

solidly immune, the second one showed a febrile reaction which persisted for five days, while in the third animal the fever subsided after ten days. Koch bodies in a relatively small number were demonstrable in the latter two animals. Both made a rapid recovery. The reason for using East Coast fever infective nymphae for the immunity test was that the available Corridor disease infective *Rh. appendiculatus* adults often failed to attach readily. It was feared that inconclusive results would have been obtained had the latter infective ticks been employed for the immunity tests. In the untreated controls typical reactions of this disease were produced, followed by death within eighteen days after attachment of ticks.

Thus aureomycin administered in repeated doses during the incubation period of *G. lawrencei* infection is a schizonticide which acts on the early schizonts by suppressing nuclear division. The result is that the development of schizonts is inhibited and the clinical disease fails to develop. Nevertheless, a solid or a partial immunity against East Coast fever is produced.

Consideration of these observations makes it apparent that aureomycin cannot be employed in practice. However, this form of treatment is of great value for the production of immune animals required for immunological studies.

#### *Prognosis.*

Prognosis should always be guarded as the mortality rate may be higher than 80 per cent.

#### *Prophylaxis.*

1. *Elimination of arthropod vectors.*—Systematic dipping and hand-dressing, as employed for the control of East Coast fever (*vide supra*), should be equally effective in checking Corridor disease. In the Union of South Africa this disease has remained confined to regions inhabited by the African buffalo. As these areas are situated within the potential East Coast fever regions, compulsory dipping may have largely contributed to its control.

2. *Removal of cattle from enzootic regions.*—Lawrence (1934) has recorded his experience on the control of Corridor disease. He stated that when cattle were moved from areas in which buffalo disease occurred to even nearby areas which were regarded as always being free from danger, mortality would cease immediately after all the animals which were showing signs of the disease when moved had died, and further, that if they were herded with susceptible cattle these would not become infected, even though ticks were prevalent. His observations have been confirmed by Neitz, Canham and Kluge (1955) in Zululand. An outbreak of malignant syncerine gonderiosis followed when cattle were introduced into the Corridor. The mortality ceased within a period of three weeks after removing these animals to the sections of the Corridor not frequented by buffaloes. Furthermore it was observed that the disease also failed to establish itself when these animals, which included several recovered cattle, were retransferred to the farms of origin (*vide supra*—History).

The problem of the African buffalo as a reservoir of Corridor disease is fully realized by the Veterinary Authorities of Southern Africa.

#### *Immunity.*

Recovered cattle develop an immunity but do not harbour the erythrocytic stage of *G. lawrencei*. The immunity is solid for periods of up to four months after recovery. (The end point has not yet been determined). Observations on a naturally recovered buffalo calf have shown that the endoglobular parasites are retained for at least a year, as determined by biological transmission experiments.

## THEILERIOSIS, GONDERIOSES AND CYTAUXZONOSIS.

Splenectomy of two calves, which had recovered from a natural infection of Corridor disease, was followed by the appearance of erythrocytic parasites after a period of 70 days in one and 124 days in the other. The number of parasitized red blood cells, as observed over a period of three months, was less than 0·1 per cent. It is possible that the organisms actually appeared earlier in both calves but that their exceedingly low incidence in the peripheral blood did not permit ready detection. It needs to be determined whether these erythrocytic parasites are those of *G. lawrencei* (see footnote on page 275).

Benign bovine gonderiosis recovered cattle are fully susceptible to Corridor disease. A variable degree of cross-immunity exists between Corridor disease and East Coast fever. It may be partial after a period of three months or alternatively remain solid for periods of up to four months. (The end point has not been determined). The reason for the immunogenic relationship between *Th. parva* and *G. lawrencei* needs to be determined.

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## 5. RHODESIAN MALIGNANT BOVINE GONDERIOSIS.

### GONDERIA BOVIS INFECTION.

#### *Definition.*

Rhodesian malignant bovine gonderiosis is a highly fatal peracute, acute or subacute tick-borne disease caused by *Gonderia bovis* Neitz, 1957. It is characterized by pyrexia, anorexia, malaise, a variable degree of lymphadenitis, general weakness, prostration and dyspnoea before death. Recovered animals develop a durable premunity.

#### *Synonyms.*

"Theileriosis", January disease: Rhodesiese kwaadaardige gonderiose van beeste (Afrikaans); Rhodesische bösartige Gonderiose der Rinder (German); Gonderiose a *Gonderia bovis* (French).

#### *History.*

A brief historical review on the occurrence of East Coast fever, benign bovine gonderiosis and malignant syncerine gonderiosis in Southern Rhodesia has been given by the writer under the heading of Corridor disease (*vide supra*). Lawrence (1933, 1934) who initiated the studies on the differential diagnosis of these diseases

was struck by the fact that the epizootology, of what is now known as Corridor disease, differed greatly from that of the other two tick-borne diseases. Outbreaks of Corridor disease ceased when cattle were moved from enzootic areas into disease-free regions. It thus became evident that this disease is a self-limiting disease in the absence of the African buffalo. However, in his subsequent annual veterinary reports Lawrence (1935-1953) stated that a disease which he named "Theileriosis" is widely distributed in Southern Rhodesia in the complete absence of buffaloes, and that affected as well as recovered cattle serve as reservoirs for the infection of ticks.

#### *Distribution.*

The occurrence of Rhodesian malignant bovine gonderiosis has so far been diagnosed with certainty only in Southern Rhodesia. There is some but no complete evidence that it also occurs in Nyasaland.

Southern Rhodesia:— Rhodesian malignant bovine gonderiosis (= "Theileriosis") has been recorded from the districts of Bulawayo, Gwelo, Lomagundi, Melsetter, Mrewa, Makoni, Nuanetsi, Salisbury, Umtali and Victoria by Lawrence (1935, 1936, 1937, 1939, 1940, 1946, 1947, 1953), Adamson (1950), Hooper-Sharpe (1936, 1938), Huston (1946, 1948), King (1942), Mackinnon (1951), Myhill (1938, 1940) and Nixon (1953).

The writer is of opinion that this disease may also occur in Nyasaland. He bases his assumption on the fact that spleen smears submitted by Wilson (1947) from Nyasaland revealed schizonts in relatively small numbers and morphologically indistinguishable from those of either *G. lawrencei* or *G. bovis*. Wilson (*loc. cit.*) made no mention that this form of gonderiosis occurred in association with the African buffalo.

#### *Aetiology.*

*Gonderia bovis* spec. nov. Neitz, 1957.

(a) *Morphology.* (i) Erythrocytic parasites:— Morphologically the erythrocytic stages of the protozoon responsible for Rhodesian malignant bovine gonderiosis resemble *G. mutans* very closely. In blood smears fixed with May-Grünwald and stained with Giemsa, the protozoon appears in the red blood cells as pear-shaped, comma-shaped, oval or round organisms. The pear-shaped forms are 0.6 micron in width and 1.8 microns in length; comma-shaped 0.5 micron in width, 2.0 microns in length; oval forms 0.6 micron in width and 1.5 microns in length and round forms 0.6 to 1.8 microns in diameter.

The cytoplasm stains light blue. The nucleus appears as a deeply stained minute reddish purple granule situated at the wider end of the pear-shaped, comma-shaped and oval forms, and on the margin of the round parasites. When reproduction takes place the nucleus divides into two and finally into four granules.

(ii) Histiotropic parasites:— Morphologically the Koch bodies of the protozoon responsible for Rhodesian malignant bovine gonderiosis are indistinguishable from those of *G. lawrencei*. In organ and blood smears fixed with May-Grünwald and stained with Giemsa the schizonts (Koch bodies) appear as masses of blue staining cytoplasm containing 1 to 16, and sometimes up to 32 reddish purple granules varying from 0.5 to 2.0 microns in size. The Koch bodies vary in size from 1.0 to 10.0 microns with an average of 5.0 microns.

They are seen either free or within the lymphocytes. Two types of schizonts commonly referred to as agamonts (macroschizonts) and gamonts (microschizonts) can be recognized. The latter type is not often seen. The common forms are the macroschizonts varying from 2·0 to 5·0 microns in diameter. When fully formed the macroschizonts liberate macromerozoites varying from 2·0 to 2·5 microns in diameter. The mature microschizonts liberate micromerozoites 0·7 to 1·0 micron in diameter when round. Some of the forms are ovoid in shape, while others are rod-like, pear-shaped or comma-shaped.

(b) *Multiplication.*—The protozoon of the disease under discussion multiplies by schizogony. When schizonts are fully formed they break up into merozoites which enter lymphocytes to grow and reproduce by schizogony again, or they penetrate the erythrocytes in which they are found in ordinary blood films. The writer observed that multiplication occurs within the erythrocytes. Division into four takes place resulting in cross forms, in which four minute pear-shaped individuals radiate from a central point.

(c) *Habitat.*—The erythrocytic stages of this parasite have been observed in an ox on the eighth day after the initial rise in temperature. Approximately five per cent of the erythrocytes were parasitized. The host cell may harbour one to four parasites. Schizonts parasitize lymphocytes. In smears prepared from the spleen, lymphatic glands, lungs, kidneys and liver approximately five per cent of lymphocytes harbour Koch bodies.

