refusal of food, creeping into corners; by the second or third day their gait was already staggering, balance was disturbed and the animals ran against the wall as though unable to see it. After this, the illness took a fairly swift course with attacks of tonic-clonic cramps, which quickly led to paralysis and death on the fourth or fifth day. Post-mortem examinations of the animals revealed the following:

Cheetah No. 1 ♂. Toxic degenerative changes of the liver.
Cheetah No. 2 ♂. Toxic dystrophy of the liver.
Cheetah No. 3 ♀ (young animal). Laennec's cirrhosis of the liver.
Cheetah No. 4 ♀ (young animal). Laennec's cirrhosis of the liver.

Serological tests on all four animals were negative for toxoplasmosis and leptospirosis.

Thus post-mortem examination showed that all animals had died from a similar cause, namely a degenerative disease of the liver. Virus diseases cannot be completely ruled out as causing the degenerative changes in the liver, but it is improbable. However, the possibility of bacterial diseases and toxoplasmosis or leptospirosis can definitely be excluded. In our opinion, the cause of the changes in the liver must be sought in a toxic agent present in the food, or in parasites. The animals' food consisted of the meat diet described by Wackernagel (1961) and up to three or four times a week, a freshly slaughtered rabbit. Infection with internal parasites seems to be the most probable condition associated with their death. The animals frequently suffered from ascarid infections which were repeatedly treated with anthelminthics. This raises the question as to whether the elimination of the ascarids with the frequent anthelminthic treatment (with Piperazine) had a toxic effect and was thus responsible for the degenerative changes in the liver cells. On the other hand, the very insignificant ascarid infection found in one of the male animals that died is partial evidence against this since the animal in question was only treated with Piperazine on two occasions.

The findings of the histological examination made by the State Veterinary Testing Service of the testicles and ovaries of two of the animals that died were as follows:

Animal No. 2 ♂. The tissues of the testicles showed spermatogonia and spermiocytes in great numbers. However, spermiogenesis was deficient in its prespermid form (i.e. spermatids and sperm were not present). In addition, Leydig cells and Sertoli sustentacular cells were small and atrophic.

Animal No. 4 ♀. The germinal epithelium of the ovary appeared to be generally lacking in primordial follicles. However, primary, secondary and medium-sized vesicular follicles were present. There was no fertile vesicular follicle.

No general conclusion can be drawn from these findings. It is necessary to obtain more data from examination of other animals. Of the cheetahs at Krefeld Zoo, however, one can conclude that further births would not have occurred because of the degeneration in the male sexual organs. Moreover, although oestrus occurred in the original female, it was never observed in the two young animals.

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TREATMENT OF CUTANEOUS GRANULOMATA IN THE BLACK RHINOCEROS

Diceros bicornis

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MICROFILARIAE and adult filarids were found in the Black rhinoceros, *Diceros bicornis*, and described as the cause of cutaneous granulomata (Schultz and Kluge, 1960). The incidence of this condition is very high among Black rhinoceroses in South Africa. Some of these granulomata acquire enormous dimensions, giving the animals a repulsive appearance.

The first sign of a developing lesion is necrosis and subsequent sloughing of the

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superficial layers of the skin. The absence of flies and regular spraying with insecticides do not prevent the appearance of new lesions. If the animal, however, is sprayed regularly with insecticides, the development from an ulcer to a granuloma appears to be slower. This may be due to the filaricidal effect of the insecticide or to the absence of flies.

All efforts to treat our affected animals proved unsuccessful until we employed daily local treatment with the following combination of drugs.

Idoform (Bactericidal and fly repellent)	10 per cent
Sulfanilamide (Bacteriostatic)	10 per cent
BHC (Insecticide. Formulation Multibenhex containing 75 per cent BHC)	10 per cent
Zinc oxide (Mild astringent)	20 per cent
Stockholm tar *qs ad* (Antiseptic and antipruritic)	

When the growths had already reached large dimensions, 10 per cent of the zinc oxide in the mixture was replaced by copper sulphate. After most of the granulation tissue had been removed by the copper sulphate, treatment with the mixture described above was continued. As a rule, most of the lesions healed within seven days after the start of local treatment. New lesions appeared repeatedly and we had to treat our rhinos continuously during the warmer months when they suffered most from this skin condition.

Some of the granulomata disappeared slowly after the daily local application of the 'Onderstepoort Blowfly Remedy' which contains 65 per cent alcohol (96 per cent), 32·35 per cent benzol, 2·5 per cent cresol (98 per cent) and 0·15 per cent sulphuric acid (*conc. comm.*).

It seems possible that these lesions may be an external manifestation of systemic filariasis. Local treatment is only of temporary help and systemic treatment may prove to be more effective. Further experiments must be conducted to find a safe and effective remedy by systemic treatment.

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WHITE MUSCLE DISEASE IN A BREEDING HERD OF NYALA ANTELOPE *Tragelaphus angasi* AT NEW YORK ZOO

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IN 1962 an extensive investigation was undertaken to determine the cause of a relatively high death rate among the younger animals in the nyala herd, *Tragelaphus angasi*, at New York Zoological Park. Most of the animals that died were under five months of age and though all showed signs of terminal pneumonia it was felt that another and more definitive factor must be present. Our analysis led to the conclusion that white muscle disease (WMD) was the basic cause.

Following this conclusion, all nyala born at New York Zoo were injected with 1 cc of BO-SE at one week of age. This is a product containing selenium and Vitamin E, manufactured by H. C. Burns Co, Oakland, Calif., USA, and is used extensively for treatment of white muscle disease in domestic stock. White muscle disease is basically a deficiency of selenium in the diet and though the role of Vitamin E in this deficiency disease is not entirely understood at present, Vitamin E is known to be intimately associated with the utilisation of selenium.

This regimen gave extremely good results and we then decided to change the nyalas' diet so as to supply selenium without having to inject it into the animals. A complete trace mineral supplement was added to the pelleted food fed to the nyalas and they were given good quality clover hay instead of high quality alalfa hay. It was felt that selenium deficiency might arise from the feeding of high quality legume hays grown on soils that are intensively fertilised and that the sulphates in the fertiliser might render the selenium inaccessible.

All injections of selenium and Vitamin E were stopped when the new diet was instituted but it must be reported that the change in feeding did not achieve the desired results. In the first three months of 1965, five nyala were born. Three were not given any BO-SE