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Flaccid Trunk Paralysis in Free-ranging Elephants (*Loxodonta africana*) in Zimbabwe

N. D. Kock,[1,2] S. A. Goedegebuure,[3] E. P. Lane,[1] V. Lucke,[4] D. Tyrrell,[1] and M. D. Kock[2,5] [1] Faculty of Veterinary Science, University of Zimbabwe, P.O. Box MP167 Mount Pleasant, Harare, Zimbabwe; [2] International Wildlife Veterinary Services, 1850 North Main Street, Salinas, California, USA; [3] Department of Veterinary Pathology, University of Utrecht, P.O. Box 80185, 3508 TD Utrecht, The Netherlands; [4] Department of Pathology, School of Veterinary Science, University of Bristol, Langford, Bristol, BS18 7DU England; [5] Department of National Parks and Wildlife Management, P.O. Box 8365, Causeway, Harare, Zimbabwe

ABSTRACT: An epizootic of flaccid trunk paralysis began in free-ranging Zimbabwean elephants (*Loxodonta africana*) on the southern shore of Lake Kariba in 1989. It involved a selective neuropathy of peripheral nerves supplying the trunk, with axon and myelin degeneration, muscle atrophy, compensatory hypertrophy, and fine endomyseal fibrosis, without inflammatory changes.

Key words: Loxodonta africana, flaccid trunk paralysis, elephants.

At least six free-ranging elephants from Fothergill Island (28°67′E, 16°75′S) and others from nearby Matusadona National Park, Zimbabwe (28°50′–28°80′E, 16°25′–16°75′S), on the southern shore of Lake Kariba, developed flaccid paralysis of the trunk. The first case was noticed by a Department of National Parks and Wildlife Management warden in 1989 on Fothergill Island. A second case was noted about a year later, and four others followed closely thereafter. In July and August 1992, more were reported from nearby Matusadona National Park. The earliest cases all were in older bulls, but affected females recently were documented in Matusadona National Park.

The condition appeared to be progressive and irreversible, although there was question about its progressive nature. Loss of prehension seemed to occur first, followed by ascending flaccid paralysis, finally involving as much as three-quarters of the trunk in some cases. These changes appeared to develop slowly, over some months, although a more exact rate of change was not known. The animals developed remarkable adaptive responses to cope with their impaired feeding and drinking abilities. Initially, the front feet were used in conjunction with the unaffected part of the trunk to scoop forage off the ground, and later on, the trunks were thrown over branches in order to bring forage in close enough proximity to the mouth for feeding. While walking, the trunks often were flung over the tusks in order to avoid being tread upon (Fig. 1). Severely affected animals needed to walk into the lake in order to drink. Behavioral abnormalities and other neuromuscular deficits were not apparent.

Surgical biopsies from the paralyzed trunks of two adult bull elephants ("Crooked Tusk 1" and "Crooked Tusk 2") were submitted to the Faculty of Veterinary Science on 18 May 1989. The animals had been immobilized with etorphine, but no records are available on doses or manufacturer. "Crooked Tusk 2" was humanely shot when wasting became profound; a necropsy was performed on 9 April 1992. A third adult bull, "Mr. Perfect," similarly was killed; a necropsy was performed on 27 November 1991.

The surgical biopsies and sections of skeletal muscles (masseter, temporal, dorsolateral and ventral trunk, biceps, triceps, extensor carpi radialis, vastus, quadriceps, cranial tibialis), peripheral nerves (dorsolateral and ventral trunk, subcutaneous, radial, optic), brain, and visceral organs from the culled elephants were fixed in 10% buffered formalin, embedded in paraffin, sectioned at 10 μm, and stained with hematoxylin and eosin (H&E). Nerves were stained with Luxol fast blue-cresyl violet and nerves and muscles were stained with van Gieson stain (Culling, 1974). Kidney sections were stained with Ziehl-Nielsen stain (Culling, 1974) to test for acid-fast

FIGURE 1. Adaptive response of an elephant with trunk paralysis, preventing injury to the flaccid trunk by carrying it over the tusk.