(d) *Life-cycle.*—In the vertebrate host the protozoon multiplies by schizogony in the lymphocytes. The forms in the erythrocytes reproduce by division into four daughter individuals. The final stage of the parasite is possibly a gametocyte or a gamete. No attempts have yet been made to study the development of the parasite in the invertebrate host.

(e) *Action of physical and chemical agents.*—In a single instance Lawrence (1936) found that blood, collected in sodium citrate from two affected cattle, remained potent for a period of 24 hours. The temperature at which the specimens were stored is not mentioned.

(f) *Biological characteristics.*—The occurrence of immunologically different strains has not yet been established. Cross-immunity tests, however, have shown that there is an immunogenic relationship between the causal agent of Rhodesian malignant bovine gonderiosis and East Coast fever (Lawrence, 1939).

In cattle a sporozoite induced infection results in the protozoon of Rhodesian malignant bovine gonderiosis completing its vertebrate life-cycle, and finally erythrocytic parasites appear which are capable of infecting ticks. No information is available whether or not the African buffalo is susceptible.

(g) *Taxonomy.*—It has become apparent from the description of the protozoon responsible for Rhodesian malignant bovine gonderiosis that it is morphologically indistinguishable from *G. lawrencei*, and that both parasites possess an immunogenic relationship to *Th. parva*. Consideration of the behaviour of the two parasites in cattle shows that there is a significant difference which has a direct bearing on the epizootology of the diseases produced by them. A sporozoite-induced *G. lawrencei* infection in cattle results in the development of schizonts but not of the erythrocytic stages; cattle can thus not serve as reservoirs, and in the absence of premune African buffaloes malignant syncerine gonderiosis is a self-limiting disease. However, the protozoon responsible for Rhodesian malignant bovine gonderiosis is not only capable of completing its vertebrate life-cycle in cattle

(Lawrence and Neitz, 1957), but recovered animals retain the erythrocytic parasites for the infection of ticks (Lawrence, 1935-1953; Adamson, 1950, 1952; Hooper-Sharpe, 1936, 1938; Huston, 1944-1949; King, 1942; Mackinnon, 1951, 1953; Myhill, 1939, 1940, 1941; and Nixon 1953). The development of a premunity in animals is a characteristic feature of the genus *Gonderia*.

Consideration of the difference in the behaviour of both these protozoa in the bovine host, and also the marked difference between the epizootology of the two diseases produced by them, clearly indicates that the two parasites are not identical. It is, therefore, proposed to name the protozoon responsible for Rhodesian malignant bovine gonderiosis *Gonderia bovis* spec. nov.

#### *Transmission.*

##### *A. Natural transmission.*

(a) *Biological transmission.*—It has been established by Lawrence and Neitz (1957) that *Rh. appendiculatus* nymphae that engorged on an animal reacting to Rhodesian malignant bovine gonderiosis in the vicinity of Salisbury, transmitted this disease to an ox in the ensuing stage.

(b) *Mechanical transmission.*—There is no evidence that this form of transmission occurs in nature.

(c) *Intra-uterine transmission.*—This form of transmission has not yet been observed.

##### *B. Artificial transmission.*

This type of transmission is possible but not always successful. Lawrence (1936) attempted to transmit the disease to four oxen. Infective blood (quantity used not mentioned) was injected into two animals by the intravenous, intraglandular, subcutaneous and intracutaneous routes, and into the remaining two animals by the subcutaneous route. One of the former two animals developed a thermal reaction (105° F) 19 days later. The fever persisted eight days. Koch bodies were demonstrable in lymphatic gland smears three to seven days after the initial rise in temperature. The erythrocytic stages of a *Gonderia* sp. were observed three days after the first appearance of Koch bodies. The identity of this parasite was not determined. It should be borne in mind that the donor and possibly also the recipient in all probability also harboured *G. mutans*. The animal recovered. Two of the remaining animals developed an inapparent infection. This was evidenced by the fact that, when their immunity was challenged with East Coast fever infective ticks 19 months later, both proved to be solidly immune (Lawrence, 1939). [There is an immunogenic relationship between *Th. parva* and *Gonderia bovis* (*vide infra*—Immunity)].

#### *Epizootology.*

Rhodesian malignant bovine gonderiosis is confined to regions in which *G. bovis* premune cattle and vectors occur. The density of infective ticks determines the incidence of the disease. At present it is known that *Rh. appendiculatus* ticks serve as transmitters (Lawrence and Neitz, 1957). It needs to be determined whether or not other *Rhipicephalus* spp. can act as vectors.

Lawrence (1934-1953), Adamson (1950), Hooper-Sharpe (1936, 1938), Huston (1944-1949), King (1942), Mackinnon (1951, 1953), Myhill (1939, 1940, 1941), and Nixon (1953) have concluded from their epizootological observations that Rhodesian malignant bovine gonderiosis ("Theileriosis") is widely distributed in

Southern Rhodesia, and that it occurs in cattle in the complete absence of the African buffalo (*Syncerus caffer* Sparrman). They draw the inference that recovered cattle develop a premunity, and thus serve as reservoirs for the infection of ticks. [Milne (1956) describes an atypical form of East Coast fever in Tanganyika which may be related to Rhodesian malignant bovine gonderiosis or malignant syncerine gonderiosis).].

Cyclical variations in the seasonal incidence of Rhodesian malignant bovine gonderiosis have been observed. Lawrence (1935-1945) states that it makes its appearance most commonly during the period from December to March. The highest incidence is in January, and farmers often refer to it as "January disease". Its almost invariable association with gross tick infestation explains its seasonal incidence. Sporadic cases may, however, occur out of season even on farms where tick control is conscientiously practised. Sporadic cases have been encountered in winter and in spring before the commencement of the rainy season.

Rhodesian malignant bovine gonderiosis often occurs in association with anaplasmosis, babesiosis, heartwater and benign bovine gonderiosis. It may also occur within the enzootic East Coast fever areas. In these circumstances it is difficult to estimate the direct losses due to Rhodesian malignant bovine gonderiosis unless systematic smear examinations are made from all animals within an area. The Veterinary Authorities in Southern Rhodesia have collected such data from potential East Coast fever areas, where stock-owners regularly submit smears from all their dead cattle. They point out that the annual losses from Rhodesian malignant bovine gonderiosis may be great unless prophylactic measures such as dipping and handdressing are practised. Prophylaxis against East Coast fever has undoubtedly been of enormous value in controlling Rhodesian malignant bovine gonderiosis.

#### *Pathogenicity.*

Cattle are susceptible to *G. bovis*. The ox is a perfect host in that this protozoon can complete its vertebrate life-cycle in this animal. Observations in Southern Rhodesia have shown that calves are as susceptible as adult cattle. The mortality rate may be as high as 90 per cent (Mackinnon, 1953).

It needs to be determined whether or not the African buffalo, sheep and goats are susceptible.

#### *Pathogenesis.*

The lesions present in the lungs, spleen, kidneys, liver, lymphatic glands and alimentary tract suggest that they are due to a toxin produced by the infectious agent. The endothelial lining of the blood vessels is also affected resulting in an oedema of the lungs, and a variable degree of oedema of the subcutaneous tissues, and hydrothorax, hydropericardium and ascites.

#### *Symptomatology.*

Rhodesian malignant bovine gonderiosis may be classified according to its symptoms into four types: (1) the acute, (2) the subacute, (3) the mild and (4) the inapparent form.

After exposure to infective ticks the incubation period is about eleven days. In a single instance it was found that after infection by the injection of infective blood, the period was 19 days (Lawrence, 1936). The duration of the disease varies from 5 to 15 days with an average of 10 days.

## TIBERIOSIS, GONDERIOSIS AND CYTAUXZONOSIS.

- (1) *The acute form.*—This form is commonly encountered. The first indication of the disease is a swelling of the parotid lymphatic glands which is followed two or three days later by a rise in temperature (104° to 107° F). The fever may be continuous for five to ten days, or alternatively it returns to normal after six to ten days, and rises again after one to two days to 106° F for a period of five to seven days. The temperature becomes subnormal shortly before death. Koch bodies may be demonstrable in small numbers one or two days before the initial rise in temperature. As in the case of East Coast fever, and the different types of gonderiosis the lymphocytes show active mitosis.

Clinical symptoms usually appear a few days after the commencement of the fever. The affected animal shows inappetence, cessation of rumination, serous nasal discharge, lachrymation, drooping ears, sometimes swelling of the eyelids and ears, swelling of the superficial lymphatic glands, muscular tremors, groaning, grinding of the teeth, swaying gait, cessation of milk-production and loss in condition. At the beginning of the pyrexial period the faeces are firm but diarrhoea often sets in six to seven days after the onset of fever. The evacuations may be mixed with blood and mucus. The patient becomes markedly emaciated, is inclined to lie down, and when forced to rise it may cough. Towards the end of the disease the respiration becomes accelerated and distressed. The animal collapses, froth escapes from the nostrils, and death due to asphyxia supervenes.

