

STUDIES ON SPECIFIC OCULO-VASCULAR MYIASIS OF DOMESTIC  
ANIMALS (UITPEULOOG): I. HISTORICAL REVIEW

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NAME AND SYNONYMS

A characteristic of the Afrikaans language is its facile descriptive potential. This has enabled South African farmers to coin most apt and descriptive names for several diseases not necessarily confined to Southern Africa, e.g. the name lamsiekte (literally translated as lame disease) is far more apt than botulism for the condition caused by the toxin of *Clostridium botulinum* type C and D and is in general use in the literature today; knopvelsiekte which has been freely translated as lumpy skin disease; bewerasiesiekte (literally trembling disease) for epidemic tremor of poultry; and geeldikkop (yellow thick head) for the well-known photosensitivity syndrome in sheep are further examples. In the present instance South African farmers have coined the name "uitpeuloog" (literally bulging or protruding eye). This name, the pronunciation of which may occasion some difficulty to the unaccustomed tongue, is far more apt than the English synonym "bulging eye disease" for the condition which is to be described and of which protrusion of the eye-ball was at one time believed to be the sole pathognomonic symptom.

An additional synonym is blue wildebeest eye (blouwildebeesoog), a term frequently used by farmers because of their association of the disease with wildebeest (*Gorgon taurinus taurinus*) which in the region concerned, are largely migratory.

It will be shown that the disease is caused by the invasion of an aberrant host by larvae of one of the Oestridae and, therefore, the name specific oculo-vascular myiasis is suggested for the disease commonly called uitpeuloog. Later it will be seen that a more fully descriptive name would be oculo-vascular-neuro myiasis but it is felt that this is unnecessarily clumsy.

HISTORICAL REVIEW

*Occurrence*

As early as 1927 reports were received of a peculiar disease of domestic ruminants and horses from the Kalahari region of South West Africa. The reports were scanty but it appeared that the disease was characterised by exophthalmia which might be unilateral or bilateral. Frequently, concurrent affection of the central nervous system was noticed.

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The incidence of the disease was irregular in that it did not appear to have any seasonal occurrence and high incidence one year might be followed by complete absence for several ensuing years. Rather was the occurrence quite unpredictable, though there appeared to be some correlation with the density of migrating game such as the blue wildebeest (*Gorgon taurinus taurinus*), springbuck (*Antidorcas marsupialis marsupialis*) and gemsbuck (*Oryx gazella*).

When the disease did appear the morbidity and mortality might be so high as to make sheep and Karakul breeding unprofitable and thus to force farmers to abandon this type of animal husbandry in favour of the less lucrative cattle breeding. It is reported that over the years many sheep breeders were ruined as a result of the decimation of their flocks by the disease.

#### *Area affected*

The first reports of uitpeulooq were received from the Kuruman district of the Republic of South Africa and the eastern Sandveld area of South West Africa. After the first reported serious enzootic in 1927 further outbreaks occurred in 1935, 1937, 1941 and 1958, during which times adjacent areas in the Bechuanaland Protectorate were involved, viz. Tsabon, Khuis and Bokseputse. In 1947 and 1948 the regional veterinary officers investigated extensive outbreaks in the Olifantshoek area and again directed attention to a possible association with blue wildebeest.

Once attention had been directed to the great economic importance of the disease, more attention was paid to it by regional veterinary officers and a scrutiny of their annual reports shows reference to outbreaks of varying severity in the Gobabis district in 1929, 1933, 1941, 1942, 1943, 1945, 1946, 1948, 1949, 1950 and 1954; in the Grootfontein district in 1934, 1935, 1936 and 1941; in the Okahandja district in 1941; in Keetmanshoop (Koës-Aroab area) in 1947 and 1954; in Otjiwarongo in 1945; in Eastern Gibbon in 1947, 1948, 1949, 1950, 1953 and 1954. It is seen, therefore, that the infected area is fairly widespread and that the occurrence of outbreaks are variable and unpredictable.

