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SHORT NOTES

Optometric examination of the Kudu *Tragelaphus strepsiceros*

by
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I INTRODUCTION

Motoring in certain parts of South West Africa is hazardous. A number of accidents, some with fatal results, occur each year, mainly at night, as a result of kudu jumping into or colliding with cars. These accidents occur on country roads as well as on highways.

In view of this, an investigation into the visual state of the kudu's eyes has been undertaken. Gemsbok (*Oryx gazella*), wildebees (*Gorgon taurinus*), horse (*Equus caballus*) and hyrax (*Procavia capensis*) were examined so that a comparative analysis could be made. Except for the horse, as far as is known, research of this nature has not been undertaken before.

Theories abound about the cause of these accidents; the speed of the car; the intensity of the headlights; the kudu's own shadow which it attempts to avoid; and migration to the highways in winter seeking vegetation at the shoulder of the road, have been postulated.

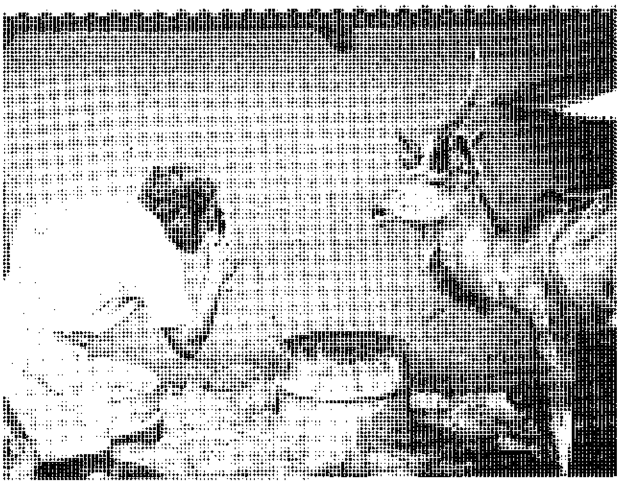
II MATERIALS AND METHOD

Five kudu, two gemsbok, a horse, a wildebees and a hyrax were studied in this analysis. Examinations took place in the Etosha National Park and on a farm 90 kilometres south west of Windhoek in the Khomas Hochland. All but one of the animals, a kudu cow, were living in captivity, but for no longer than a month. The examination of these took place in animal enclosures, while the examination of the kudu cow (labelled Yellow 14, for research purposes) was performed in the open veld. Yellow 14 was darted and drugged prior to her examination, the other animals were examined in an undrugged state. Results differed somewhat due to the effects of these drugs which will be discussed later in this article.

Yellow 14 was immobilized after a dart was fired into her rump by Dr Ebedes, the veterinary surgeon in the Etosha National Park. The dart contained the following chemicals: Etorphine hydrochloride 4 mg, phencyclidene hydrochloride 60 mg, acetyl promozine maleate 20 mg, and 5 mg hyoscine hydrobromide to counter the constriction of the pupil that etorphine (a morphine derivative) causes.

Some 65 minutes before Yellow 14 was hit, a herd of kudu in the vicinity of the Ombika waterhole, about 10 kilometres south of the Okaukuejo rest-camp in the Etosha National Park, was observed. Practical difficulties and the apparent extrasensory perception of our motives kept the kudus at a safe distance from the research party. (Possibly the herd had spotted us.)

When an immobilizing dart was eventually fired at Yellow 14 and a direct hit scored, she showed no reaction for 60 seconds and then, seeming to notice the dart for the first time, took fright and charged off into a wooded area. With the aid of Joseph, a Bushman tracker, we located our kudu some 60 minutes later.



Optometric examination of kudu.

All the animals examined were held down during the examination, including Yellow 14, who was not completely unconscious. The smaller kudu and the two gemsbok were examined in a small dark cellar with only one helper holding the animal's horns. The larger kudu were examined in bright sunlight. The horse and wildebees were examined at dusk. Examinations were conducted with various rulers, high intensity torches, ophthalmoscope retinoscope and trial lenses.

III RESULTS

The results obtained from examining five kudu, two gemsbok, one horse, one wildebees and one hyrax are summarised in Table I.

IV DISCUSSION

4.1 External examination

Location: The enormous eyes located at the sides of the head pointed in diverging directions and resulted in the huge interpupillary distances. The eyes being so placed would give the kudu, and the other animals to a lesser extent, a large field of vision. To a hunted animal this is obviously a distinct advantage. However, with the eyes so placed, binocular vision at most angles would be difficult to attain. Binocular vision is a prerequisite for good stereoscopic vision and distance judgement, and hence the kudu's distance judgement would be decidedly poor. This would apply to the other animals studied, but to a lesser extent as their eyes were placed more to the front of the head. The kudu would tend to look with one eye at a time.

THE PUPIL: The hyoscine injected into Yellow 14 more than counteracted the effects of the etorphine. However, Yellow 14's stage of anaesthesia and fright also caused dilatation of her pupils. Her pupil size was almost double that of the kudu which were not drugged.

Pupil reactions in all but one of the kudu were negligible. This could be indicative of some occular or nerve pathway lesion. Sustained dilatation of the pupil may have been caused by fear but as an extremely intense light was used, and in some cases in complete darkness, some sort of pupil reaction should have been elicited.