During the course of Rhodesian malignant bovine gonderiosis, particularly when the course of the disease persists for longer than ten days, relapses due to *Babesia bigemina* and *Anaplasma marginale* may appear. The symptoms of babesiosis and anaplasmosis may obscure the typical clinical manifestations of Rhodesian malignant bovine gonderiosis.

- (2) *The subacute form.*—This form is encountered from time to time. The symptoms resemble those of the acute form but are not so pronounced. The fever is either continuous or irregularly intermittent, and persists for five to ten days. Koch bodies in relatively small numbers can be demonstrated in the lymphatic gland and usually also in blood smears. Cattle usually recover from this form but it may take several weeks before they regain their former condition.
- (3) *The mild form.*—This form has been described by Lawrence (1936) in an artificially infected animal. The symptoms were a mild fever (105° F) which persisted for eight days, listlessness and a swelling of the superficial lymphatic glands. Koch bodies were demonstrable in lymphatic gland smears three to seven days after the initial rise in temperature. The patient made an uninterrupted recovery.
- (4) *The inapparent form.*—This form has been described by Lawrence (1936, 1939) in two artificially infected oxen. They received infective blood by the intravenous route. On challenging their immunity 19 months later they were found to be solidly immune.

### *Pathology.*

The lesions of Rhodesian malignant bovine gonderiosis are fairly uniform but may vary somewhat according to the duration and the severity of the disease.



### A. Macroscopical lesions.

Generally speaking, the lesions of Rhodesian malignant bovine gonderiosis resemble those of East Coast fever very closely. The carcasses show a variable degree of emaciation. The skin may show decubital wounds. The perineal region may be soiled with faeces. The visible mucous membranes are cyanotic. The subcutaneous and intermuscular tissues may be infiltrated with serous fluid. Degenerative changes and haemorrhages in muscles have, however, not been described.

The myocardium is flabby, and a variable number of petechiae appears on the epicardium and endocardium. Hydrothorax and hydropericardium may be present. The lungs show a variable degree of oedema and hyperaemia; the mucous membrane of the bronchi and bronchioli usually show a large number of petechiae; the trachea may contain froth. The liver is swollen, friable and brownish yellow in colour; degenerative changes are evident. The gall bladder may be markedly distended with dark green viscid bile. The spleen may be enlarged and the pulpa soft; the Malpighian corpuscles may be prominent. The lymphatic glands are as a rule swollen, and may show a variable degree of hyperaemia. The kidneys are either congested or pale yellow in colour; a variable number of haemorrhagic "infarcts" or greyish white lymphomata is often present in the cortex. The urinary bladder is usually markedly distended with clear yellow urine; petechiae may be present in the mucous membrane. The meninges may be slightly congested but the brain does not show any lesions.

The rumen and reticulum contain a relatively small amount of ingesta, while the contents of the omasum may be firm and partially dehydrated. A variable number of superficial erosions is present in the mucous membrane of the abomasum. Similar erosions as well as irregularly disseminated red streaks or patches may be encountered in the intestinal tract. The contents of the small and large intestine may show a distinct yellow discoloration. Peyer's patches may be swollen.

### B. Microscopical lesions.

No information is available in the literature on the nature of the histopathological lesions.

#### Diagnosis.

Epizootologically, *G. bovis* infection is a disease of place. When investigations are being made it is essential to determine the presence of the vector, the local incidence of Rhodesian malignant bovine gonderiosis and other diseases likely to be confused therewith, the occurrence of *G. bovis*-pre-mune cattle, the presence or absence of *G. lawrencei*-pre-mune African buffaloes, and the origin of the sick animals. These considerations should guide the investigational procedures.

Although a tentative diagnosis of Rhodesian malignant bovine gonderiosis can be made by considering the epizootology, the clinical symptoms and lesions at autopsy, a definite diagnosis depends upon demonstrating the schizonts of *G. bovis* in blood and organ smears. The Koch bodies of *G. bovis* vary from 1·0 to 10·0 microns with an average diameter of 5·0 microns, and are morphologically indistinguishable from those of *G. lawrencei*. The presence of *Gonderia* erythrocytic stages in cattle may be of assistance in making a differential diagnosis but it should be remembered that cattle on the African continent frequently, if not always, harbour *G. mutans*. In these circumstances the presence of erythrocytic parasites cannot be relied upon when making a differential diagnosis between Rhodesian malignant bovine gonderiosis and malignant syncerine gonderiosis.

The endoglobular parasites can only be identified by applying the xenodiagnosis. The Koch bodies of *Th. parva* and *G. mutans* vary from 1·0 to 15·0 microns with an average diameter of 8·0 microns. In Rhodesian malignant bovine gonderiosis and in malignant syncerine gonderiosis approximately five per cent of lymphocytes are parasitized with Koch bodies but mature microschizonts (gamonts) liberating merozoites are only encountered on rare occasions. The percentage of infected lymphocytes in benign bovine gonderiosis may be the same as in Rhodesian malignant bovine gonderiosis but in East Coast fever usually more than 60 per cent of lymphocytes harbour macro- and microschizonts. The latter stage is often seen liberating merozoites.

A differential diagnosis between malignant syncerine gonderiosis and Rhodesian malignant bovine gonderiosis is possible if the epizootology is taken into consideration. In an outbreak of malignant gonderiosis a diagnosis of Corridor disease is permissible if the investigations show that *G. lawrencei*-preimmune buffaloes occur in that region; in their complete absence a diagnosis of Rhodesian malignant gonderiosis is justified. Should both diseases be suspected to occur in an area inhabited by the African buffalo, the causal agents can be differentiated by making a xenodiagnosis. It should be remembered that practically all cattle in such areas harbour a latent infection of *G. mutans*, and that benign bovine gonderiosis may also be transmitted by the ticks employed for the test. The xenodiagnosis involves feeding *Rh. appendiculatus* larvae and nymphae on several reacting animals showing erythrocytic parasites in the peripheral circulation. The ensuing stages should be allowed to engorge on at least four fully susceptible calves. The appearance of benign bovine gonderiosis in these animals indicates that Corridor disease is responsible for the outbreak. However, should one or more of the experimental calves develop a malignant form of gonderiosis then a diagnosis of Rhodesian malignant bovine gonderiosis is justified. For further confirmation of the diagnosis, it is recommended that the immunity of all the survivors be challenged with either *Th. parva*, *G. lawrencei* or *G. bovis* infective ticks. There is an immunogenic relationship between these three protozoa but not between them and *G. mutans*. A complete or a partial immunity is additional evidence that the outbreak was due to a *G. bovis* infection.

#### *Treatment.*

##### *A. Specific treatment.*

The efficacy of several chemotherapeutic agents in the treatment of Rhodesian malignant gonderiosis has been tested during the last two decades. It has been established by Lawrence (1940, 1945, 1947, 1953) that uleron, sulphapyridin, nivaquine, penicillin, aureomycin and calcium chloride are of no value when administered during the reaction period.

It needs to be determined whether or not repeated administration of either aureomycin or terramycin during the incubation period of this disease will be as effective as in the case of East Coast fever (Neitz, 1953). It is possible that daily injections of either aureomycin or terramycin in combination with pamaquin may bring about a cure if treatment is started on the first day of fever as in the case of East Coast fever (*vide supra*—*Theileria parva* infection). In analogy with other forms of gonderiosis, pamaquin should have a specific and selective action on the erythrocytic stages of *G. bovis*.

##### *B. Symptomatic treatment.*

Good nursing should accompany the specific treatment.

*Prognosis.*

The prognosis should always be guarded as the mortality rate may be as high as 90 per cent (Mackinnon, 1953).

*Prophylaxis.*

Systematic dipping and hand-dressing as employed for the control of East Coast fever (*vide supra*) is equally effective in Rhodesian malignant bovine gonderiosis. This prophylactic measure was adopted by the Veterinary Authorities in Southern Rhodesia soon after "Theileriosis" was recognized as a distinct tick-borne disease (Lawrence, 1936, 1945, 1947; Huston, 1948; Mackinnon, 1953; Adamson, 1954). Huston (*loc. cit.*) states that dipping of cattle in arsenical dip (0.16 per cent  $As_2O_3$ ) at short intervals is effective but that the results obtained with B.H.C. formulations in dipping tanks are not satisfactory. Mackinnon (*loc. cit.*) comes to the conclusion "that the policy of effective dipping and the hand-dressing of stock on infected farms have much to recommend it. However, the treatment of infected animals instead of destruction has little to recommend it, inasmuch that the recovered animal is a carrier, and when in course of time such is sold and passes into hands of the inefficient dipper a new focus of infection is readily set up".

The problem of premune *G. bovis* cattle as reservoirs of the disease is fully realized by the Veterinary Authorities of Southern Africa. The writer recommends that in order to counteract its spread, cattle from enzootic regions must not be moved into potential Rhodesian malignant areas. Slaughter stock should be dipped and hand-dressed before transferring them either by rail or motor transport to abattoirs.

