

Rinderpest in Game.

A Description of an Outbreak and an Attempt at Limiting its Spread by Means of a Bush Fence.

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INTRODUCTION.

WITH the present day methods of immunization against rinderpest, by the use of attenuated goat virus or formolised spleen vaccine, or a combination of both, cattle can be satisfactorily protected: and were they the only susceptible animals concerned, complete eradication in a comparatively short time might be anticipated by the intensive use of these inoculation methods together with strict police control.

Unfortunately in Africa there is a widespread and varied game population, in many places in intimate contact with domestic stock. Many of these species are susceptible to rinderpest in varying degrees and are, therefore, capable of acting as transmitting agents between different groups of cattle and game.

Veterinarians in this continent have been aware of this danger for many years but the full significance of game as a factor complicating control measures was again emphasized during the recent epidemic in Tanganyika Territory (1938-41).

Previously it had been held that rinderpest appeared as epizootics in game populations, dying out comparatively rapidly with the rapid disappearance of susceptibles (owing to abortion and the high mortality rate the "calf link" tending towards enzooticity was removed). However, with a "milder" form of the disease and an apparently slower rate of transmission such as was noted in the Lake Rukwa area there were indications that, apart from the cattle factor, the disease might assume a state of enzooticity in game alone and that the vast game areas might act as reservoirs of the disease liable to overflow into contiguous cattle or game areas, whenever conditions were favourable.

BRIEF HISTORY LEADING UP TO PRESENT POSITION.

During the War (1914-18), owing to the dislocation of veterinary control services in what was then German East Africa, rinderpest spread southwards and in 1918 was threatening to invade the Rhodesias. It is more than likely that game suffered concurrently with cattle, although records on this point are lacking. Fortunately, despite the fact that no attempt to limit the movement of infected game was made, the disease died out locally following on

the active double inoculation campaign instituted by the "Rinderpest Commission" appointed by the Southern States. Thereafter the Tanganyika Civil Veterinary Department took up the struggle and gradually pushed the disease northwards until in 1935 there existed but two small outbreaks situated on the Northern Tanganyika-Kenya border. The methods of control employed during this period were inoculation with hyperimmune serum plus stringent quarantine control.

With the financial depression of 1930-35 funds for the control of the disease were reduced and the ground gained was gradually lost, until in 1938 the disease was once more invading the highlands of Southern Tanganyika. There had been a change-over of inoculation policy during this period to the double-inoculation method. During the quiescent period a large susceptible population of cattle and game had grown up and there is ample evidence of the part the latter played in the southward spread of the disease.

The outbreak was temporarily halted by immunizing, by triple and double inoculation, a belt of cattle numbering about 150,000 immediately south of the infection. This belt lay across the Iringa highlands and was flanked on both sides by tsetse infested bush, free from cattle but containing a widely scattered game population.

Late in 1939 reports of game dying in the Kilombero valley on the Eastern Game flank and in the Saba Game Reserve on the Western Game flank preceded the re-appearance of the disease in cattle in the Usangu plains south of the belt vaccinated in October, 1939. In 1940 all cattle in the southern half of Tanganyika were immunized by triple vaccination or by attenuated goat virus in combination with vaccine (a total of over a million and a half inoculations). It was assumed the disease was stamped out in cattle although it smouldered on in game in the south-eastern "flank" well into 1941. In June, 1941, notwithstanding the fact that there had been no evidence of infection in game in the "Western flank" for approximately one year, rinderpest suddenly re-appeared in susceptible calves and game in the Saisi valley, an area adjoining the huge western fly belt. It was at this point that our investigations, now to be detailed, were carried out.

THE RUKWA TROUGH OF SOUTH-WESTERN TANGANYIKA.

The Rukwa trough in which most of our investigations were carried out is a huge depression approximately 120 miles long by 40 miles wide extending south-eastwards from about the middle of Lake Tanganyika. Lake Rukwa itself is brackish and has no outlet. Its size varies tremendously depending on the seasons and in September, 1941, it was rapidly drying back to the south-eastern end leaving large mud flats. The Lake occupies but a small portion of this trough the rest forming alluvial plain or "mbuga", covered for the most part with stretches of coarse grass and sedges in which are interspersed stands of sweet grasses such as *Cynodon dactylon*, etc. Fringing the water courses of the plains along the lower foothills are belts of acacia which merge into the deciduous bush ("Miombo") covering the sides of the trough.

Running down from the surrounding hills were numerous semi-permanent streams; these in the rainy season inundate the plains and actually submerge large areas, causing a general movement of most species of game upwards to higher ground. As the dry season progresses the flooded areas gradually dry up, and eventually the streams retreat to the foot of the

escarpment. Around these waters are clustered native villages, each with its herds of cattle, goats and sheep competing with game for water and grazing.

