

Clinical features of avian vacuolar myelinopathy in American coots

R. Scott Larsen, DVM, MS; Felicia B. Nutter, DVM; Tom Augspurger, MS; Tonie E. Rocke, DVM, PhD;
Lindsay Tomlinson, DVM, DVSc, DACVP; Nancy J. Thomas, DVM, MS, DACVP;
Michael K. Stoskopf, DVM, PhD, DACZM

Objective—To characterize clinical features of avian vacuolar myelinopathy (AVM) in American coots.

Design—Case-control study.

Animals—26 AVM-affected American coots and 12 unaffected coots.

Procedures—Complete physical, neurologic, hematologic, and plasma biochemical evaluations were performed. Affected coots received supportive care. All coots died or were euthanized, and AVM status was confirmed via histopathologic findings.

Results—3 severely affected coots were euthanized immediately after examination. Seventeen affected coots were found dead within 7 days of admission, but 5 affected coots survived > 21 days and had signs of clinical recovery. Abnormal physical examination findings appeared to be related to general debilitation. Ataxia (88%), decreased withdrawal reflexes (88%), proprioceptive deficits (81%), decreased vent responses (69%), beak or tongue weakness (42%), and head tremors (31%), as well as absent pupillary light responses (46%), anisocoria (15%), apparent blindness (4%), nystagmus (4%), and strabismus (4%) were detected. Few gross abnormalities were detected at necropsy, but histologically, all AVM-affected coots had severe vacuolation of white matter of the brain. None of the control coots had vacuolation.

Conclusions and Clinical Relevance—Although there was considerable variability in form and severity of clinical neurologic abnormalities, clinical signs common in AVM-affected birds were identified. Clinical recovery of some AVM-affected coots can occur when supportive care is administered. Until the etiology is identified, caution should be exercised when rehabilitating and releasing coots thought to be affected by AVM. (*J Am Vet Med Assoc* 2002;221: 80–85)

Avian vacuolar myelinopathy (AVM) is a neurologic disease that has been diagnosed in wild birds in the southeastern United States. It was first documented in bald eagles (*Haliaeetus leucocephalus*) in 1994 and has also been found in American coots (*Fulica americana*).¹

Bald eagles with AVM are frequently found dead.¹ Affected eagles have difficulty flying and can crash into or overfly perches. Affected coots appear reluctant to fly or are wobbly in flight; they are uncoordinated on land, and may swim in circles or on their backs.

Birds with AVM that die usually have no gross lesions of the nervous system. Histologically, the disease is characterized by diffuse, spongy degeneration throughout the white matter of the CNS, with the optic tectum most severely affected.¹ No cellular inflammatory response has been observed in association with the vacuolar lesions, and there have been no consistent histologic lesions reported in non-neural tissue.¹ The etiology of this condition remains unknown. Many compounds are known to cause intramyelinic vacuoles, including hexachlorophene, triethyltin, bromethalin, anthelmintics, cuprizone, and isonicotinic acid hydrazide²⁻⁹; however, extensive tissue analyses for these compounds have not produced insight into the etiology of this disease.¹

A more complete clinical description of the disease could help direct investigation into the etiology of AVM. The purpose of the study reported here was to characterize the clinical course of AVM in American coots and compare findings with data obtained from unaffected American coots from a site where the disease has not been reported.

Materials and Methods

Twenty-six American coots were collected from Woodlake, North Carolina (35° 14'N, 79° 11'W), a site where birds affected with AVM have been identified each of the past 4 years. Eleven birds were captured in late November 2000 by use of a net from the shore. Twelve additional birds were captured 4 days later by use of a net from a boat, and 3 coots were captured in mid-December 2000 by use of a net from the shore. All of the coots captured at Woodlake were unable to swim or fly normally. They were transported to a holding facility in Raleigh, NC (35° 50'N, 78° 40'W) within 4 hours of capture, where they remained for up to 60 days.

