 1980

Case I - A-18946 - (AFIP 1667112).

History: A 6-month-old female Siberian Husky presented with a history of right forelimb lameness for a few days. The clinical diagnosis of panosteitis was made. The dog was treated with steroids, analgesics and Vitamin C. A few days later, the dog was presented non-ambulatory, with tetraparetic cervical spine pain, and upper motor neuron signs of the hind limbs. Cervical cord neoplasm (glioma) was the clinical diagnosis.

Laboratory Results: CSF analysis, Hematology and SMA were unremarkable.

Contributor Diagnosis & Comments: Cavernous angioma.

Comments: Cavernous angioma is a vascular malformation in the brain or spinal cord reported in human patients. The anomaly is characterized by the presence of blood vessels lined with endothelial cells supported by collagenous connective tissue. These channels are not separated by neural tissue.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Hemangioma, cavernous, spinal cord, Siberian husky, canine. (Vascular malformation).

Comments: The diagnosis listed was preferred as classified by WHO.[1] Attendees agreed with the contributor's comments. Histological features commented on were the large endothelial-lined vascular spaces, the multifocal acute and subacute hemorrhage in the cord, and the neuronal and axonal degeneration present. Some reported observing focal gliosis and satellitosis in the remaining gray matter of the cord. The eosinophilic fibrillar material located beneath the endothelial cells was felt by many to be compatible with collagen together with some smooth muscle cells. The neuronal changes in the cord were considered secondary to the expansile nature of the anomaly. The lesion was believed to be congenital based on the age of the animal and the features of the lesion. It was considered incompatible with telangectasis because normal neuropil was not present between the vascular spaces and the blood filled vascular spaces were very large. The lesion also was incompatible with sclerosing hemangioma of man based on the small amount of connective tissue present and the relatively low number of vascular channels present.[2] The vessels involved in this lesion were considered to be of ventral subdural origin.[4]

Contributor: The Animal Medical Center, Pathology Department, 510 E. 62 St., New York, NY 10021.

References:

1. Weiss, E.: Tumors of the Soft (Mesenchymal) Tissues. Bull. WHO 50: 101-110, 1974.
2. Neoplasm of the Central Nervous System. AFIP Tumor Fascicle.
3. Zaki, F. A.: Vascular malformation (cavernous angioma) of the spinal cord in a dog. J. Sm. Anim. Pract. 20: 417-422, 1979.
4. Cordy, D. R.: Vascular malformations and hemangiomas of the canine spinal cord. Vet. Path. 16: 275-284, 1979.

Case II - 13747 - (AFIP 1713077).

History: Tissue is from one of a male C57BL mice out of a group of 10 that died suddenly. The survivor was dyspneic. Sixteen of 20 male ICR mice from the same room died at the same time.

Laboratory Results: Bacteriologic and parasitologic exams as well as virologic titers of the surviving ICR mice were negative.

Contributor Diagnosis & Comments: Kidney, tubular necrosis, probably due to chloroform toxicity.

Comments: There is marked necrosis and degeneration of the cortical tubular epithelium with generalized sparing of the cuboidal epithelium of Bowman's capsule and upper segments of the proximal tubule. Protein casts in the tubules are prominent. These lesions are typical of brief exposure to chloroform in certain strains of male mice. Investigation showed that chloroform apparently was used in the animals' room in the days before the deaths of these mice.

Etiologic Diagnosis: Toxic tubular necrosis.

Etiology: Compatible with chloroform toxicosis in male mice.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Necrosis, tubular, acute, multifocal, severe, cortex, kidney, C57BL mouse.

Comments: The morphologic diagnosis was agreed upon after some discussion of the lesions. Several attendees preferred tubular nephrosis as their preconference choice based on the etiology. Histologic features commented on were the acute tubular necrosis characterized by hyperchromasia, pyknosis, karyorrhexis and karyolysis of the nuclei in the involved tubules, the coagulative or hyaline change of the cytoplasm of these tubular cells, formation of casts, and the presence of small numbers of PMN's. We agreed that the distribution of the lesions was restricted to the proximal tubules and that the cuboidal epithelium of the Bowman's capsule was spared. Differential etiologic diagnoses considered were heavy metal and chloronated hydrocarbon toxicosis. The pathogenesis of the lesion was discussed including the apparent predilection for the proximal tubular epithelium based on the high metabolic activity of the cells and the role of androgens in strain susceptibility.[1,2] Other lesions reported in chloroform toxicosis in mice are mineralization of renal tubules and centrilobular fatty change and hydropic degeneration of hepatocytes. Mice differ from other species of animals in the excretion of chloroform metabolites in the presence of testosterone in male mice and in ovariectomized female mice treated with testosterone.[3] The strain of mouse involved in this case is considered less susceptible than those highly susceptible strains reported in the literature.[2]

Contributor: Division of Comparative Medicine, Johns Hopkins University School of Medicine, 720 Rutland Ave., Baltimore, MD 21205.

References:

1. Deringer, M. et al.: Results of exposure of strain C3H mice to chloroform. Proc. Soc. Exper. Biol. 83: 474-479, 1953.

2. Benirschke, K. et al.: Pathology of Laboratory Animals, Springer-Verlag, New York, 1978, p. 148.

3. Taylor, D. C. et al.: Metabolism of chloroform II sex difference in the metabolism of (C^{14}) chloroform in mice. Xenobiotica 4: (3) 165-174, 1974.

4. Hill, R. N.: Differential toxicity of chloroform in the mouse. Ann. N.Y. Acad. Sci. 298, 170-175, 1978.

Histories
AFIP Wednesday Slide Conference - No. 13
7 January 1981

Case I - 27178 - (AFIP 1755279).
History: Macaca fascicularis, the cynomolgus monkey from Malaysia, one of a group of 100 in a quarantine facility.

Case II - R 27979 - (AFIP 1720287).
History: The tissue is from a control adult male Fischer rat that died spontaneously. At necropsy, the rat was found to have a unilateral abdominal mass.

Case III - N78-577 - AFIP 1711325).
History: Tissue from a moustached monkey (Cercopithecus cephus) housed at a zoo. The adult, female monkey had a history of a previous lumbar vertebral fracture. An infection in the bone of the tail was treated 4 months prior to signs of severe diarrhea and seizures. Seizures continued for one day and the animal died.

Case IV - 0070 - (AFIP 1758837).
History: 5-month-old DSH male cat. The cat was presented to the referring veterinarian with the complaint of anorexia, depression, and icterus. The animal died despite supportive therapy and was necropsied.

Laboratory Results: At necropsy the carcass was markedly icteric. A small quantity of straw-colored fluid was present in the thoracic cavity. Liver was enlarged and had a mottled appearance.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Results
AFIP Wednesday Slide Conference - No. 13
7 January 1981

Case I - 27178 - (AFIP 1755279).

History: Macaca fascicularis, the cynomolgus monkey from Malaysia, one of a group of 100 in a quarantine facility.

Contributor's Diagnosis & Comments:

Capillaria hepatica. The detection of Capillaria eggs in the liver is often an incidental finding in macaques from Southeast Asia. The eggs are distinctive and are the most important factor in the production of the lesion.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Hepatitis, granulomatous, multifocal, moderate, liver, Macaca fascicularis.

Etiologic Diagnosis: Parasitic hepatitis.

Etiology: Capillaria hepatica.

Comments: Conference participants agreed with the contributor's diagnosis and comments. Participants felt the distribution of the lesions was quite random. A few reported observing eggs within bile ducts. A few others felt there were areas of centrilobular necrosis multifocally and all described a diffuse vacuolar change within hepatocytes. The vacuolar changes may have been due to mild autolysis. The widely scattered pigment in the Kupffer cells was considered to be either hemosiderin or malarial pigment. This species of monkey commonly has chronic malarial infection which is refractory to therapy. Many of the eggs were in multinucleated giant cells. Other inflammatory cells scattered in these granulomatous lesions were lymphocytes, plasma cells, and eosinophils. Considerable fibrosis accompanied these foci.

The eggs were of the Trichuroid type, i.e., had bipolar plugs and were elongate. The egg shells had striations and those that were not degenerate contained a single cell. Other Trichuroidea with similar eggs were listed. Trichuris eggs are more ovoid and without striations in the shell; Trichisomoides is found in the urogenital system of rodents and its eggs may be found in tissue. Anatrichisoma also is found in primates (i.e., macaques). It lives in the nasal or mucosal epithelium of the oral cavity, and its eggs are passed in feces. These eggs may be differentiated from C. hepatica since eggs of Anatrchisoma contain a larva.

The life cycle of Capillaria hepatica was reviewed. Rodents are the usual final hosts but other mammals (e.g., monkeys, man) also may serve as final hosts since this parasite is not very host specific. When embryonated eggs are ingested by a suitable final host, the larvae hatch and make their way to the liver via the hepatic portal circulation. In the hapatic parenchyma, the larvae molt four times and become adults in approximately 1-2 weeks. The fertilized females lay eggs in the hepatic parenchyma where they incite a pyogranulomatous response with eosinophils. Eggs remain viable in hepatic tissue for long periods of time after the death of the worms. These eggs must get into the soil for embryonation before they are infective. This is accomplished in one of two ways. The animal may die and subsequent decomposition of its body liberates the eggs and these fall to the soil. Another mechanism is when the host is eaten by a predator. The eggs

digested from the liver of the host are passed in the predator's feces. Such predators disperse the eggs and thus may be termed transport hosts. Unembryonated eggs are non-infective. Final hosts are infected by ingestion of embryonated eggs with food or water.

Contributor: Bureau of Biologics, FDA, 8800 Rockville Pike, Bethesda, MD 20205.

References:
1. Benirschke, K. et al.: Pathology of Laboratory Animals, Vol. II, Springer-Verlag, 1978, pp 1670-1671.
2. Graham, G. L.: Parasitism in monkeys. Ann. NY Acad. Sci. 85: (3), 849-851, 1960.

Case II - R 27979 - (AFIP 1720287).

History: The tissue is from a control adult male Fischer rat that died spontaneously. At necropsy, the rat was found to have a unilateral abdominal mass.

Contributor's Diagnosis & Comments: Teratoid nephroma. This tumor is well circumscribed and comprised of tubules lined by columnar epithelial cells, some of which bear cilia. We have seen 2 other similar tumors in a series of about 500 F344 rats. Both of these were smaller than this one and appeared histologically to be benign. There appears to be only one report in the literature.

Attendees' Diagnosis & Comments: Mixed embryonal renal tumor, origin undetermined. This diagnosis was reached after some discussion of the lesion. Other preconference diagnoses submitted were nephroblastoma, teratoma, papillary adenocarcinoma, and mixed adenocarcinoma. The origin of the tumor was speculated by many and included paramesonephric duct, Mullarian and Wolffian duct reminants, prostate and seminal vesicles. The exact origin was not determined. Another lesion reported in the remaining renal tissue present on some of the slides was a mild multifocal interstitial nephritis. Characteristics of the tumor discussed were the large tubular-ductular structures lined by a simple to pseudostratified ciliated columnar epithelium and the loose to dense connective tissue stroma surrounding these structures. Large areas of the stroma appeared myxomatous. Nuclei of the epithelial component were basally located and mitoses were rare. A pale eosinophilic to amphophilic material was present in the lumens of several tubules. Scattered mitotic figures were observed within the stroma. The stroma blended into the adjacent renal stroma. Two germ layers were represented in the tumor, not 3. Classification of the lesion as a teratoma therefore would be inappropriate. The tumor is consistent with the reported teratoid nephroma (1). Primary renal neoplasms of the rat are uncommon.

Contributor: Carnegie-Mellon Institute of Research and FDA.

Reference: Thamavit, W., et al.: Spontaneous teratoid nephroma in a rat. Vet. Pathol. 16: 130-131, 1979.

Case III - N78-577 - AFIP 1711325).

History: Tissue from a moustached monkey (Cercopithecus cephus) housed at a zoo. The adult, female monkey had a history of a previous lumbar vertebral fracture. An infection in the bone of the tail was treated 4 months prior to signs of severe diarrhea and seizures. Seizures continued for one day and the animal died.

Laboratory Results: Culture of granulomatous masses were positive for Mycobacterium tuberculosis.

Contributor's Diagnosis & Comments: Meningoencephalitis, granulomatous, focal, severe, cerebrum with extensive caseation necrosis: etiology Mycobacterium tuberculosis.

Comments: Necropsy revealed large caseating lesions in the mediastinum, mesentery, uterus, cerebrum, and skin at the tail head. These lesions all contained acid-fast bacilli. The liver, intestine and spleen were free of lesions. Ulceration of the skin has been reported in tuberculosis in primates and the skin may be a portal of entry for the organism. Tubercles in the cranial cavity have been reported but are not common.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Meningoencephalitis, granulomatous, focal, severe, cerebrum, brain, Cercopithecus cephus.

Etiologic Diagnosis: Tuberculosis.

Etiology: Mycobacterium tuberculosis.

Comments: The attendees agreed with the contributor's diagnosis and comments. The submeningeal and cerebral expansile necrotic mass was considered characteristic of the disease. Complete encapsulation was not present in most sections. The remaining neuropil was unremarkable with the exception of a large focus of gitter cells and loss of parenchyma in some sections. The route of infection was considered hematogenous to the meninges with subsequent spread into the cerebral tissue. Within the center of the granuloma there were foci of mineralization and nondescript necrotic debris. Mineralization is uncommon in tuberculous lesions in nonhuman primates. The inflammatory cells along the periphery of the lesion consisted of lymphocytes, plasma cells, macrophages and occasional multinucleated giant cells. The role of these cells in the pathogenesis of the lesions was discussed, along with the significance of the caseous necrotic material in the center of the lesion. Plasma cells play a signifcant part through release of lymphkines and MIF and aid in cytolysis through activation of the complement cascade. Lymphocytes play a role in the necrosis of tissue through direct cytotoxicity of cells and activate macrophages and may aid in the formation of giant cells. Macrophages in turn elaborate monokines which enhance the activation of lymphocytes. Macrophages also are responsible for the phagocytosis of the organism. Mycobacteria can persist and divide within these macrophages by preventing fusion with lysosomes. Lipopolysaccharrides (waxes) in the cell wall may prevent this fusion. The mycolic acid on the cell surface is responsible for acid fast staining characteristic of these organisms. This mycolic acid is not degraded easily by macrophages. It has been reported that the caseous material contains few mycobacterial organisms due to the presence of proteolytic and other enzymes released by inflammatory cells at the periphery of the lesion. Liquefactive necrosis on the other hand may contain large numbers of proliferating extracellular organisms in the absence of these enzymes, and thus enhance the

spread of organisms (3). Host response determines the outcome of the disease. An acid fast stain provided demonstrated intracellular acid-fast organisms with occasional macrophages at the periphery of the lesion. Primary cerebral tuberculosis in nonhuman primates is considered uncommon but has been reported (2). Tuberculosis in the cranial cavity of humans usually involves the basal meninges and occasionally produces tuberculoma formation (4).

Contributor: USAF School of Aerospace Medicine, VSP, Brooks AFB, TX 78235.

References:

1. Carlton, W. W. & Hunt, R. D.: Bacterial Diseases. In Pathology of Laboratory Animals, Col. II, pp 1418-1421, Benirschke, K., Garner, F. M. & Jones, T. C., Eds., Springer Verlag, NY, 1978.

2. Dastur, D. K. et al.: The pathology and pathogenesis of tuberculosis encephalopathy. Acta Neuropathologica 6: 311-362, 1966.

3. Dannenburg, A. M.: Liquefaction of caseous foci in tuberculosis. Amer. Rev. of Resp. Disease 113: 257-259, 1976.

Case IV - 0070 - (AFIP 1758837).

History: 5-month-old DSH male cat. The cat was presented to the referring veterinarian with the complaint of anorexia, depression, and icterus. The animal died despite supportive therapy and was necropsied.

Laboratory Results: At necropsy the carcass was markedly icteric. A small quantity of straw-colored fluid was present in the thoracic cavity. Liver was enlarged and had a mottled appearance.

Contributor's Diagnosis: Feline cytauxzoonosis.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: 1) Necrosis and hemorrhage, acute, diffuse, severe, liver. 2) Pneumonia, interstitial, subacute to chronic, diffuse, marked to severe, lung. 3) Vasculitis, multifocal, mild to moderate, lung and liver. 4) Parasitemia, monocytic and erythrocytic, multifocal, lung and liver, DSH cat (feline).

Etiologic Diagnosis: Feline cytauxzoonosis.

Etiology: Cytauxzoon sp.

Comments: The diagnoses listed above were reached after some discussion of the lesions present in these two tissues. Other preconference diagnoses listed by participants included phelebitis and necrohemorrhagic hepatitis which were equally acceptable. Some attendees reported observing a few parasitized red blood cells containing ring forms. A few reported lesions within small arteries in the lung whereas most observed the lesions in the thick and thin walled veins in the lung and liver. The cause of the lesions in the liver were not specifically determined but felt to be due to anoxic change. Scattered histiocytic/monocytic and Kupffer cells in the liver were parasitized. There was some question as to whether hepatocytes also were parasitized. Discussion of the interstitial and vascular lesions in the lung included the diffuse interstitital thickening, hypertrophied alveolar lining cells, macrophages within alveolar spaces -- some of which

contained pigment (hemosiderin), vasculitis, and the presence of large numbers of organisms within circulating cells in vessels. Occasionally it appeared alveolar lining cells also were parasitized. Some confusion existed to whether the cells parasitized in the vessels were monocytes and/or endothelial cells. Hypertophy of endothelial cells was prominant in most large vessels. Multifocal necrosis of vessel walls with hemorrhage into the adjacent parenchyma was present in some sections. The life cycle of the parasite was discussed and compared with the disease in wild African ungulates. A fatal disease attributed to this organism has been reported in a giraffe in Africa. An ixiod tick is suspected as the biological vector of the disease in cats in the United States. The cat seems an unlikely natural host of the organism in that the disease is invariably fatal. Numerous wild sylvatic rodents have been inoculated experimentally with this organism but have proven unsusceptible to the infection (unpublished work). The bobcat may serve as the reservoir host in this country as a carrier state has been observed (unpublished material).

Differential diagnosis includes Hepatozoan sp. that have been observed in white blood cells, lung, bone marrow, and liver of naturally infected cats. Hepatozoan, however, does not infect red blood cells and gametogeny occurs in neutrophils in the circulation. Schizonts of this parasite have been observed in the spleen, bone marrow, and liver. Schizonts of Cytauxzoon have been reported in hypertrophied cells (probably histiocytes) lining the walls of blood vessels in many organs (4). Merozoites are found in erythrocytes.

Contributor: Veterinary Diagnostic Center, University of Nebraska-Lincoln, Lincoln, NE 68583.

References:

1. Wagner, J. E.: A fatal cytauxzoonosis-like disease in cats. JAVMA 168: 588-596, 1976.
2. Wightman, S. R. et al.: Feline cytauxzoonosis: Clinical features of a newly described blood parasite disease. Feline Pract., May 24-26, 1977.
3. Ferris, D. H.: A progress report on the status of a new disease of American cats: Cytauxzoonosis. Comp. Immun. Microbiol. Infect. Dis. 1: 269-276, 1979.
4. Wagner, J. E. et al.: Experimentally induced cytauxzoonosis-like disease in domestic cats. Vet. Parasitol. 6: 305-311, 1980.
5. McCully, R. M. et al.: Cytauxzoonosis in a giraffe in Zululand. Onderstepoort J. Vet. Res. 37: 7-10, 1970.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Histories
AFIP Wednesday Slide Conference - No. 14
14 January 1981

Case I - 79-727 - (AFIP 1716400).

History: Seven-year-old Siamese cat. Presented because of depression and anorexia of 2 weeks duration progressing to convulsions. Side stepping and placing responses absent on left side, depressed on right. On necropsy the right frontal sinus contained gelatinous, carmel colored material. The cranial portion of the right cerebral hemispnere covered with the same material as in sinus. On cut section there was a firm, whitish area replacing normal brain parenchyma of the right cranial cerebral hemisphere.

Case II - 7913-80 - (AFIP 1757255).

History: One-month-old foal in herd with several other foals. Outbreak of diarrhea in foals and all foals responded to antibiotic treatment except this one which died.

Case III - N79-420 - (AFIP 1757300).

History: A young male crossbred barrow in good nutritional condition and weighing 89 pounds was presented for examination. Owner put group of pigs on a ration with 500 lbs per ton of ground whole oats for a treatment of ulcers. He lowered the oats to 300 lbs per ton and pigs started to do poorly. The ratio was 16% crude protein made with corn and 38% supplement. Lately, the pigs were fed new corn from owner's crop that looked very good. Despite this, pigs were growing slowly and developed respiratory problems. Some died.

Case IV - 80-2687/18686 - (AFIP 1764666).

History: Tissue from a 10-year-old male Welsh Corgi with a history of polyuria and polydipsia. Two microslides.

Laboratory Results: (See next page).

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Results
AFIP Wednesday Slide Conference - No. 14
14 January 1981

Case I - 79-727 - (AFIP 1716400).

History: Seven-year-old Siamese cat. Presented because of depression and anorexia of 2 weeks duration progressing to convulsions. Side stepping and placing responses absent on left side, depressed on right. On necropsy the right frontal sinus contained gelatinous, carmel colored material. The cranial portion of the right cerebral hemisphere covered with the same material as in sinus. On cut section there was a firm, whitish area replacing normal brain parenchyma of the right cranial cerebral hemisphere.

Laboratory Results: Impression smears at necropsy showed a preponderance of the same cell type later seen in the neoplasm on H&E sections.

Contributor's Diagnosis: Ependymoma.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Primitive neuroectodermal neoplasms with some ependymomal differentiation, right cerebral hemisphere, brain, Siamese, feline.

Comments: The majority of the preconference diagnoses submitted included ependymoma or ependymoolastoma as the primary diagnosis. Differential diagnoses submitted included adenocarcinoma of the ethmoidal epithelium, neuroblastoma, metastatic adenocarcinoma, and pinealcytoma. Histological features of the neoplasm commented on were the invasive nature, compression, formation of numerous rosettes, neoplastic cells lining up in rows, lack of normal ependymal lined ventricle, and multifocal necrosis present in the tumor. Cellular characteristics included the tall columnar epithelial rosettes formed around small lumens, basally located nuclei, the presence of cilia at the apice of the cells, and high mitotic rate in several areas of the tumor. There was an apparent lack of secretory product in both the lumens and within cell bodies. Many rosettes morphologically resemble those described in ependymomas of man termed Flexner's rosettes which also are described in retinoblastomas. Pseudorosettes, which also were present, form around small blood vessels and stroma within the tumor. This neoplasm is more complex than ependymomas or ependymoblastomas. An olfactory neuroblastoma could not be completely ruled out based on the clinical history, however, rosette formation is not characteristic for this tumor. Other differentials which must be included are tumors of the choroid plexus and astroblastomas. Malignant ependymomas are considered rare in domestic animals and may metastasize via the cerebrospinal fluid.

Contributor: Veterinary Diagnostic Laboratory, Kansas State University, Manhattan, KS 66506.

Reference:

Rubenstein, L. J.: Tumors of the CNS, 2nd series, Fascicle 6, Atlas of Tumor Pathology, AFIP, 1972, pp 104-126.

Case II - 7913-80 - (AFIP 1757255).

History: One-month-old foal in herd with several other foals. Outbreak of diarrhea in foals and all foals responded to antibiotic treatment except this one which died.

Contributor's Diagnosis & Comments: Granulomatous colitis, diffuse, severe hepatic necrosis, acute, multifocal.

Comments: The Gram positive organisms in the foci of hepatocellular necrosis are consistent with the diagnosis of Corynebacterium equi.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: 1) Colitis, granulomatous, ulcerative, diffuse, severe, colon, foal, equine. 2) Necrosis, acute, multifocal, moderate, liver.

Etiologic Diagnosis: Bacterial colitis and hepatic necrosis.

Etiology: Corynebacterium equi.

Comments: Other morphologic diagnoses submitted prior to the conference included necrohemorrhagic colitis/enteritis and necrotizing hepatitis. There was some question as to whether the tissue represented large or small intestine. This was resolved by discussion of the histological appearance of the tissue and disclosure of the contributor's observations. Features of the colonic lesions included diffuse severe congestion of vessels in all layers, diffuse marked edema of the submucosa, diffuse infiltration of the mucosa with histiocytic cells with multifocal granuloma formation, and multifocal ulceration of the mucosa present in many sections. A few attendees reported observing thrombi in small mucosal vessels in addition to the changes reported above. Bacterial organisms were observed in the lamina propria, usually associated with the granulomas, and within the lumen of the organ. The lesions in the liver were compatible with foci of acute necrosis. The distribution of the lesions was considered random. Bacterial organisms were observed within hepatocytes at the periphery of these necrotic foci. The majority of the participants suggested Salmonella sp. as the probable etiologic agent responsible for the lesions. Other bacteria discussed differentially included: Shigella sp., E. coli, and Bacillus piliformis. However, a Gram stain then was projected which demonstrated Gram positive beaded coccobacilli both within the colonic and hepatic lesions. Based on the morphology and staining characteristics of the organisms it was concluded that the organisms were compatible with those of Corynebacterium. C. equi has been reported primarily as a pathogen of the respiratory tract of horses. Recently lesions attributed to this organism have been described in the intestinal tract of horses (1). Other lesions attributed to this organism in horses are seropurulent arthritis and hepatic abscesses (2).

Contributor: Department of Veterinary pathology, College of Veterinary Medicine, University of Missouri, Columbia, MO 65211.

Reference:

1. Bruner, D. W. and Gillespie, J. H.: Hagan's Infectious Diseases of Domestic Animals, 6th Ed., Cornell University Press, 1973, pp 321-323.

2. Cimprich, R. E. and Rooney, J. R.: Corynebacterium equi enteritis in foals. Vet. Pathol. 14: 95-102, 1977.

Case III - N79-420 - (AFIP 1757300).

History: A young male crossbred barrow in good nutritional condition and weighing 89 pounds was presented for examination. Owner put the group of pigs on a ration with 500 lbs per ton of ground whole oats for a treatment of ulcers. He lowered the oats to 300 lbs per ton and pigs started to do poorly. The ratio was 16% crude protein made with corn and 38% supplement. Lately, the pigs were fed new corn from owner's crop that looked very good. Despite this, pigs were growing slowly and developed respiratory problems. Some died.

Laboratory Results: Fibrinogen 700 mg/dl.
1+ Tricuris suis ova and a few large coccidia oocysts on fecal floatation. 5 seeds per lb. of crotolaria in feed and 1 seed per lb. of Sassid obtusiofolia.

Contributor's Diagnosis & Comments:
Pneumonia, proliferative-interstitial, chronic-active, multifocal and coalescing, severe with multifocal adenomatous change of alveoli.
Comment: Lesions present are consistent with pyrrolizidine alkaloid toxicosis.

Attendees' Diagnosis & Comments:
Morphologic Diagnosis: Pneumonia, interstitial, proliferative, chronic-active, diffuse, severe, lung, crossbred barrow, pig.
Etiologic Diagnosis: Toxic pneumonitis.
Etiology: Pyrrolizidine alkaloids.
Comments: The morphologic diagnosis and etiology were reached after considerable discussion of the lesions and disclosure of the laboratory findings. A wide variety of preconference morphologic diagnoses and suspected etiologic agents were submitted by the attendees. The majority of the participants favored a proliferative and suppurative bronchopneumonia, while others preferred a chronic-active bronchopneumonia with adenomatous change. The etiologic diagnosis most often listed by the participants was enzootic pneumonia of pigs. Other etiologic diagnoses suggested by attendees were foreign body pneumonia, verminous pneumonia, and combined bacterial and mycoplasma pneumonia. Histopathologic features included diffuse prominant hypertrophied/hyperplastic alveolar lining cells, diffuse interstitial fibrosis and infiltration with mixed inflammatory cells, multifocal presence of neutrophils within alveoli and bronchioles, multifocal emphysematous change, the formation of large numbers of giant cells and the presence of lymphoid follicles adjacent to bronchioles. The mixed inflammatory infiltrate within the interstitium consisted of lymphocytes, plasma cells, macrophages, neutrophils, and eosinophils along with the proliferating fibrous connective tissue. The most striking feature of the lesion was the multifocal to diffuse adenomatous change within the alveoli. It was pointed out that this is a nonspecific change in lungs and can be produced by a variety of agents (4). The origin of the multinucleated giant cells was discussed, but remained unresolved. Several attendees felt the giant cells resembled foreign body giant cells and others felt many of these cells resembled syncytial giant cells. Some question remained in the minds of many of the participants as to whether an interconcurrent bacterial or viral infection was present in this pig. The peribronchiolar lymphoid follicles were suggestive of mycoplasma infection and the adenomatous change could be attributed to infection with an influenza virus. The discussion turned to the actions of pyrrolizidine alkaloids. This toxin is known to prevent mitosis and thus may account for the hypertrophied alveolar lining cells in this case (1).

