

EPILEPTIFORM SEIZURES IN CAPTIVE AFRICAN VULTURES

Authors: MUNDY, P.J., and FOGGIN, CM.

Source: Journal of Wildlife Diseases, 17(2) : 259-265

Published By: Wildlife Disease Association

URL: <https://doi.org/10.7589/0090-3558-17.2.259>

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.

EPILEPTIFORM SEIZURES IN CAPTIVE AFRICAN VULTURES

P.J. MUNDY, Endangered Wildlife Trust, P.O. Box 4190, Johannesburg 2000, South Africa.

C.M. FOGGIN, Veterinary Research Laboratory, P.O. Box 8101, Causeway, Salisbury, Zimbabwe.

Abstract: African vultures are held in captivity at Salisbury, Johannesburg, and Durban, and in each place a number of birds showed epileptiform seizures. Of 17 griffon vultures (*Gyps africanus* and *G. coprotheres*) in Salisbury, three recovered and 11 died after one or more seizures. Of eight vultures of three other species, one Lappetfaced Vulture (*Torgos tracheliotus*) recovered and one Whiteheaded Vulture (*Trigonoceps occipitalis*) died. A variety of diagnostic tests, in particular levels of serum calcium and blood glucose, and histological examination of brains, has so far failed to reveal a cause.

INTRODUCTION

As part of a comparative ecological study on vultures in southern Africa, one of us (PJM) was permitted by various nature conservation authorities to keep a number of birds in captivity. To date (December, 1979), a total of 25 has been taken and kept in a Salisbury aviary, 17 as nestlings, five as first-year birds, and three as adults. Being hand-reared they all became tame and easily handled. Early in this program, a first-year Cape Vulture (*Gyps coprotheres*) developed epileptiform seizures and died nine months later. A number of other birds later showed seizures, and the majority died. Nervous disorders, called "fits" by falconers, are well known in apparently healthy birds of prey, and short-winged hawks (*Accipiter* spp.) are particularly vulnerable (I.R. Edwards pers. comm.). These hawks are considered to be nervous and highly strung, which is not the case with vultures. Although several factors may produce fits in hawks,¹ we have been unable as yet to establish a cause of seizures in the vultures. This paper is therefore intended as a preliminary communication on what appears to be a newly recognised disease in vultures.

REARING AND HOUSING

Vultures were kept in a small low aviary of 120 m³ from 1972 to 1977. Earlier nestlings were reared on a varied diet of lean meat, liver, lung, spleen, and whole guinea pigs, with a mineral and vitamin supplement[□] added daily. Nestlings reared from 1974 onwards remained on the varied meat diet, but bone fragments were added instead of the supplement. From April, 1977, vultures were kept in an aviary of 2400 m³ which allowed the birds ample flight opportunity. Fully grown birds were fed twice a week on a diet of cows' heads and whole rabbits, with bone fragments from the crushed heads. The body mass of the vultures was monitored and the food supply regulated so that the birds remained at the same average mass as wild vultures.

OBSERVATIONS

A Cape Vulture that had been found as a fledgling in November, 1973, at the base of the cliff where it had been raised, had a seizure in July, 1974. For the remaining nine months of its life this bird had many seizures, which occurred

□ Nutri-Cal, Vetex S.A. (Pty) Ltd., Johannesburg, South Africa.

both spontaneously and during handling or feeding. The "fits" in this bird and in most of the other affected vultures followed a typical course. Initially the bird appeared tense for a few seconds, and a series of clonic spasms of the neck followed, resulting in dorsal opisthotonos. The bird would then turn in circles until caught by an obstruction, falling to its side and showing clonic spasms of the wings, legs, bill and neck. Involuntary sounds, or "cackling," accompanied these spasms, which lasted from a few seconds to about 1 min before ceasing abruptly. Lesser seizures, consisting only of a few spasms of the neck, with or without some cackling, also were observed. Following a seizure birds showed a depressed habitus for up to an hour. Slight twitching of the head, neck and body was shown continuously by two birds during the 24 h prior to their deaths.

Two birds showed lethargy and apparent somnolence. A Whiteheaded Vulture (*Trigonocephalus occipitalis*) was taken as a nestling in 1972. During the two weeks prior to its death in April, 1975, the bird could be pushed over and would lie recumbent for a short period. This bird was not seen to have muscle spasms. A Whitebacked Vulture (*G. africanus*), also taken as a nestling, showed somnolence for a few days before dying in October, 1978. Eight months earlier it had a number of seizures but then appeared to recover (no. 13 in Table 1).