It was found that when the disease did occur it was encountered usually during the period August to September. This coincided with the time when certain trees such as *Albizzia anthelmintica* ("Aru"), *Acacia detinens* (swarthuak) and other acacias were flowering. It will be shown that the significance of this observation was investigated carefully. In addition, at that season of the year insect life is most prolific, an observation which will be shown to be highly significant. That there is no definite seasonal incidence is shown by reports of outbreaks during the period March to June, usually associated with plentiful rains. A change of grazing from one camp to another on the same farm, or from one farm to an adjacent farm had no constant effect upon the incidence or severity, but constant phenomena were the quite remarkably sudden onset, the rapid course of the disease and its equally dramatic and sudden disappearance after variable periods of time. Nowhere is there any report of a slow build-up of infection from a few cases to a local or widespread enzootic or a delayed waning. This factor very considerably hampered investigations into the aetiology because the affected area is vast, communications are slow and transport is difficult. The result was that on receipt of a report the investigating officer would arrive on a farm to find that the last case had occurred some days previously. Alternately, if an investigational centre was established, the staff might have to wait for years to be on hand when an outbreak did suddenly occur.

The enzootic area may be described as semi-arid, sandy, partly duned sand veld, though several severe outbreaks were reported from the limestone region of the Keetmanshoop district. The average annual rainfall is eight to ten inches. The vegetation varies from dominantly grassveld to a well balanced grassy bushveld with *Acacia*, *Albizzia* and *Grewia* species as the more important perennials. Sand dunes in the virgin areas are on the average well covered with vegetation.

For the greater part of the year drinking water is pumped from boreholes and it is around the troughs and drinking places supplied by these pumps that the domestic animals congregate.

#### *Animals affected*

Sheep, cattle, goats and equines are all susceptible but a survey has shown that Merino sheep, cattle, horses and goats are more resistant than Karakuls, cross-bred Karakuls, Persians and Afrikaner sheep. Sporadic cases have been seen in springbuck and steenbuck, and farmers in the area insist that cases have occurred in dogs and man. Although the incidence of the disease appears to be associated with the presence of large numbers of migratory blue wildebeest, or at least with their recent presence, there is no record of a definite case amongst this species of game.

A constant observation was that the flocks and herds of an individual farmer might be divided into groups maintained in fenced camps on different portions of the same farm under conditions which were apparently identical, yet one or more groups would be severely affected while the others remained completely free.

There is no correlation between the incidence of infection and either age or sex. It is worthy of note that lambs even on the day of birth but frequently at three to five days of age have been found affected.

#### *Morbidity and mortality*

According to different observers the morbidity varied from 15 per cent to as high as 75 per cent. The mortality among the more susceptible Karakul and Merinos is variable but may reach 75 per cent of affected animals.

#### *Aetiology*

The aetiology remained obscure and numerous theories, usually not supported by any concrete evidence, experimental or otherwise, were advanced by different investigators.

Maag (1933) suggested that the condition was the result of some metabolic disturbance which, however, could not be explained logically.

Maybin (1936) considered the possibility of an extension of sinusitis caused by the larvae of *Oestrus ovis* to the eye and the central nervous system. Even a cursory analysis of the epizootiology could not support this theory.

Fourie & Snyman (1942) were of the opinion that the blue wildebeest was the reservoir of an infectious agent which was the cause of the condition but were unable to throw any light upon either the agent itself or the method of transmission to domestic animals.

Hellberg (1945), Holtz (1947) and Watt (1949) considered that some poisonous plant endowed with peculiar toxic properties, might be the chief aetiological factor. Adelaar & Codd (1949) carried out a detailed botanical survey of an extensive portion of the enzootically affected area but were unable to incriminate any single plant or group of plants.