Hepatic lesions consisting of fibrosis, bile duct proliferation, megahepatocytes, and infiltration of inflammatory cells around foci of degenerating and necrotic hepatocytes and fibrous connective tissue are the classical lesions of this plant poisoning (3). The lesions present in the lung in this case have been described and are compatible with this toxicosis (1,2).

Contributor: Division of Comparative Pathology, Box J-145, College of Veterinary Medicine, University of Florida, Gainesville, FL 32610.

References:

1. Harding, J. D. J. et al.: Experimental poisoning by Senecio jacobaea in pigs. Path. Vet. 1: 204-220, 1964.
2. Huxtable, R. J.: Pyrrolizidine alkaloids and the lung endothelium: A paradigm of lung damage resulting fron circulating toxins.
3. Keeler, R. F. et al.: Effects of Poisonous Plants on Livestock. Academic Press, New York, 1978, pp 161-187.
4. Omar, A. R.: The characteristic cells of the lung and their reaction to injury. Part I. Vet. Bull. 34: No. 7, 371-443, July 1964.

Case IV - 80-2687/18686 - (AFIP 1764666).

History: Tissue from a 10-year-old male Welsh Corgi with a history of polyuria and polydipsia. Two microslides.

Laboratory Results: (See next page).

Contributor's Diagnosis & Comments: Adrenal cortical carcinoma, with necrosis and mineralization, adrenal, canine.

Comments: Based on the clinical signs and laboratory findings, the practitioner treated the dog for Cushing's Syndrome with o,p' - DDD. Approximately one month later the dog was killed at the owner's request. Select tissues were submitted for histologic evaluation to include an enlarged right adrenal, the left adrenal, liver, and lung which had numerous white nodules of varying sizes. The pituitary was not examined. The diagnosis of adrenal cortical carcinoma was confirmed by the presence of numerous metastatic foci within the lungs. Other findings included necrosis of the zona fasciculata and reticularis in the left adrenal and severe fatty infiltration of the liver with hepatocellular damage.

Adrenal cortical tumors are an uncommon cause of canine hyperadrenocorticism. The most common cause is bilateral cortical hyperplasia - either idiopathic or resulting from a functional pituitary adenoma. Clinical signs and laboratory findings are due to the long term effects of excessive glucocorticoids.

Attendees' Diagnosis & Comments: 1) Carcinoma, cortical, adrenal, with metastasis to the lung, canine. 2) Necrosis and mineralization, multifocal to diffuse, adrenal.

Comments: The majority of the conference attendees listed the diagnoses given above. Other preconference morphologic diagnoses listed were adenocarcinoma with metastasis to the lung and carcinoma of endocrine origin metastatic to the lung.

HEMATOLOGY	4/9/80	5/3/80	5/12/80
RBC ($x10^6/\mu l$)	6.44	5.26	5.03
Hb (g/dl)	15.6	12.7	13.2
WBC (/µl)	10,134	35,700	33,855
Seg. neutrophils (%)	-	91	94
Nonseg. neutrophils (%)	-	6	1
Lymphocytes (%)	-	2	4
Monocytes (%)	-	0	0
Eosinophils (%)	-	1	0
Basophils (%)	-	0	1

SERUM	4/9/80	5/12/80
Fasting glucose (mg/dl)	106	117
BUN (mg/dl)	12	3
Creatinine (mg/dl)	.5	.6
Cholesterol (mg/dl)	590	730
Alkaline phosphatase (mU/ml)	413	840
SGPT/ALT (mU/ml)	735	107
SGOT/AST (mU/ml)	51	31
Total protein (gm/dl)	6.0	5.3
Albumin (gm/dl)	3.2	2.5
Na^+ (mEq/l)	155	153
K^+ (mEq/l)	4.1	2.3

URINALYSIS	4/9/80	5/12/80
Color	Clear	Clear
pH	6.0	6.0
Specific gravity	1.006	1.005
Protein	30+	trace
Ketones	neg.	neg.
Glucose	neg.	neg.
Bile pigments	neg.	neg.
Blood	neg.	neg.

PLASMA CORTISOL (ng/ml)	4/28/80
pre-ACTH	80
2 hr. post-ACTH	290

Hyperplasia of the adrenal cortex also was mentioned by a few. Considerable discussion centered around the clinical laboratory results given by the contributor. This included changes in the hematogram such as the mild to moderate neutrophilia, absolute lymphopenia and eosinopenia. Serum chemistry abnormalities included increased fasting glucose, elevated serum cholesterol, markedly elevated serum alkaline phosphatase on both occasions, marked SGPT elevation initially and later a mild increase, low total protein and albumin on the second sample, and low serum potassium on the last sample. Abnormal findings in the urinalysis included low specific gravity and high urinary protein excretion. This apparent proteinuria led some attendees to suspect a concurrent renal disease in this case. The plasma cortisol levels were felt to be exaggerated before ACTH challenge and diagnostic of hyperplasia/neoplasia after ACTH challenge. Histologic features of the primary and metastatic lesions included the endocrinoid appearance of the cells and the presence of neoplastic cells within vessels along the capsule of the gland in some sections. Some participants mentioned observing tumor cells within foci of mineralization and necrosis in the adrenal. The specific cause of the necrosis and mineralization in the adrenal may be attributed to the tumor itself or to the treatment which was given to the animal. The persistence of the zona glomerulosa present in many sections indicated therapy as the possible cause, as o,p' - DDD is known to spare this layer of the cortex. Correlation between serum chemistries and the neoplasm was discussed with specific reference to the elevated liver enzymes and their pathogenesis. The elevated SGPT could be attributed to hepatocellular degeneration and necrosis subsequent to fatty metamorphosis which is commonly described in hyperadrenocorticism. Elevated serum alkaline phosphatase levels are attributed to increased production of alkaline phosphatase via compression of the bile ducts caused by the fatty metamorphosis in the liver, however, the exact reasons for this increased SAP production are not completely known (2). The hypercholesterolemia could be attributed to the hyperadrenocorticism and/or renal disease.

Contributor: Department of Comparative Pathology, Walter Reed Army Institute of Research, Washington, DC 20012.

References:

1. Capen, C. C.: Tumors of the endocrine gland. In Tumors in Domestic Animals, 2nd ed., University of California Press, Berkeley, 1978.

2. Cohen, S. J. et al.: Hyperadrenocorticism in a dog with adrenal and pituitary neoplasia. J. Amer. Anim. Hosp. Assoc. 16: 259-262, 1980.

3. Feldman, E. C. et al.: The synthetic ACTH stimulation test and measurement of endogenous plasma ACTH levels: Useful diagnostic indicators for adrenal disease in dogs. JAAHA 14: 524-531, 1978.

4. Meijer, J. C.: Canine hyperadrenocorticism. In Current Veterinary Therapy VII, W. B. Saunders Co., Philadelphia, 1980.

5. Owens, J. M. et al.: Hyperadrenocorticism in the dog: Canine Cushing's Syndrome. Vet. Clinics of N. America 7: No. 3, W. B. Saunders Co., Philadelphia, 1977.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Histories
AFIP Wednesday Slide Conference - No. 15
21 January 1981

Case I - 80-68 - (AFIP 1758395).

History: Tissue from a 5-month-old male red kangaroo (Megaleia rufa) which was ejected from its pouch when the mother was frightened by dogs and broke her leg. An attempt was made to hand rear the joey, but it died several months later.

Case II - 80-10933 - (AFIP 1758391).

History: Lung and spleen from a 4-year-old bull. Three bulls died in a 3-day period in a pasture with 2 other bulls and 25 cows. All animals were found dead without previous signs of illness being observed.

Laboratory Results: Marked splenomegaly, enlargement and reddening of prescapular lymph nodes and disseminated serosal hemorrhages were found at necropsy.

Case III - AFIP #2 - (AFIP 1667519).

History: These tissues are from a 13-month-old female Saint Bernard with mild signs of cystitis, prolonged time to void urine and more frequent urination. The dog occasionally passed blood clots (2-week duration).

Laboratory Results: Urinalysis: 2+ for blood, 2-3 WBC/LPF
Culture negative
Radiography showed a mass at the bladder neck on pneumocystography.

Case IV - 80-214 - (AFIP 1761490).

History: 10-year-old mare - first pregnancy. Abortion at 9 months gestation. Mare had been vaccinated three times during pregnancy.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

HEMATOLOGY	4/9/80	5/3/80	5/12/80
RBC ($x10^6/\mu l$)	6.44	5.26	5.03
Hb (g/dl)	15.6	12.7	13.2
WBC (/µl)	10,134	35,700	33,855
Seg. neutrophils (%)	-	91	94
Nonseg. neutrophils (%)	-	6	1
Lymphocytes (%)	-	2	4
Monocytes (%)	-	0	0
Eosinophils (%)	-	1	0
Basophils (%)	-	0	1

SERUM	4/9/80	5/12/80
Fasting glucose (mg/dl)	106	117
BUN (mg/dl)	12	3
Creatinine (mg/dl)	.5	.6
Cholesterol (mg/dl)	590	730
Alkaline phosphatase (mU/ml)	413	840
SGPT/ALT (mU/ml)	735	207
SGOT/AST (mU/ml)	51	31
Total protein (gm/dl)	6.0	5.3
Albumin (gm/dl)	3.2	2.5
Na^+ (mEq/l)	155	153
K^+ (mEq/l)	4.1	2.3

URINALYSIS	4/9/80	5/12/80
Color	Clear	Clear
pH	6.0	6.0
Specific gravity	1.006	1.005
Protein	30+	trace
Ketones	neg.	neg.
Glucose	neg.	neg.
Bile pigments	neg.	neg.
Blood	neg.	neg.

PLASMA CORTISOL (ng/ml)	4/28/80
pre-ACTH	80
2 hr. post-ACTH	290

Results
AFIP Wednesday Slide Conference - No. 15
21 January 1981

Case I - 80-68 - (AFIP 1758395).

History: Tissue from a 5-month-old male red kangaroo (Megaleia rufa) which was ejected from its pouch when the mother was frightened by dogs and broke her leg. An attempt was made to hand rear the joey, but it died several months later.

Contributor's Diagnosis & Comments: Mycotic gastritis, compatible with candidiasis.

Comments: Heavy yeasts, determined to be Candida albicans, were present in the feces of the joey for several weeks prior to death. The animal was treated with mycostatin but apparently it was not effective. The joey also had moderate splenic and lymph node atrophy and moderate bronchopneumonia, but no evidence of any dissemination of the candidiasis. The difficulty of orphan-rearing and subsequent candidiasis has recently been reported in grey kangaroos.

Attendees' Diagnoses & Comments: 1) Gastritis, ulcerative, acute to subacute, diffuse, moderate to severe, nonglandular stomach, red kangaroo (Mygaleia rufa). 2) Lymphadenitis, subacute, diffuse, moderate, lymph node.

Etiologic Diagnosis: Mycotic gastritis.

Etiology: Candida albicans.

Comments: Other equally acceptable preconference morphologic diagnoses submitted were necropurulent gastritis, necrotizing gastritis, or simply mycotic gastritis. A few attendees thought the tissue represented esophagus and submitted a morphologic diagnosis of esophagitis, but the contributor was present and identified the tissue as nonglandular stomach of the kangaroo. Lymphadenitis was submitted only by a few of the participants as this tissue was not present in all sections. Characteristics of the lesion included the erosion and multifocal ulceration of the mucosa, the large numbers of fungal organisms, the cellular response of the host, and the abrupt ending of the lesion at the junction of the glandular and nonglandular stomach. Fungi were observed extending to the basal layers of the stratified squamous epithelium and occasionally into the submucosa. Microabscess formation and ulceration along the mucosa were observed by all. Characteristics of the fungi include the masses of fungal hyphae and numerous blastospores. The septate hyphal forms were uniform in diameter and had parallel walls and regular branching. Blastospores were small, round to oval, and often demonstrated budding. The cellular response was mixed within the submucosa and within the lamina propria and consisted of lymphocytes and neutrophils. An almost pure neutrophilic response occurred around fungal organisms in the mucosa. Changes in the lymph node included necrosis of the germinal centers, depletion of lymphoid follicles, and diffuse infiltration of the node by moderate numbers of PMN's. The appearance of the node was described by a few as reactive. There was some discussion of the pathogenesis of this lesion and several, possibly dependent, factors were proposed. These included a change in bacterial flora of the alimentary tract due to antibiotic administration, stress and immunosuppression, stasis of the stomach. Local factors such as IgA production and local immunity may account for the cessation of the lesions at the junction of the glandular and

nonglandular stomach. It was pointed out that the cow's milk formula given these orphaned animals which is dissimilar to that of kangaroo milk in galactose content may aid in the production of this disease as kangaroos lack enzymes to digest galactose. Caution was expressed that on gross examination of the stomach these fungal mycelia may be mistaken for milk curd.

Contributor: National Zoological Park, Washington, DC 20008.

Reference:

Obendorf, D. L.: Candidiasis in young hand-reared kangaroos. J. Wildlife Dis. 16: 135-140, 1980.

Case II - 80-10933 - (AFIP 1758391).

History: Lung and spleen from a 4-year-old bull. Three bulls died in a 3-day period in a pasture with 2 other bulls and 25 cows. All animals were found dead without previous signs of illness being observed.

Laboratory Results: Marked splenomegaly, enlargement and reddening of prescapular lymph nodes and disseminated serosal hemorrhages were found at necropsy.

Contributor's Diagnosis: Septicemia, anthrax. Bacillus anthracis was isolated from the tissues.

Attendees' Diagnoses & Comments:

Morphologic Diagnoses: 1) Splenitis, necrohemorrhagic, acute, diffuse, severe, bovine. 2) Hemorrhage, congestion and edema, acute, diffuse, severe, lung. 3) Pleuritis, fibrinohemorrhagic, acute, segmental, mild, pleura, lung.

Etiologic Diagnosis: Bacterial pneumonia and splenitis. Anthrax.

Etiology: Bacteria compatible with Bacillus anthracis.

Comments: Other diagnoses given by conference attendees were pneumonitis and pleuritis, lymphoid necrosis of the spleen, and congestion and hemorrhage of the lung and spleen. Some participants described a capsulitis associated with the spleen and vasculitis was listed by one. Characteristics of the lesions included pleural thickening and hemorrhage; fibrinous material within alveoli and adhering to pleural surfaces; and edema, congestion and hemorrhage in alveolar septae and spaces. A mild inflammatory response of small numbers of neutrophils accompanied these changes in the lung. Lesions in the spleen consisted of foci of necrosis, depletion and hemorrhage in the corpuscles and diffuse congestion/hemorrhage in the red pulp. Moderate numbers of PMN's accompanied these changes in the spleen. Numerous bacterial organisms observed in the lungs and spleen were characterized as long bacilli with square ends lined up in a "boxcar" fashion. A bacterial stain demonstrated these organisms to be Gram positive. The clinical signs and pathogenesis of the disease was discussed. It was felt that death which is almost always peracute in the bovine is attributed to a shock-like condition. The production of the shock is probably mediated by the release of a toxin most probably an exotoxin which produces edema, hemorrhage and necrosis. Three toxic factors have been identified in anthrax: a lethal factor, an edema factor, and a protective antigen (2). Another possibility may be neurotoxin production by the bacteria. Other features of the bacteria discussed included the aerobic vegetative phase which produces the toxins and the necessity of exposure to atmospheric air or

oxygen for spore production. Spores persist for decades in the environment and remain infective. Opening or necropsy of carcasses in which anthrax is suspected is strictly discouraged (illegal in some countries) because of sporulation that results. Although the vegetative bacilli in internal organs of unopened carcasses are destroyed in 2-3 days by putrefaction, the blood exuded from orifices is loaded with bacilli that sporulate.

Contributor: Department of Veterinary Science, South Dakota State University, Brookings, SD 57007.

References:

1. Jubb & Kennedy: Pathology of Domestic Animals, Vol. I, Academic Press, 1970, p. 374.

2. Robbins, S. L.: Pathologic Basis of Disease - Infectious Disease. Saunders & Co., Philadelphia, 1980, p. 474.

Case III - AFIP #2 - (AFIP 1667519).

History: These tissues are from a 13-month-old female Saint Bernard with mild signs of cystitis, prolonged time to void urine and more frequent urination. The dog occasionally passed blood clots (2-week duration).

Laboratory Results: Urinalysis: 2+ for blood, 2-3 WBC/LPF
Culture negative
Radiography showed a mass at the bladder neck on pneumocystography.

Contributor's Diagnosis & Comments: Embryonal botryoid rhabdomyosarcoma.

Comment: On surgical removal this tumor presented as grapelike clusters around the neck of the bladder and down the urethra. The dog was later euthanatized because of inability to function normally after surgery and no further evidences of the tumor were found in the cadaver.

Attendees' Diagnosis & Comments: Rhabdomyosarcoma, urinary bladder, Saint Bernard, canine.

Comments: Rhabdomyosarcoma was the preferred diagnosis by all the participants and several included descriptive terms such as botryoid, polypoid, and embryonal. Differential diagnoses given by some were leiomyosarcoma, and myxomatous fibrosarcoma. Features of the neoplasm discussed were the numerous polypoid and papilliferous transitional lined projections, the obvious spindle cell component in both dense and myxomatous (loose) arrangement, formation of interlacing bundles of these spindle cells, and the presence of multifocal congestion and hemorrhage associated with the tumor in the bladder wall. Some attendees reported observing ulceration and necrosis of the mucosa by neoplastic invasion. A few attendees reported observing mononuclear inflammatory cells associated with these foci. Cellular morphology of the neoplastic cells discussed were the pleomorphism of the cell nuclei, the rare appearance of nucleoli, the variation in mitotic rate (0-4/hpf) within different areas of the tumor, the eosinophilic fibrillar cytoplasm some of which contained cross striation or so-called "strap cells" and paranuclear

vacuoles present in some cells. The discussion then turned to the possible tissue cell of origin for this tumor. It was noted that a large number of these tumors occur at the neck of the bladder in dogs. Multipotential mesodermal cells of Wolfian duct remnants in the female and Mullarian duct remnants in the male were speculated as the tissue of origin. There is an apparent breed and sex predilection for this tumor; it occurs most often in Saint Bernards and more often in females. Metastasis is rare and has been associated with multiple surgeries. In humans the tumor occurs most commonly in males less than 1-1/2 years of age. In dogs this tumor most often is reported in animals less than 2 years of age. Another feature pointed out was the apparent predilection for the urinary bladder. The reason the embroyonic remnants proliferate may be associated with transitional epithelium of the bladder as urothelium stimulates fibrous connective tissue proliferation when transplanted to sites of bone fracture. The association with hypertrophic osteoarthropathy and this tumor was discussed. The specific cause is not known but may be associated with neurogenic pathways as this seems to be the case with the thoracic pulmonary space occupying lesions via the vagal nerve (1). Direct simulation via a factor produced by the transitional epithelium of the bladder also was speculated as a possible cause.

Contributor: Department of Veterinary Microbiology and Patnology, Wasnington State University, Pullman, WA 99163.

References:
1. Halliwell, W. H. and Ackerman, N.: Botryoid rhabdomyosarcoma of the urinary bladder and hypertrophic osteoarthropathy in a young dog. JAVMA 165: 911-913, 1974.
2. Roszel, J. F.: Cytology of urine from dogs with botryoid sarcoma of the bladder. Acta Cytol. 16: 443-446, 1972.
3. Stamps, P. et al.: Botryoid rhabdomyosarcoma of the urinary bladder of dog. JAVMA 153: 1064-1068, 1968.

Case IV - 80-214 - (AFIP 1761490).

History: 10-year-old mare - first pregnancy. Abortion at 9 months gestation. Mare had been vaccinated three times during pregnancy.

Contributor's Diagnosis & Comments: Abortion, equine viral rhinopneumonitis.
Comments: Focal necrosis in the liver, lung and adrenal with areas of mineralization. Intranuclear eosinophilic inclusion bodies are present, particularly in bronchial epithelium. Equine herpes virus isolated from tissues.

Attendees' Diagnoses & Comments:
Morphologic Diagnosis: 1) Pneumonia, interstitial, acute to subacute, with multifocal necrosis, and numerous intranuclear eosinophilic inclusion bodies, lung, foal, equine. 2) Hepatitis, necrotizing, acute, multifocal, moderate, liver. 3) Necrosis and hemorrhage, with mineralization, multifocal, severe, adrenal gland.
Etiologic Diagnosis: Viral pneumonia, hepatitis, and adrenalitis.
Etiology: Compatible with Equine Herpes Virus-1 Infection.
Comments: There was general agreement with the contributor's diagnoses and comments. The morphologic diagnoses listed were reached after discussion of the

lesions and further consultation. Other equally acceptable morpnologic diagnoses given by participants prior to the conference included necrotizing pneumonitis, multifocal hepatocellular necrosis, and necrotizing adrenalitis. Some participants also listed an acute to subacute pleuritis, and RE cell hyperplasia within the liver. Features of the lesions in the lung were characterized as diffuse moderate to severe congestion of the pulmonary parenchyma, the consolidated appearance (considered almost normal for fetal lung), diffuse edema of the alveolar septa, cellular infiltrates consisting of PMN's and macrophages within septa and alveolar spaces, necrosis of alveolar and small bronchiolar lining cells, and the presence of large number of eosinophilic intranuclear inclusion bodies in several cell types. Lesions in the liver consisted of multifocal necrotic foci with a random distribution, infiltration of primarily neutrophils into these foci and the presence of occasional eosinophilic intranuclear inclusion bodies at the periphery of these lesions. Lesions in the adrenal are described as multifocal hemorrhage and necrosis with mineralization. There was complete lack of an inflammatory response within this tissue. Eosinophilic intranuclear inclusions were not obvious and were mentioned by only a few participants. Several participants mentioned foci of hypercellularity within the parenchyma of the liver with cells described as myeloid precursors. These foci were interpreted as foci of extramedullary hematopoiesis which is considered normal for fetal liver. The pathogenesis of the lesions in the fetal equine was discussed. Viral infection of the fetus is considered to be via infection of circulating fetal lymphocytes which cross the placental barrier and gain access to the maternal circulation and return to the fetus. Fetal tissues are then infected with the virus through infected fetal lymphocytes. The ability of the foal to mount an inflammatory response is present in the last half of the pregnancy and thus explains the apparent inflammatory infiltrate in the affected tissues in this case. The specific cause of abortion may be mediated through elevated fetal cortisol levels and breakdown of placental attachments with subsequent expulsion of the fetus or premature birth. Foals born live are weak and usually die in 48 hours. The virus most commonly isolated is Equine Herpes Virus 1. EHV 2 is not reported to cause abortion, and EHV 3 is not isolated commonly in abortions but can cause abortion. EHV 1 is known to produce respiratory disease and/or abortion in horses (3). Vaccination of the mare with equine rhinopneumonitis vaccine (a fact omitted from the history) was the suspected route of infection. Older EHV 1 vaccines were derived from hamster cell line and have been reported to be abortigenic (1,2).

Contributor: B.C.M.A., Veterinary Laboratory, Box 100, Abbotsford, B.C., Canada, V2S 4N8.

References:

1. Eaglesome, M. D., et al.: Equine Herpesvirus 1 Infection in mares vaccinated with a live-virus rhinopneumonitis vaccine, attenuated in cell culture. Can. Vet. J. 20: 145-147, 1979.

2. Osborne, B. I. et al.: Response of fetal adrenal cortex to congenital infection. Am. J. OB/GYN 114: 622-627, 1972.

3. Purdy, C. W. et al.: Equine rhinopneumonitis vaccine - Immunogenicity and safety in adult horses, including pregnant mares. Am. J. Vet. Res. 39: 377-383, 1978.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Histories
AFIP Wednesday Slide Conference - No. 16
28 January 1981

Case I - 80-1038 (1758463).

History: A 6-year-old female poodle was presented 10/17/79 with nystagmus, difficulty with ambulation, and a rectal temperature of 104.2°F. She was treated repeatedly with gentamycin and dexamethasone with no improvement. A head-tilt was noted 11/14/79. She was referred to a University Veterinary School for evaluation. Slight narrowing of cervical vertebrae 2-3 and 3-4, with no evidence of herniation, was demonstrated radiologically. No other abnormalities could be found. Disc fenestration was performed from C_{2-3} to C_{5-6}. Ten days later, after an apparent normal recovery from surgery, the dog died suddenly. Rabies and "DHL" vaccinations had been administered routinely in March 1979.

Case II - 0065 (AFIP 1758834).

History: Ulcerative lesion measuring 2 cm in diameter removed from the anterior portion of front leg of 8-year-old male cat. Origin of lesion is unknown but thought to be due to a bite wound from another cat.

Case III - A28109 (AFIP 1666535).

History: Tissue from a rat killed and necropsied 14 days after receiving a single intraperitoneal dose of an experimental compound.

Case IV - AT-1367-79 (1758462).

History: Tissue from an 11-week-old turkey with history of recent ill-defined respiratory disease in flock of 5,000.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Results
AFIP Wednesday Slide Conference - No. 16
28 January 1981

Case I - 80-1038 (1758463).

History: A 6-year-old female poodle was presented 10/17/79 with nystagmus, difficulty with ambulation, and a rectal temperature of 104.2°F. She was treated repeatedly with gentamycin and dexamethasone with no improvement. A head-tilt was noted 11/14/79. She was referred to a University Veterinary School for evaluation. Slight narrowing of cervical vertebrae 2-3 and 3-4, with no evidence of herniation, was demonstrated radiologically. No other abnormalities could be found. Disc fenestration was performed from C_{2-3} to C_{5-6}. Ten days later, after an apparent normal recovery from surgery, the dog died suddenly. Rabies and "DHL" vaccinations had been administered routinely in March 1979.

Contributor's Diagnosis & Comments: Granulomatous meningoencephalitis.

Comment: Fixed brain tissue and other visceral tissues were submitted. Lesions were limited to the brain. All slides may not demonstrate representative lesions of meningitis. Special stains (PAS, Gram) failed to demonstrate an etiologic agent. Although the spinal cord was not available for examination, the brain lesions are compatible with the described entity---canine granulomatous meningoencephalomyelitis.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Meningoencephalitis, granulomatous, multifocal, moderate, cerebral cortex and midbrain, brain, poodle, canine.

Etiologic Diagnosis: Canine granulomatous meningoencephalomyelitis syndrome.

Etiology: Unknown.

Comments: The morphologic diagnosis was the unanimous concensus of the attendees. The preconference etiologic diagnoses given by participants encompassed canine granulomatous encephalomyelitis and primary reticulosis of the central nervous system. The features of the lesions consisted of multifocal granulomatous lesions located within the white matter of the cortex and to some extent the gray matter, the internal capsule, and hippocampus depending on the section. Meningeal involvement was evident in some sections. Perivascular distribution was prominent in many of the lesions. For the most part the lesions were not accompanied by changes in the adjacent neuropil but some attendees reported foci of rarefaction and gliosis in addition to the granulomatous meningoencephalitis. Cell types involved in these lesions included macrophages/histiocytes, lymphocytes, plasma cells, and occasionally neutrophils. The macrophagic/histiocytic cells were described as blastic appearing by a few participants, and mitotic figures were reported by a few. A reticulum stain was projected that demonstrated discontinuous silver positive reticulum fibers around vessels and dissecting between the cells in these foci. The discussion then turned to the etiologic diagnosis of the lesions present. Designation of lesions as "canine granulomatous meningoencephalomyelitis" denotes an inflammatory nature of the condition whereas primary reticulosis infers a neoplastic involvement. Some attendees who listed granulomatous encephalitis suggested a viral etiology, but viral particles have not been reported with this entity. Special stains by the contributor and AFIP for fungal, bacterial, and protozoal organisms were negative. It was pointed out that these two entities may

in fact represent the same condition but in different stages. The infiltration of these inflammatory/neoplastic cells is considered to be from the circulating mononuclear phagocyte system. Histogenesis studies of the brain have shown that macrophages and possibly microglial cells arise from this same system. Microglial cells do not appear in the brain until after vascular penetration occurs, and autoradiography studies have shown that macrophages migrate to perivascular spaces in the brain. This disease in dogs has been compared to the condition in man termed reticulum cell sarcoma of the CNS which also is a disease without a known etiologic agent. Correlation between these two conditions has not been established. This disease condition in dogs appears to involve two age groups: young dogs 1-2 years of age and dogs over 6 years of age. Corticosteroids are reported to alleviate signs in affected animals but death is inevitable. Clinically, signs must be differentiated from other diseases involving the CNS including other inflammatory and neoplastic conditions.

Contributor: Veterinary Diagnostic & Investigational Laboratory, College of Veterinary Medicine, P. O. Box 1389, Tifton, GA 31794.

References:

1. Brand, K. G. et al.: Granulomatous meningoencephalomyelitis in six dogs. JAVMA 172: 1195-1200, 1978.
2. Cordy, D. R.: Canine granulomatous meningoencephalomyelitis. Vet. Pathol. 16: 325-333, 1979.
3. Russo, M. E.: Primary reticulosis of the central nervous system in dogs. JAVMA 174: 492-500, 1979.
4. van Furth, R. et al.: The mononuclear phagocyte system: A new classification of macrophages, monocytes, and their precursor cells. Bull Wld Hlth Org. 46: 845-851, 1972.