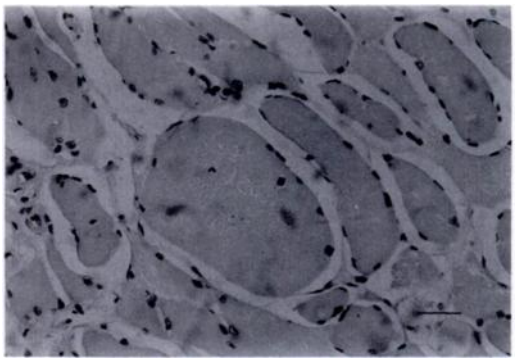

FIGURE 2. Muscle fibers from the paralyzed trunk of an elephant showing great variation in cross-sectional diameters. H&E. Bar = 25 μm.

lead inclusions. The brains were removed whole and sections from the frontal lobe, levels of the corpus callosum, internal capsule, hippocampus, cerebellum, and medulla oblongata were stained with H&E and examined.

Blood and serum were collected from all animals, and 1.5 ml of whole blood from "Mr. Perfect" was injected intraperitoneally into four 6-wk-old Balb/c mice. Complete blood counts (CBC) were done on ethylenediaminetetraacetic acid (EDTA) preserved whole blood, and serum was evaluated for total protein, albumin, globulin, creatine phosphokinase, aspartate transaminase (AST), alanine transaminase, lactic dehydrogenase, alkaline phosphatase, gamma gluteryl transaminase, bilirubin, creatinine, and blood urea nitrogen using an Electro-Nucleonics Biochemical Analyzer (Altaire, Electro-Nucleonics International Ltd., Breda, The Netherlands). Quality control of Altaire procedures was done with Gemcal Electro-Nucleonics references. Ciba-Corning (Ciba-Corning Diagnostic Corp., Irvine, California, USA) normal and abnormal assay sera were used as controls.

All three animals sampled were about 3 m at the shoulder and 30 to 40 yr old. Neither behavior nor gait appeared abnormal just prior to immobilization or culling. The two elephants killed were in extremely poor body condition, evidenced by prominent bony protuberances due to muscle atrophy and scanty visceral fat. The distal half to three-quarters of the trunks were thin and flaccid compared to the proximal parts.

Affected muscles of the trunk from all three animals were similar, and were composed of small angular or rounded fibers and aggregates of nuclei in addition to normal and hypertrophied fibers (Fig. 2). Fine endomyseal fibrosis also was evident with van Gieson stain. Although degenerative changes were minimal, occasional vacuolation of fibers and internally placed nuclei were seen in some sections. Muscle proximal to the level of paralysis was normal in all three animals, as were the skeletal muscles from the two culled elephants, although marked myofiber pleomorphism was seen in the heart of one animal.

Peripheral nerves from the atrophied trunks from all three animals were sparsely cellular when compared to normal proximal parts and other peripheral nerves. Based on van Gieson staining, abundant fibroblast nuclei and fine, immature fibrosis were observed. Enlarged empty axonal sheaths, occasionally containing debris or gitter cells, were seen without inflammatory changes (Fig. 3). Based on Luxol fast blue-cresyl violet staining, there was evidence for myelin loss. Other peripheral nerves from the culled animals were unaffected and central nervous system lesions were not apparent. Significant lesions were

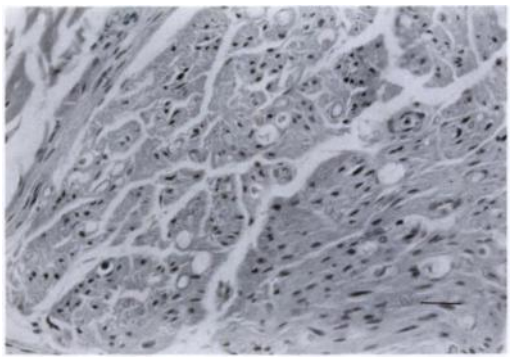

FIGURE 3. Peripheral nerve from an elephant with trunk paralysis. Enlarged axonal sheaths, usually empty but sometimes containing debris or gitter cells were seen without evidence of inflammation. H&E. Bar = 1 mm.

not seen in any visceral organs, and acid-fast inclusions characteristic for lead were not seen in the kidney with Ziehl-Nielsen staining.

