BioOne COMPLETE

CAPTURE MYOPATHY IN WILD TURKEYS (MELEAGRIS GALLOPAVO) FOLLOWING TRAPPING, HANDLING AND TRANSPORTATION IN COLORADO

Authors: Spraker, T. R., Adrian, W. J., and Lance, W. R.

Source: Journal of Wildlife Diseases, 23(3) : 447-453

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-23.3.447

BioOne Complete (complete.BioOne.org) is a full-text database of 200 subscribed and open-access titles in the biological, ecological, and environmental sciences published by nonprofit societies, associations, museums, institutions, and presses.

Your use of this PDF, the BioOne Complete website, and all posted and associated content indicates your acceptance of BioOne's Terms of Use, available at www.bioone.org/terms-of-use.

Usage of BioOne Complete content is strictly limited to personal, educational, and non - commercial use. Commercial inquiries or rights and permissions requests should be directed to the individual publisher as copyright holder.

BioOne sees sustainable scholarly publishing as an inherently collaborative enterprise connecting authors, nonprofit publishers, academic institutions, research libraries, and research funders in the common goal of maximizing access to critical research.

Downloaded From: https://bioone.org/journals/Journal-of-Wildlife-Diseases on 28 Mar 2024
Terms of Use: https://bioone.org/terms-of-use

Journal of Wildlife Diseases, 23(3), 1987, pp. 447–453
© Wildlife Disease Association 1987

CAPTURE MYOPATHY IN WILD TURKEYS (*MELEAGRIS GALLOPAVO*) FOLLOWING TRAPPING, HANDLING AND TRANSPORTATION IN COLORADO

T. R. Spraker,[1] W. J. Adrian,[2] and W. R. Lance[3]

[1] Department of Pathology, College of Veterinary Medicine, Colorado State University, Fort Collins, Colorado 80523, USA
[2] Colorado Division of Wildlife, 317 W. Prospect, Fort Collins, Colorado 80526, USA
[3] Wildlife Laboratories, Inc., 1401 Duff Drive, Suite 600, Fort Collins, Colorado 80524, USA

ABSTRACT: Sixty wild turkeys were necropsied following trapping, transporting and handling during the winters of 1980–1981, 1981–1982, and 1982–1983 in order to determine the incidence of subclinical capture myopathy. Gross lesions characterized by small to large patchy, pale white streaked areas within skeletal muscle were found in 13 of 27 birds trapped with a drop net in the winter of 1982–1983. Microscopic lesions within myocardium characterized by irregular areas of coagulative necrosis, collapse of intercellular stroma and myocardial nuclear proliferation were found in two of 14 birds in 1980–1981, five of 19 birds in 1981–1982 and 11 of 27 birds in 1982–1983. Microscopic lesions within skeletal muscle characterized by rhabdomyolysis were found in 16 of 19 birds in 1981–1982 and 25 of 27 birds in 1982–1983. These findings suggest that wild turkeys are susceptible to capture myopathy and particular caution should be exercised in capturing and handling these birds.

Key words: Meleagris gallopavo, wild turkey, capture myopathy, pathology, skeletal muscle, heart, transportation, trapping.

INTRODUCTION

Capture myopathy (CM) is a commonly occurring condition in mammals following trapping and transportation. CM was first described in a Hunter's hartebeest (*Damaliscus hunteri*) in South Africa (Jarrett et al., 1964). Following Jarrett's description, the syndrome was observed in many other wild African ruminant species (Chalmers and Barrett, 1982). Herbert and Cowan (1971) described a condition similar to CM following trapping of Rocky Mountain goats (*Oreamnos americanus*) and thought that selenium deficiency was part of the problem. They referred to the condition as white muscle disease. Subsequently, capture myopathy was described in white-tailed deer (*Odocoileus virginianus*) (Wobeser et al., 1976), moose (*Alces alces*) (Haigh et al., 1977), pronghorn antelope (*Antilocapra americana*) (Chalmers and Barrett, 1977), Rocky Mountain bighorn sheep (*Ovis canadensis canadensis*) (Spraker, 1977), and elk (*Cervus canadensis*) (Lewis et al., 1978).