Case II - 0065 (AFIP 1758834).

History: Ulcerative lesion measuring 2 cm in diameter removed from the anterior portion of front leg of 8-year-old male cat. Origin of lesion is unknown but thought to be due to a bite wound from another cat.

Contributor's Diagnosis & Comments: Diffuse pyogranulomatous dermatitis.

Comments: Organisms found in macrophages are morphologically compatible with Trichosporon sp. Futher evidence for the identification of the organisms as Trichosporon sp. was obtained from the Center for Disease Control by means of fluorescent antibody techniques using specifically labelled antiserum applied to deparaffined tissue sections.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Dermatitis, pyogranulomatous, chronic, focally extensive, moderate, subcutis and skin, cat, feline.

Etiologic Diagnosis: Mycotic dermatitis.

Etiology: Undetermined by attendees (see contributor's comments).

Comments: The majority of the attendees listed pyogranulomatous dermatitis as their preconference morphologic diagnosis. Some preferred a granulomatous dermatitis as only a few neutrophils were present in their sections. There was

general agreement with the etiologic diagnosis as a mycotic dermatitis. The etiologic diagnosis given by many was sporotrichosis with differentials that included phycomycosis, helminthosporosis, chromomycosis and phaeomycosis. Protozoal dermatitis also was mentioned as a differential diagnosis. Features of the lesion included breakdown of collagen in the superficial dermis and infiltration of the deep dermis and subcutaneous tissue with a mixed cell population of histiocytic cells, lymphocytes, plasma cells and depending on the section large or small numbers of neutrophils. In addition to the infiltrate, necrotic cellular debris also was present. Features of the intra and extracellular cigar shaped organisms included budding yeast forms, chains of organisms, and forms suggestive of hyphae. A GMS and PAS substantiated these findings. Some attendees observed fine filamentous processes at the ends of the organisms and thus the possible confusion with protozoal organisms. A few participants and other consultants within the Institute did not feel the organisms were characteristic of sporotrichosis. Features inconsistent with sporotrichosis included the large number of organisms present, chains of organisms, cigar shaped forms which were too long for sporotrichosis, and hyphae with apparent branching. Sporotrichosis more closely resembles histoplasmosis in H&E tissue sections, and the presence of large numbers of organisms is uncommon in sporotrichosis. In the discussion of the immunologic findings in this case, several attendees wondered if cross reactivity with other fungal organisms occurs. Caution was expressed that diagnosis cannot be based purely on the morphologic appearance in tissue section. Clinical pathology also is important. This case is considered unique in that deep dermal involvement of Trichosporon has not been documented. One case of disseminated Trichosporon mycosis was alluded to in one reference (2). Trichosporon is reported to involve exclusively the epidermis and hair shafts of man, horses, reptiles and a spider monkey (1,2). Trichosporon beigelli is the causative agent of white piedra in man. These lesions are restricted to the hair shafts and resemble parasitic nits grossly. T. cutaneum involves both the epidermis and hair shafts in man. The agent identified in the single case in the monkey was T. cutaneum. The organism reported in reptiles was T. species (3). The organism described in horses was designated as T. equinum.

Contributor: Veterinary Diagnostic Center, University of Nebraska, Lincoln, Nebraska 68583.

References:

1. Emmons, C. W. et al., Editors: Black Piedra, white piedra and trichomycosis axillaris. IN Medical Mycology, Lea & Febiger, Philadelphia, 1977, pp 181-184.

2. Kaplan, W.: Piedra in lower animals: A case report of white piedra in a monkey and a review of the literature. JAVMA, 113-117, Feb 1959.

3. Kuttin, E. S. et al.: Mycoses in Crocodiles. Grosse Verlag, 1978, pp 39-48.

Case III - A28109 (AFIP 1666535).

History: Tissue from a rat killed and necropsied 14 days after receiving a single intraperitoneal dose of an experimental compound.

Contributor's Diagnosis & Comments: Multifocal granulomatous nephritis associated with birefringent crystalline material.

Comments: It appears that the experimental compound or a metabolite has precepitated within the renal tubules and elicited a response similar to that of a foreign body. Although precipitation within the renal tubules is not uncommon, the granulomatous reaction is, in our experience, unusual.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Nephritis, granulomatous, multifocal, moderate to severe, kidney, rat, rodent.

Etiologic Diagnosis: Chronic toxic nephritis.

Etiology: Undetermined.

Comments: Other preconference morphologic diagnoses given by some participants were necrogranulomatous nephritis and tubulo-interstitial nephritis. The diagnosis listed was considered to encompass all of the changes present in the tissue and was not restricted to just tubular and interstitial involvement. Changes included multiple foci of granulomatous inflammation in the cortex and medulla, interstitial and tubular hemorrhage primarily in the medulla, and minimal inflammatory changes in the pelvis in some sections. Granulomatous inflammation involved foci of tubules and interstitium in the cortex and medulla and occasional glomeruli. A mild subacute infiltrate also was present in the pelvis. Some attendees reported hyperplastic regenerative changes scattered in cortical tubules. Another feature reported by some was the presence of casts and mineralized material in lumens of cortical and medullary tubules. The inflammatory foci were described as singular and coalescing infiltrates of macrophages, lymphocytes, neutrophils, plasma cells, and occasional multinucleated foreign body type giant cells. The center of many of these foci contained birefringent crystalline material. This material appeared as faint greenish structures in nonpolarized light. The polarized material was suggestive of calcium oxylate crystals. The etiologic agent(s) speculated by most participants was the experimental compound and differentially included various antibiotic compounds and heavy metals. The exact nature of the compound was not disclosed by the contributor. Histochemical analyses included microincineration and special stains for urates and calcium. The results suggested a mixture of inorganic and organic deposits in the tissue with a strong positivity for calcium complexes and a weak reaction for urates. The disappearance of birefringent material upon microincineration indicates the presence of organic deposits. Further discussion included the pathogenesis of the lesions. It was speculated that damage to the tubules of nephron could have resulted in the deposition of insoluble metabolites of the compound or products of cellular degradation in the interstitium with subsequent granulomatous response. It was surprising that only selected components of the cortex and medulla were affected and not the entire kidney as is expected with agents causing toxic tubular nephrosis. Some attendees felt the glomerular involvement was due to extension from primary tubular lesions. A suggestion of immune complex disease also was entertained by the contributor (personal communication), due to breakdown of basement membranes with exposure of antigenic substances and subsequent inflammatory response.

Contributor: Pfizer Inc. Central Research, Groton, CN 06340.

Case IV - AT-1367-79 (1758462).

History: Tissue from an 11-week-old turkey with history of recent ill-defined respiratory disease in flock of 5,000.

Laboratory Results: Electron microscopy of organisms on epithelial surface revealed morphology and attachment zones consistent with Cryptosporidia sp.

Contributor's Diagnosis & Comments: Respiratory cryptosporidiosis.

Comments: Tracheitis present is characterized by chronic inflammatory thickening of the lamina propria, mucosal epithelial proliferative change and deciliation, moderate glandular attenuation and the presence of epithelial surfaces of large numbers of protozoan organisms consistent morphologically with Cryptosporidia sp.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Tracheitis, subacute and proliferative, diffuse, moderate, trachea, turkey.

Etiologic Diagnosis: Respiratory cryptosporidiosis.

Etiology: Cryptosporidia sp.

Comments: There was general agreement with the contributor's diagnosis and comments. Histopathological features commented on were the diffuse thickening of the tracheal mucosa, the infiltration of the lamina propria with inflammatory cells, the formation of cystic structures lined by tracheal epithelium, and the presence of numerous small round organisms along the ciliated borders of the epithelium and free in cystic spaces. The inflammatory cells consisted of a mixed population of lymphocytes, plasma cells and heterophils. Features of the organisms in H&E sections were variability in size and shape and vacuolation of some forms with dense granular bodies to one side of the vacuole. Parasitophorus vacuoles also were present.

The entire life cycle of Cryptosporidium sp. is unknown. Due to its small size (2-5 microns) it is often difficult to differentiate the various stages in the life cycle. The following is a compendium of the most probable life cycle (note: we know Cryptosporidium is a coccidian so we should compare what we see with what we know to be true in other coccidian life cycles, i.e., Eimeria, Isospora). The mode of infection is unknown but it is believed due to ingestion (or possibly inhalation) of sporulated oocysts. Sporozoites probably escape from the oocyst and then infect epithelial cells. There is difference of opinion as to whether the trophozoites (the infecting sporozoite) are intra or extracellular. Electron micrographs show four layers (i.e., membranes) surrounding the parasite and thus many feel this is indicative of two inner membranes of the protozoa and the outer membranes of the epithelial cell. It is thought the sporozoite burrows right under the epithelial cell membrane and thus is intracellular. The subsequent stages of the protozoa are similar to that seen with other coccidia: Trophozoites give rise to schizonts (with eight merozoites due to three cell divisions), these rupture and give rise to other merozoites or gametes. Oocysts have been reported by a few workers. Micrographs are not consistent with that of oocysts, however, and thus they may be schizonts, either intracellular or passing in feces due to disruption of epithelial cells. It is suggested, however, that oocysts do form, are passed out in the feces, sporulate, and are then infective to other hosts.

Cryptosporidiosis has been reported in a variety of species including reptiles, turkeys, geese, mice, rabbits, guinea pigs, lambs, calves, swine, monkeys, and man (4). Diseases are not always associated with these protozoa. They inhabit the intestinal tract in most of these species.

Contributor: Animal Disease Diagnostic Laboratory, Purdue University, West Lafayette, IN 47907.

References:

1. Bird, R. G. & Smith, M. D.: Cryptosporidiosis in man: Parasite life cycle and fine structural pathology. J. Path. 132: 217-233, 1980.

2. Fletcher, O. J. et al.: Cryptosporidiosis of the Bursa of Faoricius of chickens. Avian Dis. 19: 630-639, 1975.

3. Hoerr, F. J. et al.: Respiratory cryptosporidiosis in turkeys. JAVMA 173: 1591-1593.

4. Pohlenz, J. et al.: Bovine cryptosporidiosis: A transmission and scanning electron microscopic study of some stages in the life cycle and of the host-parasite relationship. Vet. Pathol. 15: 417-427, 1978.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Histories
AFIP Wednesday Slide Conference - No. 17
4 February 1981

Case I - OL00201 (AFIP 1758400)

History: A 15-month-old female BHE rat was not using her hind legs for about a week, but was alert and eating. The animal was killed. A very firm, gray-pink protrusion, 2.0 X 0.5 X 0.5 cm, firmly attached to the dorsal area of the base of the tail was observed. The ventral lumbosacral spinal area was diffusely enlarged, tan-gray and moderately firm. The tan-gray tissue extended into the adjacent muscles. The right ovary was 4.0 cm in diameter, cream-pink and very firm. Similar tissue, 2.0 cm in diameter, was embedded in the caudal pole of the right kidney, and round masses, 0.3 - 1.0 cm in diameter, were in the lungs.

Case II - PA-089-13-80, PA-089-21-80 (AFIP 1757379).

History: A 9-week-old male mixbreed pup died after a three day course of lethargy, depression, anorhexia, nasal discharge and some vomition. There was a transient response to supportive therapy.

Laboratory Results:
CBC - 4/28/80 - at onset of initial signs
WBC - 10,800 PCV - 31%
PMN - 60%
Band - 2%
Lymph - 11%
Eos. - 27%

CBC - 5/1/80 - prior to death
WBC - 220
PMN - 24%
Band - 1%
Lymph - 25%
Eos. - 50%

Gross Necropsy Lesions: Segmental enteritis, small bowel, characterized by turgid swollen loops, and patchy areas of serosal reddening.

Case III - 2 (AFIP 1757295).

History: Tissue from a wild caught rabbit with a subcutaneous tissue mass in the inguinal region.

CASE IV - CP77-1159 (AFIP 1673404.
HISTORY AND LABORATORY DATA:

8-25-77 - A 6 month old female Irish Setter was presented to a practicing veterinarian with chief complaints of lethargy and anorexia.

Rectal temperature - 100°F
PCV - 20%
Fecal - hookworm ova

The animal was treated with dinitrophenyl (DNP), ampicillin, & a vitamin supplement.

9-7-77 - The animal was returned because she was depressed, vomiting and anorectic. Auscultation of the thoracic cavity revealed pulmonary congestion.

Temperature - 104°F
SMA 12 - normal
PCV - 30%
WBC - 25,000

Ampicillin treatment continued.

9-14-77 - Temperature - 106°F. Animal was provided fluid therapy and Gentamicin (4 mg/kg b.i.d. first day; 4 mg/kg daily thereafter).

9-22-77 - Temperature dropped to 101°F but no improvement in general condition of animal was noted. Animal was given a blood transfusion and Cepholosporin.

9-23-77 - Animal was found dead and necropsied by practicing veterinarian, who reported white plaques on parietal and visceral pleural surfaces.

DIAGNOSIS:

Tentative clinical DX: Systemic mycotic infection

TIMOTHY P. O'NEILL
Capt., USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Results
AFIP Wednesday Slide Conference No. 17
4 Feburary 1981

Case I - 0L00201 (AFIP 1758400)

History: A 15-month-old female BHE rat was not using her hind legs for about a week, but was alert and eating. The animal was killed. A very firm, gray-pink protrusion, 2.0 X 0.5 X 0.5 cm, firmly attached to the dorsal area of the base of the tail was observed. The ventral lumbosacral spinal area was diffusely enlarged, tan-gray and moderately firm. The tan-gray tissue extended into the adjacent muscles. The right ovary was 4.0 cm in diameter, cream-pink and very firm. Similar tissue, 2.0 cm in diameter, was embedded in the caudal pole of the right kidney, and round masses, 0.3 - 1.0 cm in diameter, were in the lungs.

Contributor's Diagnosis & Comments: Osteosarcoma.
Comments: The submitted sections are from the mass of the right ovary. Normal right ovarian tissue could hardly be found in the sections. All changes found at necropsy were osteosarcoma. The primary site of the neoplasm was most likely in the vertebrae of the lumbar spinal column.

Attendees' Diagnosis & Comments: Osteosarcoma.
Comments: There was unanimous agreement with the contributor's diagnosis. Osteosarcomas are considered rare in the rat. Experimentally these tumors have been produced by certain chemicals, radiation, and by mechanical means (1). Osteosarcomas have been induced by inoculation of rats with the Maloney sarcoma virus. This virus is antigenically indistinguishable from Harvey's sarcoma virus of rats. The Maloney sarcoma virus also is referred to as the Maloney murine leukemia virus or murine sarcoma virus. Tumor production with this virus is rapid and occurs in approximately 80% of inoculated rats. Other tumors reported in rats inoculated with this virus are plasmacytomas and fibrosarcomas (4). This same virus also has produced fibrosarcomas in newborn hamsters and rhabdomyosarcomas in mice. Osteosarcomas in the rat have been studied as an animal model of the human tumor, as there is similar distribution in both species (1,3).

Contributor: Searle Laboratories, Chicago, Illinois.

References:

1. Litvinov, N. N. et al.: Tumors of the bone. In Pathology of Tumors in Laboratory Animals, Vol. I, Part I, IARC Scientific Publication No. 6, 1976, pp 169-175.
2. Misdorp, W.: Skeletal osteosarcoma. Model No. 198. In Handbook: Animal Models of Human Disease, Fascicle 9, Registry of Comp. Path., AFIP, 1980.
3. Olson, H. M. et al.: Osteosarcoma. Model No. 114. In Handbok: Animal Models of Human Disease, Fascicle 6, Registry of Comp. Path., AFIP, 1977.
4. Squire, R. A. et al.: Tumors. In Pathology of Laboratory Animals, Ed. by Benirschke, K. et al. Vol. II, Springer-Verlag, New York, 1978. pp 1249-1252.

Case II - PA-089-13-80, PA-089-21-80 (AFIP 1757374).

History: A 9-week-old male mixbreed pup died after a three day course of lethargy, depression, anorhexia, nasal discharge and some vomition. There was a transient response to supportive therapy.

Laboratory Results:
CBC - 4/28/80 - at onset of initial signs
WBC - 10,800 PCV - 31%
PMN - 60%
Band - 2%
Lymph - 11%
Eos. - 27%

CBC - 5/1/80 - prior to death
WBC - 220
PMN - 24%
Band - 1%
Lymph - 25%
Eos. - 50%

Gross Necropsy Lesions: Segmental enteritis, small bowel, characterized by turgid swollen loops, and patchy areas of serosal reddening.

Contributor's Diagnoses & Comments: 1) Enteritis, characterized by epithelial cell necrosis and sloughing, crypt dilation and plugging and lymphoid depletion (some sections), subacute, diffuse, severe, small intestine, canine. 2) Bone marrow hypoplasia, myeloid and erythroid, diffuse, severe, femur, canine.

Comments: The gross and microscopic findings are characteristic of canine parvovirus infection. Such lesions are similar to those of feline panleukopenia, including a relative increase in numbers of eosinophils in the otherwise granulocyte depleted bone marrow.

Attendees' Diagnoses & Comments:

Morphologic Diagnoses: 1) Enteritis, necrotizing with villous atrophy, blunting and fusion, subacute, multifocal to diffuse, moderate to severe, small intestine, mixbreed, canine. 2) Lymphoid depletion, diffuse, moderate, Peyer's patches, small intestine. 3) Depletion, erythroid and myeloid, diffuse, severe, bone marrow, long bone.

Etiologic Diagnosis: Viral enteritis.

Etiology: Canine parvovirus.

Comments: There was unanimous agreement with the contributor's diagnoses. Attendees agreed with the contributor's comparison of these histopathological lesions to those seen in feline panleukopenia. Several participants mentioned that whole body radiation can produce essentially similar changes in the intestinal mucosa. Participants preferred bone marrow depletion to hypoplasia based on the assumption that normal bone marrow cellularity was present at one time versus a developmental hypocellularity. The two forms of canine parvovirus infection were discussed, namely the myocardial and enteric forms. The myocardial form is reported to occur in dogs less than 8 weeks of age corresponding to the developing myocardium. The intestinal form occurs in all age groups of dogs and varies from inapparent to fulminating hemorrhagic enteritis. Histories of dogs with canine

parvovirus infection often include recent boarding, kennel exposure, or association with other infected animals. Many reports describing the clinical and pathological findings of canine parvovirus infection have been generated in the past few years.

Contributor: University of Pittsburgh, Central Animal Facility, Pittsburgh, PA 15261.

References:

1. Carpenter, J. L. et al.: Intestinal and cardiopulmonary forms of parvovirus infection in a litter of pups. JAVMA 176: 1269-1273, 1980.
2. Pletcher, J. M. et al.: Histopathological evidence for parvovirus infection in dogs. JAVMA 175: 825-828, 1979.
3. Woods, C. B. et al.: Canine parvovirus enteritis. JAAHA 16: 171-179, 1980.

Case III - 2 (AFIP 1757295).

History: Tissue from a wild caught rabbit with a subcutaneous tissue mass in the inguinal region.

Contributor's Diagnosis: Subcutaneous coenurosis (Coenurus seralis) of Multiceps seralis.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: 1) Coenurus, subcutis, rabbit. 2) Cellulitis, granulomatous, focally extensive, moderate, subcutis, skin. 3) Panniculitis, granulomatous, focally extensive, skin.

Etiologic Diagnosis: Coenuriosis.

Etiology: Coenurus species probably Coenurus serialis.

Comments: The parasite has a tegument for a limiting body wall. The tegument has microvilli to increase surface area for absorption of nutrients. The body is filled with a loose parenchyma and has no coelom or pseudocoelom. The parasite is thus identified as a platyhelminth. The two major groups of parasitic platyhelminths are trematodes and cestodes. This parasite can be differentiated from trematodes since it contains no digestive tract but does contain calcareous corpuscles embedded in the parenchyma (most numerous in the neck region). The parasite is thus a larval cestode. Present are numerous scoleces invaginating from a bladder; these are characteristics of a coenurus. Each scolex has four suckers and a rostellum with numerous hooks. The scoleces are attached to the bladder by a neck. Specific identification of a coenurus is not possible on histological section. There are about 12 species of Multiceps or coenurus reported in the literature. Although hydatid cysts have cyst walls and numerous scoleces, they can be differentiated from coenuri. Hydatid cysts have much smaller scoleces. Unilocular hydatid cysts have very thick laminated walls. Multilocular hydatid cysts have very thin walls. These scoleces are often grouped in daughter cysts inside the larger parent cyst and thus do not morphologically resemble a coenurus. Coenuri are identical to cysticerci in all ways except the multiple number of scoleces. It is often wise to step section through bladders and count the number of scolices present. If only one is present it is cysticercus; more than one a coenurus.

Contributor: Syntex Research Laboratories, 3401 Hillview Ave., Palo Alto, CA 94304.

CASE IV - CP77-1159 (AFIP 1673404).

HISTORY AND LABORATORY DATA:

8-25-77 - A 6 month old female Iris Setter was presented to a practicing veterinarian with chief complaints of letharqy and anorexia.

Rectal temperature	- 100°F
PCV	- 20%
Fecal	- hookworm

The animal was treated with disophenyl (DNP), ampicillin, and a vitamin supplement.

9-7-77 - The animal was returned because she was depressed, vomiting and anorectic. Auscultation of the thoracic cavity revealed pulmonary congestion.

Temperature	- 104°F
SMA 12	- normal
PCV	- 30%
WBC	- 25,000

Ampicillin treatment continued.

9-14-77 - Temperature - 106°F. Animal was provided fluid therapy and Gentamicin (4 mg/kg b.i.d. first day; 4 mg/kg daily thereafter).

9-22-77 - Temperature dropped to 101°F but no improvement in general condition of animal was noted. Animal was given a blood transfusion and Cepholosporin.

9-23-77 - Animal was found dead and necropsied by practicing veterinarian, who reported white plaques on parietal and visceral pleural surfaces.

DIAGNOSIS:

Tentative clinical DX: Systemic mycotic infection

Contributor's Diagnoses & Comments: Kidney, acute tubular necrosis with regeneration. Kidney, mild nephrosclerosis. Kidney, metastatic calcification.

Comments: Etiologic Diagnosis: Nephrotoxicity due to gentamicin toxicity. Metastatic calcification was present in the heart, lungs, kidney, and pleura. The morphologic changes in this case suggest that the animal died in a uremic state resulting from renal failure. The renal lesions are nonspecific and occur as a result of a variety of nephrotoxic agents. The renal lesions are both acute and chronic. The tubular regeneration and immature interstitial fibrosis are consistent with the 14-day duration of gentamicin therapy. Although there are no laboratory data to confirm our suspicion, we suggest that anemia and/or dehydration may have been severe enough to produce inadequate renal perfusion, thus imparing normal renal metabolic processes and excretion of gentamicin. This may have allowed normal therapeutic doses of the antibiotic to accumulate to toxic levels, culminating in tubular necrosis and uremia and death.

Attendees' Diagnoses & Comments:

Morphologic Diagnoses: 1) Necrosis, tubular, acute, diffuse, severe, with severe multifocal dystropic mineralization, kidney, Irish setter, canine. 2) Mineralization, metastatic, multifocal, moderate, basement membranes, kidney. 3) Fibrosis, interstitial, chronic, multifocal, mild to moderate, kidney.

Etiologic Diagnosis: Nephrotoxicosis (toxic nephrosis).

Etiology: Gentamicin and cephalosporin nephrotoxicity.

Comment: There was general agreement with contributor's diagnoses and comments. Aminoglycoside antibiotics most notably gentamicin are known nephrotoxins. Cephalosporins also are nephrotoxic. A cumulative toxic effect of these two antibiotics was suspected by many of the attendees. Cells initially affected in the nephron are the proximal tubular cells. Lesions also occur in the collecting ducts and parts of the distal tubules.

Contributor: Division of Comparative Medicine, Department of Pathology, Southwestern Medical School, Dallas, TX 75235.

References:

1. Appel, G. B. et al.: The nephrotoxicity of antimicrobial agents. New Eng. J. Med. 296: No. 12, 663-670, 722-728, 784-787, 1977.

2. Houghton, D. C. et al.: Gentamicin and tobramycin nephrotoxicity. Amer. J. Pathol. 93: No. 1, 137-146, 1978.

3. Spangler, W. L. et al.: Gentamicin nephrotoxicity in the dog: Sequential light and electron microscopy. Vet. Pathol. 17: 206-217, 1980.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Histories
AFIP Wednesday Slide Conference - No. 18
11 February 1981

Case I - C-80-02 (2 slides) (AFIP 1757377).

History: This male Sprague-Dawley rat was one of a group of animals on an acute toxicity study killed on the second day of the study.

Laboratory Results: Blood urea nitrogen: 350 mg/dl
Glucose (fasting): 1600 mg/dl

Case II - Dog-Case 1 (AFIP 1757415).

History: 2-year-old male Doberman Pinscher --anorexia, emesis, obstipation for 1 week. Exploratory laparotomy revealed a dark red, flaccid ileum with dark red contents. An intussusception of the terminal ileum was easily reduced. The dog did not improve and was euthanized one week later.

Case III - 80-902 (3) (AFIP 1757303).

History: Three week old Holstein calf treated for 1 week for pneumonia and enteritis.

Laboratory Results: Cranial-ventral portions of the lung were dark red and of increased consistency. Multiple small grey foci were scattered throughout the lung.

Case IV - 77/106 (AFIP 1757044).

History: The tissue is from a 14 week old German shepherd dog. On April 4th the animal was presented with vomiting and an elevated temperature. This responded to Penicillin and Streptomylin therapy. On April 9th the dog showed a sudden deterioration. The temperature was elevated, the hind limbs and tail showed partial paresis. The animal slowly improved but never regained full use of its hind limbs. One month later it was found to be unable to walk and hyperaesthetic.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Results
AFIP Wednesday Slide Conference - No. 18
11 February 1981

Case I - C-80-02 (2 slides) (AFIP 1757377).

History: This male Sprague-Dawley rat was one of a group of animals on an acute toxicity study killed on the second day of the study.

Laboratory Results: Blood urea nitrogen: 350 mg/dl
Glucose (fasting): 1600 mg/dl

Contributor's Diagnoses and Comments: Pancreatic islet B-cell degeneration and renal tubular toxicity due to alloxan.

Comments: Alloxan was administered subcutaneously at 450 mg/kg and initially produced severe hypoglycemia due to B-cell degranulation and subsequently marked hyperglycemia as B-cells underwent further degeneration. Alloxan was initially used to study renal toxicity until Dunn and colleages discovered its dramatic effects on islet B-cells. Histologic effects on the islet B-cells include degranulation within minutes of dosing followed by cell shrinkage, nuclear pyknosis and cytolysis. Renal changes are predominately in proximal tubular epithelial cells and include swelling, pallor of staining, vacuolization, exfoliation and mineralization of cellular debris within the tubules.

Attendees' Diagnoses & Comments:

Morphologic Diagnoses: 1) Degeneration and necrosis, islet cells, acute, multifocal, mild to marked, Islets of Langerhans, pancreas, rat. 2) Degeneration and necrosis, tubular, acute, multifocal, mild to moderate, proximal tubules, kidney.

Etiologic Diagnosis: Islet cell and renal tubular toxicosis.

Etiology: Compatible with alloxan toxicity.

Comments: There was general agreement with the contributor's diagnoses and comments. Representative lesions were not present on all of the submitted sections of pancreas and kidney. Lesions ranged from mild to marked in both organs. Lesions in the pancreas were confined to the islets and involved from 10 to 50% in any given section. Renal necrosis was confined to the proximal tubules with multifocal mineral deposits in the proximal tubules and some deposits in the distal and collecting tubules. Nephrosis was used to encompass the lesions present. A differential etiologic agent listed by a few attendees was streptozoan, another diabetogenic and nephrotoxic compound. The two serum chemistry findings were discussed: 1) The marked fasting hyperglycemia corresponds well with the lesions in the pancreatic islets; and 2) The severe azotemia. Most participants did not feel the lesions present in the kidneys adequately explained the elevated BUN. Massive protein catabolism of other tissues was proposed as the underlying prerenal cause of the increased BUN. Caution was expressed that this strain of rat has a high incidence of spontaneous renal disease. Glomerulonephrosis is the term most commonly applied to these lesions. Lesions first appear at 8 to 9 months of age and become severe by 2 years of age.

Contributor: Toxicology Section, Health, Safety & Human Factors Laboratory, Kodak Park, B-320, Eastman Kodak Co., Rochester, NY 14650.

References:

1. Rerup, C. C.: Drugs producing diabetes through damage of the insulin secreting cells. Pharmacol. Rev. 22: No. 4, 485-518, 1970.

2. Weaver, D. C. et al.: Alloxan uptake by isolated rat Islets of Langerhans. Endocrinology 102: No. 6, 1547-1855, 1978.

Case II - Dog-Case 1 (AFIP 1757415).