Of the 25 vultures taken into captivity 16 developed seizures (Table 1). Fourteen of 17 griffon vultures (*Gyps* spp.) were affected, and three of these recovered. The Whitebacked Vulture (no. 15 in Table 1) that recovered is kept in a separate enclosure within the large aviary; a first-year Cape Vulture and a first-year Whitebacked Vulture have been caged with it since January, 1979, and neither of these latter birds have had seizures. Two vultures from eight of the other three species kept in the aviary, were affected;

a Whiteheaded Vulture died and a Lappetfaced Vulture (*Torgos tracheliotus*) recovered. The remaining six birds of these three species have not had seizures, but two Hooded Vultures (*Necrosyrtes monachus*) and one Whitebacked Vulture were killed by larger birds. Thus, since a seizure was first observed in a Cape Vulture, all 14 griffon vultures caged in the main aviary have developed seizures, whereas only two birds of six of the other species of vulture were affected. Also caged with the vultures since April, 1977, are two Fish Eagles (*Haliaeetus vocifer*), one Black Eagle (*Aquila verreauxi*), and one Bateleur Eagle (*Terathopius ecaudatus*); none of these have had seizures.

A typical sequence of events can be illustrated by the histories of two adult Cape Vultures, which had lived together for years in a small aviary in South Africa. They came to the Salisbury aviary in April, 1977, and bred together unsuccessfully in mid-1978. Both developed seizures in December, 1978; the male died immediately, and the female recovered.

Blood chemistry analyses were done on several samples from 17 vultures. The results for serum calcium and for glucose are shown in Table 2, and it must be emphasised that the glucose values are for whole blood. Calcium was determined by atomic absorption spectrometry using a Varian Tectron single beam instrument (non protein precipitation), and glucose by the modified Hultman method.³

The vultures form four groups. Samples were obtained from griffon vultures of both species that have recovered or not had seizures (group no. 1 in Table 2). Other samples came from griffons that later died (group no. 2), and a few samples came from some of these birds while they were having a seizure (group no. 3). Finally, the fourth group comprised vultures of the other three species. The results for calcium are remarkably uniform, and there are no significant intergroup differences (*t*-

TABLE 1. Histories of vultures that exhibited epileptiform seizures in a Salisbury aviary.

	Species*	Date into captivity	Age**	Origin***	Date into aviary	First seizure observed	Result
1.	CV	Nov. 73	fl.	RSA	Dec. 73	July 74	Died Apr. 75
2.	WBV	Nov. 72	ch.	ZR	Nov. 72	Oct. 74	Died Dec. 74
3.	WBV	Aug. 74	ch.	ZR	Dec. 74	Dec. 74	Died Dec. 74
4.	WHV	Nov. 72	ch.	ZR	Nov. 72	Apr. 75 (somnolence)	Died Apr. 75 (somnolent)
5.	LFV	Aug. 74	ch.	ZR	Jan. 75	Mar. 75	Recovered
6.	CV	Jan. 75	fl.	RSA	Jan. 75	Mar. 75	Recovered
7.	WBV	Aug. 76	ch.	ZR	Dec. 76	Jan. 77	Died July 77 (during seizure)
8.	WBV	Aug. 76	ch.	ZR	Dec. 76	Jan. 77	Died July 77 (during seizure)
9.	WBV	Aug. 76	ch.	ZR	Dec. 76	Mar. 77	Died Oct. 77 (during seizure)
10.	CV	?	ad.	RSA	Apr. 77	Dec. 78	Recovered
11.	CV	?	ad.	RSA	Apr. 77	Dec. 78	Died Dec. 78 (during seizure)
12.	WBV	Oct. 77	ch.	ZR	Jan. 78	Feb. 78	Died Feb. 78 (during seizure)
13.	WBV	Oct. 77	ch.	ZR	Jan. 78	Feb. 78	Died Oct. 78 (somnolent)
14.	CV	Jan. 78	fl.	RSA	Feb. 78	Apr. 78	Died June 79 (during seizure)
15.	WBV	June 78	1st yr	ZR	June 78	Oct. 78	Recovered
16.	CV	?	ad.	RSA	June 79	Oct. 79	Died Oct. 79 (during seizure)

*Species are the Cape Vulture (CV), Whitebacked Vulture (WBV), Whiteheaded Vulture (WHV), and Lappetfaced Vulture (LFV).