CM has been observed in avian species, but is not common. CM has been described in flamingos (Young, 1967) and in a greater sandhill crane (Windingstad et al., 1983). A syndrome associated with drive-trapping, handling and translocation of Canada geese (*Branta canadensis*) and characterized by leg paralysis and death has also been described briefly by W. Wishart (Chalmers and Barrett, 1982). CM has been noted in several finches following transportation (Spraker, 1983 unpubl. data). The pathogenesis of CM has been described by Bartsch et al. (1977), Spraker (1980, 1982) and Chalmers and Barrett (1982).

MATERIALS AND METHODS

Wild turkeys were trapped during the winters of 1980–1981, 1981–1982, and 1982–1983 from four different areas in Colorado. These birds were part of a survey of pathogens in wild turkeys (Adrian, 1984). During winter of 1980–1981, 15 wild turkeys were trapped in the Montrose area in southwestern Colorado using clover traps (Myers, 1973). In the winter of 1981–1982, 20 birds were trapped in the same area using clover traps or alpha chloralose treated corn

Downloaded From: https://bioone.org/journals/Journal-of-Wildlife-Diseases on 28 Mar 2024
Terms of Use: https://bioone.org/terms-of-use

TABLE 1. Summary of gross and histological lesions found in wild turkeys with capture myopathy.

	Number of birds examined	Gross lesions observed (%)	Microscopic myocardial lesions (%)	Microscopic skeletal muscle lesions (%)			Severity of microscopic skeletal muscle lesions (%)			
				Type A	Type B	Type C	None	Mild	Moderate	Severe
1980–1981	14	0	14	N[a]	N	N	N	N	N	N
1981–1982	19	0	26	16[b]	84	47	11	73	16	0
1982–1983	27	48	41	22	93	85	0	30	30	40

[a] Data not available.
[b] Birds had one or more types of lesions within skeletal muscle.

(Williams et al., 1973). In winter of 1982–1983, 44 birds were trapped using a drop net technique (Glazener et al., 1964) from the Montrose, Pagosa Springs, Trinidad and Pueblo areas in Colorado. Sixty of these 79 turkeys were boxed (provided by the National Wild Turkey Federation) and transported to the Diagnostic Laboratory, Department of Pathology, College of Veterinary Medicine, Fort Collins, Colorado. Birds were removed from transportation boxes, examined, and either (1) were euthanized by exsanguination and necropsied or (2) were maintained in 3 × 5 m wire isolation pens for up to 3 wk following capture. Birds that arrived in Fort Collins during the weekend were held temporarily (1 to 2 days) until they could be necropsied. During the winter of 1980–1981 some birds were maintained in captivity for 3 wk to allow exacerbation of *Mycoplasma* spp. under stress, thus increasing the likelihood of their isolation (Adrian, 1984). Tissues selected for histological examination included heart, skeletal muscle (from thigh, wing and pectoral region) and sciatic nerve. Tissues were fixed in 10% neutral buffered formalin, trimmed, embedded in paraffin, sectioned at 5–6 μm and stained with hematoxylin and eosin.

RESULTS

Clinical signs of capture myopathy were generally not observed because the majority of birds were euthanized as soon as they were removed from transportation boxes. However, one bird trapped during winter of 1982–1983 was found to be paralyzed following removal from its box. This bird could neither fly nor walk, but would lie on its side, and paddle and flap its wings.

Gross lesions compatible with capture myopathy were noted in 0/14 birds during 1980–1981, 0/19 birds in 1981–1982 and 13/27 birds in 1982–1983 (Table 1). Of all birds trapped in 1982–1983, 73% of the immature and 17% of the mature birds had visible lesions of capture myopathy. Gross lesions were characterized by small to large, patchy, pale, white streaked areas within skeletal muscle (Fig. 1). Lesions were noted most commonly in breast muscles (*Pars thoracica* of the *Muscle pectoralis*), wing muscles (*Muscle biceps brachii* and *Muscle triceps cubit*), and leg muscles (*Muscle femorotibialis externus, medius,* and *internus, Muscle caudoilioflexorius, Muscle ischioflexorius, Muscle iliofibularis, Muscle ischiofemoralis, Muscle obturator, Muscle puboischiofemoralis, Muscle flexor perforatus digit II, Muscle flexor perforans* et *perforatus digit III* and *Muscle gastronemius*) (Koch, 1973). These pale areas were accentuated by small white foci that were located regularly along individual muscle bundles. These lesions were commonly bilateral, but not symmetrical. Occasionally, ruptured muscles associated with hemorrhage were noted. Fractured bones were not found in these birds.