History: 2-year-old male Doberman Pinscher --anorexia, emesis, obstipation for 1 week. Exploratory laparotomy revealed a dark red, flaccid ileum with dark red contents. An intussusception of the terminal ileum was easily reduced. The dog did not improve and was euthanized one week later.

Contributor's Diagnosis & Comments: Ileum--exhaustion myositis. Diffuse subacute to chronic inflammation of muscularis externa, muscularis mucosa, and submucosal plexus, degeneration of smooth muscle fibers.

Comment: The lamina propria contains some mixed inflammatory cells, but is relatively unaffected. Numerous capillaries in the muscularis externa are surrounded by neutrophils. The epithelium is not involved. Brown and Brenn stain demonstrated Gram positive rods in the lumen. PAS and GMS were negative for organisms. This lesion can be seen with obstructions of the intestinal tract, and resulted in a complete paralytic ileus in this case.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Degeneration and necrosis, with subacute to chronic inflammation, diffuse, severe, muscular tunics and submucosa, small intestine, Doberman Pinscher, canine.

Etiologic Diagnosis: Paralytic ileus.

Etiology: Prolonged intestinal obstruction.

Comments: Participants agreed with the contributor's description of the lesions. The etiologic diagnosis given by the contributor was considered the most plausable in view of the changes present. These lesions were unfamiliar to many of those attending the conference. Differential etiologies listed by participants included pressure necrosis and ischemia in addition to paralytic ileus. Separation of the lamina propria from the epithelial covering of the villi was felt to be a fixation shrinkage artifact and not part of the submucosal and muscular degenerative process. Vasculitis and fibrin thrombi formation were observed in the small vessels of the muscular tunics and were felt to be secondary to the degenerative changes. Necrosis of ganglion cells in the submucosal and myenteric plexuses was observed by all and was considered to be a contributing factor in the flaccid state of the gut at necropsy and in the muscular degeneration seen microscopically.

Contributor: Sterling-Winthrop Research Institute, Dept. of Toxicology, Columbia Turnpike, Rensselaer, NY 12144.

Case III - 80-902 (3) (AFIP 1757303).

History: Three week old Holstein calf treated for 1 week for pneumonia and enteritis.

Laboratory Results: Cranial-ventral portions of the lung were dark red and of increased consistency. Multiple small grey foci were scattered throughout the lung.

Contributor's Diagnosis & Comments: Pulmonary aspergillosis.

Comments: Histologic features are typical for mycotic pneumonia. The presence of both hyphae and fruiting bodies are interesting features of this case.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Pneumonia, pyogranulomatous, chronic, diffuse, severe, lung, Holstein, bovine.

Etiologic Diagnosis: Mycotic pneumonia.

Etiology: Aspergillus sp.

Comments: There was unanimous agreement with the contributor's diagnosis. Participants also agreed with the contributor's comments, however, fruiting bodies were not observed. A large number of degenerating hyphal forms surrounded by a radiating eosinophilic material characterized as the Splendore-Hoeppli phenomenon were present in most sections. These may be confused with fruiting bodies. A GMS stain highlighted morphologic features of the fungus which are characteristic of Aspergillus sp.; septated and dicotymous branching hyphae 5-7 microns in diameter. A concurrent or predisposing bacterial infection was suspected in this case by many of the attendees. The small grey foci described at necropsy within the pulmonary parenchyma were likened to those of lungworm lesions of cattle but the young age of the calf made this an unlikely assumption.

Contributor: University of Tennessee, Department of Pathobiology, P. O. Box 1071, Knovville, TN 37901.

References:

1. Eggert, M. J & Romberg, P. F.: Pulmonary aspergillosis in a calf. JAVMA 137 (10): 595-596, 1960.

2. Jubb, J.V.F., and Kennedy, P.: Pathology of Domestic Animals, Academic Press, New York, 1970, pp 250-251.

Case IV - 77/106 (AFIP 1757044).

History: The tissue is from a 14 week old German shepherd dog. On April 4th the animal was presented with vomiting and an elevated temperature. This responded to Penicillin and Streptomylin therapy. On April 9th the dog showed a sudden deterioration. The temperature was elevated, the hind limbs and tail showed partial paresis. The animal slowly improved but never regained full use of its hind limbs. One month later it was found to be unable to walk and hyperaesthetic.

Laboratory Results: Larvae of the nematode Angiostrongylus cantonensis were recovered from the meninges at necropsy.

Contributor's Diagnosis & Comments: Granulomatous meningoencephalomyelitis due to Angiostrongylus cantonensis.

Comments: In the rat the lung worm Angiostrongylus cantonensis migrates to the brain and then to the pulmonary arteries where it reaches sexual maturity. In other mammals the migration is not completed and the worms remain and cause damage in the central nervous system.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Meningoencephalitis, granulomatous, chronic, multifocal, moderate, cerebellum, brain, German shepherd dog, canine.

Etiologic Diagnosis: Verminous meningoencephalitis.

Etiology: Angiostrongylus cantonensis.

Comments: There was unanimous agreement with the contributor's diagnosis and comments. Granulomatous lesions were not present in the neuropil of some sections. Lesions present in the neuropil were confined to the white tracts of the medulla. These and the meningeal lesions were considered primarily vascular in distribution. The granulomas in the brainstem were thought to be a foreign body reaction to degenerating larvae or larval sheaths. The parasite is a larval nematode that has polymyarian coelomyarian musculature and a large intestine composed of a few multinucleated cells. In addition there are accessory hypodermal chords. These features are characteristic of a metastrongyle. The size, morphology of the intestine, and the lateral chords are consistent with Angiostrongylus sp. Specific identification is not possible in histologic sections but the probable species is A. cantonensis. A. vasorum, a lungworm of dog, may resemble this larva but it does not undergo migration through the central nervous system.

Contributor: Division of Veterinary Biology, School of Veterinary Studies, Murdoch University, Murdoch, Western Australia 6150.

References:

1. Jindrak, K. et al.: Experimentally induced Angiostrongylus cantonensis infection in dogs. Am. J. Vet. Res. 31: No. 3, 449-456, 1970.

2. Mason, K. V. et al.: Austr. Vet. J. 52: 205, 1976.

3. Rosen, L. et al.: Life history of the canine lungworm Angiostrongylus vasorum (Baillet). Am. J. Vet. Res. 31: No. 1, 131-143, 1970.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Histories
AFIP Wednesday Slide Conference - No. 19
18 February 1981

Case I - G2-14 (AFIP 1764737).

History: Tissues taken from a young crossbred goat 21 months after it received an experimental inoculation as a young adult. No clinical abnormalities were appreciated.

Case II - 80-76 (AFIP 1771338).

History: Tissue is submitted from a 9-week-old, female, B6D2F1 mouse, which was one of many in a group that died suddenly. The mice had received 850 R of whole body radiation (cobalt) and intravenous syngeneic bone marrow cells 7 days prior to death in a study of stem cell graft response. Spontaneous post-irradiation deaths were expected at 14-21 days, generally from bacterial septicemia. These mice died prematurely at 5-8 days following exposure.

Case III - OARB001 (AFIP 1773292).

History: Tissue is from an 8-week-old meat type chicken.

Case IV - 38/UCD (AFIP 1727246).

History: Tissue from an 18-year-old quarterhorse mare having equine Cushing's disease (hyperadrenalcorticism secondary to a tumor of the pars intermedia).

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Results
AFIP Wednesday Slide Conference - No. 19
18 February 1981

Case I - G2-14 (AFIP 1764737).

History: Tissues taken from a young crossbred goat 21 months after it received an experimental inoculation as a young adult. No clinical abnormalities were appreciated.

Contributor's Diagnosis & Comments: Arthritis due to the agent of viral leukoencephalomyelitis of goats.

Comments: Microscopic lesions were recognized in brain, joint and lung tissues in this group of animals which were inoculated by the intracerebral route. Joint lesions were noted as early as one week post-infection and both continued and progressed.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Synovitis, proliferative, chronic, diffuse, moderate, synovial membranes, joint, crossbred goat, caprine.

Etiologic Diagnosis: Caprine arthritis-encephalitis virus.

Etiology: Caprine retrovirus.

Comments: Attendees agreed with the contributor's etiologic diagnosis. Lesions were described as villous proliferation with diffuse formation of lymphoid follicles and infiltration primarily by plasmocytes and ulceration of the synovial membrane. The retrovirus also causes lesions in the lungs, kidneys, vessels and central nervous system of goats. The joint is considered an immunologically priviledged site and thus favorable for viral sequestration. Other factors which may be involved include antigenic drift of surface markers and persistent infection. Viral replication is dependent on RNA dependent DNA polymerase. Persistent infection is due to incorporation of viral genome with host-cell DNA. The disease occurs sporadically in goat herds and is passed horizontally. Viral particles can be recovered from the mammary gland of infected animals. The caprine arthritis-encephalitis virus is antigenically and morphologically similar to the Visna virus of sheep.

Contributor: Division of Comparative Medicine, The Johns Hopkins University, School of Medicine, Baltimore, MD 21205.

References:

1. Cork, L. C. et al.: The pathogenesis of viral leukoencephalomyelitis-arthritis of goats. I. Persistent viral infection with progressive pathologic changes. Lab. Invest. 42: 596-602, 1980.

2. Cork, L. C. et al.: Chronic arthritis in goats caused by a retrovirus. Science 207: 997-999, 1980.

3. Crawford, T. B. et al.: The connective tissue component of the caprine arthritis-encephalitis syndrome. Am. J. Pathol. 100: 443-454, 1980.

Case II - 80-76 (AFIP 1771338).

History: Tissue is submitted from a 9-week-old, female, B6D2F1 mouse, which was one of many in a group that died suddenly. The mice had received 850 R of whole body radiation (cobalt) and intravenous syngeneic bone marrow cells 7 days prior to death in a study of stem cell graft response. Spontaneous post-irradiation deaths were expected at 14-21 days, generally from bacterial septicemia. These mice died prematurely at 5-8 days following exposure.

Laboratory Results:

Mouse hepatitis virus (MHV) was isolated from a sample of fresh frozen liver from this mouse. A polyvalent antiserum for MHV antigens was used; thus the subgroup of MHV was not determined.

Contributor's Diagnosis & Comments: Necrosis, acute, multifocal, severe, liver, etiology - mouse hepatitis virus.

Comments: Grossly this liver was enlarged with multifocal white spots throughout. More typically the livers of affected mice were diffusely necrotic, grossly appeared enlarged and uniformly pale and friable. Stress and immunosuppression from exposure to radiation were considered to be precipitating factors resulting in excerbation of latent MHV infection. The B6D2F1 mice were the only strain in our institute which developed the disease despite other strains from the same and different suppliers being used in similar studies. Serological testing (CF) similarly indicated only this strain to be infected. Communication and quality control data from the supplier showed no evidence of MHV in their colony; hence the source of infection was not totally solved. The lack of any inflammatory cell response was attributed to radio-immunosuppression. Diffuse lymphoid depletion also was present in the spleen and intestinal Peyer's patches.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Necrosis, hepatocellular, coagulative, acute, multifocal, liver, B6D2F1, mouse.

Etiologic Diagnosis: Viral hepatitis.

Etiology: Mouse hepatitis virus.

Comments: There was unanimous agreement with the contributor's diagnosis and comments. The acute nature of the lesions and past history of irradiation were postulated as the cause for the lack of an inflammatory response. Four strains of MHV are known. The virus is a coronavirus. Other conditions associated with MHV are LIVIM of neonatal mice and demyelinating leucodystrophy of mice. The virus is ubiquitous and usually silent in colonies of mice. Carriers are important sources of infection. Rats also are susceptible to infection with MHV. Persistent viral shedding occurs from intestinal mucosal epithelium of infected animals. Shedding also occurs from infected Kupffer cells of the liver. Hepatocellular necrosis occurs after destruction of Kupffer cells and subsequent invasion of adjacent hepatocytes. Nude mice often are used as sentinel animals for infection in colonies. Multiple lesions and syncytial cells occur in many tissues in nude mice. Syncytial cells form in the intestinal tract of weanling conventional mice infected with MHV. Pseudomonas free mice are selected for irradiation studies in order to eliminate a common cause of post-irradiation septicemia. The mice are monitored for this bacteria by using sterile water bottles which are cultured periodically after use.

Contributor: Armed Forces Radiobiology Research Institute, National Naval Medical Center, Bethesda, MD 20014.

References:
1. Broderson, J. R. et al.: Lethal enteritis in infant mice caused by mouse hepatitis virus. Lab. Anim. Sci. 26: 824, 1976.
2. Carthew, P.: Lethal intestinal virus of infant mice is mouse hepatitis virus. Vet. Rec. 101: 465, 1977.
3. Carthew, P.: Peroxidase-labeled antibody technique for rapid detection of mouse hepatitis virus in cases of natural outbreaks. J. Infect. Dis. 138: 410-413, 1978.
4. Gledhill, A. W. et al.: Mouse hepatitis virus and its pathogenic action. J. Path. Bact. 69: 299-309, 311-320, 1955.
5. Hierholzer, J. C. et al.: New strain of mouse hepatitis virus as the cause of lethal enteritis in infant mice. Infect. & Immunity 24: 508-522, 1979.
6. Ishida, T. et al.: Pathology of diarrhea due to mouse hepatitis virus in the infant mouse. Jap. J. Exp. Med. 49 (1): 33-41, 1979.
7. Pedersen, J. C. et al.: Antigenic relationship of the feline infectious peritonitis virus to coronaviruses of other species. Arch. Virol. 58: 45-53, 1978.
8. Taguchi, F. et al.: Pathogenesis of mouse hepatitis virus infection. The role of nasal epithelial cells as a primary target of low-virulence virus. MHV-S. Microbiol. Immunol. 23 (4): 249-262, 1979.
9. Ward, J. M. et al.: Naturally occurring hepatitis virus infection in the nude mouse. Lab. Anim. Sci. 27: 372-376, 1977.
10. White, R. J. et al.: Pathogenesis of murine hepatitis: Route of infection of the susceptible host. Am. J. Vet. Res. 25: 1236-1240, 1964.

Case III - 0ARB001 (AFIP 1773292).

History: Tissue is from an 8-week-old meat type chicken.

Contributor's Diagnosis & Comments:

Morphologic Diagnosis: Squamous cell carcinoma.

Comments: There was general agreement with the contributor's diagnosis. Skin was identified in many sections by the presence of feather follicles. The tissue had been scalded before fixation. Attendees noted multifocal epidermal ulceration and invasion of the epidermis and dermis by neoplastic squamous epithelial cells. Neoplastic cells formed cords and nests of cells with intercellular bridges and keratin pearls. Mitotic figures occasionally were observed. The origin of these neoplastic cells was postulated to be the feather follicle. Invasion and destruction of skeletal muscle occurred around feather follicles. The incidence of this lesion is about 2/10,000 in broiler chickens at slaughter. Metatastasis is not reported. These neoplasms are locally invasive, may be single or multiple and occur on any part of the body. A seasonal incidence in the spring is associated with this lesion. The cause of these neoplasms has not been determined.

Contributor: Bureau of Veterinary Medicine, Food & Drug Administration, Beltsville, MD 20705.

Reference: Turnquest, Robert U.: Dermal squamous cell carcinoma in young chickens. Am. J. Vet. Res. 40 (11): 1628-1633, 1979.

Case IV - 38/UCD (AFIP 1727246).

History: Tissue from an 18-year-old quarterhorse mare having equine Cushing's disease (hyperadrenalcorticism secondary to a tumor of the pars intermedia).

Contributor's Diagnosis & Comments: Schizogenous forms of a coccidia (probably Klossiella equi) are present within the endothelial cells of the pineal gland. Similar structures were present in vessels in the brain and renal glomeruli. Numerous coccidial forms were present in the renal tubules. There was no cellular response to these intravascular organisms nor clinical signs of their presence.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Endothelial cell parasitism, multifocal, mild to moderate, pineal gland, horse.

Etiologic Diagnosis: Parasitic vasculopathy.

Etiology: Protozoan, possibly Klossiella sp. or Sarcocystis sp.

Comments: An etiomorphologic diagnosis of parasitic vasculopathy was preferred by most attendees. A tenuous association between endothelial cell parasites in the pineal gland and kidney tubules was drawn by participants. Many participants list Sarcocystis sp. as differential etiologic agents. Differentiation between Klossiella and Sarcocystis could not be made. Electron photomicrographs of the parasite provided by the contributor demonstrated coccidian parasites within endothelial cells. Parasites were 1.5 X 6 microns in size. Up to 50 merozoites are present within parasitophorus vacuoles in endothelial cells of the pineal gland. Also commented on was the obvious lack of host response to the presence of the parasites. The reported Cushing's disease condition may have contributed to this immunosuppression. The life cycles of both Sarcocystis and Klossiella were reviewed. Three endothelial replicative stages occur in bovine Sarcocytosis. A third replication also occurs in circulating blood cells but is considered a minor component in the life cycle. The life cycle of Sarcocystis in the equine has not been fully elucidated. The possible role of Sarcocystis sp. in Equine Protozoal Encephalomyelitis also was discussed. The entire life cycle of Klossiella equi is not known. A schizont generation develops in the endothelial cells of Bowman's capsule. Merozoites pass down to the proximal convoluted tubules with the renal fluids and penetrate the epithelial cells. There they develop a second generation of schizonts. The merozoites from these rupture from the host-cell into the renal fluid. At this point these merozoites either pass outward or distally to the loop of Henle where they penetrate the epithelial cells and undergo gamete formation. Sporocysts are formed from these gametes and are passed in the urine. Sporocysts are ingested by the host, while grazing, and excyst in the digestive tract. Sporozoites presumably pass through the intestinal wall, enter the blood, eventually reach the glomeruli, enter the endothelial cells, and the cycle is repeated.

Contributor: Department of Pathology, School of Veterinary Medicine, University of California, Davis, CA 95616.

References:

1. Levine, N. D. et al.: The Coccidian Parasites (Protozoa, Sporozoa) of Rodents. The Univ. of Illinois Press, Urbana, 1965, pp 180-185, 342-345.

2. Taylor, Jack L. et al: Klossiella parasites of animals: A literature review. Vet. Parasitol. 5: 137-144, 1979.

3. Todd, K. R. et al.: Klossiella equi Baumann, 1946 (Sporozoa: Eucoccidiorida) from an Illinois horse. Vet Med/Sm Anim Clin 72: 443-448, 1977.

4. Vetterling, J. M. et al.: Klossiella equi Baumann, 1946 (Sporozoa: Eucoccidia: Adeleina) from equids. J. Parasitol. 58: 589-594, 1972.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Histories
AFIP Wednesday Slide Conference - No. 20
25 February 1981

Case I - #1 - (AFIP 1716398

History: A 6-month-old colt had multiple subcutaneous nodular masses in the left inguinal region. Initial biopsy indicated possible lymphatic hamartoma. The masses progressively enlarged and began infiltrating the left rear leg. Due to difficulty of complete surgical excision a poor prognosis was given. The colt was euthanatized and an abdominal and retroperitoneal mass was identified at postmortem. The mass was adherent to the left lumbar musculature, the craniodorsal aspect of the pelvic inlet, the mid jejunum, the left testicle and the left kidney. Infiltrative outgrowths traversed the left inguinal canal and extended into the deep intermuscular fascia of the left hindlimb.

Case II - 79P2240 (AFIP 1757311).

History: Male, 7-month-old, Labrador retriever presented dead after being listless for 1 or 2 days and 41 C temperature 24 hours prior to death.

Laboratory Results:

Necropsy revealed reddened gastrointestinal mucosa and congested liver. The myocardium was pale with small white subendocardial spots.

Case III - 80-15072 (AFIP 1758403).

History: Tissue from a full term bovine fetus which was found in the field of a small beef cattle farm.

Case IV - AN 80/254, SN 80/1310 (AFIP 1761486.

History:

AN 80/254 - A mortality involved a group of 476 cattle 296 of which had been grazing kikuyu pasture for 2 weeks and 180 of which had been introduced to the property and had not previously grazed kikuyu.

The cattle were introduced to a paddock which had been heavily fertilized and irrigated over the previous 2 months and contained an overgrowth of feed over 2 feet high.

Day 3 - 1 dead
Day 4 - 5 dead, all cattle removed from paddock
Day 5 - 19 dead
Day 6 - 10 dead
Day 7 - 2 dead
Day 8 - 1 dead

SN 80/1310 - Similar history of losses 4-7 days after access to lush kikuyu pasture.

Clinical signs included excessive salivation and dehydration, with depression, weakness, recumbency and death within 24-36 hours.

At autopsy mucous membranes were cyanotic and carcases were markedly dehydrated. There was consistently agonal ecchymotic epicardial hemorrhage. There were usually no gross lesions in the forestomachs. In the abomasum and small intestine there was occasionally patchy mucosal congestion. Liver and kidney were congested. The pH of ruminal contents in 2 animals was 8 and 9.

LABORATORY RESULTS:
(3 animals)

	Sample 1	Sample 2	Sample 3
Calcium mg/dL	9.9	12.8	11.1
Magnesium mg/dL	3.1	3.8	2.9
Total Protein g/dL	8.1	9.7	8.8
Albumin g/dL	3.7	4.3	3.9
Urea mg/ml	112	88	108
Urea Nitrogen ug/ml	52.2	41.0	50.3
Glucose mg/dL	421	132	149

TIMOTHY P. O'NEILL
Capt., USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Results
AFIP Wednesday Slide Conference - No. 20
25 February 1981

Case I - #1 - (AFIP 1716398

History: A 6-month-old colt had multiple subcutaneous nodular masses in the left inguinal region. Initial biopsy indicated possible lymphatic hamartoma. The masses progressively enlarged and began infiltrating the left rear leg. Due to difficulty of complete surgical excision a poor prognosis was given. The colt was euthanatized and an abdominal and retroperitoneal mass was identified at postmortem. The mass was adherent to the left lumbar musculature, the craniodorsal aspect of the pelvic inlet, the mid jejunum, the left testicle and the left kidney. Infiltrative outgrowths traversed the left inguinal canal and extended into the deep intermuscular fascia of the left hindlimb.

Contributor's Diagnosis: Cystic lymphangioma.

Attendees' Diagnosis & Comments:
Morphologic Diagnosis: Cystic lymphangioma, horse.
Comments: Attendees agreed with the contributor's diagnosis. Chronic multifocal cellulitis also was present. No organisms were observed in these foci. Lymph filled cystic spaces lined by endothelial cells and surrounded by dense connective tissue comprised the majority of the neoplasm. Mitotic figures were rare. Loose slightly myxomatous areas also were present along with dense areas of connective tissue. The large and invasive nature is consistent with that described for cystic lymphangioma in man. Several participants considered lymphangiosarcoma as a differential diagnosis but histologic criteria for malignancy were lacking. Many felt that this tumor was congenital in origin, arising from embryonic rests of vascular tissue, with subsequent growth after birth. These tumors are considered rare in animals. Hemangiomas and hemangiosarcomas occasionally are observed in young animals. Lymphangiomas are placed into three categories in man: 1) Simple or capillary lymphangiomas, which are hard to differentiate from lymphangectasis; 2) Cavernous lymphangiomas, which are characterized by thin walled dilated lymph vessels surrounded by variable amounts of fibrous connective tissue; and 3) Cystic lymphangiomas or hygromas, which are large dilated and cystic lymphatic vessels surrounded by thick fibrous connective tissue. Normal tissue structures may be interposed between cystic spaces in this last category as well as growth along fascial planes. The inflammatory foci observed in this case was felt to be secondary to the surgical intervention, and not a part of the tumor.

Contributor: Dept. of Veterinary Pathology & Microbiology, Washington State University, Pullman, WA 99163.

Reference: Turk, J. M. et al.: Cystic lymphangioma in a colt. JAVMA 174: 1228-1230, 1979.

Case II - 79P2240 (AFIP 1757311).

History: Male, 7-month-old, Labrador retriever presented dead after being listless for 1 or 2 days and 41 C temperature 24 hours prior to death.

Laboratory Results:

Necropsy revealed reddened gastrointestinal mucosa and congested liver. The myocardium was pale with small white subendocardial spots.

Contributor's Diagnosis & Comments: Myocarditis due to Trypanosoma cruzi.

Comments: This dog was native to South Louisiana and had not been outside the area since birth. The number of amastigotes was excessive. Identification was made using light and electron microscopy.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Myocarditis, granulomatous, diffuse, chronic, severe, heart, Labrador retriever, canine.

Etiologic Diagnosis: Myocardial Trypanosomiasis (protozoal myocarditis).

Etiology: Trypanosoma cruzi.

Comments: There was unanimous agreement with the contributor's diagnosis. Attendees' diagnosis was based on the tissue, inflammatory response, and large numbers of trypanosomal amastigotes within myocardial fibers.

The amastigotes measured 2-5 microns in diameter and contained a large nucleus and basophilic kinetoplast. A comment was made in the differentiation of the parasite from other closely related members of the group. Amastigotes of Leishmania spp. parasitize cells of the reticuloendothelial system and thus are not found intracellular in myocardial fibers. Trypanosoma rhodesiense, T. congelense, and T. vivax (i.e., causative agents of African Trypanosomiasis in animals) do not have the amastigote stage in their life cycle. Mammals are most commonly infected with T. cruzi by one of several modes: 1) Ingestion of the triatomid bug or its feces; 2) Contamination of wounds, bites or mucous membranes with the feces of the bug; or 3) Ingestion of tissues of infected animals. Transmission may also be accomplished blood transfusions. Numerous wild animals particularly opossums, raccoons, and armadillos in the Southern U. S. harbor the organism.

Contributor: Dept. of Veterinary Pathology, School of Veterinary Medicine, Louisiana State University, Baton Rouge, LA 70803.

References:

1. Anseline, A. et al.: Pathogenic mechanisms in Chagas' cardiomyopathy. In Trypanosomiasis and Leishmaniasis with Special Reference to Chagas Disease, Ciba Foundation Symposium 20 (new series), Associated Scientific Publishers, Amsterdam, London, New York, 1974, pp 123-136.

2. Snider, T. G. III, et al.: Myocarditis caused by Trypanosoma cruzi in a native Louisiana dog. JAVMA 177: 247-249, 1980.

3. Williams, G. D., et al.: Naturally occurring Trypanosomiasis (Chagas Disease) in dogs. JAVMA 171: 171-177, 1977.

Case III - 80-15072 (AFIP 1758403).

History: Tissue from a full term bovine fetus which was found in the field of a small beef cattle farm.

Laboratory Results: Brucella abortus was cultured from gastric contents, spleen, and lung. FA tests and virus isolation attempts for IBR and BVD were negative.

Contributor's Diagnosis & Comments: Meningitis, suppurative, multifocal, marked, brain. Etiologic Agent: Brucella abortus.

Comments: Gross examination revealed no significant findings. Microscopically, marked multifocal cellular infiltrates were in the leptomeninges. The infiltrates varied from one area to another and, in most areas, were composed of polymorphonuclear cells. Coccobacillus bacteria were observed in macrophages. The neuroparenchymal change was very limited. Extracerebral lesions included marked fibrinopurulent exudates in the joint, mild multifocal accumulation of mononuclear cells in the interstitium of the kidney and in the portal tracts of the liver. Meningitis and encephalitis are not uncommon findings in bovine fetuses aborted due to Brucella infection.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Meningitis, granulomatous, multifocal to diffuse, moderate, meninges, brain, fetus, bovine.

Etiologic Diagnosis: Bacterial meningitis.

Etiology: Brucella sp. (probably B. abortus).

Comments: The majority of participants listed pyogranulomatous meningitis with the etiologic diagnosis of Enzootic Bovine Abortion (foothill abortion) caused by Chlamydial organisms as their preconference choice. Differential diagnoses given were Brucellosis and Listeriosis. The cellular response was predominantly large and small mononuclear cells with only a few neutrophils. Degenerating histiocytes mimicked polymorphonuclear cells and resulted in misinterpretation of the inflammatory response. Some histiocytic cells contained small pale staining bacilli that could be seen in the H&E stained section. The majority of the participants were unfamiliar with this fetal lesion of Brucella abortion. Classically a granulomatous pneumonia is the lesion most commonly reported. Articular, renal and hepatic lesions also are reported.

Contributor: Livestock Disease Diagnostic Center, University of Kentucky, 1429 Newtown Pike, Lexington, KY 40511.

References:

1. Guarda, F. Ulteriori Ricerche Sulla Neuropatologia Dei Feti Bovini Abortiti, (Further research on the neuropathology of the aborted fetuses). Annali Fac. Med. Vet. Torino. 24: 56, 1977.

2. Guarda, F.: Problemi Attuali Degli Aborti Bovini. Schweiz. Arch. Tierheilk. 121: 323-326, 1979.

Case IV - AN 80/254, SN 80/1310 (AFIP 1761486) 2 slides.