*Immunity.*

Naturally recovered cattle develop a premunity which persists for as yet undetermined periods.

Benign bovine gonderiosis-recovered cattle are fully susceptible to Rhodesian malignant bovine gonderiosis. However, a cross-immunity exists between the latter disease and East Coast fever as determined by Lawrence (1939) in Southern Rhodesia and Wilson (1947) in Nyasaland with *Th. parva*-infective ticks supplied by the writer. Lawrence (*loc. cit.*) found that a solid cross-immunity still existed 19 months after recovery. The reason for the immunogenic relationship between *Th. parva* and *G. bovis* needs to be determined.

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## OVINE AND CAPRINE GONDERIOSIS.

## 1. GONDERIA HIRCI INFECTION.

*Definition.*

Malignant ovine and caprine gonderiosis is a highly fatal, acute, subacute or chronic tick-borne disease, caused by *Gonderia hirci* (Dschunkowsky and Urodschevich, 1924). It is characterised by pyrexia, malaise, anorexia, progressive anaemia, icterus and a transitory haemoglobinuria in some cases, frequent pulse, nasal discharge, dyspnoea, constipation, swelling of the lymphatic glands, tumor splenis, tumor hepatis, and "lymphomata" in the kidneys. Recovered animals develop a durable premunity.

*Synonyms.*

Malignant ovine and caprine Theileriosis; Kwaadaardige Gonderiose of Theileriose van skape en bokke (Afrikaans); Gonderiose of Theileriose van schapen en geiten (Netherlands); Gonderiose oder Theileriose der Schafe und Ziegen (German); Gonderiose ou Theileriose du mouton et de la chèvre (French); Kisdërma et Istura (Russian).

*History.*

Littlewood (1914, 1915) and Mason (1915) encountered a form of piroplasmosis distinct from *Babesia ovis* infection in Egyptian and Sudanese sheep. They also determined that the disease was fairly widely distributed in Egypt. Microscopic smear examination revealed the presence of erythrocytic parasites and schizonts in various organs. Mason (1915) placed this parasite in the genus *Theileria* but did not give it a specific name. Du Toit (1918) in his classification of the piroplasms gave the parasite the name *Theileria ovis*, and attributes the name to Littlewood, 1914. In the revision of the classification of the piroplasms, Wenyon (1926) pointed out that as the specific name *ovis* had, however, been already used for the non-pathogenic form *Theileria ovis* by Rodhain in 1916, it would seem that the correct name for the pathogenic protozoon in sheep and goats is *Theileria hirci* Dschunkowsky and Urodschevich, 1924. Recently Neitz and Jansen (1956) revised the classification of the Theilerias. They differentiate between the genera *Theileria* and *Gonderia* on the basis that the former genus multiplies only by schizogony within the lymphocytes, while the latter reproduces by schizogony in the lymphocytes and by binary fission in the erythrocytes. Since the life-cycle of *Th. hirci* is typically that of a *Gonderia* sp., they renamed the protozoon *Gonderia hirci* (Dschunkowsky and Urodschevich, 1924).

*Distribution.*

The distribution of *G. hirci* is given below in Table XVI. (See also Map No. 5).

*Aetiology.*

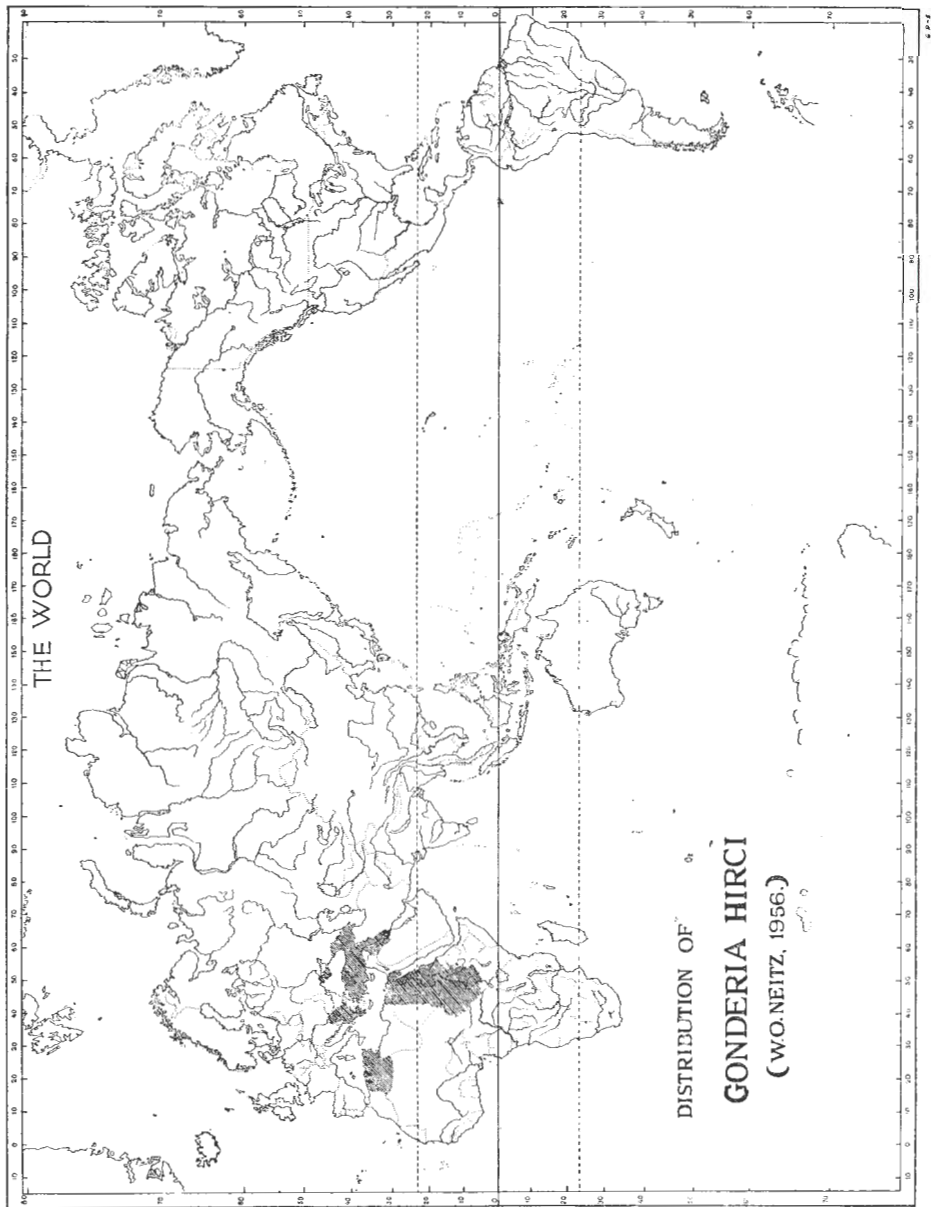
*Gonderia hirci* (Dschunkowsky and Urodschevich, 1924).

Synonyms: *Theileria ovis* du Toit, 1918.

*Theileria hirci* Dschunkowsky and Urodschevich, 1924.

(a) *Morphology.*—(i) Erythrocytic parasites:—In blood smears fixed with May-Grünwald and stained with Giemsa, *G. hirci* appears in the red blood cells

THEILERIOSIS, GONDERIOSES AND CYTAUXZONOSIS.



as pear-shaped, comma-shaped, oval, round or anaplasma-like organisms. The pear-shaped forms are 0.5 micron in width and 1.5 microns in length; comma-shaped forms 0.3 micron in width and 1.7 microns in length; oval forms 0.5 micron in width and 1.0 micron in length; round forms 0.6 to 2.0 microns in diameter, and anaplasma-like forms 0.5 to 1.2 microns in diameter.

TABLE XVI.

Continent.	Country.	Animal.	References.
Africa.....	Algeria.....	Sheep.....	Lestoquard, 1924; 1926.
	Egypt.....	Sheep.....	Littlewood, 1914; 1915.
	Egyptian Sudan...	Sheep.....	Mason, 1915. Littlewood, 1914, 1915. Mason, 1915.
Asia.....	Iraq.....	Sheep.....	Khayyat and Gilder, 1947.
	Turkey.....	Sheep.....	Sprehn, 1939.
		Goat.....	Baumann, 1939.
Europe.....	Crimea (Russia)..	Sheep.....	Markoff 1929.
	Greece.....	Sheep.....	Lestoquard, 1926.
	North Caucasia...	Sheep.....	Yakimoff, 1929.
	Serbia.....	Goat.....	Dschunkowsky and Urodschevich, 1924.
	Transcaucasia....	Sheep.....	Dschunkowsky and Luhs, 1929.

The cytoplasm stains light blue. The nucleus appears as a deeply-stained, minute, reddish purple granule situated at the wider end of the pear-shaped, comma-shaped and oval forms, and on the margin of the round parasites. When division takes place two, three or four chromatin granules are observed. In the anaplasma-like forms the cytoplasm can hardly be recognized. (See Fig. 3).