The function of the pupil is principally optical. Contraction reduces the amount of light entering the eye. It acts as an emergency mechanism, giving the retina time to adapt to the illumination. Narrowing of the pupil cuts off the peripheral parts of the refracting system. Hence it diminishes spherical and chromatic aberration and astigmation caused by oblique pencils of light. It also increases the depth of focus — e.g. in man from f2,5 to f13. The shape of the pupil has no effect on the clarity of the image provided enough light enters the eye and the eye is corrected to emmetropia. Whenever the intensity of illumination increases above a threshold value within a certain minimum time the pupil contracts, provided everything is normal. The chief factor which determines what effect a change of illumination will have on the pupil is the state of adaptation of the retina.

The kudu's pupils not reacting to light could be caused by lesions in the following pathways:— (a) The afferent pathway of the light reflex which includes structures from the rods and cones up to the pretectal region. (b) The intercalated fibres from the synapse in this region into the posterior commisure from which the fibres bifurcate and run to the Edinger-Westphal nuclei of both sides. (c) Supranuclear afferent pathways coming from undetermined regions of the brain and having to do with associated movements of the pupil e.g. near or obicularis reaction. (d) The occulomotor nucleus itself, i.e. the Edinger-Westphal nucleus. (e) The occulomotor nerves from the Edinger-Westphal nucleus to the cilliary ganglion. (f) The cilliary ganglion and the short cilliary nerves and their endings in the sphincter muscle, both cholinergic and adrenergic. (g) The myoneural junction of the 3rd cranial nerve in the sphincter muscle. (h) The cells of the sphincter muscle themselves. (i) Any part of the afferent sympathetic pathway from the hypothalamus through the brain stem to the superior cervical ganglion and thence into the eye by way of the short cilliary nerves. However, as there was also no observable consensual reaction the most likely site of a lesion, if a lesion existed at all, would be in the vicinity of the retina.

An interesting observation was made at the Okahandja Zoo. Two very tame fully grown kudu, one bull and one cow, were fed peanuts by hand while an observation of their pupils was made. The pupil in the eye which was facing a bright setting sun was contracted into a slit, the size measuring 25 × 5 mm. The fact that such a marked contraction was observed with these tame kudu seems to indicate fear as being a significant factor in causing the pupil dilatation of the other kudus examined which were not tame.

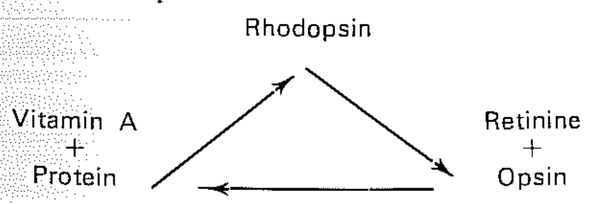
4.2 Internal examination

Ophthalmoscopic examinations revealed clear cornea, anterior chamber, crystalline lens and vitreous in all animals included in this analysis. A tapetum lucidum was not observed in the younger kudu and hyrax. The tapetum lucidum is an opaque membrane situated posterior to the retina. It reflects light, hence the green glow that many motorists have seen at night in South West Africa. The function of the tapetum is to re-transmit the image that has already been formed on the retina. It brightens the image without causing dazzle.

4.3 The retina

The retina in all the kudu showed black choroidal pigmentry patches interspersed throughout. It has been postulated that often with degeneration of rods and cones there is a migration of pigment into the retina. Pigmentary degeneration may be intimately connected with night blindness.

The retina is only stimulated by those rays reaching it. The intrinsic sensitivity of rods (and cones) is changed by the presence of retinal pigment. Rhodopsin is a magenta coloured pigment rapidly bleaching in light to become colourless. Rhodopsin = Carotene + Porphyropsin. In scotopic (dark adapted) vision, the amount of energy required to stimulate vision is least with light of wave length 510 mu. This supports the contention that the primary photochemical process in scotopic vision is the absorption of light quanta by rhodopsin and a certain number of quanta must reach the retina in order to evoke a given visual sensation. Pigments which absorb light are necessary for the function of the retina and the action of light on these pigments is to break them down into one or more active products which stimulate the photoreceptors. These then discharge an impulse in the optic nerve fibres. The constant breakdown of pigment in light and its reformation in the dark establishes a "steady state" depending on the degree of illumination. This steady state is produced as a result of balance between catalysis and synthesis and exists as long as the illumination remains unchanged. The synthesis of rhodopsin from retinene and scotopsin requires no external source of energy, being a spontaneous reaction. Whenever these two substances are present together in the dark, they form rhodopsin and yield energy spontaneously. The synthesis of rhodopsin from Vitamin A plus scotopsin requires energy. The basic mechanism of rhodopsin synthesis therefore is energy demanding oxidation of Vitamin A to retinene coupled with energy, yielding condensation of retinene and opsin to form rhodopsin.