History:

AN 80/254 - A mortality involved a group of 476 cattle 296 of which had been grazing kikuyu pasture for 2 weeks and 180 of which had been introduced to the property and had not previously grazed kikuyu.

The cattle were introduced to a paddock which had been heavily fertilized and irrigated over the previous 2 months and contained an overgrowth of feed over 2 feet high.

Day 3 - 1 dead
Day 4 - 5 dead, all cattle removed from paddock
Day 5 - 19 dead
Day 6 - 10 dead
Day 7 - 2 dead
Day 8 - 1 dead

SN 80/1310 - Similar history of losses 4-7 days after access to lush kikuyu pasture.

Clinical signs included excessive salivation and dehydration, with depression, weakness, recumbency and death within 24-36 hours.

At autopsy mucous membranes were cyanotic and carcases were markedly dehydrated. There was consistently agonal ecchymotic epicardial hemorrhage. There were usually no gross lesions in the forestomachs. In the abomasum and small intestine there was occasionally patchy mucosal congestion. Liver and kidney were congested. The pH of ruminal contents in 2 animals was 8 and 9.

LABORATORY RESULTS:

(3 animals)	Sample 1	Sample 2	Sample 3
Calcium mg/dL	9.9	12.8	11.1
Magnesium mg/dL	3.1	3.8	2.9
Total Protein g/dL	8.1	9.7	8.8
Albumin g/dL	3.7	4.3	3.9
Urea mg/ml	112	88	108
Urea Nitrogen ug/ml	52.2	41.0	50.3
Glucose mg/dL	421	132	149

Contributor's Diagnoses & Comments: Omasitis, reticulitis, (usually diffuse). Rumenitis (usually focal). Epithelial necrosis with neutrophil infiltration. Acute patchy cortical nephrosis. Kikuyu poisoning.

Comments: Martinovich and Smith (1973) noted a decrease in abomasal acidity and, in contrast to grain engorgement, that ruminal pH was within the normal range in cattle with Kikuyu poisoning. The similarity between so-called Kikuyu poisoning of cattle and 2 experimental mycotoxicoses has been noted (Martinovich et al. 1972).

Attendees' Diagnoses & Comments:

Morphologic Diagnoses: 1) Necrosis, tubular, acute, multifocal, mild to moderate, kidney. 2) Necrosis, epithelial, lamellar, acute, multifocal, omasum, rumen and reticulum, bovine.

Etiologic Diagnosis: Toxic nephrosis, omasitis, reticulitis and rumenitis.

Etiology: Kikuyu grass toxicity (Pennisetum clandestinum).

Comments: There was general agreement with the contributor's diagnoses and comments. Urea poisoning was listed most often as a differential diagnosis by participants. Attendees noted a slight rumen alkalosis, a hyperglycemia, and slightly elevated blood urea level in the clinical pathology results provided by the contributor. Attendees agreed that the histological lesions were confined to the superficial layers of the mucosa. Ulceration was considered artifactual because inflammation, necrosis and hemorrhage in the lamina propria did not accompany this. Renal lesions were confined to the cortex, and consisted of necrosis and degeneration of tubular epithelial cells. Interstitial nephritis has also been reported with this toxicosis. A single toxic factor has not been isolated in this disease. At least two and possibly three interdependent factors exist: 1) The Kikuyu grass which may contain a toxic principle; 2) The presence of army worms (army catapillars), Spodoptera exempta; and 3) Mycotoxins elaborated from fungi which grow in the litter under the grass. Studies have shown that elimination of either the fungus or the army worms reduces the indidence of the disease. A few attendees commented on the similarity of lesions present in this case to those observed in sweating sickness in calves reported in South Africa.

Contributor: Regional Veterinary Laboratory, Wollongbar, N.S.W., Australia.

References:

1. Cordes, D. O. et al.: Acute ruminal indigestion, alkalosis and death of cattle grazing Kikuyu grass. New Zealand Vet. J. 17: 79, 1969.

2. Busch, J. et al.: Acute ruminal indigestion, alkalosis and death of cattle grazing Kikuyu. New Zealand Vet. J. 17: 182, 1969.

3. Martinovich, D. et al.: Similarities between so-called Kikuyu poisoning of cattle and two experimental mycotoxicoses. New Zealand Vet. J. 20: 57. 1972.

4. Martinovich, D. et al.: Kikuyu poisoning of cattle. 1. Clinical & pathological findings. New Zealand Vet. J. 21: 55, 1973.

5. Smith, B. et al.: Kikuyu poisoning of cattle. 2. Epizootiological aspects. New Zealand Vet. J. 21: 85, 1973.

6. Gabeedy, B. J. et al.: Kikuyu poisoning of cattle in Western Australia. Austr. Vet. J. 50: 369, 1974.

7. Bryson, R. W. et al.: Kikuyu grass poisoning of cattle in Natal. J. South African Vet. Assoc. 49 (1): 19-21, 1978.

8. Van Heerden, J. et al.: An outbreak of "Kikuyu Poisoning" in Western Transvaal. J. South African Vet. Assoc. 49 (1): 27-30, 1978.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Histories
AFIP Wednesday Slide Conference - No. 21
18 March 1981

Case I - 84484 (AFIP 1757061).
History: A 9-year-old male Golden retriever was presented with a history of illness of 10 days duration. It was anorectic, weak, and showed lameness in the right rear leg. There was enlargement of the right hock to about 6X9 cm, and atrophy of muscles of the leg. X-ray revealed lytic lesions in the right tibia, fibula, tarsus and metatarsus. The dog had a draining fistula below the right hock. The dog died and tissues were taken at necropsy examination.

Case II - 1889 (AFIP 1744737).
History: Sprague-Dawley rat (Charles River, CD); control animal; male; birth date 5/78; sacrificed 1/14/80; sac weight 600 g.; sacrificed because of inappetence and general unthrifty appearance.

Laboratory Results:

RBC	7.12×10^6	HGB	14.5
MCV	64.5	Plasma Protein	5.8
HCT	42		
WBC	5490		

Case III - 80-0356 (AFIP 1757048).
History: 3-year-old Suffolk ewe with history of weight loss, patchy loss of wool, pruritis manifested by rubbing, scratching, nibbling fleece; mild ataxia, lip smacking and nibbling when scratched.

Case IV - 9-208-80 (AFIP 1758839).
History: Multiple spontaneous abortions occurred in a research herd of African Pygmy Goats. 2 microslides (H&E and Giemsa) included.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Results
AFIP Wednesday Slide Conference - No. 21
18 March 1981

Case I - 84484 (AFIP 1757061).

History: A 9-year-old male Golden retriever was presented with a history of illness of 10 days duration. It was anorectic, weak, and showed lameness in the right rear leg. There was enlargement of the right hock to about 6X9 cm, and atrophy of muscles of the leg. X-ray revealed lytic lesions in the right tibia, fibula, tarsus and metatarsus. The dog had a draining fistula below the right hock. The dog died and tissues were taken at necropsy examination.

Contributor's Diagnosis & Comments: Coccidioidomycosis.

Comments: There was a granulomatous osteomyelitis affecting the tibia, fibula and hock of the right rear leg, with cellulitis of the soft tissues over the hock joint area. There was also a granulomatous synovitis in the hock joint itself. Numerous spherules of the fungal organism Coccidioides immitis were present in the bone lesions. There were focal areas of granulomatous pneumonia present also, the site of initial infection. Initial presentation of osteomyelitis or draining fistulas associated with bone is not uncommon in the dog, and in endemic areas of this fungus caution should be exercised in cultureing such lesions, because of the hazard to laboratory personnel.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Osteomyelitis, pyogranulomatous, chronic, diffuse, severe, bone marrow, right hindlimb, canine.

Etiologic Diagnosis: Coccidioidomycotic osteomyelitis.

Etiology: Coccidioides immitis.

Comments: There was unanimous agreement with the contributor's etiologic diagnosis. All observed large thick walled spherules some of which contained many endospores. Occasional double contoured and ruptured organisms were also observed. Ruptured forms incited an intense neutrophilic response with microabscess formation. The pathogenesis of this disease was briefly reviewed. Arthrospores of C. immitis detach easily from the hyphal form and become airborne. The arthrospores are inhaled by the host and result in a granulomatous pneumonia. These arthrospores in turn develop into spherules with development of endospores. The pulmonary form of the disease is usually self limited in most individuals with destruction of the organisms and resolution of the lesions. Radiographically so-called "coin" lesions are seen in man and the dog with the pulmonary form. Local lymph node involvement may accompany the pneumonia. Disseminated disease results from the hematogenous spread of the endospores to various tissues of the body most notably skin and bone. Meningeal infection is reported in the disseminated disease in humans (3). In cattle and horses a mediastinal and tracheobronchial lymphadenitis is sometimes observed and may be confused with a tuberculous lesion at necropsy or slaughter. Cell-mediated immunity is necessary for recovery from the disease. Circulating antibody to the organism is not protective and may alone or in conjunction with soluble substances released by the fungal organisms, suppress the cell-mediated immunity (2). Alveolar macrophages alone, from rhesus monkeys have been shown to be incapable of destroying C. immitis organisms in vitro (1). Also recent studies suggest a complement deficiency in both classical and alternative pathways in infected individuals (4).

Contributor: Los Angeles County Dept. of Health Services, Comparative Medical & Veterinary Services, 12824 Erickson Ave., Downey, CA 90242.

References:
1. Beaman, L. et al.: In vitro response of alveolar macrophages to infection with Coccidioides immitis. Inf. & Immun. 28:, 594-600, 1980.
2. Catanzaro, A.: Pulmonary coccidioidomycosis. Med. Clinics of North America 64: 461-473, 1980.
3. Coccidioidomycosis. Chapt. 17, Medical Mycology, pp 230-253.
4. Galgiani, J. N. et al.: Complement activation by Coccidioides immitis: In vitro and clinical studies. Inf. & Immun. 28: 944-949, 1980.

Case II - 1889 (AFIP 1744737).

History: Sprague-Dawley rat (Charles River, CD); control animal; male; birth date 5/78; sacrificed 1/14/80; sac weight 600 g.; sacrificed because of inappetence and general unthrifty appearance.

Laboratory Results:

RBC	7.12×10^6	HGB	14.5
MCV	64.5	Plasma Protein	5.8
HCT	42		
WBC	5490		

Contributor's Diagnosis & Comments: Histiocytic lymphosarcoma.
Comments: This large tumor on the top of the head and swelling extended down the dorsal surface of the neck and showed signs of erosion through the skull. Additionally there was fluid between the brain and the roof of the skull. There were raised nodules on the lung and on the left lobe of the liver which were histologically similar to the mass on the head.

Attendees' Diagnosis & Comments:
Morphologic Diagnosis: Lymphosarcoma, histiocytic, subcutis and bone, Sprague-Dawley rat.
Comment: There was a wide variety of preconference diagnoses submitted by the participants for this neoplasm. Poorly differentiated or undifferentiated sarcoma was most often listed. The final morphologic diagnosis was reached after some discussion. The tumor was characterized as an expansile solid mass which in some sections invaded muscle and bone. Cellular morphology remained fairly uniform consisting of large pleomorphic histiocytic cells with abundant eosinophilic cytoplasm. Multinucleated giant cells were observed frequently. The mitotic rate varied from 1 to 3 per high power field. Stroma and accompanying vasculature were sparce. This neoplasm was considered uncommon in the rat by those familiar with rodent neoplasms.

Contributor: Environmental Protection Agency, Mail Drop 72, Research Triangle Park, NC 27711.

Case III - 80-0356 (AFIP 1757048).

History: 3-year-old Suffolk ewe with history of weight loss, patchy loss of wool, pruritis manifested by rubbing, scratching, nibbling fleece; mild ataxia, lip smacking and nibbling when scratched.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Neuronal vacuolization, multiple, brain stem nuclei, and gray matter spinal cord, Suffolk, sheep, ovine.

Etiologic Diagnosis: Viral encephalomyelopathy.

Etiology: Scrapie virus (unclassified virus).

Comments: Several preconference morphologic diagnoses were submitted by the conference attendees including spongiform encephalomyelopathy, spongiform polioencephalomyelopathy and vacuolating neuropathy. Etiologic diagnoses included viral neuronal degeneration, viroid encephalomyelopathy, scrapie and viral encephalomyelitis. Vacuolated neurons were present in the spinal cord and brain stem. The swollen axon sheaths containing enlarged axons were considered to be postmortem and/or fixation and processing artifacts. The history coupled with the histologic features found in the neurons is strongly suggestive of the diagnosis of scrapie. Evaluation of suspected scrapie infected CNS tissue should also include special strains for astrocytic proliferation and hypertrophy of astrocytic processes. The etiologic agent of scrapie infection is listed as an unclassified virus. The infection is initiated early in post natal life in non-neural tissues most notably lymphatic and intestinal tissues. After many months of replication in lymphatic tissues, the virus (presumably by hematogenous spread) appears in the CNS especially the brain stem. Other CNS sites include the cerebellum, cortex and spinal cord. Viral replication proceeds slowly in neural tissue. The pathologic lesions are restricted to the nervous system, with resultant clinical signs. Infection via the alimentary tract is the most plausable mode of transmission. Vertical transmission has not been documented. Goats raised with infected sheep are susceptible to the disease and may develop similar lesions and signs (1). Experimental animals used in the study of scrapie include hamsters, mice and mink. Many papers have been published in the past few years which review the disease.

Contributor: Dept. of Pathology, College of Veterinary Medicine, Oklahoma State University, Stillwater, OK 74074.

References:

1. Hadlow, W. J. et al.: Virologic and neurohistologic findings in dairy goats affected with natural scrapie. Vet. Pathol. 17: 187-199, 1980.

2. Narayan, O.: Proceedings of ACVP Meeting, New Orleans, Louisiana, p. 99, 1980.

3. Marsh, R. F.: Slow virus infections. Amer. J. of Pathol. 69: 209-212, 1972.

4. Marsh, R. F.: Slow virus diseases of the central nervous system. Advances in Vet. Res., 155-177, 1976.

Case IV - 9-208-80 (AFIP 1758839).

History: Multiple spontaneous abortions occurred in a research herd of African Pygmy Goats. 2 microslides (H&E and Giemsa) included.

Laboratory Results: All fetuses were near term, autolysis was slight and no significant gross lesions were present. Placentas were covered with a tan, mucoid exudate, cotyledons were hemorrhagic and necrotic villi were noted. Cultures of fetal stomach contents did not result in any bacterial growth.

Contributor's Diagnosis & Comments: Rickettsial abortion, due to Coxiella burnetti.

Comments: An initial diagnosis of Chlamydial abortion was based on the necrotizing placentitis and the presence of what were thought to be elementary bodies in the Giemsa stained section. The possibility of rickettsia as an etiologic agent was not considered and samples were sent to Dr. Herb Stoenner at the Rocky Mountain Laboratory where FA-stained impression smears were positive for Coxiella burnetti.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Placentitis, necrotizing, subacute, diffuse, severe, placenta, pigmy goat.

Etiologic Diagnosis: Rickettsial placentitis.

Etiology: Coxiella burnetti.

Comments: There was general agreement with the contributor's diagnosis and comments after disclosure of the etiologic agent. Most participants, however, listed a Chlamydial species as their preconference etiologic agent. Bacterial agents listed for differential diagnosis included Camphylobacter fetus, Brucella abortus, Brucella melitensis and Listeria sp. Myriads of tiny intracellular organisms and large numbers of free bacilli measuring approximately 0.5 by 1.0 microns; all were Giemsa positive. The two forms were considered to represent different stages of the rickettsial replicative cycle. Mineralization was noted by all and felt to be a normal finding in post parturient placentas. A primary lymphoplasmacytic inflammatory infiltrate also was noted in the placental tissue. A photograph provided by the contributor depicted the gross appearance of the placenta as previously described. Photomicrographs of rhotamine and fluorescene conjugated antibody to C. burnetti were projected which substantiated the contributor's diagnosis. This condition generally is not reported in animals. Coxiella burnetti is commonly considered nonpathogenic for domestic livestock (3). The organism also is of zoonotic importance in that it causes Q fever in humans.

Contributor: Marsh Veterinary Laboratory, Box 997, Bozeman, Montana 59715.

References:

1. Lennette, E. H. et al., Eds.: Diagnostic Procedures for Viral, Rickettsial and Chlamydial Infections, 5th Ed., Amer. Pub. Health Assoc., Wash. DC, 1979.

2. Storz, J.: Chlamydia and Chlamydia-Induced Diseases, Charles C. Thomas, Springfield, IL, 1971.

3. Waldhalm, D. G. et al.: Abortion associated with Coxiella burnetti infection in dairy goats. JAVMA 173: 1580-81, 1978.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Patnology

Histories
AFIP Wednesday Slide Conference - No. 22
25 March 1981

Case I - 186-00386 (AFIP 1761485).
History: A 535-day-old Sprague-Dawley male rat had a 3.0 cm diameter mass on the right mandible. The mass was red and very firm. The rat was given .01 millimoles of N-hydroxy Formylaminofluorene/100 grams body weight intraperitoneally three times a week for four weeks.

Case II - 63-1435 (AFIP 1727492).
History: Tissue from a chicken.

Case III - 80-2 (1757054).
History: Femoral bone marrow smear taken at necropsy from an adult German shepherd dog. Clinical signs included lethargy, lymphadenopathy and hyphema.

Case IV - H76-461 (AFIP 1667029).
History: This was a poor-developed pig, 7 months old, weighing 24 kg in body weight. The animal was present in clinic, treated with kanamycin for one day and died on second day.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Results
AFIP Wednesday Slide Conference - No. 22
25 March 1981

Case I - 186-00386 (AFIP 1761485).

History: A 535-day-old Sprague-Dawley male rat had a 3.0 cm diameter mass on the right mandible. The mass was red and very firm. The rat was given .01 millimoles of N-hydroxy Formylaminofluorene/100 grams body weight intraperitoneally three times a week for four weeks.

Contributor's Diagnosis & Comments: Ameloblastic odontoma.

Comments: Although much of the mass is made up of dentin trabeculae lined by small basophilic cells interpreted as odontoblasts, the periphery of the section has areas containing a mixture of dentin and enamel. The dentin is eosinophilic often with internal small linear tubular structures whereas the enamel is either a darkly amphophilic amorphous substance or a lightly basophilic fibrilar material. Adjacent to the enamel in a few areas are elongate columnar epithelial cells interpreted as ameloblasts. Although odontoblasts and ameloblasts often are difficult to distinguish within the neoplasm, the presence of enamel and dentin is evidence of both cell types. Since the mass is proliferative and from a 535-day-old rat, it was interpreted as a neoplasm of mixed cell population (ameloblastic odontoma) rather than a hamartoma (congenital odontoma). Although the rat was injected with an aromatic amine, the neoplasm was considered an incidental finding.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Odontoameloblastoma.

Comments: There was general agreement with the contributor's diagnosis and comments. The morphologic diagnosis of odontoameloblastoma was adopted as classified by the WHO (5), which is equivalent to ameloblastic odontoma. A diagnosis of composite odontoma was preferred by oral pathologists upon consultation. These tumors often are confusing with considerable overlap of histologic features which sometimes makes classification difficult.

Contributor: Pathology Services Project, University of Arkansas, National Center for Toxicological Research, Jefferson, AR 72079.

References:

1. Baskin, G. B. & Hubbard, G. B.: Ameloblastic odontoma in a baboon (Papio anubis). Vet. Path. 17: 100-102, 1980.

2. Moulton, J.: Tumors in Domestic Animals, 2nd Edition, Ch. 7, 1978, pp 242-246.

3. Peter, C. P., et al.: Ameloblastic odontoma in a pony. Am. J. Vet. Res., 1495-1498, July 1968.

4. Splitter, G. A. et al.: Ameloblastic odontoma in a rhesus monkey. JAVMA 161: 710-713, 1972.

5. Head, K. W.: Tumors of the Upper Alimentary Tract. WHO Bull. 53: 145-166, 1976.

Case II - 63-1435 (AFIP 1727492).

History: Tissue from a chicken.

Contributor's Diagnosis & Comments: Multiple granulomas in the brain of a chicken probably due to Toxoplasma sp.

Comments: These granulomas once were believed to be gliomas but now are thought to represent a reactive response in the brain to infection by toxoplasma. These sections were prepared from paraffin blocks kindly provided by Dr. Cecil Jackson in 1963 and designated "gliomatosis and intergrades of disseminated encephalitis" as in reference (4). He originally thought such lesions were gliomas (3) and his term gliomatosis still connotes neoplasia which does not seem appropriate. Case submitted because of historical interest to honor a pioneer veterinary oncologist.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Encephalitis, granulomatous, multifocal, moderate to severe, brain, chicken.

Comments: A variety of preconference morphologic diagnoses were submitted including: Astrocytoma, pleomorphic and multicentric gliomas, and nonsuppurative meningoencephalitis with glial nodule formation. Following presentation and discussion there was unanimous agreement with the contributor's diagnosis. A neoplastic lesion was discredited based on the multifocal and compressile nature of the lesion, the mixed inflammatory and reactive cell population within the nodules, and the presence of meningeal and perivascular infiltrates in areas adjacent and removed from the nodules. An etiologic agent was not observed by the attendees. Several attendees listed Toxoplasmosis or avian encephalomyelitis virus as possible etiologies. As the contributor and references indicate, the exact cause is not known but toxoplasmosis is suspected. Another reference suggests A.E. as a possibility (3).

Contributor: Department of Veterinary Science, University of Wisconsin, Madison, WI 53706.

References:

1. Benirschke, K. et al.: Pathology of Laboratory Animals, Vol. I, Springer-Verlag, New York, 1978, p. 383.

2. Erichsen, S. et al.: Toxoplasmosis in chickens. II. So-called gliomas observed in chickens infected with toxoplasma. Acta Path. et Microb. Scandinavica. 33: 381-386, 1953.

3. Hemboldt, C. F. et al.: Tumors of unknown etiology. Diseases of Poultry, 7th Ed., Iowa State Univ. Press, 1978.

4. Jackson, C.: The incidence and pathology of tumors of domesticated animals in South Africa. Onderstepoort J., Vol. VI, No. 1: 323-328, 1936.

5. Jackson, C.: Ralationship of glioma to encephalitis in the domestic fowl, and an associated parasitic agent. Nature 161: 441-442, 1948.

6. Wight, P.A.L. et al.: The histopathology of epizootic gliosis and astrocytomata of domestic fowl. J. Comp. Pathol. 74: 373-380, 1964.

Case III - 80-2 (1757054).

History: Femoral bone marrow smear taken at necropsy from an adult German shepherd dog. Clinical signs included lethargy, lymphadenopathy and hyphema.

Contributor's Diagnosis & Comments: Malignant lymphoma.

Comments: This dog was one of several involved in a study of low dose radiotherapy of canine malignant lymphoma. Most dogs with marrow involvement at the time of initial workup and staging proved to be poor responders to radiotherapy.

Attendees' Diagnosis & Comments: Lymphosarcoma, lymphoblastic, bone marrow, canine.

Comments: There was unanimous agreement with the contributor's diagnosis. Differentiation of this neoplasm from other neoplasms found in bone marrow, such as myeloid or erythroid myeloproliferative disease is based on cellular morphology (2). This neoplastic involvement of the bone marrow was further characterized as stage IV lymphoblastic leukemia, due to its presence in the marrow cavity and primitive blastic appearance. The almost complete absence of myeloid, erythroid, and megakaryocytic cells was felt to be secondary to neoplastic invasion. The radiotherapy may account for some loss, particularly the megakaryocytic elements as these cells are very sensitive to ionizing radiation. Methyl green pyronine (MGP) stained bone marrow smears from this animal demonstrated pyrinophilic cytoplasmic granules. This finding is consistent with lymphocytic cells of B-cell origin which are the cells involved in the majority of leukemias in dogs and humans.

Contributor: Dr. G. A. Parker, 11101 Streamview Court, Great Falls, VA 22066.

References:

1. Miale, J. B.: Laboratory Medicine: Hematology, 5th Edition, C.V. Mosby Co., St. Louis, 1977.

2. Schalm, O. W. et al.: Veterinary Hematology, 3rd Edition, Lea & Febiger, Philadelphia, 1975.

3. Squire, R. A. et al.: Clinical and pathologic study of canine lymphoma: Clinical staging, cell classification, and therapy. J. Natl. Cancer Inst. 51: 565-574, 1973.

4. Cowall, D. E. et al.: Effects of low dose, total-body irradiation on canine bone marrow function and canine lymphoma. Radiation Res., 1981 (In Press).

5. Harvey, J. W. et al.: Well-differenitated lymphocytic leukemia in a dog: Longterm survival without therapy. Vet. Path. 18: 37-47, 1981.

Case IV - H76-461 (AFIP 1667029).

History: This was a poor-developed pig, 7 months old, weighing 24 kg in body weight. The animal was present in clinic, treated with kanamycin for one day and died on second day.

Laboratory Results: Culture of the kidney - positive for Staphylococcus sp.

Contributor's Diagnosis & Comments: Cortical necrosis, acute, severe, kidney, porcine.

Etiology: Staphylococcus sp. infection.

Comments: Gross findings: Numerous, flat erythemas were scattered throughout the skin, mainly on the ventral portion of the body, neck and head. The surface of the kidneys contained scattered diffuse pinpoint to matchhead sized reddish spots; the remainder of the surface was demarcated abruptly by a yellowish gray zone. Upon opening the kidney, the medulla was essentially normal. The cortico-medullary junction was red with congestion and hemorrhage which extended out along the vessels into the cortex in finger-like projections. The majority of the cortex was dull and pale.

Histologic findings: Marked fibrinous thrombi were present in several large vessels at the border of the infarct. These vessels also had fibrinoid necrosis of their walls. The necrosis of the cortical components consisted of glomeruli, tubules, interstitium and vessels. Few polymorphonuclear leucocytes were present at the border of the infarct.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Necrosis, acute, diffuse, severe, with multifocal hemorrhages, thrombi and fibrinoid necrosis of vessels, cortex, kidney.

Etiologic Diagnosis: Renal cortical ischemia secondary to disseminated intravascular coagulation. (DIC).

Etiology: Staphylococcus sp.

Comments: Attendees agreed with the contributor's diagnosis and comments. Disseminated intravascular coagulation was the preferred etiologic diagnosis. An etiologic agent was not apparent in the tissue sections viewed by the attendees, but they did concur with the etiology given by the contributor. Exotoxin mediated DIC was the probable pathogenesis involved in this lesion.

Contributor: Department of Veterinary Pathology, Pig Research Institute of Taiwan, Republic of China, Miaoli, Taiwan, 350.

References:

1. Allen, A. C.: The Kidney: Medical and Surgical Diseases, 2nd Edition, Grune & Stratton, New York, 1962, pp 635-640.

2. Boucot, N. G. et al.: Bilateral renocortical necrosis with recovery. New Eng. J. Med. 257: 416-418, 1957.

3. Casper, J. et al.: Bilateral cortical necrosis of kidneys in an infant with favism. Am. J. Clin. Path. 26: 42-46, 1956.

4. Hani, H. et al.: Bilateral renal cortical necrosis associated with esophagogastric ulcers in pigs. Vet. Path. 17: 234-237, 1980.

5. Thal, A.: Selective renal vasospasm and ischemic arenal necrosis produced experimentally with staphylococcal toxin. Observations on pathogenesis of bilateral cortical necrosis. Am. J. Pathol. 31: 233-259, 1955.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Histories
AFIP Wednesday Slide Conference - No. 23
1 April 1981

Case I - 771075 (AFIP 1709298).
History: Male F344 rat given Direct Blue #6 at 1500 ppm in the diet for 90 days from 6 weeks of age.

Case II - 11166-12 (AFIP 1713078).
History: This 11-month-old male American foxhound was euthanized after showing signs of ascites and anorexia. A 9-month-old female littermate had died 2 months previously. Both dogs had enlarged livers at necropsy.

Case III - EPL 89192 (AFIP 1713374).
History: Feline, 9 weeks old, female, Siamese presented as an emergency with severe dyspnea and abdominal breathing. Auscultation of the chest showed a muffled tachycardia, no audible lung sounds over the left lung fields and lung sounds limited to the dorsal posterior right lung fields. The animal died during radiographic procedures. At necropsy, there was serosanguinous fluid in the thoracic and abdominal cavities, the lungs were atelectatic, and the liver was severely congested. The heart was slightly enlarged and the endocardium of the left ventricle was thickened and white.

Case IV - EN-78P1132 (AFIP 1713947).
History: A bovine fetus was aborted alive between 220 to 235 days of gestation. The fetus was weak and experienced respiratory difficulty and died shortly after birth. Its lungs failed to collapse and were uniformly firmer than normal. All lymph nodes examined were 2 to 2.5 times normal size. The cut surface of lymph nodes revealed a thickened cortex. The adrenal glands were enlarged and the adrenal cortex was 3 times its expected thickness.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Results
AFIP Wednesday Slide Conference - No. 23
1 April 1981

Case I - 771075 (AFIP 1709298).