(ii) Histiotropic parasites:— In organ and blood smears fixed with May-Grünwald and stained with Giemsa, the schizonts (Koch bodies) appear as masses of blue-staining cytoplasm containing one to eighty reddish purple dots varying from 1.0 to 2.0 microns in size. The Koch bodies vary in size from 1.0 to 10.0 microns, and in some cases they may be up to 20 microns in diameter. Schizonts 5.0 microns in size are commonly seen. They are seen either free or within the lymphocytes. Two types of schizonts commonly referred to as agamonts (macroschizonts) and gamonts (microschizonts) can be recognized. When fully formed they break up into numbers of minute bodies (merozoites) situated around a residual body. They appear as minute organisms 1.0 to 2.0 microns in diameter when round. Some of the forms are ovoid in shape, others are rod-like, while some are pear-shaped or comma-shaped.

(b) *Multiplication*.—*G. hirci* multiplies by schizogony. When schizonts are fully formed they break up into merozoites, which either enter lymphocytes to grow and reproduce by schizogony again, or they penetrate the erythrocytes, in which they are found in ordinary blood films. According to Baumann (1939) multiplication also occurs within the erythrocytes. Division into four takes place resulting in cross forms, in which four minute pear-shaped individuals radiate from a central point.

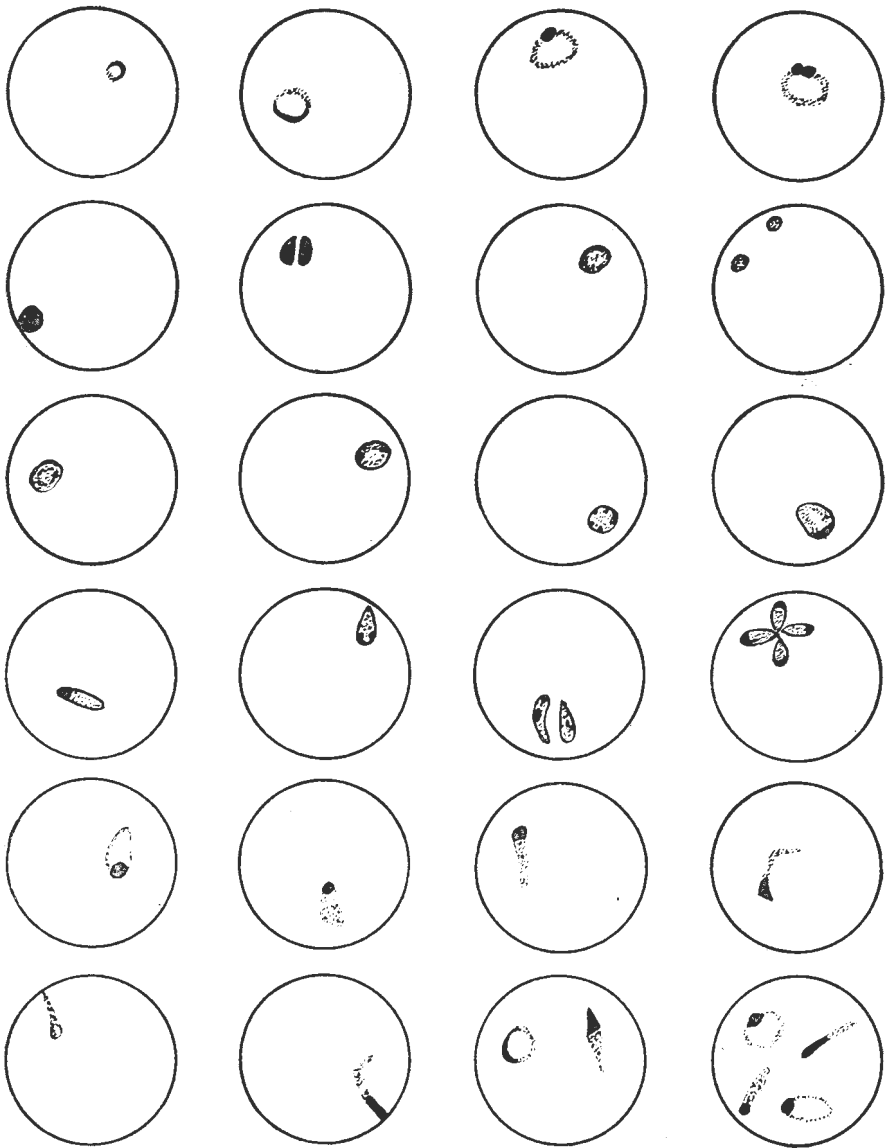


FIG. 3.—*Gonderia hirci*.

(c) *Habitat*.—The erythrocytic stages of *G. hirci* can be readily demonstrated in blood and organ smears. As many as 95 per cent of the erythrocytes may be parasitized (Lestoquard, 1926). The host cell may harbour 1 to 4 microorganisms. Schizonts have been found in fairly large numbers in the spleen, lymphatic-glands, liver, kidney and bone marrow (Lestoquard, 1926). The presence of schizonts in the peripheral blood indicates that careful microscopic examination will also reveal them in other organs.



(d) *Life-cycle*.—Although the life-cycle in the vertebrate host is known, no attempts have yet been made to study the development of the parasite in the intermediate host. (See Fig. 4).

(e) *Cultivation*.—No information is available on the cultivation. There is every reason to believe that it will be possible to grow Koch bodies in tissue culture according to the technique evolved by Tchernomoretz (1945) for the cultivation of *Gonderia annulata*.

(f) *Action of physical and chemical agents*.—The infectious agent remains potent in citrated blood for at least several hours at room temperature.

(g) *Biological characteristics*.—The occurrence of immunologically different strains has not yet been established. The variation in the mortality rate in sheep, however, suggests that strains of different virulence may exist in nature.

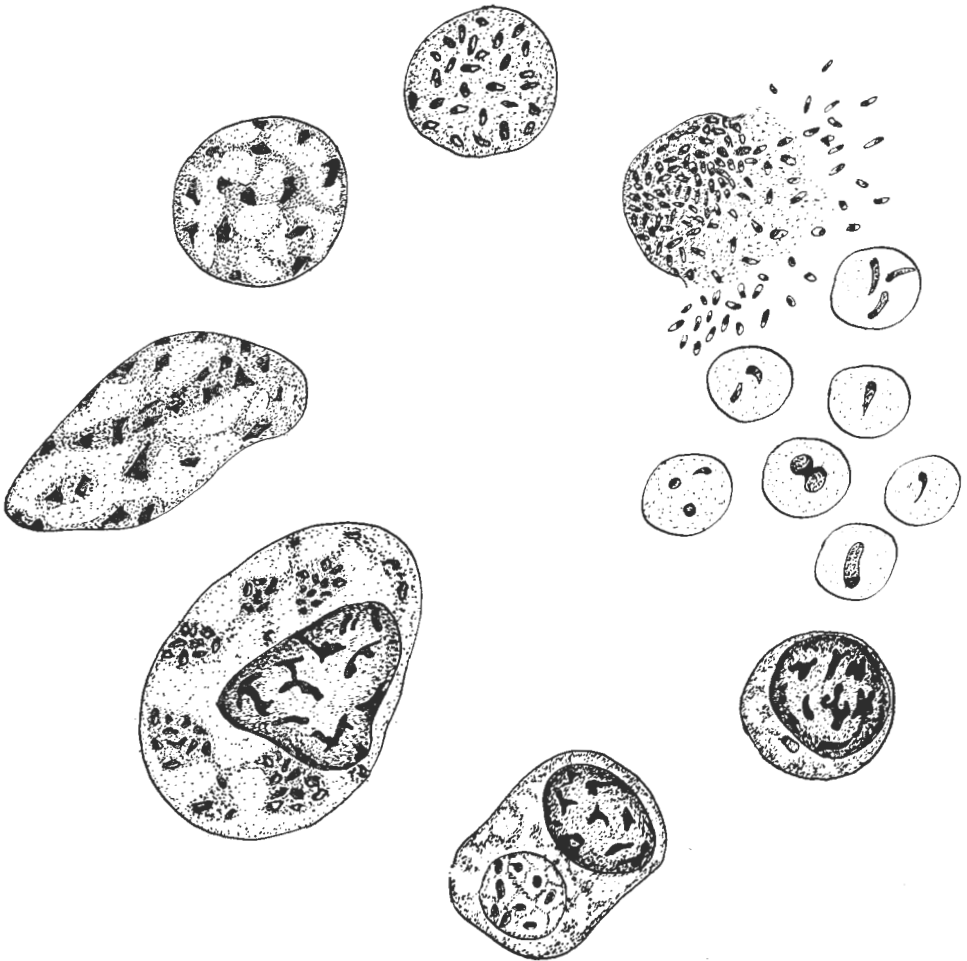


FIG. 4.—*Gonderia hirca*.

*Transmission.**A. Natural transmission.*

(a) *Biological transmission.*—The vector of *G. hirci* has not yet been established. It has been suggested by Dschunkowsky and Urodschevich (1924) that *Rhipicephalus bursa* Canestrini and Fanzago, is the transmitter in Serbia. This tick occurs in all the above-mentioned malignant gonderiosis enzootic regions (*vide supra*).