Birds received a complete physical and neurologic evaluation within 24 hours of arrival in Raleigh. Physical examination included evaluation of feathers, feather shafts, dermis, uropygial gland, head, eyes, eyelids, nares, oral cavity, phar-

From the Environmental Medicine Consortium, Departments of Clinical Sciences (Larsen, Nutter, Stoskopf) and Microbiology, Pathology, and Parasitology (Tomlinson), College of Veterinary Medicine, North Carolina State University, Raleigh, NC 27606; the North Carolina Zoological Park, Asheboro, NC 27203 (Larsen); the United States Fish and Wildlife Service, Raleigh Field Office, Raleigh, NC 27636 (Augspurger); and the US Geological Survey, Biological Resources Division, National Wildlife Health Center, Madison, WI 53711 (Rocke, Thomas). The study was conducted at the Fish and Wildlife Educational Unit and the Veterinary Teaching Hospital, North Carolina State University, Raleigh, NC 27606. Dr. Tomlinson's present address is the Department of Pathology, Bristol-Myers Squibb Co, 1 Squibb Dr, New Brunswick, NJ 08903.

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Address correspondence to Dr. Larsen.

ynx, submandibular area, neck, respiratory rate and effort, wings, legs, feet, coelom, cloaca, and pericloacal area. Body condition was subjectively evaluated by 1 of the investigators and scored by use of a standard avian body condition score index with a scale of 1 to 4 (1 being emaciated, 4 being obese).¹⁰ Birds were weighed to the nearest gram by use of an electronic scale.

Mental status was evaluated by noting whether birds appeared conscious, aware, anxious, or aggressive. **Cranial nerve (CN) II** was assessed by evaluating the ability to avoid obstacles during ambulation. The functions of CN II and III were evaluated by use of a bright penlight or directed sunlight to elicit a pupillary light response. Observation of eye movement was used to evaluate CN III, IV, VI, and VII. Abnormal function of CN III was also evaluated by checking for anisocoria. Intact CN V and X were assessed by subjectively evaluating beak strength, and CN V and VII were assessed by observing the ability to blink. The function of CN VIII was assessed by observing the reaction to an abrupt clap and shout from a person outside the field of view. Intact CN IX, X, XI, and XII were evaluated by assessing tongue movement and strength.

Body reflexes were evaluated by use of a neurologic scoring system of 0 to 4, with 0 indicating areflexia, 1 indicating hyporeflexia, 2 indicating normal reflexes, 3 indicating hyperreflexia, and 4 indicating hyperreflexia with tetany.¹¹ The left and right sides of the body were assessed separately for body postures and reflexes. Wing posture was assessed to detect drooping or weakness. Leg strength and knuckling were assessed when the birds attempted to ambulate. Withdrawal reflexes were elicited by firmly pinching the hind limb or foot with the thumb and forefinger. Several different locations on the hind limb and foot were pinched until investigators were convinced of the result and had ruled out an escape response or stoicism. Pain perception was also evaluated by assessing the reaction to hind limb and toe pinches. This assessment differentiated pain perception from withdrawal reflex by evaluating conscious recognition of the stimulus, such as movement of the bird's head, wings, or body in reaction to the limbs or toes being pinched. Proprioception was evaluated by supporting birds at the base of the wings, turning one foot over on to its dorsal aspect, and watching the response to correctly position the foot. Several attempts were made with each foot until the investigators were convinced the responses were not attributable to inadequate weight bearing, excessive weight bearing, or struggling. The vent response and cloacal sphincter tone were assessed by subjectively evaluating the speed and strength of reaction of the vent to light tactile stimulation.