**Ages are nestlings (ch., hand-reared), fledglings (fl., wild-reared), first-year (1st yr), and adults (ad.).

***Originally taken into captivity in South Africa (RSA) or Rhodesia, now Zimbabwe (ZR).

TABLE 2. Values of serum calcium and whole blood glucose for captive vultures of five species.

	Calcium (mg/dl)				Glucose (mg/dl)			
	no. of birds	no. of bleeds	mean (\pm S.D.)	range	no. of birds	no. of bleeds	mean (\pm S.D.)	range
1. Recovered/healthy griffons*	5	17	10.06 (\pm 0.52)	9.24-10.80	5	60	118.4 (\pm 12.5)	102.1-159.5
2. Griffons that died	7	16	10.13 (\pm 0.66)	8.70-11.20	7	49	121.2 (\pm 10.2)	102.3-160.4
3. Bled during seizures	5	7	9.99 (\pm 0.51)	9.30-10.70	3	4	122.5 (\pm 15.2)	101.0-136.5
4. Other species**	5	19	10.12 (\pm 0.70)	8.69-11.35	5	12	139.6 (\pm 16.5)	121.6-180.3

*Both Cape Vultures and Whitebacked Vultures.

**Lappetfaced, Whiteheaded, and Hooded Vultures.

tests, $P > 0.6$). These values are at the top end of the range for some American raptors.⁴ We could not separately determine the ionized calcium fraction, but the average of 34 values for serum total protein (g/dl) was 3.68 (S.D. \pm 0.46, range 2.89 - 4.92), and the average albumin/globulin ratio was 0.47. From a formula given us by J. Hattingh (Department of General Physiology, University of the Witwatersrand, Johannesburg), based on the serum total protein, the average level of ionized calcium was calculated at 6.13 mg/dl, or 61% of the average serum calcium.

Glucose values show more variation, and the group of recovered/healthy griffons had significantly lower values than did the other non-griffon species (*t*-test, $P < 0.001$). However, glucose values for all griffon vultures, including the four blood samples taken while the birds were in a seizure, were very similar (*t*-tests, $P > 0.2$). The whole blood glucose values for the vultures, after multiplying by approx. 2 to convert to plasma glucose, are at the very bottom of the range for some American raptors.⁴ One Whitebacked Vulture died after being in status epilepticus for about 20 h, and a blood sample was taken immediately after death from the ruptured vena cava during necropsy. The calcium and glucose values (excluded from Table 2) were 10.38 and 3.6 mg/dl respectively.

Post mortem examination was carried out on nine vultures which had developed seizures. The most significant lesions were present in the brain, with two birds (nos. 11 and 16 in Table 1) showing extensive hemorrhage in the forebrain (Fig. 1), which microscopically appeared to be perivascular. Many of the smaller vessel walls were obliterated, and others were necrotic, and the larger vessels showed karyorrhetic nuclei and lymphocyte and heterophil reaction. Moderate edema was also present. It appeared that the hemorrhage was secondary to the vasculitis, but a similar



FIGURE 1. Acute hemorrhage, forebrain of a Cape Vulture, *Gyps coprotheres*.

less marked vasculitis was present in some of the brains in the absence of any hemorrhage.

An acute hepatitis was evident in the liver of one of the birds which had cerebral hemorrhage. Small foci of necrosis with lymphocyte and polymorph reaction were present and there was some cellular infiltration in the portal tracts. No significant bacterial isolates were made from the organs of any of the dead birds. No viruses were recovered by inoculation of brain material into calf testis cell culture or suckling mice.

Four birds were given medical treatment. A Whitebacked Vulture (no. 8 in Table 1) was given intravenous Sagatal[‡] when it entered a continuous seizure but it died later the same day. Another Whitebacked Vulture (no. 13)

developed seizures in February, 1978, and was given intravenous dextrose saline and Lincocin[§].[¶] It recovered and remained healthy until October, 1978, when it died. The adult female Cape Vulture (no. 10) was given intramuscular Valium[‡] every day for seven days after it had a seizure. This bird has remained healthy. A second, immature Cape Vulture (no. 14), entered a continuous seizure and was given intramuscular Valium and Neurobion[§].[¶] It lost consciousness, but maintained some muscle tone. It died about 40 h later, still unconscious. We have not treated additional birds, because we do not believe that this will tell us the cause of the seizures. As three birds have recovered without treatment, we therefore do not know if Valium controlled or cured the seizures in the adult Cape Vulture.