(b) *Mechanical transmission.*—Blood-sucking insects have not been incriminated as vectors.

(c) *Intra-uterine transmission.*—This form of transmission has been observed by Lestoquard (1926) in two lambs in Algeria.

*B. Artificial transmission.*

*G. hirci* can be readily transmitted by means of blood and organ emulsions, collected during the course of the febrile reaction, by the intravenous route.

*Epizootology.*

Although it has not yet been established experimentally that *Rh. bursa* is a vector of *G. hirci*, it is nevertheless interesting to note that malignant gonderiosis does not occur in countries adjoining the known enzootic areas in which this species of tick is absent. *Rh. bursa* is also a vector of *Babesia ovis* (Babes, 1892), *B. motasi* Wenyon, 1926, and the non-pathogenic parasite *Gonderia ovis* (Rodhain, 1916). Malignant gonderiosis may, therefore, occur in association with babesioses. In these circumstances it may be difficult to estimate the direct losses due to *G. hirci* infection unless systematic smear examinations are made from all animals that die within an area.

In enzootic areas, it appears that young lambs and kids contract a relatively mild form of the disease, which renders them premune. This seems to be reflected in the relatively low mortality rate (16 per cent) in adult sheep born and bred in tick-infested areas of Turkey (Baumann, 1939), as compared with the high morbidity and mortality rate (100 per cent) in sheep introduced from malignant gonderiosis-free areas of North Caucasia into the highly enzootic regions of Transcaucasia (Yakimoff, 1929).

In the Mediterranean countries malignant gonderiosis occurs chiefly during the summer months (June, July and August) but fatal cases have also been recorded in October, (Lestoquard, 1926). In the Caucasian States (Yakimoff, 1929) and in Turkey (Baumann, 1939) the disease has also been observed in Spring (May).

*Pathogenicity.*

Malignant gonderiosis is transmissible from sheep to goats and *vice versa*. The mortality is high. Dschunkowsky and Urodschevich (1924) observed mortality in 46 per cent of affected goats in Serbia. Yakimoff (1929) recorded a mortality rate of 100 per cent in fully susceptible sheep introduced into Transcaucasia.

The disease has been encountered in Egypt in Egyptian, Sudanese and Syrian sheep (Littlewood, 1915; Mason, 1915), in Algeria in French Merino as well as in indigenous sheep (Lestoquard, 1936), in Turkey in sheep and goats (Baumann, 1939), in North Caucasia and Transcaucasia in sheep (Yakimoff, 1929) and in Serbia in goats (Dschunkowsky and Urodschevich, 1924). No mention is made about the variation in resistance possessed by different breeds of sheep and goats.

### *Pathogenesis.*

The lesions present in the liver, spleen, lymphatic glands, kidneys, lungs and alimentary tract suggest that they are due to a toxin produced by the infectious agent. The parasitized erythrocytes liberate haemoglobin followed by haemoglobinuria. Haemoglobinaemia results in an increased production of bile and in icterus. The endothelial lining of blood vessels is also affected resulting in an oedema of the lungs and subcutaneous tissues.

### *Symptoms.*

Observations on naturally and artificially infected animals suggest that the severity of malignant gonderiosis varies in degree according to the virulence of the *G. hirci* strain, and the individual as well as the breed susceptibility. Accordingly acute, subacute and chronic forms can be expected. It should be borne in mind that the state of nutrition and concurrent infections, namely verminoses (Baumann, 1939), and babesioses (Yakimoff, 1929), can greatly modify the clinical syndrome.

The incubation period following a natural infection has not yet been determined. In analogy with other forms of gonderioses it probably varies from 10 to 20 days. The period following an artificial infection is approximately 15 days.

1. *Acute form.*—This is the usual type observed in sheep and goats. The first signs of the disease are a gradual or sudden temperature elevation (104°—106° F) accompanied by anorexia. The fever is continuous and persists for four or five days. Death is preceded by hypothermia. Under field conditions sheep and goats may only be observed to be ill on the day preceding death.

Affected animals show listlessness, dullness, malaise, nasal discharge, hyperaemia of the conjunctiva, developing palour of the visible mucous membranes, icterus in some of the advanced cases, a full bounding pulse, dyspnoea, atony of the digestive tract, decreased milk-yield and oedema of the jowl. The superficial lymphatic glands are swollen. The urine is dark yellow in colour. A transitory haemoglobinuria has frequently been observed. Animals become very weak and show a staggering gait. If they do not die from exhaustion, the symptoms gradually subside after several weeks, and recovery eventually takes place.

2. *Subacute form.*—The symptoms resemble those of the acute form but are not so pronounced. A fever of a continuous nature persists for four to six days. After the primary thermal reaction there may be febrile exacerbations and remissions. Recovery is the usual outcome of this form but it may take several weeks before the animal regains normal condition.
3. *Chronic form.*—Although this type may be chronic from the start it is usually a continuation of either the acute or subacute forms. The course of the disease varies from four to six weeks. Fever is present only in the initial stages of the disease. The pronounced anaemia renders the animals listless and inactive. The mucous membranes are very pale but icterus is not a constant symptom. The appetite is capricious and animals are extremely emaciated. They become very weak, the gait is staggering, and finally they can scarcely maintain stance. Decubitus wounds are usually present. When affected animals do not die from exhaustion the symptom of anorexia subsides and recovery eventually takes place.

*Pathology.*

The lesions at autopsy vary according to the duration and severity of the disease. It is self-evident that concurrent infections can greatly modify the pathological picture. Lestoquard (1926) and Baumann (1939) have given the following account:—

*A. Macroscopical lesions.*

In acute and chronic cases, emaciation and marked anaemia sometimes associated with a variable degree of icterus are characteristic lesions. The subcutaneous tissues of the jawl, throat, ventral region of the neck and abdomen and intermuscular tissues may be infiltrated with clear serous fluid, giving them a gelatinous appearance. Fairly often irregularly distributed petechiae may also be seen in the subcutaneous tissues.

The myocard is flabby and a variable number of petechiae appears on the epicardium and endocardium. Hydrothorax, hydropericardium and ascites may be present. Oedema of the glottis has also been observed. The lungs show a variable degree of oedema and are pink or yellowish pink in colour. The visceral and parietal pleura and peritoneum may be spotted with petechiae. The liver is increased in size, soft, friable and yellowish brown to lemon yellow in colour; parenchymatous degeneration is evident. The gall bladder is distended with dark green viscous bile. The spleen is markedly enlarged and the pulpa is soft. The Malpighian corpuscles are prominent. Rupture of the spleen has been observed by Baumann (1939). The lymphatic glands are swollen and often red in colour. The kidneys are congested, somewhat enlarged, and frequently show a variable number of "lymphomata" or "infarcts" similar to those seen in East Coast fever. The urinary bladder contains bile-stained urine. Haemoglobinuria is not constantly present. The mucous membrane of the urinary bladder may show petechiae. The blood vessels of the brain are moderately injected. The mucous membrane of the abomasum shows a variable number of petechiae. The intestinal mucosa, particularly that of the caecum and large intestine exhibits irregularly disseminated, red patches. The intestinal contents may be mixed with blood but are usually bile-stained. Intratesticular haemorrhages have also been encountered.

*B. Microscopical lesions.*

The blood shows degenerative and regenerative changes. Lestoquard (1926) states that in the peripheral circulation up to 95 per cent of the red blood cells are parasitized. Baumann (1939), on the other hand, only found 1 to 4 per cent of the erythrocytes of the peripheral blood infected, while the red blood cells in the capillaries of the liver, kidney and myocard practically all harboured parasites.

Baumann (1939) gives the following brief account of the histo-pathological lesions:—

The myocard shows albuminous degeneration. The Malpighian corpuscles of the spleen are markedly enlarged, the pulpa is congested and haemosiderin is present in the reticulum cells. The lymphatic glands show pronounced hyperaemia, the blood vessels are not only distended but are packed with erythrocytes; the lymphatic tissue is oedematous; the lymph sinusoids are dilated and contain desquamated endothelial cells, a few polymorphonuclear leucocytes and a variable number of red blood cells. Haemosiderin is present in the endothelial cells of the sinusoids. The liver shows albuminous degeneration. In cases that had developed haemoglobinaemia, the changes resemble those of equine infectious anaemia. Cellular infiltrations consisting of histiocytes and

lymphocytes are observed. The former cells as well as the endothelial cells of the central veins and the Kupffer cells contain haemosiderin. Abnormalities in the kidneys are not very pronounced except in sheep that have suffered from haemoglobinuria. The glomerular capsules and renal tubules are distended and contain haemoglobin cylinders. Interstitial cellular infiltration may be present in the form of linear lesions which are incorrectly termed "infarcts". The epithelium of the convoluted tubules shows karyolysis of the nuclei and a fairly extensive necrosis. In cases not associated with haemoglobinuria necrosis is not often seen and very little albumin is excreted into the lumen of the tubules.

