

9. *Thelazia and hodesii theory*.—*Thelazia* and *hodesii* is the only eye worm found in South Africa, and it occurs exclusively in the ox and buffalo. It plays no role, contrary to popular belief, for thousands of animals that do not harbour the parasite contract the disease and vice versa. Thus far it will be seen we have fairly conclusively eliminated all the theories except two—the rheumatic and the virus of the influenza type. These two cannot be studied at this juncture for very obvious reasons. We cannot now come to the last and most promising theory:

10. *The theory of protein sensitization*.—Keratitis anaphylactica is a well known condition in the human subject, and we have observed it in donkeys more than once during the process of hyperimmunization against *B. anthracis*. The study of allergy is one of the romances of modern medicine and since the demonstration of Walzer of whole food proteins in the normal blood stream is likely to assume tremendous importance. It is already well known that food allergy alone accounts for many cases of migraine, eczema, bronchial asthma, intestinal obstruction, idiopathic epilepsy, angioneurotic oedema of the urogenital tract, hypotension, irregular and painful menstruation, sinus congestion, asthenia, diarrhoea, constipation and nausea, etc.—a truly formidable list.

As yet, however, nothing has been done to study systematically the allergic reactions in the domestic animals, but it is fervently hoped that such work will be undertaken here in the immediate future.

It is conceivable that in ophthalmia we have first a sensitization of the cornea and perhaps also of the conjunctiva. The subsequent absorption of the sensitizing protein would presumably, by analogy, lead to an oedematous condition of these structures. As a result of this oedema the superficial epithelial cells of the cornea would become desquamated, thereby paving the way for secondary bacterial infection. The picture would now be one of an acute case of ophthalmia.

Such a theory would preclude the possibility of a so-called incubation period. Instead of this there would be an indefinite sensitizing period.

Paper No. 8.

A HYPOTHESIS CONCERNING THE ETIOLOGY OF
“STIFF-SICKNESS.”

By Dr. C. SHEPPARD CRUZ, Veterinary Officer, Department of
Agriculture, Mozambique.

THE author of this essay has had the opportunity, in the course of the last year, to come in contact with two forms of “stiff-sickness” in an acute stage, and to observe their known symptomatology, under favourable conditions. The report of these cases was made in such a manner as to place us from the outset on the right track. Subsequent observations on these animals—in kraals on the left margin of the Incomati River (Pacuane), and in other kraals on the left margin of the Limpopo River (Moambe)—confirmed the diagnosis.

If there are, as we believe, two diseases, both bearing the name of “stiff-sickness,” the cases which we observed must be attributed to

phosphate deficiency in the soil as their initial cause, and not to the eating of the leguminous toxic plant, *Crotalaria burkeana*, which is the cause of the disease that we may name "Crotalariosis."

The pasturage area where we discovered the first cases, and where the Pacuane cattle were put to graze, adjoins the region where, about two and a half years ago, several cases of paratuberculosis (lamziekte) were observed by us. Seeing that a common origin is given to both sicknesses—phosphorosis—this proximity and the strikingly similar physiognomy of both zones (stiff-sickness at Pacuane and Paratuberculosis at Mafabaz), support such an opinion. In fact, for many years, in the Union of South Africa the similarity and probable kinship of the two diseases, occurring on the same farm or adjoining ones, has been verified. Taking the animals under identical conditions as from the osteophagy, this would lead, in the one case, to paratuberculosis, and, in the other, to what will directly determine "stiff-sickness," which we may consider still unknown to us.

Both at Pacuane and at Moambe, the sickness broke out during the period of intense drought, especially at Moambe, where for many years it had never been so great and alarming. The symptoms observed were the painful support of the feet, giving the animals, both when walking and when stationary, the peculiar character so often described, followed by the rapid and defective growth of the hoofs. These, after some time, took a spatular shape, the wider part, polished, corresponding to the healthy portion, the strangulated part, being dull and with transversal ridges, produced in the course of the sickness by an abnormal keratogenesis. The foot, in profile, resembled that of an ostrich supporting itself on its talons.

The deformation, unlike that caused by the crotalariosis, did not lead, in the numerous cases observed by us, to the superimposition of the nails (hoofs), but both rested on the ground, separated one from the other by a rather wide angle. They were, besides, flat from top to bottom. However striking this difference may have been—easily noticed by comparing the accompanying photographs, one of which was taken by the Director of Veterinary Department—it was based on the conviction that the cases to which we have been referring were ascribed to the deficiency of assimilative phosphoric acid. A more recent confirmation of this fact arose after most careful and thorough investigation, no cases of "*crotalaria burkeana*" having been found in the region of Moambe (Figure 1).