Microscopic lesions were confined to myocardial and skeletal muscle. Histological lesions were not found in sciatic nerves. Myocardial lesions were predominantly located in the left ventricle and were characterized by multifocal, irregular areas of coagulative necrosis, collapse of intercel-

Downloaded From: https://bioone.org/journals/Journal-of-Wildlife-Diseases on 28 Mar 2024
Terms of Use: https://bioone.org/terms-of-use

lular stroma and mild to moderate myocardial nuclear proliferation (Fig. 2). Inflammatory cell infiltration was not a prominent feature in the myocardial lesions.

Histological lesions in pectoral, wing or thigh muscles were similar. Three distinct types of lesions were found in skeletal muscle and will be described as Type A, B and C. Type A lesions were characterized by multifocal areas of myocytes containing basophilic sarcoplasm with distinct striations and intact sarcolemmal nuclei which had undergone mild hypertrophy and hyperplasia (Fig. 3). Inflammatory cells were not a prominent feature in Type A lesions. Type B lesions were typical of rhabdomyolysis and characterized by swollen myocytes with eosinophilic sarcoplasm and pyknotic nuclei. Sarcoplasm varied from being eosinophilic and homogeneous with loss of striations to having marked disruption including cleavage and fragmentation of myofibrils (Fig. 4). Infiltration of inflammatory cells was not a feature of this lesion. Type C lesions were characterized by severe rhabdomyolysis with marked proliferation of sarcolemmal nuclei and infiltration of macrophages and heterophils (Fig. 5). Edema was occasionally noted between myocytes. Lesions were not found in vessels.

Small basophilic elongated organisms identified as sarcocysts were found in 3 of 23 birds in 1982–1983. These sarcocysts had not elicited any type of inflammatory reaction and they appeared to be of little consequence to the birds.

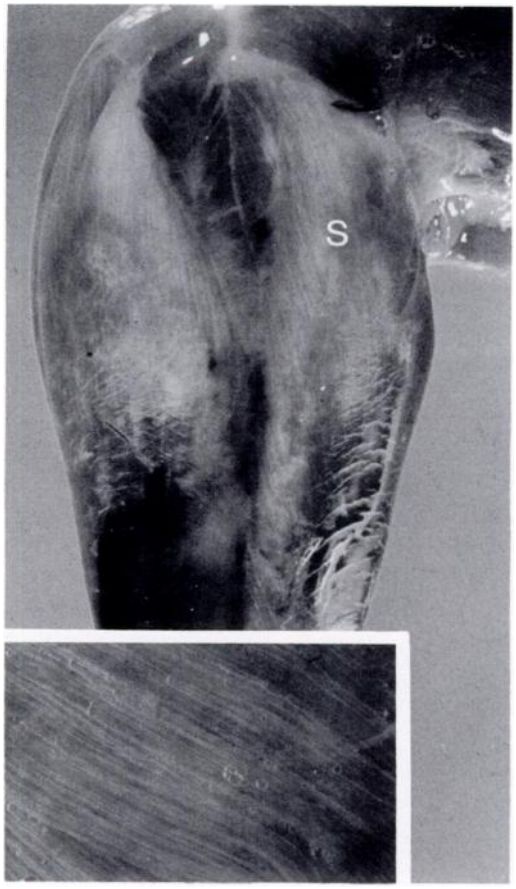


FIGURE 1. Skeletal muscle of a wild turkey with capture myopathy. Note pale streaking (S) of muscle fibers (insert).

DISCUSSION

Trapping, with or without translocation, of wild turkeys is a common management tool used by wildlife biologists. During this procedure mortality of birds due to capture stress/capture myopathy can occur and can result in high mortality.

Clinical signs of capture myopathy were infrequently observed during this study, but the prevalence of capture myopathy at the cellular level was quite high. The reason for the lack of clinical signs may have resulted from the quick and incomplete physical examination that the birds received prior to euthanasia. If they had been allowed to walk or fly more evidence of clinical CM may have been observed.