Complete blood counts and biochemical analyses were considered normal, based on comparisons with other free-ranging elephants in Zimbabwe (Kock et al., 1993). The mice injected with elephant blood were observed for 6 mo afterwards and did not show any signs of disease.

The major lesion in these elephants was a primary and selective peripheral neuropathy of the nerves of the trunk, with secondary changes in the muscles. Such changes could result from several causes, including trauma, intoxications, and infectious diseases. Local concern for these elephants has been fervent, and much speculation on the cause of the condition had been made, including trauma, either from electric fences or the use of bird shot fired to deter elephants from entering the Fothergill Island resort, toxins used by rural people to discourage crop raiding, cumulative environmental toxins, and infectious diseases. Other possible causes include nutritional deficiencies or perhaps hereditary conditions (Morris, 1989). A single electric fence surrounds the Fothergill Island resort, but the voltage is not high enough to cause significant damage on contact. The likelihood of bird shot or other traumatic injury resulting in such a selective deficit is slight. For the most part, rural people do not inhabit the shoreline at Fothergill Island or Matusadona National Park, making other forms of human intervention unlikely. The absence of inflammatory changes in the tissues likely rules out infectious causes, and while hereditary causes cannot be ruled out, they are considered least likely, leaving nutritional deficiencies and intoxication, or perhaps the two in combination. Neurotoxic properties are accorded to numerous plants and chemical compounds, although the lesions produced are typically less discriminant than those found in the elephants, and agents causing such selective peripheral neuropathy are largely unknown. Chronic lead intoxication in adult humans, however, causes segmental degeneration of axons and myelin of nerves supplying the extensors of the wrists and fingers and the peroneal muscles, muscles which are extensively used (Sullivan, 1985; Cotran et al., 1989). It similarly may affect the nerves of other groups of muscles that are used continually in work or recreation (Polson et al., 1983). Chronic intoxication of horses also may cause selective nerve paresis and paralysis, particularly of the cranial nerves, larynx, and pharynx; liver levels as little as 4 to 7 parts per million have been found in horses with chronic fatal toxicosis (Sullivan, 1985). Adequate dietary selenium can protect against lead and other heavy metal toxicosis (Levander, 1986), and since deficiency is well recognized in Zimbabwe, it is possible that the problem with the elephants involves both deficiency and toxicosis.

The possibility of a central lesion cannot be completely ruled out, for while brain was examined and found to be normal, only a relatively small part was available for examination due to damage incurred in killing the animals. If more postmortem examinations can be done, closer examination of the brain and cranial nerves will be included, and at present attempts are

being made to look into possible environmental toxins and trace mineral levels.

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LITERATURE CITED

COTRAN, R. S., V. KUMAR, AND S. L. ROBBINS (editors). 1989. Environmental pathology. *In* Robbin's pathologic basis of disease, R. S. Cotran, V. Kumar, and S. L. Robbins (eds.). W. B. Saunders, London, England, pp. 492–495.

CULLING, C. F. A. 1974. Handbook of histopathological and histochemical techniques, 3rd ed. Butterworths, London, England, pp. 219, 396, and 446.

KOCK, M. D., R. MARTIN, AND N. D. KOCK. 1993. Chemical immobilization of free-ranging African elephants (*Loxodonta africana*) in Zimbabwe, using etorphine (M99) mixed with hyaluronidase, and evaluation of biological data collected soon after immobilization. Journal of Zoo and Wildlife Medicine 24: 1–10.

LEVANDER, O. A. 1986. Selenium. *In* Trace elements in human and animal nutrition, Vol. 2, 5th ed. W. Mertz (ed.). Academic Press, Inc., London, England, pp. 212–226.

MORRIS, M. A. 1989. The nervous system. *In* Robbin's pathologic basis of disease, R. S. Cotran, V. Kumar, and S. L. Robbins (eds.). W. B. Saunders, London, England, pp. 1443–1444.

POLSON, C. J., M. A. GREEN, AND M. R. LEE. 1983. Clinical toxicology, 3rd ed. Pitman Press, Bath, England, pp. 459–470.

SULLIVAN, N. D. 1985. The nervous system. *In* Pathology of domestic animals, 3rd ed., K. V. F. Jubb, P. C. Kennedy, and N. Palmer (eds.). Academic Press, Inc., London, England, pp. 272–273.

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