### *Diagnosis.*

Malignant gonderiosis may be expected in any of the above-mentioned enzootic regions (Table XVI). Clinically it is often difficult to make a diagnosis. Not only are there several diseases (babesiosis, anaplasmosis, eperythrozoonosis, verminoses and intoxication following the ingestion of poisonous plants) which give rise to similar symptoms, but some of these may actually exist as complications.

It is essential that blood, lymphatic gland and spleen smears be examined before a diagnosis is made. The erythrocytic stages of the parasite are usually present in fairly large numbers in contra-distinction to *Gonderia ovis* infection where the parasitism of the erythrocytes seldom exceeds 2 per cent. Schizonts of *G. hirci* are readily demonstrable in organ smears, while those of *G. ovis* are very rarely seen, and when present appear in extremely small numbers.

A biological test on susceptible sheep and goats can also be resorted to for making a diagnosis. Blood or an emulsion prepared from the spleen, collected during the febrile reaction, should be used as inoculum. During the ensuing reaction blood smears should be carefully examined in order to exclude the presence of concurrent infections of *B. ovis* Starcovici, 1893, *B. motasi* Wenyon, 1926, *Anaplasma ovis* Lestoquard, 1924, *Eperythrozoon ovis* Neitz, Alexander and du Toit, 1934, and *Rickettsia ovina* Lestoquard and Donatien, 1936.

The carrier state of malignant gonderiosis may be determined by applying the xenodiagnosis. This would involve feeding larvae and nymphae of the alleged vector (*Rh. bursa*) on the suspected carrier, and feeding the adult stage on susceptible sheep or goats, preferably splenectomized animals. Careful examination of blood, lymphatic gland and spleen smears during the course of the ensuing reaction will determine whether the suspected carrier harboured *G. hirci* or *G. ovis*.

### *Treatment.*

#### *A. Specific treatment.*

No drug is known to have a specific action on *G. hirci*. The progress made in the chemotherapy of East Coast fever (Neitz, 1950, 1951, 1953), suggests that studies based on similar lines should be attempted in gonderiosis. This will entail determining whether the 8-amino-quinoline preparations (plasmoquine, pamaquin, and pentaquine) have a selective action on the erythrocytic stages of the parasite, and whether the antibiotics (aureomycin and terramycin) have a suppressive action on the schizonts when administered during the incubation period following a natural infection.

**B. Symptomatic treatment.**

Affected animals are frequently presented in the advanced stage of the disease. Good nursing is, therefore, essential. Special attention should be paid to the anaemia, and the atony of the digestive tract. The administration of saccharated iron compounds, liver preparations, Vitamin B complex compounds, and cardiac sustenants may be of value. For the prevention and treatment of the constipation the administration of 100 gm. of treacle or sugar, diluted in an adequate amount of water, together with 100 c.c. of vinegar may be beneficial. Treatment should be repeated until the evacuations are soft and regular.

From what has been said above in connection with the drop in the milk-yield of reacting animals, it follows that necessary attention must be paid to the nourishment of lambs and kids. Rams must be given a rest period of at least three months after recovery before they are used for service.

*Prognosis.*

The prognosis should always be guarded. Not only is the mortality very high but the financial losses experienced by sheep and goat breeders as the result of emaciation, poor wool-production and decreased milk-yield are also great. Several months may elapse before the animals become fit for slaughter. Milk-production may only become normal again after the next lambing or kidding.

*Prophylaxis.*

(a) *Elimination of arthropod vectors.*—The only effective weapon for the destruction of ticks is regular systematic dipping or spraying combined with careful hand-dressing. The dipping of sheep and goats (with the exception of woolled sheep and Angora goats) in arsenical dips (0·16 per cent  $As_2O_3$ ) at weekly intervals has been successfully employed in some parts of Africa. This system should, therefore, be of value for the control of ticks in other countries. Dipping fluids containing either D.D.T. in a concentration of 0·1 to 0·2 per cent para-para D.D.T. (Bekker and Graf, 1946), or the gamma isomere of benzene hexachloride (B.H.C.) in a concentration of 0·005 to 0·01 per cent plus 0·16 per cent  $As_2O_3$  (Bekker and Graf, 1946) are very effective.

Frequent dippings of woolled sheep are not conducive for the production of a high standard wool clip. For this reason the use of a shallow dipping tank or "walk-through" tank has been recommended by van Rensburg (1928). Ticks as a rule favour bare portions of the body, notably the feet and legs, the belly, perineum and axilla as sites for attachment. The shallow tank should contain sodium arsenite in combination with either D.D.T. or B.H.C. in the above-mentioned concentrations. The long duration of protection of sheep afforded by B.H.C. in the case of myiasis (du Toit and Fiedler, 1953) suggests that this acaricide should be used in preference to D.D.T. in the dipping fluid. If this process of tick control is employed, hand-dressing of ears, should not be neglected.

Sheep and goats recovered from malignant gonderiosis develop a premunity, and can thus play an important role as reservoirs for the infection of ticks. Despite systematic dipping, therefore, complete eradication of infected ticks cannot be expected.

(b) *Quarantine measures.*—The transmitters of malignant gonderiosis have not yet been established. However, it is known that the species of ticks occurring in the enzootic regions are also present beyond their boundaries. The introduction of animals from enzootic areas into countries free from infection and in which potential vectors occur, should be prohibited.

(c) *Immunization*.—This prophylactic measure is not employed in practice but will become possible when specific drugs become available. This would entail infecting animals with *G. hirci*-infective blood, and the treatment of the ensuing reaction. Whether or not *G. hirci* will lend itself to attenuation by serial passage as has been possible in the case of *Gonderia annulata* (Dschunkowsky and Luhs, 1904) by Cordier, Ménager and Delorme, 1936), needs to be determined.

#### *Immunity.*

Recovered sheep and goats develop a premunity which probably persists for several years. The occurrence of immunologically different strains has not been established. No mention is made in the literature whether splenectomy is followed by a relapse. There is no cross-immunity between *G. hirci* and *G. ovis*.

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## 2. GONDERIA OVIS INFECTION.

### *Definition.*

Benign ovine and caprine gonderiosis is a tickborne disease caused by *Gonderia ovis* (Rodhain, 1916). It is characterised by pyrexia, swelling of the superficial lymphatic glands and a mild anaemia. Recovered animals develop a premunity which persists for several years.

### *Synonyms.*

Ovine and caprine Theileriosis; Gonderiose of Theileriose van skape en bokke (Afrikaans); Gonderiose of Theileriose van schapen en geiten (Netherlands); Gonderiose oder Theileriose der Schafe und Ziegen (German); Gonderiose ou Theileriose du mouton et de la chèvre (French).

### *History.*

During the course of his investigations Rodhain (1916) encountered a small piroplasm in apparently healthy sheep in the Belgian Congo. He named this parasite *Theileria ovis*. Sergent, Parrot and Hilbert (1922) named this organism *Gonderia ovis*, but Lestoquard (1929) having described Koch bodies in such infections in sheep and goats renamed the protozoon *Theileria recondita*. In view of the fact that schizonts had not been described, Wenyon (1926) considered it justifiable to transfer this organism to the genus *Babesia*, and in doing so proposed a new name *Babesia sergenti*. Thomson and Hall (1933) reviewed the classification of the piroplasms, and arrived at the conclusion that the correct name for the non-pathogenic *Theileria* sp. of sheep and goats is *Theileria ovis* Rodhain, 1916. Neitz and Jansen (1956) revised the classification of the Theilerias. They differentiate between the genera *Theileria* and *Gonderia* on the basis that the former genus multiplies only by schizogony within the lymphocytes, while the latter reproduces by schizogony in the lymphocytes and by fission in the erythrocytes. Since the life-cycle of *Th. ovis* is typically that of a *Gonderia* sp. they renamed the protozoon *Gonderia ovis* (Rodhain, 1916).



*Distribution.*

The distribution of *G. ovis* is given below in Table XVII. (See also Map No. 6).