It is not difficult to realize that, although the contact between game and domestic stock is closer at the height of the dry season, the seasonal movement associated with the rains, causing a scattering of the game, increases the likelihood of disease transmission over wider stretches of country. It may be noted also that game observation becomes much more difficult during the latter period when the grass is high (up to 12 feet) and when large portions of country become inaccessible to observers on account of the flooding of rivers.

THE GAME POPULATION.

A general cross section of the game population around any of these watering places at the time of our visit at the height of the dry season revealed the following species:—

Buffalo in herds of a few to a hundred animals, could be found in the reeds and swamps and adjacent bush, but never very far from water. They grazed in the open plains by night, exceptionally by day.

Eland were scattered over a very large area singly or in groups up to twenty or more. They drink only every second or third day and consequently ranged very far afield in the open plains.

In the "mbugas" were vast herds of Topi and Zebra with Reedbuck interspersed in small groups or families. Kudu, Roan and Impala in small herds were found in their selected habitat, i.e., where the browsing and grazing to which they are partial occurs. A few Puku were to be seen along the lake, becoming more numerous towards the eastern end. Occasional families of Hartbeest, Waterbuck, Bushbuck, Duiker and Steenbuck were encountered. The spoor of Bushpig were frequently seen in thickets along the kloofs and water courses. Warthog and Giraffe were seen only on the plains to the west. Elephants were concentrated in the swamps of the Saisi delta and the thicker bush country of the north-western end of the lake. Lions were seen on two occasions and undoubtedly accounted for some of the carcase-remains observed. Hippo and Crocodile frequented the shores of the lake, which is noted for its abundance of fish.

THE DISEASE IN GAME.

Our arrival in September, 1941, at Sakalilo on the south-western plains of the Rukwa trough seems to have coincided with a flare-up of the disease here. Three months before this, numerous reports of game dying in the Saisi valley about 50 miles south-east had been followed by a period in which game observers patrolling the area on the look-out for sick game could find nothing unusual. Fairly close observation during a four weeks' stay showed that mortality had almost stopped before we departed. A herd of buffalo and one of eland in which sick animals could be "spotted" on our arrival appeared healthy at the time of our departure.

It is, of course, impossible to give any accurate estimate of the mortality rate. All that can be said is that a number of sick animals were seen and an unusually large number of carcases or remains were encountered. Within a

RINDERPEST IN GAME.

3 to 4 miles radius of the watering place at Kikumbi a rough count revealed the following carcase remains:—

Buffalo	10	Topi	11
Eland	24	Reedbuck	1
Kudu	10	Unidentified	3

The mortality certainly could not be said to have been very severe, and it was a striking fact that the greatest loss was among young game, 3 years and under. Many of the adult animals must have had the disease in a mild form but had recovered. For instance in the pooled blood and organs of three such adult buffalo (Experiment 2) the presence of virus was demonstrated by subinoculation. Vultures, an indication of mortality, could be seen in large numbers in the vicinity on our arrival, later they appeared to have moved elsewhere. On extending our survey towards the west, which seemed the natural direction of spread, we were able to find a few isolated sick eland, and also a limited number of carcasses and skeletons, but nothing like the obvious recent and localized epidemic at Sakalilo. It must be assumed, therefore, that in the dry season congregations of game are more or less separate round each watering place and that pockets of susceptible animals may remain free from infection until a chance contact brings about another minor local epidemic.

SYMPTOMS, LESIONS AND HISTOPATHOLOGY.

(a) *Buffalo*.—As a rule normal buffalo remained grouped in compact herds all the time. When alarmed they bunched together even closer with the calves in the centre, and maintained this formation even when running. The herd instinct is thus very strong and breaking up with scattering of individuals, as is described when severe mortality occurs, was not seen or heard of in this instance. Occasionally isolated sick animals remained behind in the shade near the watering places when the main herd was out grazing, but there appeared to be no tendency to migrate deliberately. It is probable, therefore, that a mild form of the disease will cause less disruption in the herd and will spread more slowly than would a sudden virulent outbreak.

One herd of about fifty was kept under almost daily observation at Sakalilo. It remained grazing in the same locality out in the plains even during the day and thus it was possible to approach it by lorry and examine it at close range through field glasses. The herd consisted mainly of adults and yearlings; only 3 of the previous year's calves were to be seen.

This herd was constantly accompanied by egrets which usually stalked around the grazing animals to catch grasshoppers and other insects. It was quite a common sight, however, to see several of these birds riding on the backs of the buffalo, a practice rarely noted in the case of other species, e.g., zebra and eland. Very few real tickbirds (*Buphagoides erythrorhynchus*) were observed. The possible rôle played in transmission by such close contact between birds and game infected by disease is, of course, worth bearing in mind.

Unfortunately the herd must have been in the "recovery stage" on our arrival although some of the full grown animals were purging still. In the three animals shot, although virus was demonstrated to be present in the pooled blood, the lesions were very mild and atypical. A few days before, a game observer had shot obviously sick animals and actually speared one *in extremis*. Carcase remains of others were picked up.