Blood was collected from the right jugular vein from 20 of the affected coots. One to two milliliters of blood was collected into heparinized syringes and placed in a clean collection tube. Digital pressure was applied to the phlebotomy site for 3 to 5 minutes to minimize hemorrhage. Blood smears for differential cell counts and cytochemical stains were prepared immediately and air-dried. Packed cell volume was measured after standard centrifugation of microhematocrit tubes. Total WBC counts were performed within 18 hours of collection on samples from 15 affected coots using the eosinophil unopette method.¹² White blood cell counts could not be performed for 5 affected coots because of clot formation. Blood smears were stained with a modified Wright giemsa stain.¹² Leukocytes were categorized into 1 of 6 groups: heterophils, lymphocytes, monocytes, eosinophils, basophils, or immature band cells. Biochemical assays were performed within 72 hours of collection with an automated analyzer.^a These included measurement of plasma glucose, **blood urea nitrogen (BUN)**, uric acid, phosphorus, calcium, total protein, albumin, globulin, cholesterol, bile acid con-

centrations, and activities of **alkaline phosphatase (ALP)**, **aspartate aminotransferase (AST)**, **creatinine kinase (CK)**, and **lactose dehydrogenase (LDH)**. Plasma indices for icterus, hemolysis, and lipemia were calculated with absorbance measurements.

Three severely affected coots were euthanatized immediately after admission by exsanguination after anesthesia with isoflurane.^b While anesthetized, these 3 birds were perfused with modified Karnovsky solution.^c Another affected coot died within 20 minutes of being examined. The remaining 22 affected birds were housed in a 7 × 11-m outdoor pen with a 3 × 5-m pond. The pond bottom sloped to provide a depth from 5 cm to 30 cm. The coots were housed with 8 captive-raised Mallard ducks for the first 3 to 7 days after admission as part of an exposure trial. They were fed water-soaked commercial poultry diet^d with bean sprouts laid on top. Bean sprouts were also floated on the surface of the pond. Birds were checked twice daily and were physically examined and weighed at 3, 7, 14, 19, and 55 days after admission. Birds that were not ambulatory and weighed < 95% of admission body weight were tube fed twice daily with 15 ml of a slurry composed of 5 cm³ of blended diet, 5 ml of water, and 5 ml of an electrolyte solution^e formulated for oral administration. Tube-fed birds were weighed daily and tube feeding was discontinued when birds were ambulating, weighed ≥ 95% of admission body weight, and were observed eating. After the completion of the Mallard duck exposure trials, coots were placed in portable canine transport kennels for shelter during periods of rain or snow. Kennels were brought into a heated building if ambient temperature was < 0 C.

Sixteen of the 22 affected coots in the exposure trial were found dead within 7 days of admission and another died 21 days after admission. The other 5 affected coots survived > 21 days and were euthanatized with 39 mg of sodium pentobarbital^f administered IV via metatarsal, brachial, or jugular vein when they developed severe self-inflicted trauma attempting to escape their enclosure. All gross postmortem examinations were conducted within 24 hours after the birds died. Cadavers were weighed, brains and kidneys were removed and fixed in neutral-buffered 10% formalin for histologic examination, and livers were frozen. Additional body tissues from 6 birds that had signs of neurologic improvement during captivity were sampled and fixed in formalin. Tissues in formalin were embedded in paraffin, sectioned at 5 μm, and stained with H&E for histologic examination by use of standard methods. Brain tissue from the 3 severely affected birds in which tissues were perfused with modified Karnovsky solution was examined via electron microscopy.

In February 2001, 12 additional coots were captured at Mattamuskeet National Wildlife Refuge, NC (35° 30'N, 76° 15'W) by use of a bait station and a rocket net. Lake Mattamuskeet is a site where AVM has not been reported and these 12 birds served as unaffected controls for comparison with affected coots. Complete physical, neurologic, and clinical pathology examinations were conducted on 10 of these birds, which were held for 18 to 24 hours and then euthanatized by cervical dislocation. Brains were removed, fixed in neutral-buffered 10% formalin, and processed routinely for histologic examination. Two of these control coots received physical and neurologic examinations, after which they were anesthetized with isoflurane, euthanatized by exsanguination, and perfused with modified Karnovsky solution. The brains of these 2 coots were evaluated via electron microscopy.