The "bone test" was made, and we were unable, after the acute stage had passed, to detect any well-defined cases of osteophagia among the herds in which this sickness had raged. We only managed to verify, among a great number of the animals of those herds, that they continually licked for a long time the white bones which had a slight smell. The phenomenon was, however, known to the natives of the region of Moambe, who had noticed it with a well-defined character of osteophagia among their own cattle.

But what chiefly suggested the present considerations to us, was the following observation, which was made on the same occasion and at only about one hundred metres from the place where we were examining the animals attacked by stiff-sickness, at Moambe: The chickens of a foreign Missionary, who had his huts there, had for some time been dying from a strange disease. We were shown a

sick fowl: It walked with difficulty, as if feeling the ground for support of the feet, and we noticed, across the discoloured skin of the tarsus, metatarsus, and phalanges, an intensive congestion, translated into a strikingly pinkish colour. The claws, then already excessively grown—about one and a half centimetres—showed, by their transparency, the same congestion of the adjoining tissues.

In another dead fowl, the claws of which are illustrated, Figure 2, showed also that the nails took a spiral form, being a modification perhaps accounted for by retraction after death, as it had not been noticed with the sick animal above quoted. We do not think any food was given to these birds, beyond a few handfuls of mealies now and then.

It was impossible not to connect the phenomena observed among the cattle (bovines) and not to attribute the two diseases to a common cause, both having identical symptomatology, apparently only one circumstance distinguishing them: While the mortality among the bovines was limited to two cases, which were accounted for as due to starvation (as the animals were, for many days, unable to rise from the ground), on the other hand, the mortality among the chickens was great, although somewhat similarly attributed to the difficulty in procuring food.

The great scientist, Sir Arnold Theiler, in his studies re “stiff-sickness,” was impressed by the clinical resemblance between the cases of this disease and those of laminitis or podophilitis of horses, attributed by him to a toxicosis of vegetable origin. That led him then (see Report of Pretoria Laboratory of 1909-1910) to the conclusion that stiff-sickness might be due to the eating of the *Crotalaria burkeana*, according to the testimony of farmers. Experimental feeding with the plant reproduced the symptoms of the disease, which was, evidently crotalariosis. Lately, the generalized verification of cases of stiff-sickness, where the plant did not exist, gave rise to Hutcheon's theory, which, in 1884, without any reference to *Crotalaria burkeana*, considered this disease dependent on the deficiency of phosphates in the food. As the experiments of Theiler were of indisputable reliability, the hypothesis of the existence of two diseases to which the same name has been given, would have resulted as a consequence thereof.

From this we may give an opinion which appears to us to be quite logical: As the congestion of the tissues of the foot—initial stage in the inflammatory process which leads to the osteitis of stiff-sickness, and which is the cause of the keratogenous activity which produces the deformation of the nails—is due, in one of the cases (crotalariosis) to a toxicant vegetable, why should we not admit the hypothesis that, in the other case, stiff-sickness, an identical deformation with symptoms should not be caused by a toxicant of microbial origin? We base ourselves precisely on the same kinship as between stiff-sickness and the podophilitis of the horses, which, as is known, is also connected with infectious stages, that is, a microbial origin. Why should not the stiff-sickness which we observed (giving it that name in the absence of a more rigorous designation) be a toxemia of saprophytic origin, as the paratuberculosis of the bovines, thus explaining the relation of one with the other of these diseases, by a frequent association of their casual agents in the organic matter—bones, putrified flesh of carcasses found in the grazing ground—

which the animals lick or swallow during the most intense drought seasons? Given that simultaniety, as verified by us, of the disease of the bovines and the chickens, and knowing, as we do, that these animals eat all the organic detritus they can find, it is not difficult to admit that the bovines got their toxicosis from the same source, provided it were actually a case of toxicosis.

These are simply hypotheses, which laboratorial research only can either invalidate or confirm. The following observations relating to the epidemiology of the disease, will further give them verosimilarity, viz: That the disease attacks a great number of animals of the same kraal, whereas neighbouring kraals are left absolutely immune, this leading us to believe that, in reference to the attacked kraals, there occurred certain conditions, limited to small areas which did not occur in the other though larger areas. The case of lamsiekte already referred to by us justifies exactly the same observation, as, in fact, it seems characteristic of this sickness. The epidemiological distribution would correspond, in stiff-sickness, as well as in lamziekte, to the stains of diffusion of the causal microbe brought from afar, the simple, verified vice of bone-licking implying the deglutition of saliva loaded with bacteria, being sufficient to explain the toxicosis.