The post-mortem findings included the following:—emaciation, excoriation of the skin of the back, slight conjunctivitis with slight mucopurulent discharge. Shallow abrasions of the buccal mucosa and papillae, some possibly mechanical. Localized diphtheroid omasitis, paramphistomes in rumen. Ulcerative or erosive abomasitis. These erosions or depressions were found in the fundus portion of all three buffaloes. They were sharply circumscribed, rounded or angular excavations, varying in size from 0.2 to 4 cm. The cavity had clean sides, the floor only occasionally showing a small haemorrhage in its depth.

These depressions, on histological examination, appear to be brought about by a disintegration of the tubular glands of the mucosa. The epithelial cells lining these glands, chief cells as well as oxyntic, seem to swell up and burst, dispersing their cytoplasm and nuclear matter so that only the stroma of the glands remain and this collapses to the propria mucosa. The lumen of some tubular glands outside such a depression may be choked with cellular debris and desquamated cells. No inflammatory reaction seemed to accompany this type of lesion. (In a fourth buffalo, believed healthy, similar erosions were found in the abomasum. This has raised some doubt in our minds as to whether these lesions have any specific significance.)

On the edges of the leaves of the omasum (in one animal) localized raised and necrotic looking patches were seen. Microscopically the horny layer was greatly thickened and porous and was loosely attached to the underlying zone of swollen, vacuolated, degenerated cells of the Malpighian layer. Small groups of neutrophils infiltrated the epithelium and propria. The lung of one buffalo showed a gelatinous and partly consolidated area about 6 cm. in diameter. This proved to be a localized catarrhal bronchopneumonia with rather marked degeneration and desquamation of the bronchial epithelium.

(b) *Eland*.—The herd instinct appeared to be weaker than in buffalo and the herd was a much looser and more casual aggregation. One or more animals would often break away and wander off on their own even under normal conditions.

Sick animals were usually found singly, often far away from others and in poor condition, although one could not describe them as emaciated. On approach, they would stand for some time and then move off in a hesitating or mildly stupid way, at once recognized as abnormal by anyone familiar with the shy disposition of this animal. It seemed as if the animal's senses were dulled and their sight impaired but not entirely lost. Several cases of purging were seen in the herd. In such animals the tail was often carried in an elevated position also in the intervals between defecation. Possibly this was due to the "scalding" or excoriation in and around the anus.

In some cases there were hairless patches on the belly and numerous "paint brush" eruptions along the flanks, back and shoulders. These are scattered small tufts of hair, standing erect due to the fact that the epidermis at this spot is crusty. These tufts can be scratched off readily, leaving a shallow depression. Some ticks were present.

A very constant feature seemed to be the profuse discharge from the eyes. A sticky, turbid fluid ran from the inner canthus and spread over a wide portion of the skin of the face, cheek and jaw. Dust and ash from burnt veld combined with the drying discharge, to cake the eyelashes and form crusts on the skin. The cornea usually presented a smoky opacity of varying severity.

The mucosa of the nostrils was reddened and swollen and the outer margin caked with drying discharge, dust and cell debris. Small excoriations of the epidermis could be seen when this was scratched off. The mouth may have a foetid odour, the saliva being thick, turbid and stringy. The inner surface of the lips was covered with a patchy, greyish yellow deposit which was easily rubbed off leaving a mat instead of the normal glistening appearance. The tongue might be similarly affected and the papillae at the angle of the mouth were often swollen and red.

The omasal contents were usually very hard and dry and adherent to the leaves. The mucosa in places showed a reddish to brown thickening near the edge of the leaves due to a form of parakeratosis as seen in one of the buffaloes. The abomasum usually contained a little watery ingesta. The mucosa, especially of the fundus portion, was a deep brick-red colour (inflamed). There were numerous very shallow erosions especially toward the omasal groove, and some of them contained streaks of blood. Slight ascites and fibrous peritonitis was seen in one case.

Apart from rather prominent Peyer's patches the intestines showed only patchy and streaky hyperaemia. Diarrhoea was not present in all cases, but slight excoriation along the folds of the anus was noted in some. Other organs did not reveal any change of importance.

Three affected eland were examined in all, but fairly complete material for histological study was collected from one only, lesions being found in the following organs: Disseminate pin-point centres of cell disintegration, not unlike those seen in acute paratyphoid in calves, were seen in the liver. The lungs were congested, neutrophiles were frequent in the alveolar capillaries and a slight bronchial catarrh was present. A striking feature seen in the spleen and some lymphatic glands was the accumulation of neutrophiles in large numbers around the lymphoid follicles. The reticulum cells in parts of the spleen appeared swollen and even hyalinized. Slight parakeratosis with flaky desquamation and infiltration with exudate was seen in the skin. Quite extensive inflammatory infiltration with necrosis and desquamation occurred in the lachrymal and Meibomian and sebaceous glands. The epithelium of the conjunctiva also showed vacuolation, swelling and desquamation, but no serious organic change could be seen in the rest of the optic apparatus and nerve. The greyish deposit seen in the mouth and nostrils consisted of the swollen and softened upper layers of the epidermis which were being shed in the form of shreds or flakes sometimes mixed with a little exudate. The loss of these layers was, however, only superficial and rarely affected the Malpighian layer proper.