Statistical analyses—Statistical analyses of hematologic and plasma biochemical values were performed with commercially available software^g with comparison between samples from affected coots and from reference coots in an unmatched control population design. Mann-Whitney rank-sum tests were used for nonparametric comparison of the ranks

of values for hematologic and biochemical data and equivalency of distribution of sample sets was assumed. Differences between groups were considered significant at $P < 0.05$.

Results

Affected coots had crusting of urates around the vent (14/26), accumulation of uropygial discharge around the uropygial gland (7/26), wet feathers (3/26), and severe feather damage (3/26). None of these abnormalities were found in the 12 unaffected coots. Lice (unidentified species) were found on 5 of the affected coots and on 1 of the unaffected coots. Mean \pm SD initial body weight of affected birds was 448 ± 46 g and mean weight of unaffected birds was 539 ± 106 g. Median body condition score was 2 for affected birds and 3 for unaffected birds. Weight loss was observed in all affected birds except those that survived < 1 day. Surviving birds began to gain weight after 7 days in captivity.

Coots were generally aggressive towards handlers, pecked with their beaks, and attempted to scrape with their feet. Nonaggressive birds appeared to have decreased awareness of their surroundings. Anisocoria was observed in 4 (15%) birds at admission. Two of these birds died within 2 days, but the anisocoria resolved by day 3 in the other 2. Nystagmus was observed at admission in a coot that died on day 3, and strabismus was detected at admission in a bird that died on day 5. One coot appeared blind and died within 1 day. Normal eye movement was difficult to assess and was determined to be abnormal in only 1 bird. Head tremors were observed in 8 (31%) of the affected birds and 2 of these birds also had torticollis. Only 1 bird with head tremors survived more than 5 days. Fifteen affected coots and 7 control coots had lack of response to noise. Beak and tongue weakness were detected in 10 and 11 AVM-affected coots, respectively.

Most (23/26) affected birds had moderate-to-severe ataxia, knuckling, or limb weakness in 1 or both hind limbs. Six birds (23%) had 1 hind limb trailing when trying to swim or ambulate, and these birds propelled themselves forward using their wings. Ataxia and knuckling were generally bilateral, but 4 birds had abnormal function in only 1 leg. One bird that was completely recumbent at admission became ambulatory (but ataxic) by day 3 and was ambulating normally after day 19. This bird had no further signs of ataxia through day 60. Three birds with moderate bilateral ataxia and weakness had clinical improvement during a 2-week period and ambulated normally by day 19. These 3 birds also had normal gaits until euthanatized. One coot that developed ataxia on day 3, had mild clinical improvement through day 7, but then became completely recumbent on day 12. The right tibiotarsal joint was warm and swollen but the foot was cold. Septic arthritis of the tibiotarsal joint was diagnosed after arthrocentesis and cytologic examination of the joint fluid. This bird was treated with antibiotics but died on day 21. Tibiotarsal swelling was also observed on the right hock joint of another coot on day 56. Only 1 of the control coots had any gait abnormalities. This bird was moderately lame on 1 leg and had a laceration on the ventral aspect of the foot web, but did not have signs of incoordination or ataxia.

There was no obvious pattern in withdrawal reflexes. Some affected birds that had normal withdrawal reflexes at admission developed weak or absent withdrawal reflexes on subsequent examinations and some birds that had no withdrawal reflexes at admission developed weak or normal withdrawal reflexes. Decreased withdrawal reflexes were detected in 23 of 26 affected coots and 4 control coots. Decreased pain responses were detected in 7 affected coots and 1 control coot. Proprioceptive deficits were observed at admission in 19 of 26 (73%) affected coots and developed later in 2 coots. Two birds with proprioceptive deficits became normoreflexive by 7 to 14 days. Two other coots with proprioceptive deficits had transient improvement but continued to be hyporeflexive. Two control coots had proprioceptive deficits. Eighteen affected coots had decreased vent response and cloacal hypotonia, whereas 2 control coots had these findings.