The sensitivity of the retina is at a maximum when the concentration of rhodopsin is at a maximum. This occurs in the state of dark adaptation when the sensitivity of the eye is more than ten thousand times as great as when the eye is light adapted. Breakdown of rhodopsin occurs when light is admitted to the dark-adapted eye. The bleaching of the first molecules of rhodopsin has a relatively enormous effect on the sensitivity of the retina and results in a large fall. Much of the light and dark adaptation involves the first and the last small portions of rhodopsin that is bleached and resynthesised.

A blue-green light is absorbed strongly by rhodopsin whereas an orange light is scarcely absorbed at all. Vitamin A deficiency, liver disease, abnormalities associated with bile absorption and disorders of the large intestine are causes of night blindness.

4.4 Retinoscopy

This examination to determine the lens prescription was performed on all the animals. Only Yellow 14 appeared to be myopic, and in her case an arbitrary 0,75 was deducted from the prescription found, due to the hyoscine instillation.

V CONCLUSION

In the case of the kudu examined, poor vision is indicated by their large unreacting pupils, aggravated by a small refractive error. Retransmission of the retinal image by the tapetum lucidum would tend to offset this. Unlike the findings on a survey conducted on African elephant, none of the animals in this survey exhibited any conjunctival or corneal pathology of traumatic or other aeteology. Vision must therefore be good enough to cause an avoidance of objects which might injure the eyes, such as thorn trees and bushes. However, the kudu must rely on its other senses, such as hearing, to react quickly enough to an on-coming car. (Depending on the direction from which the wind blows, this may not always be possible.)

Binocular vision and night blindness seem to be very significant factors in this survey. In the case of kudu poor distance judgement is indicated by the following facts:—

1. Collisions with kudu have taken place in daylight.
2. Kudu often become entangled in fences when they misjudge their jumps.
3. When collisions have taken place at night, the kudu have jumped on to the vehicle and not over it. It is likely that the kudu views the oncoming car with one eye only. This eye would be temporarily blinded by the bright headlights of the car. Vision would be suppressed in that eye and switched over to the other eye, which is not viewing the car, and is consequently not blinded. This eye sees its own shadow moving and the animal jumps away from it in fright, on to the car.

It is claimed that if the interior lights of the car are burning, the kudu will vault the car. This probably happens as the kudu will assess the size of the car more accurately. Furthermore, the contrast of the headlights and the dark night would not be as great. This would shorten the time required to resynthesise rhodopsin i.e. lessen it's night blindness. While the kudu is browsing in the dark, its rhodopsin balance would be in a "steady state". As soon as the headlights become visible the "steady state" is affected and rhodopsin breakdown takes place. If the kudu lacks Vitamin A (as it probably does in winter) resynthesis of rhodopsin is delayed and the possibility of a collision is increased. As the ahsorption maximum of rhodopsin varies in certain animals (497 mu in man) the colour of the headlights could be of significance.

VI SUMMARY

Of the ten animals examined in this programme, the kudu's vision is undoubtedly the worst. Unreacting large pupils, small refractive errors and night blindness seem to be strong contributory factors in collisions between motor vehicles and kudu at night. Fear and hyoscine had a dramatic effect on the kudu pupil far outweighing the constriction caused by the etorphine hydrochloride. Undrugged animals produced the best results as no arbitrary estimations due to the effect of the drug on the eye had to be made.

VII ACKNOWLEDGEMENTS

The writer wishes to express his appreciation to the Department of Nature Conservation and Tourism SWA for allowing the darting of a kudu in the Etosha National Park. Sincere appreciation is expressed to Mr Attie Port of the farm "Valencia", where the greater part of this survey was conducted. I wish to thank Dr H. Ebedes of the Etosha National Park, and Prefessor Hank Hoffstetter of Indiana University, Bloomington USA, for their constructive comments and for encouraging me to publish this paper.

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Species	Identity No.	Age Months	Sex	Inter Pupil Dist. mm	Palperbral Apperture Size mm	Pupil Size mm	Pupil Reactions	Pupil Shape	Ametropia Correction D	Pigmentry patches on retina
Kudu	Yellow 14	Adult	F	165	45x28	30x25	Nil		R. −0.25 L. −0.75	Present
Kudu	Port 1	1	M	90	43x20	15x10	Nil		R. + 2.00 L. + 2.00	Present
Kudu	Port 2	1	F	110	43x20	15x6	Fair		R. + 1.00 L. + 1.00	Present
Kudu	Port 3	15	M	160	46x25	15x6	Nil		R. + 1.00 L. + 1.00	Present
Kudu	Port 4	30	M	175	50x30	15x10	Nil		R. + 2.50 L. + 2.50	Present
Horse	Bingo	52	Mg	145	35x25	25x16	Good		R. + 1.00 L. + 1.00	Not Present
Gemsbok	Port 1	20	F	120	30x25	20x10	Good		R. + 0.50 L. + 0.50	Not Present
Gemsbok	Port 2	9	F	140	30x25	20x10	Good		R. + 1.00 L. + 1.00	Not Present
Wilde-bees	Lashes	9	M	100	30x23	25x20	Good		R. + 0.75 L + 0.50	Not Present
Hyrax	Port 1	24	F	40	5x3	4x2	Good		R & L + 0.50 −1.00 × 90°	Not Present