The abomasal mucosa was hyperaemic throughout and there was a slight infiltration with neutrophiles. Groups of tubular glands seemed to have shed the upper part of their structure leaving shallow depressions or erosions. Both oxyntic and chief cells were swollen and disintegrated but the process did not extend as far and as deep as was the case with the buffalo.

(c) *Kudu*.—These animals were not numerous in the Sākalilo region; and owing to their acute sense of hearing, observation was difficult.

Kudu bulls, as is well known, usually browse on their own, while the cows and calves keep more or less together. One young and obviously sick bull was observed fairly closely. It was emaciated and was wandering about aimlessly in the bush, with head hanging, swaying gait, and appeared anything but alert. However when it became aware of human beings at close

quarters, it took flight and was able to run a good distance. When tracked next day it was found dead in a shallow hole into which it must have fallen and from which it had been unable to extricate itself. Another young bull was found dead alongside a path one morning.

In both these animals profuse lachrymation was present with soiling of the skin of the inner canthus and face. The mouth and mucosa of the nostrils were reddened in parts and covered with some greyish dirty flaky substance, easily rubbed off. The lungs were somewhat congested. The abomasum was intensely inflamed and reddened and covered with mucus, and small shallow erosions were present. The rumen and small intestine showed moderate hyperaemia.

Material from the one case only was examined microscopically. The epidermis for the greater part was "slipping" (autolysis), but extensive necrobiotic changes were still visible in the sebaceous glands and Meibomian glands of the eyelids. The glandular cells were swollen, vacuolated and disintegrated, so that the glandular lumina were often packed with cellular debris and exudate. Great thickening of the buccal mucosa was evident, the keratin layer of the epidermis particularly being swollen, spongy, vacuolated and coming off in flakes and shreds. Inflammatory exudate was only slight.

There was intensive hyperaemia of the abomasum almost to the propria mucosae. The chief cells first and then the oxyntic cells seem to disintegrate by lysis, leaving the stroma to collapse and form the bed of the depression. The edges may be sharp, abrupt or slope inward. Inflammatory exudate and bacteria did not seem to play a part in the process.

The more constant features seen in these three species can, therefore, be summarized as follows:—

- (i) Parakeratosis of skin with extensive vacuolation and degeneration and shedding of squamous epithelia in general, e.g., mouth, nostrils, conjunctiva, fore-stomachs and anus. This may be accompanied by inflammatory complications of varying degree.
- (ii) Extension of the above process to certain glands, e.g., lachrymal, Meibomian and sebaceous, resulting in an exaggerated discharge of secretion mixed with exudate and cellular debris from the eyes soiling the face.
- (iii) Severe gastritis—in which degeneration and disintegration of glandular cells to form shallow erosions seems a common occurrence.
- (iv) Mild enteritis, with prominent Peyer's patches, occasional necrotic ulcers, and usually diarrhoea.

EXPERIMENTS.

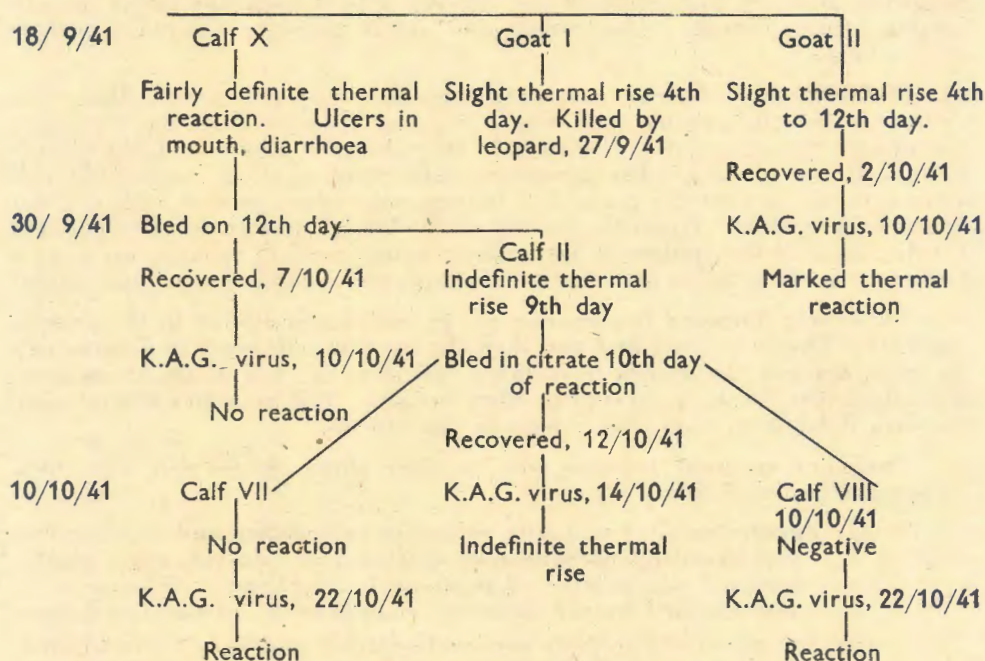
In order to confirm the diagnosis of rinderpest made in respect of this disease in game in the Lake Rukwa area, a few subinoculations from game to calves and goats were undertaken. In judging the results of these experiments due allowances should be made for the difficulties attending such work in the bush, where no special preparation for it had been made. At first, there was some difficulty in procuring recipient animals which could be regarded as susceptible, as all bovines in the vicinity had been immunized either by vaccine or goat virus in 1940 and calves and goats, as is well known, are unsatisfactory animals to work with.