Few abnormalities were detected at gross post-mortem examination. A large area of hemorrhage was found near the jugular vein in 6 birds, and a large blood clot was found in the cranial thorax in 2 birds. Blood was evident in the distal portion of the trachea of 1 bird. One coot had multifocal pinpoint white foci on the mesentery and on thoracic and abdominal air sacs. This coot had a swollen right tibiotarsal joint, with edema of the subcutaneous tissue and joint capsule. The synovial fluid of this joint had low viscosity. Another bird that was euthanatized had a similarly swollen and edematous tibiotarsal joint with low-viscosity synovial fluid.

Brains of all 26 affected coots and all 12 unaffected coots were examined histologically. All of the affected coots had severe vacuolation consistent with AVM. None of the unaffected control coots had any vacuolation. The 6 birds that regained neurologic function had extensive, mild-to-severe vacuolation of white matter tracts, with vacuoles as large as $80 \mu\text{m}$ in diameter found individually and in large clusters. Vacuolation in the optic tectum was consistently observed in these 6 coots, but the degree of vacuolation in the cerebrum, cerebellum, and midbrain varied considerably.

Tissues from the birds that regained neurologic function had mild infiltration of lymphocytes, plasma cells, and occasional heterophils in the portal regions of the liver (6/6 birds), islets of the pancreas (5/5), esophageal submucosa (5/5), tunica muscularis of the ventriculus (5/6), lamina propria of the proventriculus (2/3), perivascular intestinal interstitium (2/3), kidney, (2/2), and skeletal muscle (1/1). Lacy vacuolation of hepatocytes (3/6) was also observed. The lungs (4/6) were moderately congested and erythrocytes filled numerous parabronchi. Mild periparabronchial anthracosis was evident, and small aggregates of lymphocytes were evident around pulmonary vessels (3/6).

Two birds had inflammation in the tibiotarsal joint, characterized by joint capsule edema, fibrinous exudate, synovial ulceration, and heterophilic infiltration of the joint capsule and surrounding connective tissue. In 3 birds, sections of skin from traumatized areas on the carpometacarpi had regionally extensive ulceration and erosion with serocellular exudates that

Table 1—Hematologic values (median [interquartile range]) for 14 American coots with avian vacuolar myelinopathy (AVM) and 10 control coots

Variable	AVM	Control
Hct (%)	45 (39, 48)	44 (40, 47)
WBC cell count (cells/ μ l)	5,300 (4,950, 8,075)	5,875 (4,625, 6,625)
Heterophils (cells/ μ l)*	2,533 (1,210, 3,030)	3,858 (3,362, 3,992)
Lymphocytes (cells/ μ l)*	3,298 (2,344, 4,290)	1,848 (1,550, 2,017)
Monocytes (cells/ μ l)	125 (51, 432)	41 (0, 115)
Eosinophils (cells/ μ l)*	0 (0, 37)	71 (45, 111)
Basophils (cells/ μ l)	0 (0, 0)	0 (0, 31)

*Significant ($P < 0.05$) difference between values for affected coots and control coots.

Table 2—Plasma biochemical values (median [interquartile range]) for 17 American coots with AVM and 10 control coots

Variable	AVM	Control
Glucose (mg/dl)	275 (248, 327)	251 (236, 267)
BUN (mg/dl)	6 (3, 8)	6 (4, 8)
Uric acid (mg/dl)	8 (5, 18)	12 (11, 19)
Phosphorus (mg/dl)	4.8 (3.8, 5.7)	5.8 (5.1, 7.4)
Calcium (mg/dl)	10.6 (10.3, 10.8)	10.4 (9.8, 11.0)
Total protein (g/dl)*	3.8 (3.3, 4.2)	4.2 (4.2, 4.4)
Albumin (g/dl)	1.7 (1.4, 1.8)	1.8 (1.6, 1.9)
Globulin (g/dl)*	2.1 (1.9, 2.4)	2.6 (2.4, 2.6)
Cholesterol (mg/dl)	296 (260, 332)	291 (260, 319)
Bile acid (μ mol/L)*	49 (33, 64)	28 (24, 28)
ALP (U/L)*	168 (118, 197)	274 (224, 329)
AST (U/L)	751 (534, 931)	989 (793, 1,322)
CK (U/L)*	5,128 (3,066, 7,662)	16,006 (11,642, 41,020)
LDH (U/L)	735 (422, 1640)	2,173 (794, 5,962)
Icterus index (unconjugated bilirubin [mg/dl])	1 (1, 2)	2 (2, 2)
Hemolysis index (hemoglobin [mg/dl])	34 (25, 85)	40 (14, 64)
Lipemia index* (Lipid [mg/dl])	12 (0, 16)	0 (0, 1)