History: Male F344 rat given Direct Blue #6 at 1500 ppm in the diet for 90 days from 6 weeks of age.

Contributor's Diagnosis & Comments: Oval cell hyperplasia, (also in some sections - foci of cellular alteration, neoplastic nodules, and/or cholangiofibrosis.

Comments: The chemical contains benzidine, a human carcinogen. Hepatocellular carcinomas were seen in a few rats. Later they would have developed in many other rats.

Note: The slides were prepared from several different blocks. Oval cell hyperplasia is the primary lesion of interest.

Attendees' Diagnoses & Comments:

Morphologic Diagnoses: 1) Hyperplasia, bile ductules (oval cell hyperplasia), diffuse, moderate, liver, rodent, rat. 2) Neoplastic nodules, multifocal, liver. 3) Foci of hepatocellular alteration, multifocal, liver. 4) Cholangiofibrosis, multifocal, minimal to mild, liver.

Comments: The disucssion of the changes observed in the liver centered on the oval cell hyperplasia. Most investigators agree that the origin of the oval cell is the biliary epithelium and thus the preferred term bile ductule cells. Ultrastructurally these cells differ from hepatocytes by containing small oval nuclei with inconspicuous or small nucleoli, small mitochondria, rare cilia, and a basement membrane and the absence or scant amount of rough endoplasmic reticulum and glycogen granules (2). The physiologic role of the bile ductule cell is similar to the bile ducts that is water and bicarbonate absorbtion and secretion (3). The hormone secretin influences their function. Proliferation of bile ductules has been described in several pathologic conditions (3).

Contributor: National Cancer Institute, National Institutes of Health, Bethesda, Maryland 20205.

References:

1. Brooks, Stanley E. et al.: Scanning electron microscopy of proliferating bile ductules. Lab. Invest. 33: 311, 1975.

2. Grisham, J. W. et al.: Origin and fate of proliferated hepatic ductal cells in the rat: Electron microscopic and autoradiographic studies. Expt. & Molec. Path. 3: 242-261, 1964.

3. Schaffner, F. et al.: Electron microscopic studies of normal and proliferated bile ductules. Amer. J. Path. 38 (4): 393-410, April 1961.

Case II - 11166-12 (AFIP 1713078).

History: This 11-month-old male American foxhound was euthanized after showing signs of ascites and anorexia. A 9-month-old female littermate had died 2 months previously. Both dogs had enlarged livers at necropsy.

Contributor's Diagnosis & Comments: Chronic-active hepatitis associated with leptospires.

Comments: Significant findings at necropsy were an enlarged firm liver and ascites. A diagnosis of chronic-active hepatitis was made on the basis of 1) acute and chronic inflammation of periportal areas with some extension into lobules; 2) focal disruption of periportal limiting plates of hepatocytes by this inflammatory infiltrate with spotty necrosis of individual hepatocytes; 3) mild periportal fibrosis and bile ductular proliferation. The lesions in this particular dog are more acute than those in other cases reported in this series, with a greater proportion of neutrophils and little fibrosis. Abundant spirochetes were demonstrated in the liver by Warthin-Starry stain. There were no significant kidney lesions.

Attendees' Diagnosis & Comments: Hepatitis, chronic-active, diffuse, moderate, liver, American foxhound, canine.

Etiologic Diagnosis: Bacterial hepatitis.

Etiology: Leptospiria sp.

Comments: There was unanimous agreement with the contributor's diagnosis and comments. Other changes in the liver were regenerating hepatic nodules, diffuse lymphatic and multifocal sinusoidal ectasia, and thrombosis in a few large vessels in some sections. As pointed out by the contributor, Leptospiral infection may cause this hepatic lesion (3). Chronic active hepatitis also has been produced experimentally in dogs with infectious canine hepatitis virus, obstructive jaundice, and occasionally has been associated with drug administration in both humans and dogs (1,2,4). Comparatively, this lesion resembles chronic hepatitis in humans infected with hepatitis B virus.

Contributor: Division of Comparative Medicine, Johns Hopkins University School of Medicine, 720 Rutland Ave., Baltimore, MD 21205.

References:

1. Gocke, D. J. et al.: Chronic hepatitis in the Dog: The role of immune factors. JAVMA 156 (12): 1700-1705, 1970.

2. Meyer, D. J. et al.: Obstructive jaundice associated with chronic active hepatitis in a dog. JAVMA 176 (1): 41-44, 1980.

3. Strombeck, D. R. et al.: Chronic active hepatitis in the dog. JAVMA 173 (4): 380-386, 1978.

4. Bishop, L. et al.: Chronic active hepatitis in dogs associated with Leptospires. Am. J. Vet. Res. 40 (40): 839-844, 1979.

5. Toth, D. M. et al.: Drug-induced hepatitis in a dog. VM/SAC, 421-422, March 1980.

Case III - EPL 89192 (AFIP 1713374).

History: Feline, 9 weeks old, female, Siamese presented as an emergency with severe dyspnea and abdominal breathing. Auscultation of the chest showed a muffled tachycardia, no audible lung sounds over the left lung fields and lung sounds limited to the dorsal posterior right lung fields. The animal died during radiographic procedures. At necropsy, there was serosanguinous fluid in the thoracic and abdominal cavities, the lungs were atelectatic, and the liver was severely congested. The heart was slightly enlarged and the endocardium of the left ventricle was thickened and white.

Contributor's Diagnosis: Endocardial fibroelastosis.

Attendees's Diagnosis & Comments:

Morphologic Diagnosis: Fibroelastic proliferation, endocardial, diffuse, moderate, left ventricle, heart, Siamese, feline.

Etiologic Diagnosis: Feline endocardial fibroelastosis.

Etiology: Primary or secondary.

Comments: Feline endocardial fibroelastosis is characterized by diffuse fibrous and elastic thickening of the endocardium in the absence of myocarditis or myocardial necrosis. Secondary ventricular hypertrophy, usually left sided, accompanies this change. The disease may be primary or secondary. The primary form has a familial incidence (autosomal recessive gene) and most often is seen in Burmese cats. The secondary form often is associated with cardiac anomalies (i.e. aortic stenosis). The proposed pathogenesis of the primary form involves subendocardial edema, dilation of lymphatics, endocardial fibroelastic proliferation and degeneration of Purkinje fibers secondary to the fibrosis. This latter feature is the suggested cause of acute cardiac failure. This disease in cats has been compared to several diseases in man with similar histologic features.

Contributor: Experimental Pathology Laboratories, Inc., P. O. Box 474, Herndon, VA 22070.

References:

1. Eliot, T. S. et al.: First report of the occurrence of neonatal endocardial fibroelastosis in cats and dogs. JAVMA 133: 271-274, 1958.

2. Paasch, L. H. et al.: The pathogenesis of endocardial fibroelastosis in Burmese cats. Lab. Invest. 42 (2): 197-204, 1980.

3. Zook, B. C.: Some spontaneous cardiovascular lesions in dogs and cats. Adv. Cardiol. 13: 148-168, 1974.

Case IV - EN-78P1132 (AFIP 1713947).

History: A bovine fetus was aborted alive between 220 to 235 days of gestation. The fetus was weak and experienced respiratory difficulty and died shortly after birth. Its lungs failed to collapse and were uniformly firmer than normal. All lymph nodes examined were 2 to 2.5 times normal size. The cut surface of lymph nodes revealed a thickened cortex. The adrenal glands were enlarged and the adrenal cortex was 3 times its expected thickness.

Laboratory Results: Cow-positive for Brucellosis with Card Agglutination Test. Fetus - Negative card test, negative C.F. test. Elevation of Igs in serum. Recovery of B. abortus biotype I from abomasal fluids, lung, and internal iliac lymph node.

Contributor's Diagnosis & Comments: Pneumonia, diffuse interstitial; multifocal granulomatous. Bovine Brucellosis.

Comments: This case represents the typical pattern of pneumonia in B. abortus infected fetuses which abort between the 200th to 240th day of gestation. This reaction is observed in experimental infections in which the fetus is inoculated per os or I.M. Neutrophils are not a prominent cellular component of these inflammatory reactions. Occasionally a severe arteritis is noted in pulmonary arteries. In these instances macrophages are still the predominant inflammatory cell.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Bronchopneumonia, nonsuppurative, diffuse, moderate to severe, lung, fetus, bovine.

Etiologic Diagnosis: Bacterial pneumonia.

Etiology: Brucella abortus.

Comments: There was some difference in opinion on the morphologic diagnosis. The majority, however, considered the interstitial involvement to be an extension of the bronchopneumonia. The history and lesions are quite suggestive of fetal Brucella infection. Pulmonary infection is via inhalation of infectious fetal fluids in utero. Evidence for inhalation of fluids is substantiated by the presence of occasional sloughed squamous epithelial cell within airways, a feature also observed in normal fetal lung tissue. Other lesions observed in tissues of aborted calves due to Brucellosis are granulomas in the spleen, liver, and lymph nodes (1), meningoencephalitis (2), and adrenal cortical hyperplasia due to fetal stress. A necrotizing placentitis accompanies these fetal lesions.

Contributor: Louisiana State University, School of Veterinary Medicine, Department of Pathology, Baton Rouge, LA 70803.

References:

1. Jubb, K.V.F. & Kennedy, P.C.: Pathology of Domestic Animals, Vol. I, 1970, pp 528-530, 1970.

2. Wednesday Slide Conference Results #20, Case III, 1981.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Histories
AFIP Wednesday Slide Conference - No. 24
8 April 1981

Case I - 79-2231 (AFIP 1714909).
History: This is the lower jejunum of a 6-year-old female goat which had shown a progressive loss of weight, a decreased production and soft feces over a period of several weeks. Finally, the animal became emaciated and very weak and was destroyed.

Case II - Animal Health Lab (AFIP 1713317).
History: This 1-week-old pigeon was presented (live) for necropsy with a clinical history of sickness for a period of 3-4 days, would not swallow food - stuck in crop and labored breathing. The bird was euthanized and posted.

At necropsy, there were pale subcutis and musculature, edematous heart, multiple whitish nodules in the throat region, empty stomach, greatly enlarged pale liver with multiple whitish foci over the surface and kidneys and spleen were enlarged with whitish foci over the surface.

Case III - X-12280 (AFIP 1755475).
History: Tissue from a Hippocampus hudsonius, (sea horse).

Case IV - 6497-79 (AFIP 1777264).
History: Baboon, male. A traumatized tumor, 4 cm in diameter, was removed from the right pectoral region. The tumor involved cutaneous and subcutaneous tissue, was well encapsulated and vascularized. Some fibrous tissue was noted.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Results
AFIP Wednesday Slide Conference - No. 24
8 April 1981

Case I - 79-2231 (AFIP 1714909).

History: This is the lower jejunum of a 6-year-old female goat which had shown a progressive loss of weight, a decreased production and soft feces over a period of several weeks. Finally, the animal became emaciated and very weak and was destroyed.

Contributor's Diagnosis & Comments: Johne's Disease.

Etiologic Diagnosis: Mycobacterial enteritis.

Etiology: Mycobacterium paratuberculosis, Johne's disease.

Comments: At necropsy, emaciation and mild thickening of the mucosa of the lower small intestine, cecum and colon were noted. The main microscopic intestinal lesion was in the upper and middle parts of the intestinal tract. Granulomatous enteritis was mild and multifocal in the upper and middle parts of the small intestine, and diffuse and severe in the lower small intestine, cecum and colon. The lesion was limited mainly to the lamina propria of the affected intestinal segments with only mild, multifocal involvement of the submucosa. Macrophages in intestinal lesions contained massive numbers of acid fast bacilli. Johne's disease can cause important losses in some goat herds. Diagnosticians should always keep in mind that Johne's might be present in adult goats which have been suffering a [illegible] debilitating disease with or without diarrhea. Gross lesions in the intestinal tracts of these goats are often not very evident and can be missed easily. Mesenteric lymph node enlargement with or without caseous necrosis and calcification is a common finding in these animals. For these reasons, we should always examine histopathologically the intestinal tracts and mesenteric lymph nodes of these goats even when other lesions such as caseous lymphadenitis or intestinal parasitism are present.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Enteritis, granulomatous, diffuse, moderate to severe, jejunum, caprine, goat.

Comments: There was unanimous agreement with the contributor's diagnosis and comments. In addition to the features pointed out by the contributor, some sections contained crypt abscesses and considerable numbers of eosinophils and multinucleated giant cells. Lesions of Johne's disease in goats may occur in more proximal areas of the small intestine in addition to the distal small intestine, cecum and colon as observed in cattle. Lesions may contain either few or numerous acid-fast bacilli. An additional lesion occasionally observed in sheep, cattle and goats with Johne's is calcification of the endocardium of the heart and/or aorta, the pathogenesis of which has not been elucidated. Minor antigenic variations exist between bovine and caprine Mycobacterium paratuberculosis (4). Cross reaction between M. paratuberculosis, M. bovis, M. avian, and BCG have been reported. Fecal culture for M. paratuberculosis is the most reliable test for the detection of Johne's disease in ruminants.

Contributor: Dept. Pathologie et Microbiologie, Faculte de medicine veterinarie Universite de Montreal, Case postale 5000, Saint Hyacinthe, Quebec, Canada J2S 7C6.

References:
1. Bass, E. J.: Paratuberculosis (Johne's disease) in goats. Proc. Sheep and Goat Practice Symposium. Colorado State University, pp 26-40, 1976
2. Fodstad F. H. et al.: Postmortem examination in the diagnosis of Johne's disease in goats. Acta vet. scand. 20: 157-167, 1979.
3. Gunnarsson, E. et al.: Cultural and biochemical characteristics of mycobacterium paratuberculosis isolated from goats in Norway. Acta vet. scand. 20: 122-134, 1979.
4. Gunnarsson, E. et al.: Analysis of antigens in mycobacterium paratuberculosis. Acta vet. scand. 20: 200-215, 1979.
5. Jubb, K.V.F. et al.: Pathology of Domestic Animals. Vol. 2, 1970, pp 135-140.

Case II - Animal Health Lab (AFIP 1713317).

History: This 1-week-old pigeon was presented (live) for necropsy with a clinical history of sickness for a period of 3-4 days, would not swallow food - stuck in crop and labored breathing. The bird was euthanized and posted.

At necropsy, there were pale subcutis and musculature, edematous heart, multiple whitish nodules in the throat region, empty stomach, greatly enlarged pale liver with multiple whitish foci over the surface and kidneys and spleen were enlarged with whitish foci over the surface.

Laboratory Results: Beta hemolytic Streptococcus sp., E. coli and Klebsiella sp. were isolated from the lung, kidney and liver. Also yeast was isolated from the throat mass.

Contributor's Diagnosis & Comments: Myeloblastosis.
Comments: Multifocal, nodular accumulations (both intra and extravascular chiefly around portal tracts) of neoplastic cells of the granulocytic series with many mitoses and packed with acidophilic granules are compatible with granulocytic (myelogenous) leukosis. These cells also were seen in the kidney and spleen. The natural occurrence of myeloblastosis is very rare and this would be the first natural case reported in a pigeon.

Attendees' Diagnosis & Comments:
Morphologic Diagnosis: Myeloblastosis, liver, pigeon.
Comments: An inflammatory etiology was discredited on the basis of distribution, homogenicity, mitotic activity, and lack of hepatic necrosis. Similar histologic lesions also were projected involving the kidney. Attendees familiar with the normal histology of young birds stated the hepatic infiltrates were not characteristic of inflammatory lesions or extramedullary hematopoiesis. Avian leucosis virus (retrovirus) was the proposed etiologic agent and ovarian transmission the mode of infection. Viral isolation, serum antibody, and electron microscopic studies would be helpful in this and similar cases.

Contributor: Maryland Department of Agriculture, Animal Health Laboratory, 4901 Calvert Road, College Park, MD 20740.

References:

1. Purchase, H. G. et al.: Disease of Poultry, (Hafstad, et al.) 7th edition, p. 445.

2. Moulton, J. E. et al.: Tumors in Domestic Animals, 2nd edition, p. 186.

Case III - X-12280 (AFIP 1755475).

History: Tissue from a Hippocampus hudsonius, (sea horse).

Contributor's Diagnosis & Comments: Sphaeropsidales infection, probably Phoma herbarum.

Comments: This sea horse was injured by a hermit crab. This lesion developed at the site of the injury and the sea horse died. Other fish in the aquarium are normal.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Dermatitis, cellulitis, myositis, stomatitis, and panopthalmitis, subacute to chronic, multifocal to diffuse, with numerous fungal hyphae, head, Hippocampus hudsonius, sea horse.

Etiologic Diagnosis: Disseminated mycosis, head, sea horse.

Comments: Not all the described morphologic lesions were present in some of the submitted sections. The inflammatory infiltrate consisted of heterophils, and macrophages. Morphologic features of the fungus were size (2-4 u), regular septation, dicotymous branching, and parallel walls. These characteristics are suggestive of Sphaeropsidales infection, Phoma herbarum. Culture of the fungus would be required for positive identification. The fungus was considered an opportunistic pathogen as a result of the previous trauma. Numerous other species of fungi affect aquarium fishes, some as primary pathogens and others as opportunistic organisms (1,2,3).

Contributor: Department of Pathology, Division of Toxicology, Eli Lilly Company, Greenfield, IN 46140.

References:

1. Leibovitz, L. et al.: Mycotic infections. JAVMA 177 (11): 1110-1112, 19[illegible].

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

3. Wolke, R. E.: Pathology of bacterial and fungal diseases affecting fish In The Pathology of Fishes, W. E. Ribelin et al (Eds.), Univ. of Wisconsin Press, Madison, 1975, pp 33-116.

Case IV - 6497-79 (AFIP 1777264).

History: Baboon, male. A traumatized tumor, 4 cm in diameter, was removed from the right pectoral region. The tumor involved cutaneous and subcutaneous tissue, was well encapsulated and vascularized. Some fibrous tissue was noted.

Contributor's Diagnosis: Yaba tumor virus.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Proliferative fibrohistiocytosis, dermis, skin, baboon.

Etiologic Diagnosis: Yaba tumor virus.

Etiology: Pox virus.

Comments: A number of equally acceptable preconference diagnoses were submitted. These included dermal histiocytosis, histiocytic pseudotumor, and chronic ulceroproliferative dermatitis. The exact cellular derivation of cells involved was not resolved. Proliferating mesodermal cells are primarily involved. Fibrocytic cells are most often described in the literature. Variably sized eosinophilic intracytoplasmic inclusions were observed in the cells. Regression of the tumor was evidenced by the mixed inflammatory infiltrate observed multifocally. There are four pox viral diseases affecting monkeys: Small pox, monkey pox, benign epidermal monkey pox and Yaba pox. Gross and microscopic lesions of these diseases in monkeys were discussed. In addition to skin lesions, lung tumors have been produced experimentally in monkeys with aerosolized Yaba pox virus (5). Several species of Old World monkeys are susceptible to both experimental and natural infection with Yaba pox virus (3).

Contributor: AFAMRL/THP, Wright Patterson AFB, OH 45433.

References:

1. Green, D. E.: Spontaneous viral disease of nonhuman primates. Pathology of Laboratory Animals Course, AFIP, 1980.

2. Kupper, J. L. et al.: Experimental Yaba and benign epidermal monkey pox in rhesus monkeys. Lab. Anim. Care 20 (5): 979-988, 1970.

3. Niven, Janet S. F. et al.: Subcutaneous "growths" in monkeys produced by a poxvirus. J. Pathol & Bacteriol. 81 (1): 1-14, 1961.

4. Wolfe, L. G. et al.: Immunologic response of monkeys to aerosols of Yaba virus. JNCI 41 (5): 1197-1202, 1968.

5. Wolfe, L. G. et al.: Experimental aerosol transmission of Yaba virus in monkeys. JNCI 41 (5): 1175-1195, 1968.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Histories
AFIP Wednesday Slide Conference - No. 25
15 April 1981

Case I - 8776 (1 H&E, 1 Warthin Starry) (AFIP 1771334).

History: Intermittent respiratory disease occurred in a breeding colony of LEW rats despite occasional treatment with tetracycline over a period of years. Dyspnea, unthriftiness, deaths during anesthesia, infertility and occasional stillbirths had been noted. Five adult and 4 weanling rats showing dyspnea were submitted for evaluation. 2 microslides included.

Case II - LAIR 28933 (AFIP 1758835).

History: This female rhesus monkey was used as a breeder. She arrived in the colony in April 1976. She was bred on 28 April 1978 and aborted on 13 July 1978. Tuberculin tests had been completely negative until 3 Oct 78 when there was a moderate swelling of the palpebral injection site. An intradermal test on the abdomen was negative on 18 Oct 78. On 28 Dec 78 the animal was noted to be severely ill. Thoracic radiographs revealed numerous densities in the lungs. The animal was submitted for necropsy.

Case III - 5737 (AFIP 1756981).

History: Thirty out of 40, 2-month-old turkeys, died suddenly over 2 days. All turkeys were raised in one building and had access to old chicken and duck yards.

Case IV - 78-R-245 (AFIP 1708983).

History: A 2.5 year old crl:CBS^{R} CD^{R} (SD) rat used for geriatric studies

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Results
AFIP Wednesday Slide Conference - No. 25
15 April 1981

Case I - 8776 (1 H&E, 1 Warthin Starry) (AFIP 1771334).

History: Intermittent respiratory disease occurred in a breeding colony of LEW rats despite occasional treatment with tetracycline over a period of years. Dyspnea, unthriftiness, deaths during anesthesia, infertility and occasional stillbirths had been noted. Five adult and 4 weanling rats showing dyspnea were submitted for evaluation. 2 microslides included.

Laboratory Results: Gross and/or microscopic lesions of MRM in varying degrees of severity were present throughout the respiratory tracts of all animals. Some females had suppurative salpingitis and perioophoritis. Mycoplasma pulmonis was isolated from all respiratory tracts and genital lesions. Virus serologies were positive for Sendai, rat coronavirus and Kilham rat virus. Pseudomonas aeruginosa and Pasteurella pneumotropica were isolated from trachea of a few rats.

Contributor's Diagnosis & Comments: Murine respiratory mycoplasmosis, advanced, with associated filamentous bacterium, lung.

Comments: Subsequent to the study by van Zwieten, et al. (1), the contributor has studied two additional rat colonies with similar clinicopathologic findings. The agents common to all three were: M. pulmonis, Sendai virus and the unclassified filamentous bacterium. The precise role (s) of each of these agents and environmental factors in pathogenesis of the respiratory disease have yet to be defined. M. pulmonis may have been responsible for the observed infertility and stillbirths, but rat virus also may have contributed.

The slide submitted is a section of lung with lesions of severe, advanced MRM, including: bronchiectasis, bronchiolectasis, lung abscesses, and empnysema. The Warthin Starry stained section demonstrates the silver positive filamentous bacterium in large numbers on relatively normal respiratory epithelium.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Bronchitis and bronchiolitis exudative with bronchiectasis, bronchiolectasis, chronic, multifocal, severe, associated with filamentous bacteria, lung, LEW rat, rodent.

Etiologic Diagnosis: Murine respiratory mycoplasmosis, (MRM).

Etiology: Mycoplasma pulmonis and unidentified filamentous bacteria.

Comments: There was unanimous agreement with the contributor's diagnosis and comments. The synergism between Sendai virus, Mycoplasma pulmonis and the filamentous bacterial organisms was discussed. As pointed out by the contributor the significance of the presence of the bacteria is unknown. Attendees proposed at least a mechanical obstruction of the cilia and thus a defective clearing of the airways by the bacteria, if not a direct pathogenic role in the lesions produced. The gross and microscopic lesions of acute and chronic pulmonary MRM were discussed. Synergism between ammonia levels and mycoplasmosis in rats has been established. Interactions with other agents such as various bacteria and viruses has not been clearly elucidated. In addition to the lesions produced in the pulmonary and reproductive tracts, rhinitis, otitis media, laryngitis, and tracheitis are also reported in rats infected with M. pulmonis.

Contributor: Department of Comparative Pathology, University of Alabama, School of Medicine and Dentistry, University Station, Birmingham AL 35294.

References:

1. van Zwieten, M. S. et al.: Respiratory disease in rats associated with a filamentous bacterium: A preliminary report. Lab. Anim. Sci. 30: 215-221, 1980.

2. Lindsey, J. R. et al.: Diseases due to mycoplasmas and rickettsias. Chapt. 15, IN Pathology of Laboratory Animals, Benirschke, K., Garner, F. M. and Jones, T. C. (Eds), Springer-Verlag, N.Y., 1979, pp 1481-1550.

3. Cassell, G. H. et al.: Mycoplasmal and rickettsial diseases. Chapt. 10, IN The Laboratory Rat, Vol. I, Baker, Lindsey & Weisbroth, (Eds.), Academic Press, N. Y., 1979, pp 243-269.

4. Jacoby, R. O. et al.: Viral diseases. Chapt. 11, IN The Laboratory Rat, Vol. I, Baker, Lindsey & Weisbroth, (Eds), Academic Press, N. Y., 1979, pp 271-306.

Case II - LAIR 28933 (AFIP 1758836).

History: This female rhesus monkey was used as a breeder. She arrived in the colony in April 1976. She was bred on 28 April 1978 and aborted on 13 July 1978. Tuberculin tests had been completely negative until 3 Oct 78 when there was a moderate swelling of the palpebral injection site. An intradermal test on the abdomen was negative on 18 Oct 78. On 28 Dec 78 the animal was noted to be severely ill. Thoracic radiographs revealed numerous densities in the lungs. The animal was submitted for necropsy.

Contributor Diagnosis & Comments:

Morphologic Diagnosis: Metritis and salpingitis, granulomatous, chronic, multifocal to diffuse, severe, uterus and oviducts, rhesus monkey due to mycobacterium tuberculosis.

Comments: Gross and microscopic lesions of tuberculosis were found in prescapular, bronchial, sublumbar and mesenteric lymph nodes, lung, liver, spleen, both kidneys, pericardium, myocardium, uterus, oviducts and colon. The CNS was not examined. M. tuberculosis (human strain) was isolated from lung, liver and spleen. This case is interesting in several respects. The tuberculin test was suspicious but not considered positive, two months before the animal's death. The systemic spread of the disease was very rapid since no clinical signs were noted until shortly before death. The monkey had been in an indoor breeding colony for two years prior to its terminal illness. During this time, the colony was "closed". No new animals were introduced and none had positive tuberculin tests. From the time of this animal's death (Dec 1978) until the present (Aug 1980) no newer animals in this colony have had positive tuberculin tests or clinical signs of illness.

The history of the colony and affected animal suggests that the animal had the disease in an "inactive" form from the time of its arrival. Perhaps immunologic suppression related to the pregnancy allowed exacerbation of the disease.

Attendees' Diagnoses & Comments:

Morphologic Diagnoses: 1) Metritis, salpingitis and oophoritis, granulomatous, multifocal, chronic, moderate, uterus, oviduct and ovary, Macaca mulatta, rhesus monkey, nonhuman primate. 2) Endometritis, suppurative, multifocal, mild, uterus.

Etiologic Diagnosis: Uterine and ovarian mycobacteriosis.

Etiology: Mycobacterium tuberculosis.

Comments: The additional morphologic diagnosis of suppurative endometritis was listed by some participants based on histologic evidence of ulceration of the mucosa and accompanying neutrophilic response. This was only present in some sections and the etiology was not resolved. Foci of Splendore-Hoeppli phenomenon was also observed in some sections, which is not typical of tuberculosis lesions. Attendees proposed a concurrent infection with other organisms. Differentially, along with M. tuberculosis, such agents as Actinomycosis, Nocardiosis, Yersiniosis and unidentified mycotic agents were suggested, however, such organisms could not be demonstrated. Other granulomatous lesions described by the contributor and observed by the attendees were felt to be quite characteristic of tuberculosis in monkeys. A special stain provided by the contributor demonstrated occasional acid-fast organisms within the granulomas.

Contributor: Letterman Army Institute of Research, SGRD-ULV-P, Presidio of San Francisco, CA 94129.

Reference:

Carlton, W. W. & Hunt, R. D.: Bacterial diseases. IN Pathology of Laboratory Animals, Benirschke, K. (ed), 1978, pp 1370-1371, 1419-1421 & 1463-1466.

Case III - 5757 (AFIP 1756981).

History: Thirty out of 50, 2-month-old turkeys, died suddenly over 2 days. All turkeys were raised in one building and had access to old chicken and duck yards.

Contributor's Diagnosis: Hepatitis, necrotizing, diffuse, severe, etiology - Histomonas meleagridis.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Hepatitis, necrotizing, subacute, disseminated, severe, liver, turkey, avian.

Etiologic Diagnosis: Hepatic Histomoniasis.

Etiology: Histomonas meleagridis.

Comment: The lesions in this case were felt to be an excellent example of the disease. Multinucleated giant cells observed occasionally by attendees prompted a morphologic diagnosis of necrogranulomatous hepatitis by some. Characteristic PAS positive organisms were projected from similar lesions of avian histomoniasis. The morphology and life cycle of the organism and the importance of the cecal worm Heterakis gallinarum in the transmission of Histomonads was discussed. The significance of cohabitation of turkeys and chickens, or exposure of turkeys to areas formerly occupied by chickens in the transmission of the disease was also discussed (2).