RINDERPEST IN GAME.

Another more serious obstacle was the difficulty in transporting inoculation material from sick game often shot many miles away in the bush, to experimental animals in camp, without loss of viability. This was later overcome by acquiring a refrigerator and wide-necked thermos flasks.

EXPERIMENT 1.

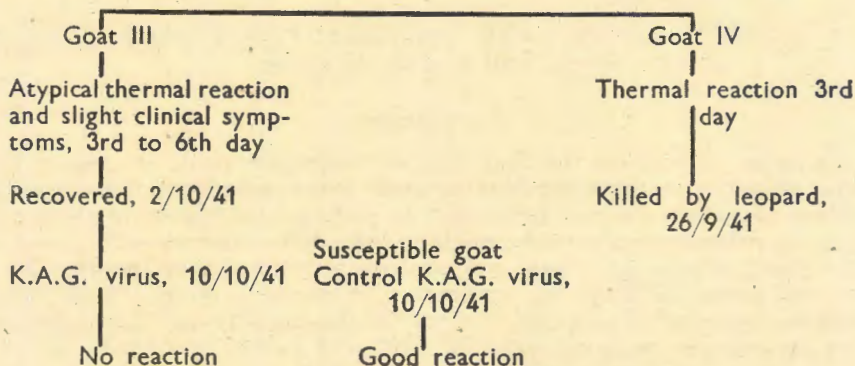
Sick Eland No. 1 shot on 18th September, 1941. Citrated Blood and Spleen Pulp Inoculated.



N.B.—K.A.G. is the dried Kenya Attenuated Goat virus as used for active immunization of cattle.

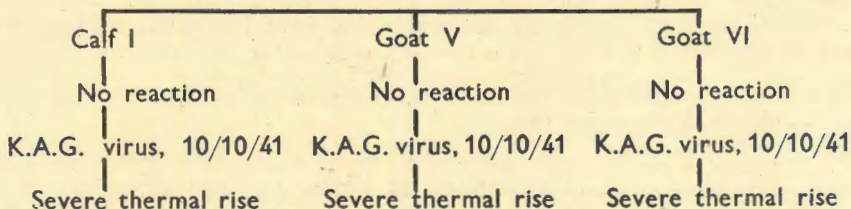
EXPERIMENT 2.

Three Buffaloes Shot (one sick), 19th September, 1941. Spleen Pulp Pooled.



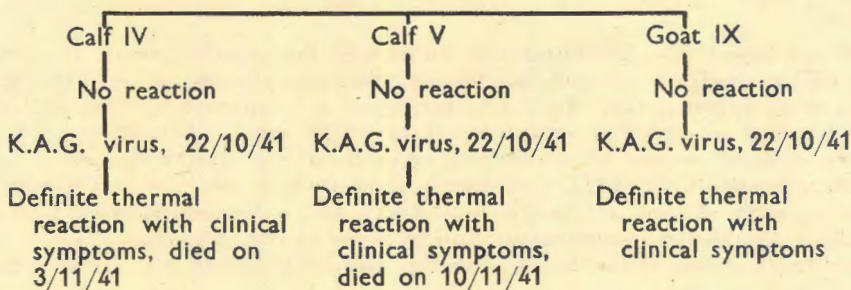
EXPERIMENT 3.

Spleen Pulp from Buffalo Shot. No Symptoms of Illness, 30th September, 1941.