[‡] Sagatal, Maybaker (S.A.) Pty Ltd., Port Elizabeth, South Africa.

[§] Lincocin[®], The Upjohn Company, Kalamazoo, Michigan 49001, USA.

[¶] Valium 10, Roche Products (Pty) Ltd., Isando, Transvaal, South Africa.

[¶] Neurobion[®], Merck Pharmaceuticals (South Africa) (Pty) Ltd., Wynberg 2199, South Africa.

DISCUSSION

Seizures occurred in all ages of birds whether taken as nestlings and hand-reared, or taken as older birds. Similar epileptiform seizures have been shown by a first-year Cape Vulture in a Durban aviary; it subsequently died (S.E. Piper pers. comm.). Two nestling and two first-year Cape Vultures in a Johannesburg aviary also developed seizures; three of these died (R. Friedman pers. comm.). Hitherto, epileptiform seizures appear unreported in vultures, even though very many birds are kept in zoos throughout the world, where they can live to ages of 30 or more years.² Indeed, 14 *Gyps* vultures have been kept for many years in an aviary at Pretoria Zoo with no occurrence of seizures (E. Marais pers. comm.).

A number of factors are known to produce nervous disorders in birds of prey, among them hypocalcemia, hypoglycemia, vitamin deficiencies (especially perhaps of thiamine), trauma, infections with bacteria or viruses, and

poisoning, particularly with pesticides.¹ The captive vultures are well fed and supplied with bone fragments, and frequent blood chemistry analyses showed constant levels of calcium and glucose, even in those birds sampled during a seizure. We therefore discount both hypocalcemia³ and hypoglycemia as causes of the epileptiform seizures. The varied diet, inclusive of whole animals, and the selectivity of the syndrome for the *Gyps* spp. militate against vitamin deficiencies, though it is accepted that this is a possibility. Similarly, because only one bird had seizures at any one time, and because the vultures' food is supplied by a local abattoir, we discount poisoning by pesticides or other compounds. However, the dead vultures have not been analysed for these. Although a few brains had lesions, we do not yet know how these are involved.

The sequence of events in the vultures suggests an infectious disease. However, bacteriological and virological investigations have so far proved negative, though tests are continuing.

Acknowledgements

The vultures were taken into captivity under various licences from the Department of National Parks and Wild Life Management (Zimbabwe) and the Nature Conservation Division of the Transvaal Province (South Africa). The vultures were originally supported by the Department of Zoology, University of Rhodesia, and currently by the Conservation Trust of Zimbabwe. Harry and Marie Scott care for them at Larvon Bird Gardens, Salisbury. B. Currell and F. Reyers did the biochemical analyses and R. Swanepoel the virology at the Veterinary Research Laboratory, and R.M. Barlow (Edinburgh), J.E. Cooper (London) and R. Plotkin (Johannesburg) examined the brains. Help was also given by I.R. Edwards, R. Friedman, C.M. Haxen, Johannesburg Zoo, E. Marais, A. Morris, C.R. Parry, S.E. Piper and R.I.E. Smith. We are particularly grateful to John E. Cooper for help and advice and for commenting on the manuscript.

LITERATURE CITED

1. COOPER, J.E. 1978. *Veterinary Aspects of Captive Birds of Prey*. Standfast Press, Saul (England), 256 pp.
2. FLOWER, S.S. 1938. Further notes on the duration of life in animals — IV Birds. *Proc. zool. Soc., Lond.* 107: 195-235.
3. HYVÄRINEN, A. and E.A. NIKKILÄ. 1962. Specific determination of blood glucose with o-toluidine. *Clin. Chim. Acta* 7: 140-143.

4. IVINS, G.K., G.D. WEDDLE and W.H. HALLIWELL. 1978. In: *Zoo and Wild Animal Medicine*, M.E. Fowler, ed. Saunders, Philadelphia, U.S.A., 951 pp.
5. WALLACH, J.D. and G.M. FLIEG. 1970. Cramps and fits in carnivorous birds. *Int. Zoo Yrbk* 10: 3-4.

Received for publication 20 February 1980