*Significant ($P < 0.05$) difference between values for affected coots and control coots.

contained colonies of coccobacilli. Heterophils, lymphocytes, and multinucleated giant cells were evident in the underlying dermis, along with vascular congestion and hemorrhage.

There were few clinical pathology values that differed significantly between the AVM-affected coots and the control coots. Although there was no significant difference in total WBC count, affected coots had significantly more lymphocytes and fewer heterophils and eosinophils than did control coots (Table 1). Affected coots had significantly lower globulin and total protein values, ALP values, and CK values, but had significantly higher concentrations of bile acids, compared with affected coots (Table 2). The lipemia index was also significantly higher in affected coots, compared with control coots.

Discussion

There was considerable variation in form and severity of the neurologic abnormalities of AVM-affected coots. This variation likely came from 3 sources: the inherent difficulty of assessing neurologic function in wild birds, natural variation in the disease process, and admission of birds into the investigation at different

points during the course of the disease. In birds, there are substantial challenges associated with performing tests of neurologic function, and it is difficult to appreciate and interpret subtle changes in CN or reflex function.¹³ Most descriptions of neurologic evaluations of birds are focused on the examination of companion psittacine species,^{13,14} and some of the components of these evaluations may not be applicable to non-psittacine birds. The neurologic examination is also confounded by the stress imparted on birds that are wild and have only recently been captured. Behavioral abnormalities may be particularly difficult to evaluate in wild-caught birds, because stressed birds alter their behavior in order to hide signs of disease.¹⁵ Thus, it was critically important to compare the AVM-affected coots with wild coots from an area where AVM has not been diagnosed. The AVM-negative control coots helped determine which findings were truly abnormal and which were confounded by the species and the conditions under which they were examined. Abnormal eye movements (anisocoria, strabismus, nystagmus), ataxia, abnormal limb movements, beak weakness, tongue weakness, and head tremors were observed only in AVM-affected coots and appeared to be strongly associated with the disease. In contrast, deficits in withdrawal reflexes, proprioceptive reflexes, responses to painful stimuli, and lack of response to noise were observed in healthy coots as well as abnormal coots, making them unreliable for determining disease status. Much of the variation in clinical signs was probably attributable to the variation of CNS lesions in AVM-affected birds, because our findings agree with an earlier report of variability in lesion distribution.¹ Interestingly, although the optic tectum was consistently affected in the coots we examined, clinical neurologic dysfunction of the eye was observed in fewer than half of the birds.

A variable that could not be controlled in this study was when a bird first acquired AVM in relation to its entrance into the study. Wild coots entered the study at an unknown point in the course of the disease. This was evident because some birds entered the study debilitated, with severe neurologic dysfunction, and others had only subtle signs. The clinical condition of all affected coots deteriorated for 3 to 7 days after being brought into captivity, and the severity of neurologic dysfunction consistently increased during this time.

Another source of variability lies in the comparison of AVM-affected coots with unaffected controls. These groups were captured at different times of the year and different stages of migration. Although these timing differences were unlikely to have affected results of neurologic examination, they may have affected differences in body weight, body size, or physical condition. Hematologic and biochemical values may also have been affected by the timing differences, suggesting caution should be used when comparing these data between the affected and control groups. The blood values for American coots we were able to locate in the wildlife and zoological medicine literature¹⁶ were derived via substantially different analytic methods, sample handling techniques, and sampling schedules, compared with those used in our investigation. Although we did not have time-matched controls,

we believe the values obtained from our control population represent a better reference data set than is otherwise available. Although our data did not permit us to definitively determine whether the values from the affected birds were truly abnormal, it did facilitate comparisons between the 2 cohorts of coots.