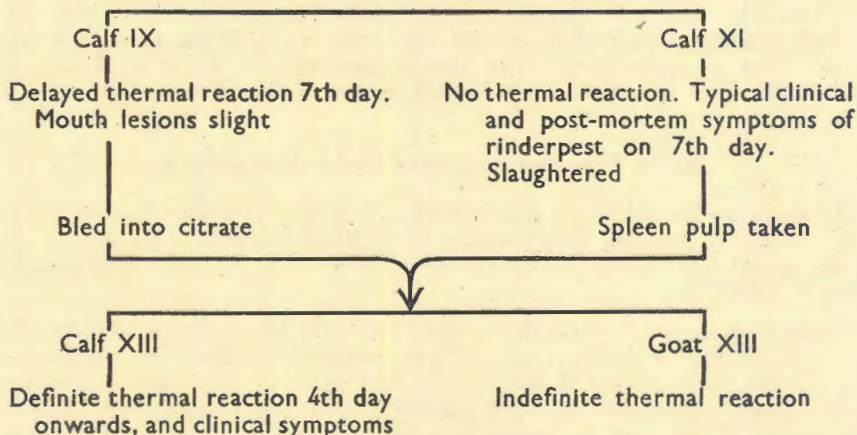
EXPERIMENT 4.

Spleen Pulp from Sick Eland, 5th October, 1941 (attempted preservation by freezing with Ethyl chloride).



EXPERIMENT 5.

Spleen Pulp from Fresh Kudu Carcase, 15th October, 1941.



NOTES ON EXPERIMENTS.

Experiment 1.—Calf X went through a fairly typical thermal reaction accompanied by ulcers in the mouth and followed by diarrhoea. It recovered and was solidly immune to K.A.G. virus. It is assumed that Calf II was immune since it gave such an indefinite reaction and was subsequently refractory to K.A.G., although it is possible that the virus was no longer present in the blood of Calf X on the 12th day after inoculation.

For some inexplicable reason Goat II did not develop an immunity as shown by the K.A.G. virus test.

Experiment 2.—The only remaining goat, although it gave an atypical reaction, developed a good immunity as shown by the subsequent K.A.G. virus test.

Experiment 3.—Gave negative results. Either the buffalo did not carry the virus or the material lost its viability during operations.

Experiment 4.—In spite of the eland showing pronounced and typical signs of rinderpest, subinoculation tests proved negative. Faulty technique (an attempt to freeze spleen pulp with ethyl chloride and transport it in a thermos flask) may have been responsible for the loss of viability of the material.

Experiment 5.—Although the kudu had been found dead, the organs were still perfectly fresh and no difficulty was experienced in inoculating two calves with spleen pulp. Calf IX developed a temperature, although when last observed no clinical symptoms were to be seen. The other, No. XI, showed typical lesions of rinderpest (mouth ulcers, diarrhoea, etc.). Subinoculation into Calf XIII produced a temperature reaction and symptoms. Unfortunately it was not possible to carry out a cross-immunity test with K.A.G. virus as the investigation had to cease at our departure. From these results there seems little doubt that the disease in game was rinderpest.

Incidentally the possibility of the disease in game being a modified form of rinderpest contracted by them from cattle actively immunized in that vicinity by Kenya Attenuated Goat virus is most unlikely. Since such transmission (if at all possible) must have taken place recently, it can be assumed that the virus when returned to goats as in our experiments would have given rise to characteristic initial temperature rises. This was not the case and, further, the history of the outbreak originating from Ivuna must be taken into consideration. The disease appeared in game at Ivuna before any K.A.G. virus inoculations had been given in the Mbeya district in 1941.

BUSH FENCING TO LIMIT GAME MOVEMENTS.

As mentioned earlier in this report, with the immunization of all cattle in the southern part of Tanganyika in 1940 rinderpest died out in cattle, and in game, the last recorded mortality was in the Usangu plains in March, 1940.

From then onwards for over a year there was no evidence to indicate that this outbreak had not died out. Then suddenly rinderpest reappeared at Ivuna on the Saisi River in both game and cattle. Its origin has not satisfactorily been explained and the possibility of the virus being maintained in an unknown host must be borne in mind; alternatively there is the question

of virus carriers in game or cattle. It is unlikely that the "Intelligence" staff working in the Rukwa trough would have missed evidence of a "normal" outbreak in game.

In this connexion, however, attention might be drawn to the extraordinarily mild clinical picture shown in cattle in the Ivuna outbreak and in a similar one in calves at Kipeta (Saisi River). The disease was, to all intents and purposes, hardly diagnosable without resort to subinoculation. It is reasonable to suppose that the same "mild" clinical picture might be shown by game. Such an outbreak would be almost undetectable by the ordinary methods of game observation.

The apparent variability both as regards virulence and infectivity of the field virus, whether in cattle or game, is a matter requiring careful observation and study.

To return to the position in July, 1941; to stop the spread of rinderpest in cattle was comparatively easy, but in game, there appeared every likelihood of the disease spreading southwards; and to prevent this the Veterinary Department of Tanganyika was faced with the necessity of doing something and doing it quickly.

To segregate game in a vast uncivilized country with few inhabitants and poor road communication might at first glance have appeared to be impossible, particularly as there was a war on and the possibility of obtaining large supplies of fencing material in time to be of any use was out of the question.