Contributor: University of Illinois, Department of Veterinary Pathobiology & Laboratories of Diagnostic Medicine, P. O. Box U, Urbana, IL 61801.

References:

1. Dhillon, A. S. et al.: Atypical histomoniasis in Bobwhite quail. Avian Dis. 24 (2): 510-516, 1979.

2. Kemp, R. L. and Springer, W. T.: Histomoniasis. IN Diseases of Poultry, Ed. Hofstad, Iowa State University Press, Ames, 1978, pp 832-840.

Case IV - 78-R-245 (AFIP 1708983).

History: A 2.5 year old crl:CBS^R CD^R (SD) rat used for geriatric studies.

Contributor's Diagnosis: Testicular interstitital (Leydig) cell tumor.

Attendees' Diagnoses & Comments:

Morphologic Diagnoses: 1) Interstitital cell tumor, testicle, Sprague-Dawley rat, rodent. 2) Atrophy, tubular, diffuse, moderate, seminiferous tubules, testicle.

Comments: There was unanimous agreement with the contributor's diagnosis. The neoplastic cells were readily identified as interstitial (Leydig) cells by attendees. Multifocal hemorrhages and lymphocytic infiltrates were also observed in some sections. The strain incidence of spontaneous tumors and experimental production of these tumors was discussed. The incidence of spontaneous I.C.T.'s may reach 80-90% in aged male Fischer 344 rats, whereas the incidence may be as low as 0.4% in Osborne-Mendel rats. Experimentally, vascular occlusion, cadmium salts and estrogens have also been shown to produce I.C.T.'s in rats and mice.

Contributor: The Pennsylvania State University, The Milton S. Hersney Medical Center, Hershey, PA 17033.

References:

1. Goodman, D. G. et al.: Neoplastic and nonneoplastic lesions in aging F344 rats. Toxicol. & Appl. Pharmacol. 48: 237-248, 1979.

2. Goodman, D. G. et al.: Neoplastic and nonneoplastic lesions in aging Osborne-Mendel rats. Toxicol. & Appl. Pharmacol. 55: 433-447, 1980.

3. McConnell, E. E. & Gardner, D. M.: Comparison of neoplasms in the ovaries of rats. J. Natl. Cancer Inst. 50, 1259, 1973.

4. Thompson, S. W. et al.: Spontaneous tumors in the Sprague-Dawley rat. J. Natl. Cancer Inst. 27: 1017, 1961.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Histories
AFIP Wednesday Slide Conference - No. 26
22 April 1981

Case I - 79-6573 (AFIP 1758840).
History: Progressive ascending paralysis in a 3-year-old female Doberman.

Case II - 79-10197 (AFIP 1711324).
History: Tissue from a 1-year-old imported Fennec fox.

Case III - 80-241 (AFIP 1777001).
History: Juvenile male Macaca fascicularis was found with aggressive cagemate. He was found comatosed and hypothermic. He responded to IV glucose but, two days later he refused food, was hypothermic and severely depressed. He was euthanized. Gross lesions were severe cachexia, severe congestion and edema of the lungs, and mild black discoloration of the liver.

Case IV - 80-708-7/8 (AFIP 1757060).
History: Three female golden hamsters (Mesocricetus auratus) 8-9 months old died or were found moribund 2-3 weeks after producing litters. These animals belonged to a small inbred breeding colony. No known pathogen was present in the colony nor were animals exposed to experimental antigens. (2 microslides - 1 H&E, 1 Congo red).

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Results
AFIP Wednesday Slide Conference - No. 26
22 April 1981

Case I - 79-6573 (AFIP 1758840).

History: Progressive ascending paralysis in a 3-year-old female Doberman.

Laboratory Results: Postmortem - extensive diffuse nonsuppurative meningoencephalomyelitis with numerous toxoplasma-like cysts in the brain and spinal cord.

Contributor's Diagnosis: Toxoplasmosis.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Meningoencephalomyelitis, granulomatous, subacute to chronic, multifocal, moderate to severe, brain and spinal cord, Doberman, canine.

Etiologic Diagnosis: Protozoal meningoencephalomyelitis.

Etiology: Toxoplasma sp.

Comments: There was general agreement with the contributor's diagnosis. However, the majority of participants favored a granulomatous form of nonsuppurative meningoencephalomyelitis or meningoencephalitis with associated areas of neuropil destruction and collapse (malacia). Not all the slides contained sections of spinal cord. Protozoal cysts consistent with toxoplasmosis were present in most, if not all sections. Some attendees reported intense aggregates of eosinophils in several foci. The inflammatory response was considered typical for toxoplasmosis, although granuloma formation also is reported (1). Multiple system involvement especially pulmonary is commonly encountered with toxoplasmosis (2). The life cycle was reviewed briefly. The most common routes of infection are ingestion and transplacental.

Contributor: B. C. Department of Agriculture, B.C. Box 100, Abbotsford, British Columbia, Canada.

References:

1. Averill, D. R. Jr. & deLahunta, A.: Toxoplasmosis of the canine nervous system: Clinicopathologic findings in four cases. JAVMA 159: 1134-1141, 1971.

2. Jubb, K.V.F. & Kennedy, P. C.: Pathology of Domestic Animals, Vol. 2, Academic Press, New York, 1970, pp 673-677.

Case II - 79-10197 (AFIP 1711324).

History: Tissue from a 1-year-old imported Fennec fox.

Contributor's Diagnosis & Comments: Granulomatous hepatitis due to Mycobacterium sp.

Comments: Numerous acid-fast bacilli compatible with Mycobacterium sp. were present within macrophages in the liver.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Granulomas, multiple, disseminated, severe, liver, Fennec fox (Fennecus zerda), canidae.

Etiologic Diagnosis: Hepatic mycobacteriosis.

Etiology: Mycobacterium sp.

Comments: Single and coalescing granulomas containing numerous acid-fast bacilli were demonstrated. Caseation necrosis was not a prominent feature in this case. The large number of organisms present suggested infection with M. bovis or M. avium. Dogs are susceptible to infection with M. bovis and M. tuberculosis and are also susceptible but more resistent to infection with M. avium and atypical mycobacteriosis (1). Horses and dogs are reported to respond to mycobacteriosis with sarcomatous lesions whereas humans and bovines exhibit the more classical tubercle formation with caseation necrosis and calcification (3). The pathogenesis of mycobacteriosis in the canidae was discussed. This case is very similar to one recently reported involving several Fennec foxes (2).

Contributor: Livestock Disease Diagnostic Center, Route #6, Newtown Pike, Lexington, Ky 40505.

References:

1. Friend, S.C.E. et al.: Infection of a dog with Mycobacterium avian serotype II. Vet. Path. 16: 381-384, 1979.

2. Himes, E. M. et al.: Tuberculosis in Fennec foxes. JAVMA 177: 825-826, 1980.

3. Jubb, K.V.F. & Kennedy, P.C.: Pathology of Domestic Animals, Vol. I, Academic Press, New York, 1970, pp 241-248.

Case III - 80-241 (AFIP 1777001).

History: Juvenile male Macaca fascicularis was found with aggressive cagemate. He was found comatosed and hypothermic. He responded to IV glucose but, two days later he refused food, was hypothermic and severely depressed. He was eutnanized. Gross lesions were severe cachexia, severe congestion and edema of the lungs, and mild black discoloration of the liver.

Contributor's Diagnosis & Comments: Malaria, chronic, moderate, P. knowlesi.

Comments: The malaria in this monkey did not cause his death but possibly contributed indirectly. P. knowlesi usually is clinically silent and overt disease can result from stress or immune suppression. On questioning the handlers it was revealed that this monkey was dominated by his cage mate and denied food. This resulted in cachexia and hypoglycemic crisis. The terminal disease resulted from a severe, peracute, septic, thrombo-embolic pneumonia and myocarditis. The only explanation found for this is a nosocomial infection associated with parenteral therapy. There also was immunosuppression; lymphoid follicles in both the spleen and lymph nodes were atrophic, probably a result of the food deprivation perpetrated by his aggressive cage mate. Other lesions noted were chronic glomerulonephritis of an immune complex type, R.E. hyperplasia of lymph nodes and liver, pigment deposition in Kupffer cells and spleen, and focal acute hydropic degeneration of the liver. Speciation of the parasite was performed at S.W. Foundation as "Most likely P. knowlesi".

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Parasitemia, erythrocytic, mild to moderate, Macaca fascicularis, nonhuman primate.

Etiologic Diagnosis: Malaria.

Etiology: Plasmodium knowlesi.

Comments: In the majority of the smears only the trophozoite form of the parasite could be identified. Fewer smears contained both trophozoites and schizonts. All of the smears contained parasitized red blood cells and fine yellow-brown granular intraerythrocytic pigment. Specific identification of the species of Plasmodium could not be made as there are a number of Plasmodia which infect Asian monkeys. Differentially Hepatocystis sp. must be included because parasitized red blood cells also contain trophozoites and gametocytic forms and pigment, however, erythrocytic schizogany does not occur. The major gross and histopatological lesions of malaria in monkeys were discussed (4). Some of the immunological parameters of the disease also were reviewed (2).

Contributor: University of Texas, Medical School at Houston, 6431 Fannin, Houston, TX 77030.

References:

1. Coatney, G. R. et al., Editors: The Primate Malarias, U. S. DHEW Publication #1744-0005, Bethesda, MD 20014.

2. Immunology of Malaria. Bull. WHO 57 (Suppl 1): 277-290, 1979.

3. Loeb, W. F. et al.: Hematologic disorders. In Pathology of Laboratory Animals, Benirschke, K. et al., Ed., Springer-Verlag, N. Y., 1978, p. 1004.

4. Voller, A.: Plasmodium and hepatocystis. In Pathology of Simian Primates Part II, Karger, Basel, 1972, pp 57-73.

Case IV - 80-708-7/8 (AFIP 1757060).

History: Three female golden hamsters (Mesocricetus auratus) 8-9 months old died or were found moribund 2-3 weeks after producing litters. These animals belonged to a small inbred breeding colony. No known pathogen was present in the colony nor were animals exposed to experimental antigens. (2 microslides - 1 H&E, 1 Congo red).

Contributor's Diagnosis & Comments: 1) Kidney, amyloidosis, moderate to severe. 2) Liver, amyloidosis. 3) Spleen, amyloidosis.

Comments: Unfortunately H&E and Congo red sections are not from the same animal, although lesions are similar. Regretably, all slides do not contain kidney, liver and spleen. Spontaneous generalized amyloidosis is common in older hamsters (greater than one year). Lesions of this severity are unexpected in animals of this age without experimental induction.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Amyloidosis, diffuse, moderate to severe, kidney, liver and spleen, Golden hamster (Mesocricetus autatus), rodent.

Comments: There was unanimous agreement with the contributor's diagnosis and comments. Diffuse glomerular and interstitial amyloid deposits were present in the kidney sections. Similar diffuse deposits also were present in the spleen and liver. A few attendees reported centrilobular necrosis and inflammation of unknown etiology in some liver sections. The incidence of spontaneous amyloidosis in the hamster is strain dependent and may reach 90% at 18 months of age in some strains (2). Deaths in these animals is attributed to renal failure secondary to amyloid deposition. Polycystic kidneys also occur in aged hamster with amyloidosis. Spontaneous amyloidosis may interfere with interpretation of experimental procedures in hamsters.

Contributor: Department of Pathology, College of Veterinary Medicine, Kansas State University, VCS Bldg., Manhattan, KS 66506.

References:

1. Chai, C. K.: Spontaneous amyloidosis in LLC mice. Am. J. Path. 90: 381-386, 1978.

2. Gleiser, C. A. et al.: Amyloidosis and renal paramyeloid in a closed hamster colony. Lab. Anim. Sci. 21: 197-202, 1971.

3. Glenner, G. G.: Amyloid deposits and amyloidosis, Parts 1 & 2, N.E.J.M. Vol. 302 (23): 1283-1292, Vol. 302 (24): 1333-1343, 1980.

4. Gruys, E. et al.: Deposition of amyloid in the liver of hamsters: An enzyme-histochemical and electron microscopy study. Lab. Anim. 13: 1-9, 1979.

5. Jakob, W.: Spontaneous amyloidosis of mammals. Vet. Path. 8: 292-306, 1971.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Histories
AFIP Wednesday Slide Conference - No. 27
29 April 1981

Case I - 79050 - (AFIP 1771039).
History: Canine, 3 years old, decreased endurance, cough, on exercise.

Laboratory Data:

WBC	16,400
Segs	11,480
Bands	0
Lymphs	2,114
Eosinophils	2,132
Monos	246
Basophils	328
Few Immunocytes	

Urinalysis Sp Gv	1.032
pH	6.2
Protein	2+
No significant sediment	
BUN	30
SGPT	30
BSP	7.5% retention 30 min
Knotts test:	Negative

Case II - 13576 (AFIP 1761484).
History: This was an approximately 4-year-old female rhesus monkey that was found prostrate with bloody diarrhea and died before a clinical examination could be performed.

Case III - 19643-B (AFIP 1783172).
History: Tissue from a 1 X 1 X 1.25 cm mass located on the cranial pole of the right kidney of a 175-day-old Spartan rat.

Case IV - BG-91 (AFIP 1783252).
History: This slide represents an incidental finding in a 6-year-old monkey (Macaca fascicularis).

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Results
AFIP Wednesday Slide Conference - No. 27
29 April 1981

Case I - 79050 - (AFIP 17/1039).

History: Canine, 3 years old, decreased endurance, cough, on exercise.

Laboratory Data:

WBC	16,400	Urinalysis Sp Gv	1.032
Segs	11,480	pH	6.2
Bands	0	Protein	2+
Lymphs	2,114	No significant sediment	
Eosinophils	2,132	BUN	30
Monos	246	SGPT	30
Basophils	328	BSP	7.5% retention 30 min.
Few Immunocytes		Knotts test:	Negative

Contributor's Diagnosis & Comments:

Morphologic: 1) Pneumonitis, interstitial, multifocal, chronic, eosinophilic moderate with an occasional microfilarial fragment present in the lesions. 2) Endarteritis, proliferative, villous, moderate, chronic lobar branches pulmonary artery. 2) Hyperplasia, medial, small pulmonary arteries, minimal, lung.

Comment: Thirty-four mature adult Dirofilaria were removed from the pulmonary artery and right ventricle at necropsy. In this study 20% of 34 infected dogs had cryptic or occult infections. Although the females were gravid and producing microfilariae they were being sequestered and destroyed in the pulmonary circulation. The eosinophilia and basophilia are consistent with chronic parasitism and immunocytes indicate probable antigenic stimulation. Proteinuria in the absence of significant urine sediment indicates glomerular dysfunction. Renal function is adequate, i.e., ability to concentrate urine and lack of azotemia. Increased BSP retension suggested diminished hepatic blood flow due to chronic passive congestion.

Attendees' Diagnoses & Comments:

Morphologic Diagnoses: 1) Proliferation, villous, intimal, multifocal, moderate, pulmonary artery, lung, canine. 2) Pneumonia, interstitial, eosinophilic, subacute to chronic, multifocal, minimal, lung. 3) Hyperplasia, medial, multifocal, minimal, pulmonary arteries, lung.

Etiologic Diagnoses: 1) Parasitic (dirofilarial) endarteritis. 2) Parasitic (dirofilarial) interstitial pneumonia. 3) Increased pulmonary resistance.

Comments: There was general agreement with the contributor's diagnoses and comments. Attendees observed that the lung had been perfused. Abnormal clinical pathology findings were leucocytosis which included absolute neutrophilia, eosinophilia, and basophilia; increased BSP retention; slightly elevated SGPT and a mild proteinuria. The eosinophilic interstitial pneumonia, microgranulomas and microfilaria were not reported by most participants in their preconference diagnoses. The prominant collateral circulation surrounding the pulmonary artery was presumed to be due to increased pulmonary resistance. The pathogenesis of the lesions were discussed. Several attendees mentioned that an immune complex mediated disease participated in the development of the arterial lesion, as is the case in the lesions observed in membranous glomerulonephritis associated with dirofilariasis. Other lesions observed with dirofilariasis in the dog are infarcts of the brain, heart, kidney and other organ and saddle thrombi.

Contributor: Animal Resources Branch, Div. Veterinary Medical Research, FDA, Beltsville, MD 20705.

References:

1. Adcock, J. L.: Pulmonary arterial lesions in canine dirofilariasis. J. Vet. Res. 22: 655-662, 1961.

2. Munnell, J. F. et al.: Intimal lesions of pulmonary artery in dogs with experimental dirofilariasis. Am. J. Vet. Res. 41: 1108-1112, 1980.

3. Rawlings, C. A. et al.: The response of the canine heart and lungs to Dirofilaria immitis. JAAHA 14: 17-32, 1978.

4. Weil, G. J. et al.: Dirofilaria immitis; Parasite specific humoral and cellular responses in the experimentally infected dog. Exp. Parasitol. 51: 80-86, 1981.

5. Whong, Ming M.: Experimental occult dirofilariasis in dogs with refetence to immunologic response and its relationship to tropical eosinophilia in man. S.E. Asian J. Trop. Med. & Public Hlth. 5: 480-486, 1974.

Case II - 13576 (AFIP 1761484).

History: This was an approximately 4-year-old female rhesus monkey that was found prostrate with bloody diarrhea and died before a clinical examination could be performed.

Contributor's Diagnoses & Comments:

Syndrome - cerebral venous thrombosis. BV - Brain - cerebral venous thrombosis. Brain - perivenular demyelination. Brain - gemistocytic astrocytosis. Brain - malacia.

Comments: Cerebral venous thrombosis (CVT) was identified in 4 rhesus monkeys. Two animals initially exhibited neurologic signs and 3 had diarrhea or dysentery. All 4 cases had severe intestinal disease, including 3 cases of ulcerative colitis; these may be related to an underlying coagulopathy. Central nervous system lesions were confined to the centrum semiovale and consisted of multiple thrombosis of internal cerebral veins, perivenular demyelination, and gemistocytic astrocytosis. The lesions were similar to those found in humans with CVT and support the hypothesis that perivenular demyelination may occur as a sequela to venous occlusion. Moreover, the lesions appeared to be identical to the pathologic changes found in "leucoencephalosis and perivascular myelosis" an entity of unknown etiology previously described in monkeys.

Attendees' Diagnoses & Comments:

Morphologic Diagnoses: 1) Thrombosis, venous, subacute, multifocal, mild to moderate, cerebral cortex, brain, rhesus monkey, (Macaca mulatta), nonhuman primate 2) Degeneration, white matter, perivenular, subacute to chronic, multifocal, moderate, midbrain and cerebral cortex, brain. 3) Mineralization, vascular, multifocal, mild, meninges, midbrain and cerebral cortex, brain.

Etiologic Diagnosis: Cerebral venous thrombosis.

Etiology: Unknown.

Comments: Not all of the lesions above were present in all of the sections. The venous thrombosis and mineralization were absent in a few slides but the perivenular white matter degeneration, gemistocytosis and astrocytosis were present in all of the sections studied by the attendees. Capillary thrombosis and sludging was observed by some. Differentially the attendees' etiologic diagnoses included Lead poisoning, leukoencephalosis and disseminated intravascular coagulation. Lead poisoning could not be ruled out, but the presence of thrombosis and lack of capillary proliferation does not fit the described lesions of this condition.

Leukoencephalosis, a disease in monkeys of unknown etiology was plausable, however, thrombosis is not reported, but as the contributors point out in their published report (2), this may have been missed in the original descriptions of the disease (3). The participants drew an association between the bloody diarrhea and the lesions in the brain. A coagulopathy (DIC) was proposed as the cause secondary to a bacterial enteritis/colitis such as shigellosis. At the close of the discussion the etiology still remained unresolved.

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

References:

1. Averback, P.: Primary cerebral venous thrombosis in young adults: The diverse manifestations of an underrecognized disease. Ann. Neurol. 3: 81-85, 1978.

2. Sheffield, W. O. et al.: Cerebral venous thrombosis in the rhesus monkey. Vet. Path. 18: 326-334, 1981.

3. Van Bogaert, L. et al.: Neurologic disease of apes and monkeys. In Comparative Neuropathology, Saunders, L. Z., Ed., Academic Press, New York, 1961, pp 67-75.

Case III - 19643-B (AFIP 1783172).

History: Tissue from a 1 X 1 X 1.25 cm mass located on the cranial pole of the right kidney of a 175-day-old Spartan rat.

Contributor's Diagnosis & Comments: Nephroblastoma, right kidney, Spartan rat. Also indiscriminately classified as an embryonal carcinoma, embryonal cell tumor, adenosarcoma and Wilms' tumor, the nephroblastoma appears to be the most commonly found primary renal tumor in rats.

Attendees' Diagnoses & Comments:

Morphologic Diagnoses: 1) Nephroblastoma, kidney, Spartan rat. 2) Glomerulonephrosis, chronic, multifocal, mild, kidney.

Comments: There was unanimous agreement with the contributor's diagnosis of nephroblastoma. Attendees also included the diagnosis of glomerulonephrosis as lesions consistent with this entity were present in many of the slides. Nephroblastomas are the most common spontaneous primary renal neoplasm of the rat kidney. They are presumed to arise from the metanephric blastema. This tumor is contrasted to the sarcomatoid tumor of the rat which is less frequently encountered spontaneously and can be induced chemically (2). The features of the latter tumor are composition entirely of mesenchymal tissue elements including fibroblastic, smooth muscle, embryonic mesenchyme cells, adipose and osteoid tissue (2,3). Preexisting renal tubules and glomeruli may be engulfed by the sarcomatoid tumor cells giving the initial appearance of a nephroblastoma (2).

Contributor: Bureau of Foods, Division of Pathology, Food & Drug Administration, 200 C Street., S.W., Washington, DC 20204.

References:

1. Benirschke, K. et al.: Pathology of Laboratory Animals, Vol. II, Springer-Verlag, New York, 1978, pp 1170-1171.

2. Hard, G. C. et al.: Nephroblastoma in the rat: Histology of a spontaneous tumor, identity with respect to renal mesenchymal neoplasms, and a review of previously recorded cases. J. Natl. Cancer Inst. 57: 323-329, 1976.

Case IV - BG-91 (AFIP 1783252).

History: This slide represents an incidental finding in a 5-year-old monkey (Macaca fascicularis).

Contributor's Diagnosis & Comments: Pentastomiasis.

Comments: The Pentastomida constitute an aberrant group of nelmintns which have lost most affinities to other major classes (1). Modern studies suggest that the pentastomes share both arthropod and annelid characters but it can not be justified to assign them to either phylum. They are, therefore, assigned the status of an independent phylum (1).

There are two orders. The Cephalobaenida are the more primitive and their life cycles involve insects, fish, amphibia, and reptiles as secondary host (1). Since some in this order can presumably complete direct life cycles in a single reptilian host species, they are considered the most primitive (1). The Porocephalida, excepting one species, require mammals as secondary and reptiles as definitive hosts (1).

Pentastomes are characterized by having: 1) an unsegmented body; 2) metamerically arranged musculature; 3) chitinous cuticle with numerous glands; and 4) neither a circulatory nor a respiratory system. Fish, mice, primates, and opossum are common intermediate hosts for parasites of reptiles while herbivorous mammals are the usual intermediate hosts of Linuatula serrata, a pentastome of dogs and occasionally man. Nymphal stages are commonly found in the lymph nodes, but rarely in the liver of cattle; in the liver of humans; and in the lymph nodes, lungs and omentum of primates (2).

At least two of the following microscopic characteristics should be present on the sections submitted. 1) Pseudosegmented body; 2) striated metamerically arranged muscles; 3) various acidophilic glands; 4) cuticle that may be spiny, smooth, or show signs of annulation with pit-like sclerotized openings to skin glands; 5) intestine with numerous villi involving supporting or connective tissue or 6) two pairs of hooks in the head region.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Pentastome parasite, periadrenal fat, Macaca fascicularis, nonhuman primate.

Etiologic Diagnosis: Pentastomiasis.

Etiology most probably Armillifer armulatus.

Comments: There was unanimous agreement with the contributor's diagnosis. The contributor's comments and references listed were felt to be a comprehensive review of parasite host relationship.

References:

1. Self, J. T.: Biological relationships of the Pentastomida: A bibliography on the Pentastomida. Exp. Parasitol. 24: 63-119, 1969.

2. Chitwood, M. et al.: Identification of parasitic metazoa in tissue sections. Exp. Parasitol. 32: 407-519, 1972. See pp 415-419.

3. Noble, E. R. et al.: Parasitology: The Biology of Animal Parasites, Lea & Febiger, Philadelphia, 1976, pp 407-410.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Histories
AFIP Wednesday Slide Conference - No. 28
13 May 1981

Case I - 80-212 (AFIP 1757728).
History: Tissue from a 4-year-old female Cuban hutia (Capromys pilorides) that was acquired from a zoo in Florida five months earlier and recently became anorectic and showed weight loss.

Case II - 15278 (AFIP 1764738).
History: Aged (11 years +) male, ringneck duck (Aythya collaris) was being readied for shipment to another zoo. Weight dropped from 750 g to 600 g in recent months. Routine blood work revealed elevated white count. No other clinical signs were noted. Bird was placed on antifungal agents and was found dead three weeks after the initial presentation.

Laboratory Results:	Initial	1 week later
Total WBC	35,900/mm^3	30,500/mm^3
Seg.	92%	93%
Lymph.	8%	7%
Hematocrit	35%	30%
Total protein	4.8 gm/dl	4.4 gm/dl

Case III - 80-002 (AFIP 1783250).
History: Mice, B6C3F1 (C57BL X C3H), females, 25 months old.
Mice were sacrificed after 2 years on a dietary chronic toxicity study. (In order to provide enough slides of the lesion, sections were prepared from 12 different mice, most of which were controls. The number in parentheses is the individaul animal code.) These lesions are regarded as spontaneous and not related to treatment.

Case IV - 8160-80 (AFIP 1757063).
History: 200 lb. yearling Columbia ewe was presented to the veterinarian depressed, off feed and weight loss. Purchased this ewe and several others in Minnesota 6 months previously; none showed signs of sickness except this one and one other that was losing weight. Sheep were fed 12-14% corn/protein, A, D & E. All sheep in dry lot. Ewe died 4 hours after presentation. At necropsy there was generalized icterus, coffee colored urine and dark brown kidneys.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Results
Wednesday Slide Conference - No. 28
13 May 1981

Case I - 80-212 (AFIP 1757728).

History: Tissue from a 4-year-old female Cuban hutia (Capromys pilorides) that was acquired from a zoo in Florida five months earlier and recently became anorectic and showed weight loss.

Contributor's Diagnosis & Comments: Lung, metastatic hepatocellular carcinoma.

Comments: The liver of the hutia is unusual in that each lobe is subdivided into numerous smaller lobes, imparting a "cubed" appearance to it. There was a large (5 cm) pedunculated mass involving the left lobe as well as small nodules in other lobes. Both lungs were more liver-like than lung-like. The tumor also metastasized to the adrenal and heart. The animal also had a small ovarian granulosa-theca cell tumor.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Hepatocellular carcinoma metastatic to lung, Cuban hutia (Capromys pilorides), rodent.

Comments: A Cuban hutia is a large (3-5 lb) omnivorous rodent from the Caribbean Islands. There was general agreement with the contributor's diagnosis. All attendees agreed that the cellular morphology was compatible with hepatic tissue and that hematogenous spread was the route of metastasis. Some attendees submitted an equally acceptable preconference diagnosis of metastatic hepatocellular adenocarcinoma based on the acinar formation by the neoplastic cells. Patterns found in hepatic tumors other than acinar are trabecular and solid sheets of tumor cells. Hepatocellular carcinomas have a high rate of metastasis to the lung in rats. Mice also may have metastatic lesions of liver tumors.

Contributor: National Zoological Park, Washington, DC 20008.

References:

1. An Overview of tumors in zoo animals. IN Comparative Pathology of Zoo Animals, R. J. Montali and G. Migaki, Eds., Smithsonian Institution Press, Washington, DC, 1980, pp 531-542.

2. Histologic typing of liver tumors of the rat. JNCI 64 (1): 180-206, 1980.

Case II - 15278 (AFIP 1764738).

History: Aged (11 years +) male, ringneck duck (Aythya collaris) was being readied for shipment to another zoo. Weight dropped from 750 g to 600 g in recent months. Routine blood work revealed elevated white count. No other clinical signs were noted. Bird was placed on antifungal agents and was found dead three weeks after the initial presentation.

Laboratory Results:	Initial	1 week later
Total WBC	35,900/mm^3	30,500/mm^3
Seg.	92%	93%
Lymph.	8%	7%
Hematocrit	35%	30%
Total protein	4.8 gm/dl	4.4 gm/dl

Contributor's Diagnosis & Comments: Avian tuberculosis.