Schulz (1949) noted the highest incidence of the condition frequently when Aru (*A. anthelmintica*) and certain species of acacias were flowering. He determined experimentally that pollen from the flowers, on intra-ocular instillation into the eyes of sheep, was capable of producing a severe local oedema accompanied by conjunctivitis and scleritis. The clinical syndrome produced, however, did not resemble the condition found in naturally occurring cases.

Badenhorst (1949) supported the contention of Fourie & Snyman that the blue wildebeest was the reservoir of infection and suggested the transmission of infection by unknown insect vectors. They were unable to produce any evidence in support of this hypothesis.

R. du Toit (1950) is credited with the suggestion that just as certain species of oestridae may be the cause of ophthalmomyiasis of man in the Mediterranean littoral, so other genera such as *Gedoelestia* may be the cause of uitpeuloog. Unfortunately this suggestion was not followed up until the present series of experiments was carried out, and their successful conclusion prompted this series of reports.

From time to time infected eyes as well as brain material from typical natural cases were subjected to careful bacteriological examination. No incriminating pathogenic organism was cultivated or isolated. Similarly, in attempts to throw some light on the possible infectious nature of the disease, transmission experiments were carried out at both Armoedsvlakte and Onderstepoort, using fresh and preserved blood, as well as emulsions of infected eyes, brain and spleen, for subinoculation into sheep, all with entirely negative results.

Histologically Schulz & Adelaar (1949) failed to find any evidence of parasitism on examination of orbital and brain material. Jackson (1949) on the other hand recorded pleomorphic bodies of exogenous origin and apparently parasitic in nature, in the hyperaemic tunica propria of the scleral conjunctiva. The bodies were ring-like, pyriform, bipolar or double oval in shape and might occur in morula-like masses. Their size was variable but the larger ones exceeded the diameter of an erythrocyte. In addition he found granular forms of various sizes, the smallest of which were only just within the range of microscopic visibility. These forms were found to occur in chains or filaments. The significance of these bodies was not elucidated, nor was their nature or identity determined.

#### *Symptomatology*

Several of the early investigators attempted an orderly classification according to the symptomatology of the various forms of the disease as encountered in the field. For instance, Hellberg (1945) described three forms of uitpeuloog.

- (1) Peracute form, where animals died suddenly with no eye or brain lesions other than a peculiar lustre in the eyes.
- (2) Acute nervous and ophthalmic form, where the symptoms were referable to the simultaneous development of lesions in one or both eyes and in the brain.
- (3) Nervous form, with very slight or the complete absence of eye lesions.

It is apparent, however, that the consensus of opinion inclined to the view that the disease occurs in two forms, the ophthalmic form and the nervous form, with one or other form predominating as an acute, subacute or chronic infection.

#### *A. The ophthalmic form*

The first symptom, characterised by very rapid onset and no rise in body temperature, is exophthalmia which may be unilateral or bilateral and without any lesion on the cornea. There is evidence of great pain, the head often being held to one side. Rapidly protrusion of the eye-ball becomes pronounced and usually the animal is unable to close the chemotic eye-lids. Haemorrhages or opacities are often noticed in the aqueous humor. Keratitis usually follows. The cornea may become desiccated and necrotic, so that the eye-ball may rupture with loss of its contents. The severity of the eye lesion may vary within the widest possible limits. Full recovery without impairment of vision may be the outcome. On the other hand permanent corneal opacity may develop, or the animal may be completely blind in one or both eyes without showing any visible defect or blemish. Secondary bacterial infections play an important role, as well as trauma, with their sequelae, caused by the animal bumping into obstacles. Rhinitis has been observed but is not constantly present.

#### *B. Nervous form*

This form of the disease has been observed in only the more susceptible species, and has not been noted in cattle, though it has been described in a mild form in a goat.

Symptoms of affection of the central nervous system are noticed either before or after the appearance of eye lesions. Frequently the animal is completely blind with no detectable eye lesion other than exophthalmos, the pupillary reflex being absent and nystagmus and oedema of the eye-lids developing later.