But the topography and nature of the country helped. The infected game area was bounded on the south-east by the almost sheer Ufipa escarpment and on the east by the highland populated country of Mbosi. Between the two there was the lower and comparatively narrow outlet to Rhodesia, namely the valley along which the Nkana and Saisi Rivers flow. These rivers find origin in the Ikamba watershed on the Northern Rhodesia border and flow northwards into Lake Rukwa. Should rinderpest have found its way Southwards along this valley, as might be expected if left to itself, then extension into the huge game country of the Chozi and Luangwa valleys might well have taken place. The valley was for the most part heavily timbered, and it was natural enough, therefore to turn to the utilization of this timber for the erection of a bush fence. Rough fences and ditches are employed locally to exclude game from native fields and gardens, and are known to be fairly efficient, but the trouble is that they are perishable. Still a bush fence, given proper care and maintenance, might be expected to last 2 to 3 years and tide over the dangerous period. During this time it was believed the disease might have spent itself and receded, or if it did not, at any rate a valuable respite would be afforded in which to evolve more lasting and effective safeguards.

The siting of the fence required care, in order to ensure that infection had been left well to the north, and at the same time giving consideration to accessibility by road.

The first game fence in this campaign against rinderpest was started on the 5th July, 1941. It stretched from Ithaka on the Mbosi highlands, across the Saisi valley at Msangano, to the Ufipa escarpment on the west, a distance of 26 miles. This fence was erected in 26 days by approximately 1,000 native labourers recruited by the Administration from

RINDERPEST IN GAME.

local tribes and supervised by 3 European members of the veterinary staff. Its cost, including European salaries, amounted to approximately £40 per mile. The construction was very simple, all material was obtained on the spot and all implements necessary were those usually carried by every native male—namely, the primitive hatchet which is also convertible to a digging tool for making holes for poles.

The fence consisted of pairs of upright poles planted into the ground to a depth of 2 feet at intervals of 5 to 10 feet. Logs were stacked horizontally between the uprights and held by them at the ends; then poles and thinner branches to a height of 8 to 10 feet. The uprights were lashed together once or twice as stacking progressed either with bark or with wire when procurable. Forked poles to act as props were placed at frequent intervals on both sides. Gates of the self-closing pattern (hanging logs) were left at suitable intervals for the use of pedestrians, and where necessary road gates closed by horizontal poles were made.

There is ample proof that such a fence when built with care and properly maintained is effective in preventing the passage of game from one side to the other. We have observed buck walking along searching in vain for a place to push through, and in some places the well worn game paths along both sides further testify to its efficacy. When the lower part of the fence is well stacked, even wild pig are completely excluded. So far, elephants and occasionally carnivora are the only animals which the fence has not succeeded in keeping back. Usually elephants either unstack the upper portion of poles and step over the remaining few feet of fence or else push the whole fence over. In either event only a short section of fence is damaged and can rapidly be made good. With a wire fence (as in the Addo Bush) many hundreds of yards of fence are liable to be pulled down by elephants and require correspondingly extensive repair each time. Obviously a bush fence has to be patrolled continuously so that gaps may be repaired as they occur and before game finds and uses them.

It is, of course, too early to say whether big game will be held in all circumstances, e.g., a frightened and stampeding herd or when a favourite watering place or grazing site is cut off. Nevertheless it is quite evident that the bush fence with its solid, wall-like appearance is far more likely to deter game from throwing themselves headlong against it than a correspondingly high wire fence, no matter how well constructed.

The only drawback to the bush fence, and it is unfortunately a serious one, is, of course, its perishable nature. On account of fire, white ants, decay and the poor quality of the wood, the useful life of such a fence will inevitably be short. With constant attention and replacements it is probable that its life could be extended to three years.

At the Veterinary Conference held in Mbeya in July, 1940, it was decided to erect a similar bush fence along the Northern Rhodesia-Tanganyika Territory border. The first section extending between Tunduma and the Saisi River (86 miles) was completed in November, 1941, at a total cost of £4,500 or a little over £50 per mile. The second section between the Saisi River and Kalambo Falls on Lake Tanganyika was erected the following year. There were a few gaps in the line of fencing in which sufficient timber could not be cut owing to scarcity of bush. This was the case, for instance, for 5 to 6 miles on both sides of the Saisi River. Timber could have been transported over this distance fairly easily but it was thought that this would have raised the cost considerably and delayed completion of the fence.

Accordingly a four-strand plain wire fence 7 feet high was erected and for additional safety was flanked on one side by a continuous ditch 6 feet wide by 4 feet deep.

According to reports these portions of the fence were not so successful. In the first place, during the rainy season the sides of the ditch tended to fall in, starting soil erosion, and silting up took place in the flat or lower stretches. Secondly, the fence itself was inadequate owing to lack of sufficient and suitable material. The plain wire strands were too few, too wide apart and not high enough to prevent large antelopes from jumping over and smaller animals from crawling through.