Macroscopic lesions characterized by pale streaked areas within muscles were found in these turkeys and they were similar to those reported in a greater sandhill crane (Windingstad et al., 1983). The gross lesions observed in more severely affected turkeys were similar to the gross lesions reported in greater and lesser flamingos

Downloaded From: https://bioone.org/journals/Journal-of-Wildlife-Diseases on 28 Mar 2024
Terms of Use: https://bioone.org/terms-of-use

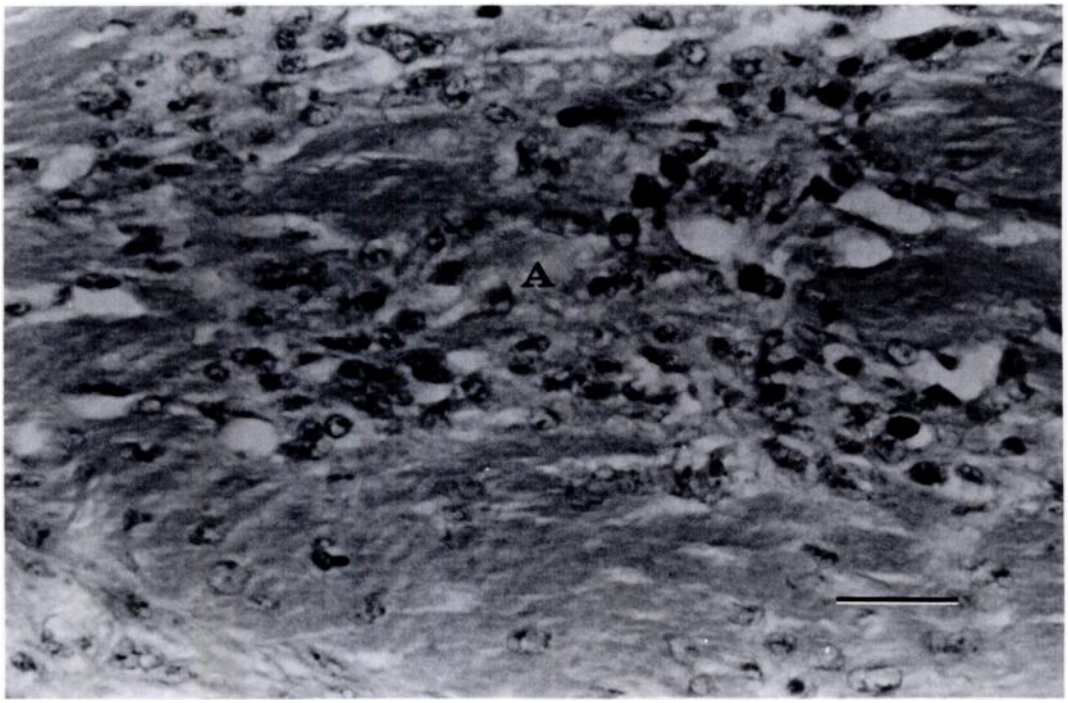


FIGURE 2. Myocardium of a wild turkey with capture myopathy. Note necrosis of myocardial cells, collapse of stroma and myocardial nuclear proliferation (A). H&E. Bar = 100 μm.

(Young, 1967). Both had extensive areas of hemorrhage, but hemorrhage was usually associated with muscle rupture in turkeys.

Gross lesions were not observed in birds during 1980–1981 and 1981–1982, whereas 48% of the birds during the winter of 1982–1983 had gross lesions of CM. Also, of the birds with gross lesions 73% of the birds were immature and 17% were mature. This suggests that immature birds may be more susceptible to CM. There was no difference in the occurrence of CM between sexes. Birds captured in 1980–1981 were trapped in clover traps and in 1981–1982 with clover traps or alpha chloralose treated corn, whereas birds in 1982–1983 were trapped with drop nets. These data suggest that CM was more severe in birds captured with the drop net, as compared to those caught in the clover trap or alpha chloralose treated corn.