Comments: Acid fast organisms were present in very large numbers in both sections and tissue imprints. Cultures and serotyping are still in process.

Attendees' Diagnoses & Comments:

Morphologic Diagnoses: 1) Cholangitis, granulomatous, chronic, multifocal, severe, liver, ringneck duck (Aythya collaris). 2) Pneumonia, interstitial and necrotizing, acute to subacute, focally extensive, moderate, lung.

Etiologic Diagnosis: Tuberculous (mycobacterial) cholangitis, hepatitis and pneumonia.

Etiology: Mycobacterium sp. probably M. avian.

Comments: Biliary proliferation, cholestasis, periportal hepatocellular necrosis, granulomas and fibrosis also were observed by the participants. Parasitic cysts (probably Leucocytozoan parasites) also were noted in some sections of lung. The unusual distribution of the lesions in this case is consistent with the variable presentation of mycobacteriosis in birds. Intracellular bacilli within macrophages were observed at the periphery of the lesions in the H&E sections. Abnormal laboratory results included a leucocytosis of primarily heterophils and a slight anemia.

Contributor: Division of Comparative Medicine, School of Medicine, The Johns Hopkins University, Baltimore, MD 21205.

Reference

Mycobacterial Infections of Zoo Animals. Montali, R. J., Ed., Smithsonian Institution Press, Washington, DC, 1978.

Case III - 80-002 (AFIP 1783250).

History: Mice, B6C3F1 (C57BL X C3H), females, 25 months old. Mice were sacrificed after 2 years on a dietary chronic toxicity study. (In order to provide enough slides of the lesion, sections were prepared from 12 different mice, most of which were controls. The number in parentheses is the individaul animal code.) These lesions are regarded as spontaneous and not related to treatment.

Contributor's Diagnosis & Comments: Fibrous osteodystrophy (fibro-osseous lesion, medullary fibrosis), vertebral.
Additional lesions noted in some slides include: mouse (25) - lymphoma; mouse (00) - epidermoid cyst of spinal cord; mice (62) and (86) - herniated intervertebral discs; mouse (37) axonal degeneration and demyelination, slight.

Comment: Presented are decalcified sections of thoracic spinal column with spinal cord and adjacent soft tissue.

This lesion is noted quite frequently in the aged female B6C3F1 mouse. In this study, fibrous osteodystrophy was diagnosed histologically on 1 of 96 (1%) control male mice and 68 of 94 (72%) control female mice. We have noted vertebral fibrous osteodystrophy in female mice as early as 15 months of age.

The pathogenesis of the lesion is unknown. The parathyroid glands were histologically normal. There was frequently a low grade chronic renal disease but this was not correlated with the bone lesions. Chronic renal disease of similar severity was also present in the male mice. As reported by Sass and Montali[1], most of the female mice have cystic endometrial hyperplasia lending further support to possible sex hormonal imbalance.

We have noted increased serum alkaline phosphatase activity in the aged female mouse. Female mice at two years of age generally have alkaline phophatase values 4-5 times higher than males. In mice under 1 year of age, the females generally have alkaline phosphatase values of 1.5-2 times those of the male mice. Additionally, the level of alkaline phosphatase appears to have a crude correlation with severity of the bone lesion.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Fibrosis, medullary, multifocal to diffuse, mild to moderate, marrow cavity, vertebrae, spinal column, B6C3F1 mouse.

Comments: As noted by the contributor, other lesions were present in some slides. The fibro-osseous lesions were the primary interest. A gradation of fibrosis in the marrow cavities was observed. Fibrous tissue replacement can range from mild to complete. The cellular origin is unknown but is believed to be a multipotential marrow stem cell. Lesions most often are found in the sternebrae, the vertebrae of the spinal column and sometimes the long bones. Strain incidence, sex predilection and hormonal influence (particularly the role of estrogens) are thought to be involved in the pathogenesis of the lesions. Vitamin and mineral imbalances and primary and/or secondary hyperparathyroidism are not thought to play a role in this condition.

Contributor: Toxicology Research Laboratory, The Dow Chemical Co., Midland, MI 48640.

References:

1. Sass, B. & Montali, R. J.: Spontaneous fibro-osseous lesions in aging female mice. Lab. Anim. Sci. 30: 907-909, 1980.

2. Ward, J. M. et al.: Neoplastic and nonneoplastic lesions in aging (C57BL/6N X C3H/HeN)F1 (B6C3F1) mice. J. Natl. Cancer Inst. 63: 849-854, 1979.

Case IV - 8160-80 (AFIP 1757063).

History: 200 lb. yearling Columbia ewe was presented to the veterinarian depressed, off feed and weight loss. Purchased this ewe and several others in Minnesota 6 months previously; none showed signs of sickness except this one and one other that was losing weight. Sheep were fed 12-14% corn/protein, A, D & E. All sheep in dry lot. Ewe died 4 hours after presentation. At necropsy there was generalized icterus, coffee colored urine and dark brown kidneys.

Contributor's Diagnosis & Comments: Chronic, nonsuppurative cholangitis, diffuse, liver; biliary stasis, severe, widespread, liver; portal fibrosis, marked, widespread, liver; tubular degeneration, necrosis and early regeneration, kidney, with numerous hemoglobin casts.

Comments: The postmortem findings are consistent with a diagnosis of hepatogenous copper poisoning. Liver copper level was 390 ppm wet weight (atomic absorption). The cause of the nonsuppurative cholangitis could not be determined.

Attendees' Diagnoses & Comments:

Morphologic Diagnoses: 1) Cholangitis, chronic, diffuse, moderate, liver, Columbian ewe, ovine. 2) Fibrosis, periportal, chronic, diffuse, moderate, liver. 3) Proliferation, biliary, diffuse, mild, liver. 4) Cholestasis, diffuse, mild, liver. 5) Nephrosis, tubular, acute, diffuse, moderate, kidney.

Etiologic Diagnoses: Liver - toxic hepatitis.
Kidney - hemoglobinuric nephrosis.

Etiology: Copper toxicosis.

Comments: There was general agreement with the contributor's diagnoses and comments. Additionally many attendees also listed centrilobular hepatocellular degeneration and necrosis as a morphologic diagnosis. Copper toxicosis was the primary diagnosis based on the history and histological lesions in the liver and kidney. A rhodamine stain for copper was positive. The material stained was confined to the centrilobular hepatocytes and sinusoidal macrophages. The attendees also proposed a concurrent disease condition responsible for the cholangitis and biliary proliferation, however, an etiology was not determined. The lesions in the kidney were attributed to a direct toxic action of hemoglobin and/or hypoxia, secondary to a hypovolemic state.

Contributor: Department of Veterinary Pathology, College of Veterinary Medicine, University of Missouri, Columbia, MO 65211.

References:

1. Gooneratne, S. R. et al.: Copper, zinc and iron levels in the cerebrospinal fluid of copper poisoned sheep. Res. Vet. Sci. 27: 384-385, 1979.

2. Gooneratne, S. R. et al.: Creatine kinase release and muscle changes in chronic copper spoisoning in sheep. Res. Vet. Sci. 28: 351-361, 1980.

3. King, T. P. et al.: Autophagy and apoptosis in liver during the prehaemolytic phase of chronic copper poisoning in sheep. J. Comp. Path. 89: 515-530, 1979.

4. Gooneratne, S. R. et al.: An ultrastructural and morphometric study of the liver of normal and copper-poisoned sheep. Am. J. Pathol. 99 (2): 429-449, 1980.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Histories
AFIP Wednesday Slide Conference - No. 29
20 May 1981

Case I - 032-02581 (AFIP 1619721).
History: This C3H mouse received 10 ppb diethylstilbestrol in the feed beginning at 6 weeks of age until sacrifice 559 days later.

Laboratory Results:
A single gray moderately hard mass, about 15 mm in maximum diameter, was found in the groin.

Case II - LAIR 26035 (AFIP 1758835).
History: Tissue from a 13-month-old female Gordon Setter that had a history of progressive CNS dysfunction. When running and going up steps she exhibited marked hypermetria and hyperextension of front legs. She was active, alert, responsive and otherwise in apparent good health. The dog was euthanized since her condition made her unsuitable as a breeding, hunting, or show animal.

Case III - AM-1593-80 (AFIP 1758461).
History: Tissue from an 18-day-old White Pekin duck with history of leg weakness and 30% mortality in group of 2,000. Gross lesions were seen as swollen livers and spleens with occasional subcapsular hemorrhages.

Case IV - VRL A-131891 (AFIP 1776777).
History: This mare aborted a 9-month fetus. The placenta was thickened. Sections of several tissues, including lung and placenta, were submitted.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Results
AFIP Wednesday Slide Conference - No. 29
20 May 1981

Case I - 032-02581 (AFIP 1619721).

History: This C3H mouse received 10 ppb diethylstilbestrol in the feed beginning at 6 weeks of age until sacrifice 559 days later.

Laboratory Results:

A single gray moderately hard mass, about 15 mm in maximum diameter, was found in the groin.

Contributor's Diagnosis & Comments: Malignant granular cell myoblastoma.

Comments: These granular cell tumors were first thought to be of muscle origin. Currently, most pathologists believe they are of Schwann cell origin (5), but some think they are derived from undifferentiated mesenchymal cells (4). In man, they have been observed in striated muscle, particularly of the tongue, but can occur almost anywhere. The common variety consists of many large cells with small nuclei and a cell body molded by pressure of its neighbors into varying shapes. The cytoplasm contains diastase resistant PAS positive granules. The malignant variety is variously called malignant organoid granular cell myoblastoma, malignant nonchromaffin paraganglioma and alveolar soft part sarcoma. They occur chiefly in muscles and often metastasize through the blood stream. They have an organoid pattern with small rounded groups of cells surrounded by a fibrovascular framework. The nuclei are relatively larger than in benign tumors and have more prominent nucleoli. Their exact nature and relation to the benign tumors is uncertain. Nonhuman granular cell tumors are rare and have been induced in the uterine cervix of mice by estrogen treatment (1).

Attendees' Diagnosis & Comments: Granular cell tumor (myoblastoma), cervix, C3H mouse, rodent.

Comments: There was general agreement with the contributor's diagnosis. Several attendees also preferred a malignant designation for the tumor. This tumor has been reported in a variety of species (3). These tumors occur most often in the oral cavity of humans, the lung of horses, the tongue of dogs and the brain of rats. There are also reports of these tumors in the cervix of mice treated with estrogens (1,2). One report describes this tumor in the cerebrum of a dog (3). As the contributor has pointed out the specific cell of origin is not known, but most pathologists favor a Schwann cell origin based on several histological ultrastructural features (3,4). Cells termed angulate bodies also have been observed in these tumors (3) and are best seen with the PAS reaction at the periphery of the lobules. These tumors should not be confused with nonchromaffin paragangliomas as there are several histologic differences between the two (5). The tumor also must be differentiated from histiocytic inflammatory reactions. The role of estrogens in the production of these tumors in mice has not been elucidated.

Contributor: National Center for Toxicological Research, Pathology Services Project, University of Arkansas for Medical Science, Jefferson, AR 72074.

References:
1. Dunn, T. B. et al.: Cysts of the epididymis, cancer of the cervix, granular cell myoblastoma, and other lesions after estrogen injection in newborn mice. J. Nat'l. Cancer Inst. 31: 425-455, 1963.
2. Highman, B. et al.: Neoplastic and preneoplastic lesions induced in female C3H mice by diets containing Diethylstilbestrol or 17B-Estradiol. J. Environ. Pathol. & Toxicol. 4:81-95, 1980.
3. Parker, G. A. et al.: Cerebral granular cell tumor (Myoblastoma) in a dog: Case Report and Literature Review. Cornell Vet. 68 (4): 506-520, 1978.
4. Sobel, H. J. et al.: Granular cells and granular cell lesions. In Pathology Annual, Sommers, S. E. (Ed.), Appleton-Century-Crofts, Prentice Hall, New York, 1974, pp 43-79.
5. Stout, A. P. & Lattes, R.: Tumors of the soft tissues. Atlas of Tumor Pathology, AFIP, Wash. DC, 1967, pp 180-186.

Case II - LAIR 26035 (AFIP 1758835).

History: Tissue from a 13-month-old female Gordon Setter that had a history of progressive CNS dysfunction. When running and going up steps she exhibited marked hypermetria and hyperextension of front legs. She was active, alert, responsive and otherwise in apparent good health. The dog was euthanized since her condition made her unsuitable as a breeding, hunting, or show animal.

Contributor's Diagnosis & Comments:

Syndrome Diagnosis: Cerebellar cortical abiotrophy of Gordon setters.

Morphologic Diagnoses: 1) Degeneration and necrosis, progressive, multifocal to diffuse, Purkinje cells, cerebellum, canine. 2) Atrophy, diffuse, moderate, granular cell layer, cerebellum.

Comments: Over the past 9 years veterinarians in New York, Pennsylvania, Virginia, North Carolina and California have observed a total of 18 Gordon setter dogs with clinical histories consistent with a slowly progressive cerebellar disease. Necropsies of ten of these dogs have revealed similar cerebellar cortical degenerative lesions. The clinical signs and lesions are those of late onset cerebellar cortical degeneration primarily affecting Purkinje cells. The term abiotrophy has been applied to the degeneration of a cell due to some intrinsic metabolic abnormality that causes premature death of that cell. In this disease, Purkinje cells form normally and apparently function for a number of months prior to their degeneration and onset of clinical signs. Examination of pedigrees of affected dogs suggests that the abnormality may be genetically determined. More animals and pedigrees must be studied before the mode of inheritance is determined. Similar lesions have been observed in other breeds such as Kerry blue terriers and rough coated collies from Australia.

Attendees' Diagnoses & Comments:

Morphologic Diagnoses: 1) Degeneration and necrosis, neuronal, multifocal to diffuse, moderate to severe, Purkinje cells, cerebellum, Gordon setter, canine. 2) Atrophy, multifocal to diffuse, mild to moderate, granular cells, cerebellum.

Etiologic Diagnosis: Hereditary cerebellar cortical abiotrophy of Gordon setters.

Etiology: Autosomal recessive trait.

Comments: There was unanimous agreement with the contributor's diagnosis and comments. Some variation in the degree of Purkinje cell loss and granular cell atrophy existed among the submitted slides. The clinical and histological features

of the condition have been published recently (4). The neuropathological changes are reported to be confined to the cerebellum, Grossly the cerebellum may be reduced in weight and the folia are thinned. The histological changes reported are similar to those observed in this case. There are other inherited neurological conditions in several breeds of dogs with some of the same histopathological features of this condition in Gordon setters (1,2,3,4).

Contributor: Letterman Army Institute of Research, SGRD-ULV-P, Presidio of San Francisco, CA 94129.

References:

1. de Lahunta, A. et al.: Hereditary cerebellar cortical and extrapyramidal nuclear abiotrophy in Kerry blue terriers. JAVMA 168: 1119-1124, 1976.
2. de Lahunta, A.: Veterinary Neuroanatomy and Clinical Neurology. W. B. Saunders, Philadelphia, 1977.
3. de Lahunta, A.: Comparative cerebellar diseases in domestic animals. Comp. Vet. Ed. II: 8-19, 1980.
4. de Lahunta, A. et al.: Cerebellar cortical abiotrophy in the Gordon setter. JAVMA 177: 538-541,1980.
5. Hartley, W. J. et al.: Inherited cerebellar degeneration in the rough coated collie. Austr. Vet. Pract. 1-7, 1978.

Case III - AM-1593-80 (AFIP 1758461).

History: Tissue from an 18-day-old White Pekin duck with history of leg weakness and 30% mortality in group of 2,000. Gross lesions were seen as swollen livers and spleens with occasional subcapsular hemorrhages.

Laboratory Results: Erysipelothrix insidiosa was isolated from the livers of the two birds received.

Contributor's Diagnosis & Comments: Erysipelas.

Comments: Histologic alterations include hepatic congestion, chronic pericholangitis and RE cell swelling with cytoplasmic vacuoles, many of which contain basophilic staining rod shaped bacteria.

Attendees' Diagnoses & Comments:

Morphologic Diagnoses: 1) Cholangitis, chronic, diffuse, mild, liver, White Pekin duck, avian. 2) Proliferation and swelling, sinusoidal lining cells, diffuse, mild to moderate, liver. 3) Bacteremia, diffuse, mild, liver. 4) Vacuolar change, diffuse, mild, liver.

Etiologic Diagnoses: Bacterial cholangitis and hepatitis.
Hypoxic change (vacuolar) secondary to hepatic congestion.

Etiology: Erysipelothrix insidiosa.

Comments: In addition to the above diagnoses, a morphologic diagnosis of hepatocellular necrosis also was given by a few. The majority of the attendees submitted a preconference etiologic diagnosis of Lankesterella sp. or other protozoa. Attendees felt there was parasitism of the sinusoidal and leukocytic cells. A protozoal etiology was discredited based on several factors, including, lack of demonstrable schizonts, pleomorphism of the organisms and free organisms within vessels and sinusoids. Many of the basophilic intracytoplasmic structures surrounded by clear spaces were interpreted as stages of erythrophagocytosis, which could be followed sequentially from whole RBC's to pyknotic nuclear forms.

Numerous Gram positive coccobacilli were present in RE cells and vascular spaces. A mixed bacterial and protozoal infection could not be ruled out completely. The gross and histologic lesions of erysipelas in birds were reviewed. Turkeys are the species most commonly affected with the greatest economic losses occurring in these birds.

Contributor: Animal Disease Diagnostic Laboratory, Purdue University, West Lafayette, IN 47907.

Reference:

Bickford, A. A. et al.: Pathology of experimental erysipelas in turkeys. Avian Dis. 22: 503-518, 1978.

Case IV - VRL A-131891 (AFIP 1776777).

History: This mare aborted a 9-month fetus. The placenta was thickened. Sections of several tissues, including [illegible] placenta, were submitted.

Contributor's Diagnosis & Comments: Coccidioidomycosis, fetal pneumonia and placentitis.

Comments: The mare was from the Nevada area. The number of organisms and the location are unusual. Many different fungal growth stages can be identified in most of the slides. Special stains were not necessary.

Attendees' Diagnoses & Comments:

Morphologic Diagnoses: 1) Placentitis, necrotizing, subacute, diffuse, severe, placenta, fetus, equine. 2) Pneumonia, interstitial, granulomatous, subacute to chronic, multifocal to diffuse, moderate, lung.

Etiologic Diagnosis: Coccidioidomycotic placentitis and pneumonia.

Etiology: *Coccidioides immitis*.

Comments: There was unanimous agreement with the contributor's etiologic diagnosis and comments. Some variation existed in the preconference morphologic diagnoses for the placenta and lung. Some attendees preferred pyogranulomatous or granulomatous placentitis and pyogranulomatous or granulomatous bronchopneumonia due to the variation in their slides. The fungal organisms (spherules and endospores) were readily seen. Gross lesions associated with this form of the disease have been reported (1,2). The route of fungal spread to the lung from the placenta was discussed. Most of the participants favored an inhalation route over a hematogenous spread as lesions in other organs were not described.

Contributor: Veterinary Reference Laboratory, P. O. Box 30633, Salt Lake City, Utah 84125.

References:

1. Smith, Jones & Hunt. Veterinary Pathology, 4th Ed., Lea & Febiger, Philadelphia, 1972, pp 646-649.

2. Langham, R. F. et al.: Abortion in a mare due to coccidioidomycosis. JAVMA 170: 178-180, 1977.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Histories
AFIP Wednesday Slide Conference - No. 30
27 May 1981

Case I - 80434 (AFIP 1777000).

History: 6-year-old Quarterhorse mare with slowly developing cutaneous nodules and progressive emaciation.

Case II - 80-83 (AFIP 1783194).

History: This tissue is from a 3-year-old, female Bedlington terrier presented for signs of lethargy, anorexia, vomition, and icterus. She had whelped 3 weeks previously. (2 microslides, H&E and rhodamine).

Laboratory Results:

Tot protein	8.1 gm/dl	BUN	84 mg/dl
Albumin	34.8%	Alk Phos	510 mu/ml
α-1 globulin	2.8%	LDH	660 mu/ml
α-2 globulin	24.8%	SGOT	830 mu/ml
β-globulin	23.8%	SGPT	3440 mu/ml
γ-globulin	14.2%	Tot bilirubin	10.9 mg/dl
A/G ratio	0.5	Direct "	10.0 mg/dl
		Glucose (fasting)	149 mg/dl

Case III - UT 80-889 (AFIP 1757481).

History: A 5-year-old castrated male cat was presented for anorexia and pale mucous membranes. Hematology revealed a non-regenerative anemia and atypical lymphocytes with dark blue cytoplasm. Platelets levels were about 2 million/mm^3. At necropsy there was generalized lymph node enlargement, an enlarged mahogany red spleen and a pale tan liver containing disseminated pin-point yellow foci. There was also mild icterus.

Case IV - BG-81 (AFIP 1783198).

History: This tissue section represents a lesion found during necropsy of an adult White Carneau pigeon from the block at our research farm used for atherosclerosis research. Two organs' samples are present.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology

Results
AFIP Wednesday Slide Conference - No. 30
27 May 1981

Case I - 80434 (AFIP 1777000).

History: 6-year-old Quarterhorse mare with slowly developing cutaneous nodules and progressive emaciation.

Contributor's Diagnosis & Comments: Histiolymphocytic lymphosarcoma.

Comments: Nodules located both in the dermis and subdermis were uniform in appearance. They were not found in other locations including viscera and regional lymph nodes. Two populations of cells in the lesions were lymphocytes and large pleomorphic reticular cells. Corynebacterium sp. was isolated from "tumor" tissue. IgG levels in the serum were normal.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Histiolymphocytic lymphosarcoma, skin, Quarterhorse, equine.

Comments: There was general agreement with the contributor's diagnosis and comments. This case closely parallels a recently reported case of cutaneous lymphosarcoma in a horse without visceral involvement (1). Other cases of equine cutaneous lymphosarcoma were accompanied by visceral involvement and may represent later stages of the same disease. Several attendees reported intracytoplasmic, linear, and crystalline forms within histiocytic cells. Their significance is unknown but may be related to degenerating Corynebacterium sp. bacterial organisms (1). The isolation of bacteria and virus from the tumor suggests a defective immune response (1). Reports indicate Coryneform bacteria stimulate macrophages and enhance growth of tumors. This case illustrates that in addition to the multicentric, thymic and alimentary forms of lymphosarcoma, a form exists in horses that is primarily cutaneous in nature.

Contributor: National Animal Disease Center, Ames, IA.

Reference:

Sheahan, B. J. et al.: Histiolymphocytic lymphosarcoma in the subcutis of two horses. Vet. Path. 17: 123-133, 1980.

Case II - 80-83 (AFIP 1783194).

History: This tissue is from a 3-year-old, female Bedlington terrier presented for signs of lethargy, anorexia, vomition, and icterus. She had whelped 3 weeks previously. (2 microslides, H&E and rhodamine).

Laboratory Results:

Tot protein	8.1 gm/dl	BUN	84 mg/dl
Albumin	34.8%	Alk Phos	510 mu/ml
-1 globulin	2.8%	LDH	660 mu/ml
-2 globulin	24.8%	SGOT	830 mu/ml
-globulin	23.8%	SGPT	3440 mu/ml
-globulin	14.2%	Tot bilirubin	10.9 mg/dl
A/G ratio	0.5	Direct "	10.0 mg/dl
		Glucose (fasting)	149 mg/dl

Contributor's Diagnosis & Comments: Chronic-active hepatitis, with bile stasis and accumulation of copper granules within hepatocytes; compatible with inherited copper disease of Bedlington terriers.

Comments: Grossly the liver was dark green, which is explained by the severe bile stasis evident microscopically. Copper analysis of the liver showed 1405 micrograms/gm. (wet tissue). The rhodanine stain demonstrates heavy accumulations of copper-containing granules within hepatocytes, principally in the centrilobular regions. Examination of other tissues revealed heavy accumulations of hemosiderin within the spleen and tubular degeneration in the kidney.

Attendees' Diagnoses & Comments:

Morphologic Diagnoses: 1) Hepatitis, centrilobular, chronic-active, diffuse, moderate, liver. Bedlington terrier, canine. 2) Cholestasis, diffuse, severe, liver.

Etiologic Diagnosis: Inherited copper disease of Bedlington terriers.

Etiology: Autosomal recessive gene.

Comments: The history incorrectly states the special stain is a rhodamine stain; this should be a "rhodanine" stain. There was unanimous agreement with the contributor's diagnosis and comments. Subacute to chronic cholangitis also was included in the diagnoses listed by some attendees. Values for the following clinical pathology parameters were elevated: total protein, alpha 1&2 proteins, BUN, SAP, LDH, SGOT, SGPT and total and direct bilirubin. Serum albumin and the A/G ratio were low. Hepatic hemosiderosis also was present in this dog. Hemosiderosis usually is present in Bedlington livers and often in large amounts. Lesions in other organs have been commented on by the contributor. This disease has been compared to Wilson's disease of man. Several dissimilarities between the condition in man and animals do exist.

Contributor: Division of Comparative Pathology, Armed Forces Radiobiology Research Institute, NNMC, Bethesda, MD 20014.

References:

1. Ludwig, J. et al.: The liver in the inherited copper disease of Bedlington terriers. Lab. Invest. 43 91): 82-87, 1980.

2. Twedt, D. C. et al.: Clinical, morphologic, and chemical studies on copper toxicosis of Bedlington terriers. JAVMA 175: 269-275, 1979.

3. Ludwig, J. et al.: Copper stains and the syndrome of primary biliary cirrhosis: Evaluation of staining methods and their usefulness for diagnosis and trials of penicillamine treatment. Arch Pathol Lab Med 103: 467, 1979.

Case III - UT 80-889 (AFIP 1757481).

History: A 5-year-old castrated male cat was presented for anorexia and pale mucous membranes. Hematology revealed a non-regenerative anemia and atypical lymphocytes with dark blue cytoplasm. Platelets levels were about 2 million/mm^3. At necropsy there was generalized lymph node enlargement, an enlarged mahogany red spleen and a pale tan liver containing disseminated pin-point yellow foci. There was also mild icterus.

Contributor's Diagnosis & Comments: Megakarocytic myelosis.

Comments: The liver contains multifocal infiltrates of reticulum-type cells (megakaryoblasts) as well as giant cells with lobulated nuclei suggestive of megakaryocytes. Central hemosiderosis of hepatocytes and foci of coagulation necrosis which are either central or paracentral in location also are present. The spleen, peripheral and internal lymph nodes, and femoral bone marrow were extensively infiltrated by cells like those present in the liver. The cat was not tested for FeLV.

Attendees' Diagnoses & Comments:

Morphologic Diagnoses: 1) Megakaryocytoid leukemia, liver, feline. 2) Necrosis, centrilobular, acute (coagulative), diffuse, moderate to severe, liver.

Comments: There was total agreement with the contributor's diagnosis. Lipofuscinosis of centrilobular hepatocytes also was present. The first diagnosis is in accordance with the WHO classification of tumors in animals (2). This is a rare condition in animals. A leukemic condition also rarely is reported (3). Anemia and/or hemorrhage also may be associated with this condition in man and animals. The hepatic necrosis was felt to be secondary hypoxic change due to anemia.

Contributor: Department of Pathobiology, University of Tennessee, P. O. Box 1071, Knoxville, TN 37901.

References:

1. Michel, R. L. et al.: Megakaryocytic myelosis in a cat. JAVMA 168 (11): 1021-1025, 1976.

2. Jarrett, W. F. H. et al.: Neoplastic diseases of the haematopoietic and lymphoid tissues. Bull Wld Hlth Org 50: 21-34, 1974.

3. Holscher, M. A. et al.: Megakaryocytic leukemia in a dog. Vet. Path. 15: 562-565, 1978.

Case IV - BG-81 (AFIP 1783198).

History: This tissue section represents a lesion found during necropsy of an adult White Carneau pigeon from the block at our research farm used for atherosclerosis research. Two organs' samples are present.

Contributor's Diagnosis & Comments: Seminoma of the testicle with metastasis to the liver.

Comments: Two other such cases are present in the files at Bowman-Gray.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Seminoma, testicle with metastasis to the liver, White Carneau pigeon, avian.

Comments: Differential preconference diagnoses also included malignant endocrine tumors of various origin. This tumor although rare has been encountered in several species of birds (1,2). A higher incidence of seminomas in birds occurs in the Budgerigars (2).

Contributor: Department of Comparative Medicine, Bowman-Gray Medical School, Winston-Salem, NC.

References:

1. Campbell, J. G.: Some unusual gonadal tumors of the fowl. Brit. J. Cancer 5: 69, 1951.

2. Petrak, M. L. et al.: Neoplasms in Diseases of Cage and Aviary Birds, Lea & Febiger, Philadelphia, 1969, pp 482-483.

TIMOTHY P. O'NEILL
Captain, USAF, BSC(VC)
Registry of Veterinary Pathology
Department of Veterinary Pathology