The shortcomings of such bush fences were, of course, fully realized at the time, nor was it expected that their erection alone constituted all the safeguards necessary. The Msangano fence for instance was intended merely as an advance and temporary check to give time to establish a more permanent defence line further south. The Northern Rhodesia border fence again formed part of a bigger scheme calculated to strengthen the existing natural barrier by creating a game-free zone at the watershed between the Rukwa and Congo Basin—each with its great game carrying plains.

The achievement of this aim was perhaps not perfect, but the following features undoubtedly helped:—

1. The presence, initially, of a large body (several thousands) of native labourers cutting bush, making a road, erecting the fence and hunting, must have frightened the game away towards either side for a time.
2. The bush fence itself, as has now been shown, actually forms an effective and absolute barrier (while it lasts). It prevents passage, migration or stampeding of game while shooting out is in progress to create a wider game-free zone.
3. A cordon of pickets (3 natives to each) was established and maintained along the whole fence. Their duties included (a) patrolling their respective sections of fence daily, reporting on the state of the fence and repairing gaps caused by animals, floods, etc., (b) to shoot any game near the fence and later to hunt within a given distance from it.
4. The siting of such a fence is also very important. It must take into account the topographical as well as ecological features of the locality. It is especially essential to know beforehand something about the diurnal and seasonal movements of game as well as the routes they follow, and their favourite or only supplies of food and water at given times of the year.

It will be realized that the last word has not been said regarding the value and possibilities presented by this by no means novel attempt at controlling rinderpest. What is urgently needed, however, is a more realistic and practical approach to the vast problems attending wild animals all over this continent and their relationship to rinderpest as well as to other important diseases, and agriculture generally. There is ample room in Africa for game in plenty as well as for livestock, but their interests need not and must not be allowed to clash. In other words it is becoming more and more evident that wild animals will have to be segregated in sanctuaries as much for their own protection as for that of livestock and agriculture. More

RINDERPEST IN GAME.

research is necessary not only in respect of the diseases wild animals carry or suffer from, but also in regard to their habits and likely means of control or segregation. The need for this may not be so obvious in the thinly populated part of Central Africa, but certainly it is making itself felt in Southern Africa.

SUMMARY.

1. The history of rinderpest and of measures taken to check its spread in Central East Africa is briefly reviewed.

2. A description is given of a disease, believed to be rinderpest, affecting buffalo, eland and kudu in the Lake Rukwa trough.

3. Attention is drawn to some of the habits of game and the close association often existing between cattle and game in the dry season.

4. Subinoculations from affected buffalo and eland into susceptible young cattle and goats show that these animals suffered from rinderpest.

5. While defensive measures for cattle, namely immunization and control of movement are eminently successful, game still remains the greatest source of danger for spreading and for maintaining rinderpest. Segregation of game on a large scale, possibly with selective destruction in certain restricted areas, is the only weapon we possess at present to counter this danger. The type of bush fencing described is an attempt at evolving a practical, effective and relatively cheap means of achieving this.

6. The need for further research into the diseases of game animals and the necessity for revision of the prevalent attitude toward the game problem in Africa is emphasized.

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Fig. 1.—Herd of Buffalo at Sakalilo. Note egrets on buffalo's backs.



Fig. 2.—Sick eland cow shot on Rukwa plain. Profuse discharge from eyes and mealy deposits on tongue and gums.



Fig. 3.—Ulcerative stomatitis in Calf X inoculated from sick eland.



Fig. 4.—The Msangano fence viewed across the Nkua valley from the Ufipa escarpment.

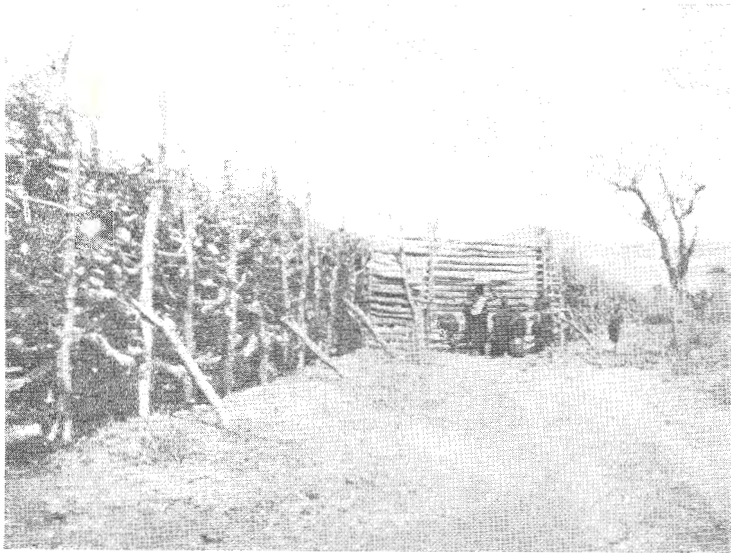


Fig. 5.—The Msungano fence, Natives crossing at road gate.