Significant differences observed in relative numbers of heterophils, lymphocytes, and eosinophils may have been caused by different levels of stress, differences in timing of sample collection, or the disease itself. Differences in CK concentrations were potentially attributable to muscle damage during capture, because affected coots were captured with dip nets, whereas control coots were captured with rocket nets. Differences in CK values attributable to different capture techniques have been described in Mallard ducks.¹⁷

The differences we observed in total protein, globulin, and lipemia index could indicate a catabolic state in affected coots. Common eiders (*Somateria mollissima*) that do not feed while nesting mobilize lipids as the primary source of energy, with consequent increases in β -hydroxybutyrate and decreases in glucose, total protein, and globulin concentrations.¹⁸ Similarly, lipid metabolites such as glycerol and β -hydroxybutyrate increase in western sandpipers (*Calidris mauri*) as they lose body mass.¹⁹ The only lipid we measured was cholesterol, which did not differ between the 2 groups. Measurement of other lipid components could elucidate whether the differences observed in our studies were related to differences in nutrition or seasonal differences in physiologic factors, or whether they were associated directly with the pathogenesis of AVM.

Plasma bile acid concentrations were significantly higher in affected coots than in control birds. Although bile acids can increase postprandially,²⁰ this seems an unlikely explanation for the difference because affected coots were in poor-to-fair body condition and had not eaten within at least 4 hours. Bile acids may also be increased in association with altered liver function,²⁰ but severe hepatic disease was not observed in the coots of this study and consistent hepatic lesions have not been reported in previous investigations of AVM.¹ Therefore, the cause for the differences in plasma bile acid concentrations that we observed was unknown.

All 6 affected coots that survived the disease had lymphocytic infiltration of multiple organs. This could have been caused by infection, stress, or trauma secondary to confinement, but future studies should be designed to evaluate this observation.

Avian vacuolar myelinopathy is not necessarily a fatal disease, as evidenced by the recovery of some of the coots in this investigation. The clinical recovery of birds affected with AVM has not previously been described, and attempts to rehabilitate clinically affected birds have been unsuccessful. Six coots in our investigation appeared to clinically recover from the disease with minimal supportive care. The surviving coots had complete or nearly complete return to normal neurologic function, despite the histologically confirmed presence of vacuoles in the white matter or the CNS. We were unable to accurately predict which coots would survive.

The clinical recovery of some of the affected coots

suggests that successful rehabilitation of AVM-affected birds is possible. However, it would be unwise to rehabilitate and release AVM-affected birds until the cause of the disease is identified and the potential for recovered birds to be disease carriers is determined. If there is the potential for AVM to be transported from 1 location to another, it may be challenging to determine whether a wild population is truly free of the disease. Screening tools, such as physical examinations, neurologic evaluations, and plasma biochemistry analyses, can help determine whether a control population is clinically normal, but histologic examination of brain tissue may still be necessary for confirmation that control birds are truly unaffected by AVM.

^aBoehringer Mannheim/Hitachi 912 Analyzer, Boehringer Mannheim Corp, Indianapolis, Ind.

^bAerane, Anaquest, Madison, Wis.

^cKarnovsky MJ. A formaldehyde-glutaraldehyde fixative of high osmolality for use in electron microscopy (abstr). *J Cell Biol* 1965;27:137A.

^dHigh Tech X-L-A, Quality Feeds Small and Special Poultry Flocks, Southern States Cooperative, Richmond, Va.

^ePedialyte, Abbott Laboratories, Abbott Park, Ill.

^fBeuthanasia-D Special, Schering Plough Animal Health Corp, Kenilworth, NJ.

^gSAS, SAS Institute Inc, Cary, NC.

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