The histological lesions in skeletal muscle of wild turkeys were similar to those described in greater and lesser flamingos (Young, 1967), in a greater sandhill crane (Windingstad et al., 1983), and in mammals (Bartsch et al., 1977). In wild turkeys our separation of lesions into 3 types (A, B, C) probably only represented severity and time lapse from muscle damage to death (euthanasia) of the bird. We believe Type A lesions represent fibers that had suffered mild, acute non-fatal damage. Type B lesions represented acute rhabdomyolysis of myocytes and Type C lesions were fibers that had undergone rhabdomyolysis with subsequent phagocytosis by macrophages for the healing process. The pathogenesis of these muscular lesions was related to cellular hypoxia and lactic acidosis (Spraker, 1982). Although skeletal muscles were examined only in 1981–1982 and 1982–1983, the characteristics of lesions were more severe in birds trapped by the drop net. Also, mild to moderate

Downloaded From: https://bioone.org/journals/Journal-of-Wildlife-Diseases on 28 Mar 2024
Terms of Use: https://bioone.org/terms-of-use

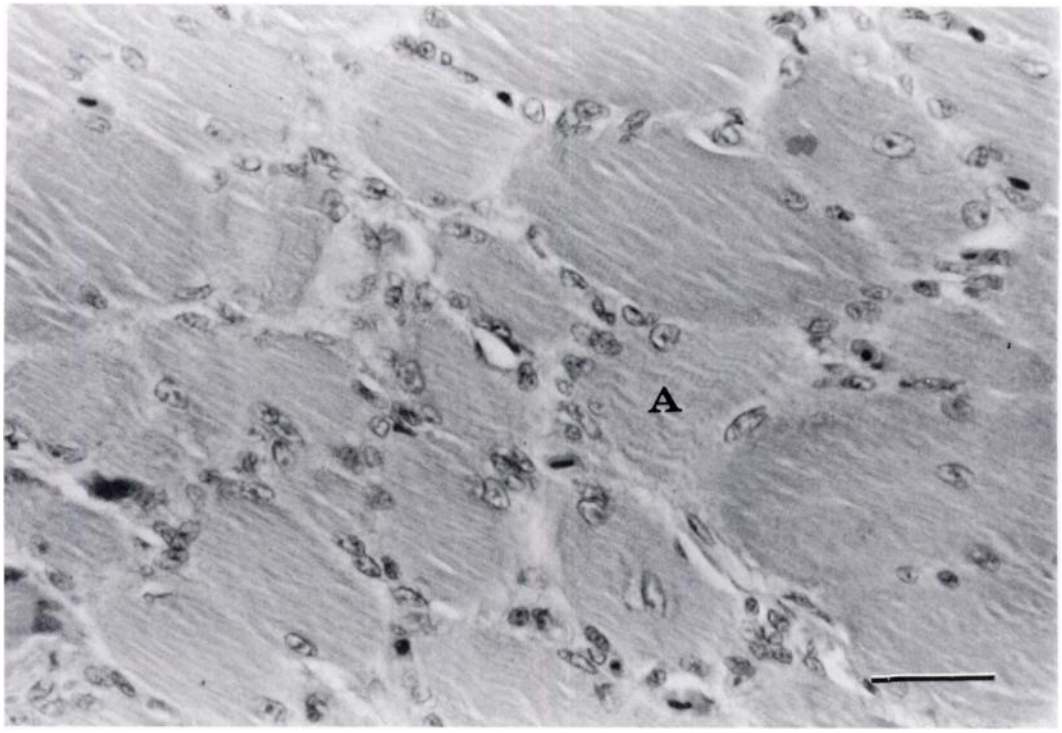


FIGURE 3. Type A (early reversible stage) lesion of skeletal muscle in a wild turkey with capture myopathy. The fibers are characterized by basophilic sarcoplasm, intact striations and hypertrophied and hyperplastic sarcolemmal nuclei (A). H&E. Bar = 100 μm.

rhabdomyolysis occurred in birds trapped during 1981–1982, but gross lesions were not observed. This indicates that without histopathology, CM could be overlooked.

Microscopic lesions in the myocardium of turkeys were similar to those described in baboons (McConnell et al., 1974). Young (1967) described "necrosis" within the myocardium, but did not give a detailed description of the type of necrosis. In the present study, myocardial lesions were more common in birds trapped with the drop net than in the other two techniques. This suggested that the drop net method resulted in greater myocardiopathy.

We feel that most of the birds in this study with myocardial and skeletal muscle lesions probably would have recovered when released into new habitat. However, these lesions could predispose affected birds to a greater chance of predation several weeks following release, or possibly to chronic heart failure. Even if recovered birds healed, myocardial lesions could result in decreased tolerance to exercise. This would alter the birds' ability to escape from predators, or affect their normal behavior patterns.