The most important symptoms are general hyper-aesthesia, a tendency to move in circles, clonic convulsions, ataxia, torticollis, loss of balance which may be partial or complete, and paresis followed by paralysis. Inco-ordination of movement is rapidly progressive, followed by the animal going down on its side showing the galloping movements of the legs so frequently seen in heartwater. These convulsive fits may be continuous or intermittent, with intervals of several hours, and the outcome is usually death after periods of three to 14 days. Invariably the appetite is unimpaired though mastication and deglutition may be interfered with to a varying degree.

### *Pathology*

#### *A. Macroscopic*

The following lesions have been described in varying degrees of severity:—

Hydrophthalmos and oedema of the peri-orbital tissues, with or without evidence of secondary bacterial invasion, conjunctivitis, keratitis, venous stasis and coagulation of the blood in the orbital and peri-orbital vessels; congestion and catarrh of the nasal mucous membrane; hyperaemia of the meninges and haemorrhages into the sclera and cervical portion of the spinal cord extending up to the ventral surface of the brain between the cerebrum and the cerebellum; peri-neural oedema and haemorrhages in the trigeminal and maxillary nerve; thickening of the pia of the brain with superficial brownish discoloration in the more chronic cases; areas

## SPECIFIC OCULO-VASCULAR MYIASIS OF DOMESTIC ANIMALS

of necrosis and softening in the cerebellum, brain stem, and thalamus; venous thrombosis in the vicinity of an affected eye but also as distal as the bifurcation of the jugular vein; slight ascites and hydropericardium; enlargement and haemorrhages of the cranial and cervical lymphatic glands; well marked degeneration of the myocard.

### *B. Microscopic*

*Eye.*—Acute conjunctivitis and keratitis which may be purulent and necrotic, iridocyclitis, retinitis, neural oedema of, and extensive haemorrhages in, the optic nerve with possible neuritis, degeneration of the lachrymal glands, oedema of the sclera and retina and infiltration of neutrophiles into the substantia propria. Jackson described an infiltration of the connective tissue with lymphocytes, plasma cells, eosinophiles and neutrophiles and the presence of some unidentified bodies possibly parasitic in nature.

*Brain.*—Lepto-meningeal oedema and hyperaemia, lepto-meningitis and encephalo-meningitis; subarachnoidal haemorrhages; haemorrhagic perineuritis and degeneration of the optic nerve with acute perineuritis; haemorrhages into the cerebellum and thalamus with areas of necrosis; non-purulent encephalitis with encephalomalacia.

*Spinal cord.*—Focal subarachnoidal haemorrhages in the anterior portion of the cervical cord with degenerative changes in the grey matter and evidence of acute myelitis.

The kidneys show slight cloudy swelling and degeneration and there was variable acute interstitial hepatitis. In the heart there might be acute interstitial myocarditis.

### *Therapy*

No treatment either prophylactic or therapeutic was found to have any value. It was claimed that non-specific protein therapy was of value but there is no record of these claims having been substantiated. Apparently local treatment of the superficial eye lesions was instrumental in reducing the incidence of secondary bacterial infection.

### *Immunity*

There is no evidence of the development of any immunity. There are authentic cases of an animal having recovered from a mild unilateral infection or a severe infection which terminated with rupture of the eye-ball only to show a subsequent infection of the same or the other eye or both eyes during the same or a subsequent season. It has also been reported that infection of one eye might be followed by infection of the other within a period of a week.

### COMMENT

It must be pointed out that the subject matter for this general and historical review of the disease has been obtained not only by reference to the available literature but also by a careful scrutiny of all the available official reports in the offices of the Director of Veterinary Services, Republic of South Africa, and the Director of Agriculture, South West Africa Administration. Consequently in the bibliography reference is made only to published reports or personal communications of particular interest and the names of officers who compiled the official reports are not cited.

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