Our gross and histological data demonstrate that wild turkeys are susceptible to CM. This is an important condition and it can cause loss of animals during trapping and transportation operations. CM is difficult to treat and every effort should be made to prevent the problem. Thus, wildlife personnel should exercise extreme care during trapping, handling and transporting wild turkeys since our results suggest that wild turkeys are extremely susceptible to CM. Moreover, the drop net caused the highest frequency of occurrence of CM, thus particular caution should be exercised

Downloaded From: https://bioone.org/journals/Journal-of-Wildlife-Diseases on 28 Mar 2024
Terms of Use: https://bioone.org/terms-of-use



FIGURE 4. Type B (acute rhabdomyolysis) lesion of skeletal muscle in a wild turkey with capture myopathy. These fibers are characterized by swollen myocytes with eosinophilic sarcoplasm, marked cleavage and fragmentation of myofibrils, loss of striations and pyknotic nuclei (B). H&E. Bar = 100 μm.

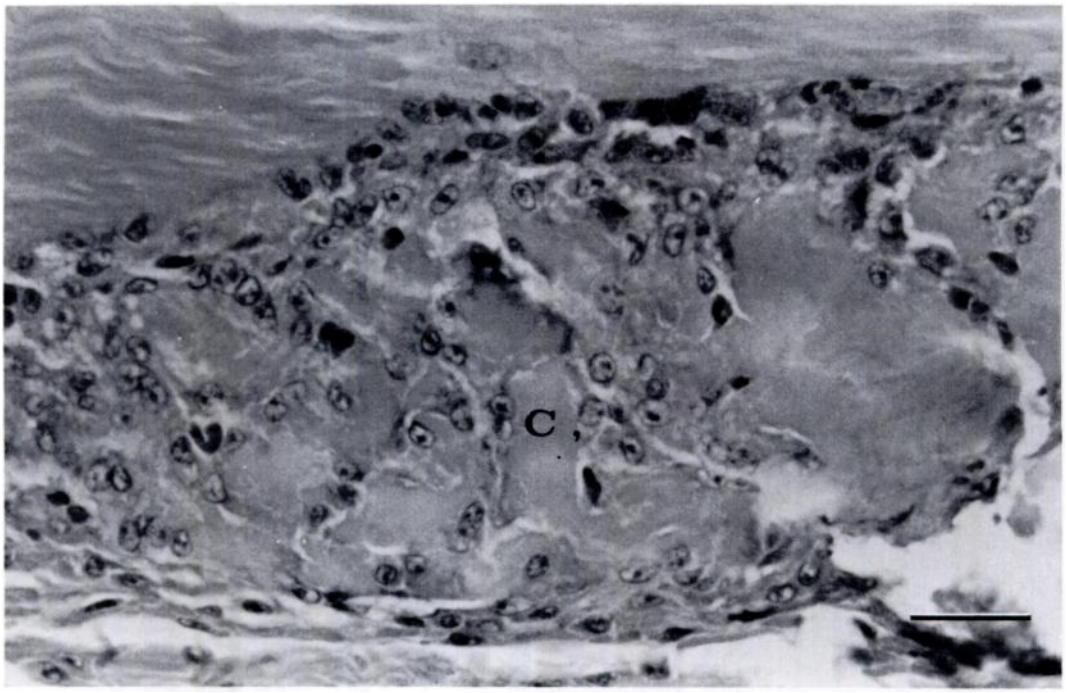


FIGURE 5. Type C (rhabdomyolysis in early healing stages) lesion of skeletal muscle in a wild turkey with capture myopathy. These fibers are characterized by severe rhabdomyolysis with marked proliferation of sarcolemmal nuclei and infiltration of macrophages and heterophils (C). H&E. Bar = 100 μm.

Downloaded From: https://bioone.org/journals/Journal-of-Wildlife-Diseases on 28 Mar 2024
Terms of Use: https://bioone.org/terms-of-use

in capturing and handling wild turkeys trapped by this method.

ACKNOWLEDGMENTS

We thank Lisa M. Monzingo for typing this manuscript and the Colorado Chapter of the National Wild Turkey Federation, the National Wild Turkey Federation, Colorado Division of Wildlife and the National Rifle Association for financial support.

LITERATURE CITED

ADRIAN, W. J. 1984. Investigation of diseases as a limiting factor in wild turkey populations. Ph.D. Dissertation. Colorado State University, Fort Collins, Colorado, 80 pp.

BARTSCH, R. C., E. E. MCCONNELL, G. D. IMES, AND J. M. SCHMIDT. 1977. A review of exertional rhabdomyolysis in wild and domestic animals and man. Veterinary Pathology 14: 314–324.

CHALMERS, G. A., AND M. W. BARRETT. 1977. Capture myopathy in pronghorns in Alberta, Canada. Journal of the American Veterinary Medical Association 171: 918–923.

———, AND ———. 1982. Capture myopathy. *In* Noninfectious disease in wildlife, G. L. Hoff and J. W. Davis (eds.). Iowa State University Press, Ames, Iowa, pp. 84–94.

GLAZENER, W. C., A. S. JACKSON, AND M. L. COX. 1964. The Texas drop-net trap. Journal of Wildlife Management 28: 280–287.

HAIGH, J. C., R. R. STEWART, G. WOBESER, AND P. S. MACWILLIAMS. 1977. Capture myopathy in a moose. Journal of the American Veterinary Medical Association 171: 924–926.

HERBERT, D. M., AND I. MCT. COWAN. 1971. White muscle disease in the mountain goat. Journal of Wildlife Management 35: 752–756.

JARRETT, W. F. H., F. W. JENNINGS, M. MURRAY, AND A. M. HARTHOORN. 1964. Muscular dystrophy in a wild Hunter's antelope. East African Wildlife Journal 2: 158–159.

KOCH, T. 1973. Anatomy of the chicken and domestic birds. Iowa State University Press, Ames, Iowa, pp. 48–51.

LEWIS, R. J., G. A. CHALMERS, W. M. BARRETT, AND R. BHATNAGAR. 1978. Capture myopathy in elk in Alberta, Canada. A report of three cases. Journal of the American Veterinary Medical Association 171: 927–932.

MCCONNELL, E. E., P. A. BASSON, V. DEVOS, B. J. MYERS, AND R. E. KUNTZ. 1974. A survey of diseases among 100 free-ranging baboons (*Papio ursinus*) from the Kruger National Park. Onderstepoort Journal of Veterinary Researach 41: 97–167.

MYERS, G. T. 1973. The wild turkey on the Uncompaghre Plateau. Colorado Department Game and Fish. Pittman-Roberts Report, Project W-37-R, Fort Collins, Colorado, 153 pp.

SPRAKER, T. R. 1977. Capture myopathy of bighorn sheep. *In* Proceedings of Desert Bighorn Sheep Council Transactions. Desert Bighorn Sheep Council, Las Vegas, Nevada, pp. 14–16.

———. 1980. Pathophysiology associated with capture of wild animals. *In* The comparative pathology of zoo animals, R. J. Montali and G. Migaki (eds.). Smithsonian Institution Press, Washington, D.C., pp. 403–414.

———. 1982. An overview of the pathophysiology of capture myopathy and related conditions that occur at the time of capture of wild animals. *In* Chemical Immobilization of North American Wildlife, L. Nielsen, J. C. Haigh, and M. E. Fowler (eds.). Wisconsin Humane Society, Inc., Milwaukee, Wisconsin, pp. 83–118.

WILLIAMS, L. E., JR., D. H. AUSTIN, T. E. PEOPLES, AND R. W. PHILLIPS. 1973. Capturing turkeys with oral drugs. *In* Wild turkey management, G. C. Sanderson and H. C. Schultz (eds.). University of Missouri Press, Columbia, Missouri, pp. 219–227.

WINDINGSTAD, R., S. S. HURLEY, AND L. SILEO. 1983. Captive myopathy in a free-flying greater sandhill crane (*Grus canadensis tabida*) from Wisconsin. Journal of Wildlife Diseases 19: 289–290.

WOBESER, G., J. E. C. BALLAMY, B. G. BOYSEN, P. S. WILLIAMS, AND W. RANGE. 1976. Myopathy and myoglobinuria in a wild white-tailed deer. Journal of the American Veterinary Medical Association 169: 971–974.

YOUNG, E. 1967. Leg paralysis in the greater flamingo and lesser flamingo. International Zoo Yearbook 7: 226-227.

Received for publication 3 June 1985.

Downloaded From: https://bioone.org/journals/Journal-of-Wildlife-Diseases on 28 Mar 2024
Terms of Use: https://bioone.org/terms-of-use