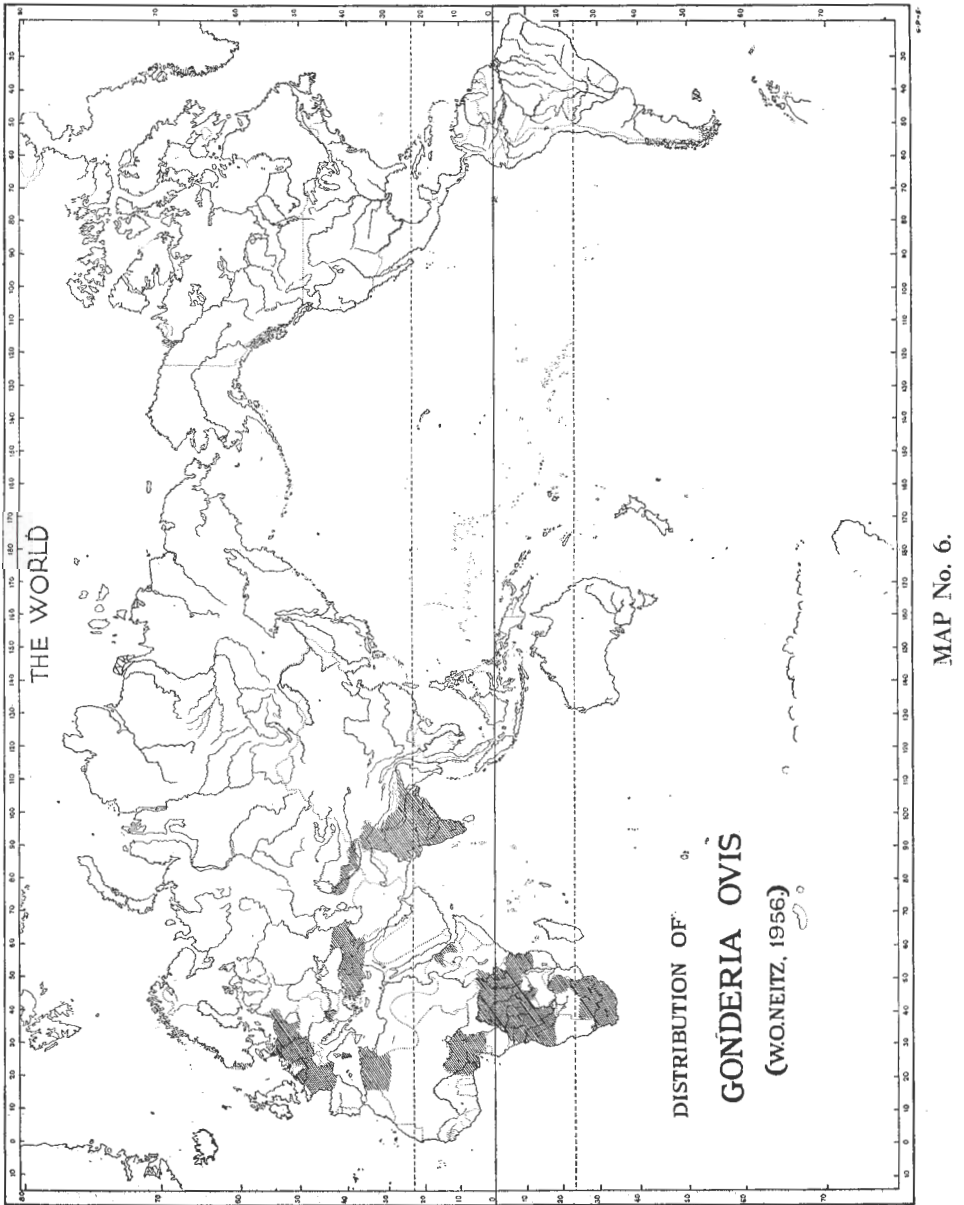


TABLE XVII.

Continent.	Country.	Animal.	References.
Africa.....	Algeria.....	Sheep..... Goat.....	Sergent, Parrot and Hilbert, 1922, 1923. Lestoquard, 1922.
	Angola.....	Goat.....	F. de Mello and Santa Paes, 1923. F. de Mello and Carbal, 1923.
	Belgian Congo...	Sheep.....	Rodhain, Pons, van den Branden and Bequaert, 1913; Rodhain, 1916.
	Cameroon.....	Goat.....	Ziemann, 1904.
	Eritrea.....	Sheep.....	Martoglio, Stella and Carpano, 1911.
	Nigeria.....	Sheep.....	MacFie, 1914, 1915.
	South Africa....	Sheep.....	De Kock and Quinlan, 1926; Jansen and Neitz, 1956.
	Southern Rhodesia	Sheep.....	Wenyon, 1915; Bevan, 1915.
	Tanganyika.....	Goat..... Sheep..... Sheep.....	Panse, 1908. Schellhase, 1913, 1914. Mettam, 1932.
Uganda.....	Sheep.....		
Asia.....	India.....	Goat.....	Lingard and Jennings, 1904.
	Turkestan.....	Sheep.....	Yakimoff, Schokhor, Koselkine and Paroisky, 1917.
	Turkey.....	Sheep.....	Lestoquard and Ekrem, 1931.
Europe.....	France.....	Sheep.....	Lestoquard and Savance, 1933.
	Germany.....	Sheep.....	Spiegel, 1927; Enigk, 1953.
	Hungary.....	Sheep.....	Ratz, 1912.
	North Caucasia...	Sheep.....	Yakimoff, 1929.
	Sardinia.....	Sheep.....	Cerruti, 1934.
	Transcaucasia....	Sheep.....	Rastegaieff, 1933.

*Aetiology.*

*Gonderia ovis* (Rodhain, 1916).

Synonyms: *Theileria ovis* Rodhain, 1916.

*Gonderia ovis* Sergent, Parrot and Hilbert, 1922.

*Theileria recondita* Lestoquard, 1929.

*Babesia sergenti* Wenyon, 1926.

(a) *Morphology.*—(i) Erythrocytic parasites:— Morphologically *G. ovis* is indistinguishable from *G. hirci*. In blood smears fixed with May-Grünwald and stained with Giemsa, *G. ovis* appears in the red blood cells as pear-shaped, comma-shaped, oval, round or anaplasma-like organisms. The pear-shaped forms are 0·5 micron in width and 1·5 microns in length; comma-shaped forms 0·3 to 0·5 microns in width and 1·8 microns in length; oval forms 0·5 microns in width and 1·0 microns in length; round forms 0·6 to 2·2 microns in diameter, and anaplasma-like forms 0·5 to 1·2 microns in diameter.

The cytoplasm stains light blue. The nucleus appears as a deeply-stained minute reddish purple granule situated at the wider end of the pear-shaped, comma-shaped and oval forms, and on the margin of the round parasites. When division takes place two, three or four chromatin granules are observed. In the anaplasma-like forms the cytoplasm can hardly be recognized. (See Fig. 5.)

(ii) *Histiotropic parasites*.—In lymphatic gland smears fixed with May-Grünwald and stained with Giemsa, the schizonts (Koch bodies) appear as masses of blue-staining cytoplasm containing one to forty reddish purple dots varying from 1·0 to 2·0 microns in size. The Koch bodies vary in size from 1·0 to 10·0 microns and in some cases they may be up to 20·0 microns in diameter. They are seen either free or within the lymphocytes. Schizonts commonly referred to as agamonts (macroschizonts) are the type commonly seen. The so-called gamonts (microschizonts) have apparently so far only been encountered by Lestoquard (1929) as shown in his illustrations. Although mature Koch bodies liberating merozoites have not yet been encountered, there is every reason to believe that this process also occurs in the development of *G. ovis*, otherwise the erythrocytes would not become parasitized. (See Fig 6.)

(b) *Multiplication*.—*G. ovis*, multiplies by schizogony. When schizonts are fully formed they break up into merozoites which either enter lymphocytes to grow and reproduce by schizogony again, or they penetrate the erythrocytes, in which they are seen in ordinary blood films. According to Sergent, Parrot and Hilbert (1922) and Lestoquard (1929) multiplication also occurs within the erythrocytes. Division into two, giving rise to two daughter cells (Lestoquard, 1929; Enigk, 1953), or alternatively into four takes place resulting in the cross forms, in which four minute pear-shaped individuals radiate from a central point.

(c) *Habitat*.—The erythrocytic stages of *G. ovis* can be fairly readily demonstrated in blood and organ smears. In non-splenectomized animals only up to two per cent of the red blood cells are parasitized (Lestoquard, 1926). In splenectomized sheep Enigk (1953) found up to 10 per cent of the erythrocytes infected, while Jansen and Neitz (1956) observed an infection not exceeding three per cent. The host cell may harbour 1 to 4 micro-organisms. Schizonts have only been demonstrated in smears prepared from lymphatic glands (Lestoquard, 1929; Jansen and Neitz, 1956). These were found after a prolonged examination.

(d) *Life-cycle*.—Although the salient features of the developmental cycle in the vertebrate host are known, no attempts have yet been made to study the development of the parasite in the vector.

(e) *Cultivation*.—No attempts have yet been made to cultivate this protozoon. It needs to be determined whether the schizonts of *G. ovis* can be grown in tissue culture according to the method evolved by Tchernomoretz (1945) for *G. annulata*,

(f) *Action of physical and chemical agents*.—The infectious agent remains potent in citrated blood for at least 24 hours at room temperature. Viability of the protozoon is lost within 12 hours when infective blood is collected in the O.C.G. solution.

(g) *Biological characteristics*.—Immunologically different strains of *G. ovis* have not been encountered. A variation in virulence between strains of different countries has not been observed.

### *Transmission.*

#### *A. Natural transmission.*

(a) *Biological transmission*.—Several species of ticks have been incriminated as vectors of *G. ovis* in Russia. Rastegaieff, (1933) working in Transcaucasia states that the two-host tick, *Rhipicephalus bursa* Canestrini and Fanzago, acquired the parasite in the larval and nymphal stages, and transmitted it in the adult stage. The incubation period was three days when the erythrocytic stages of

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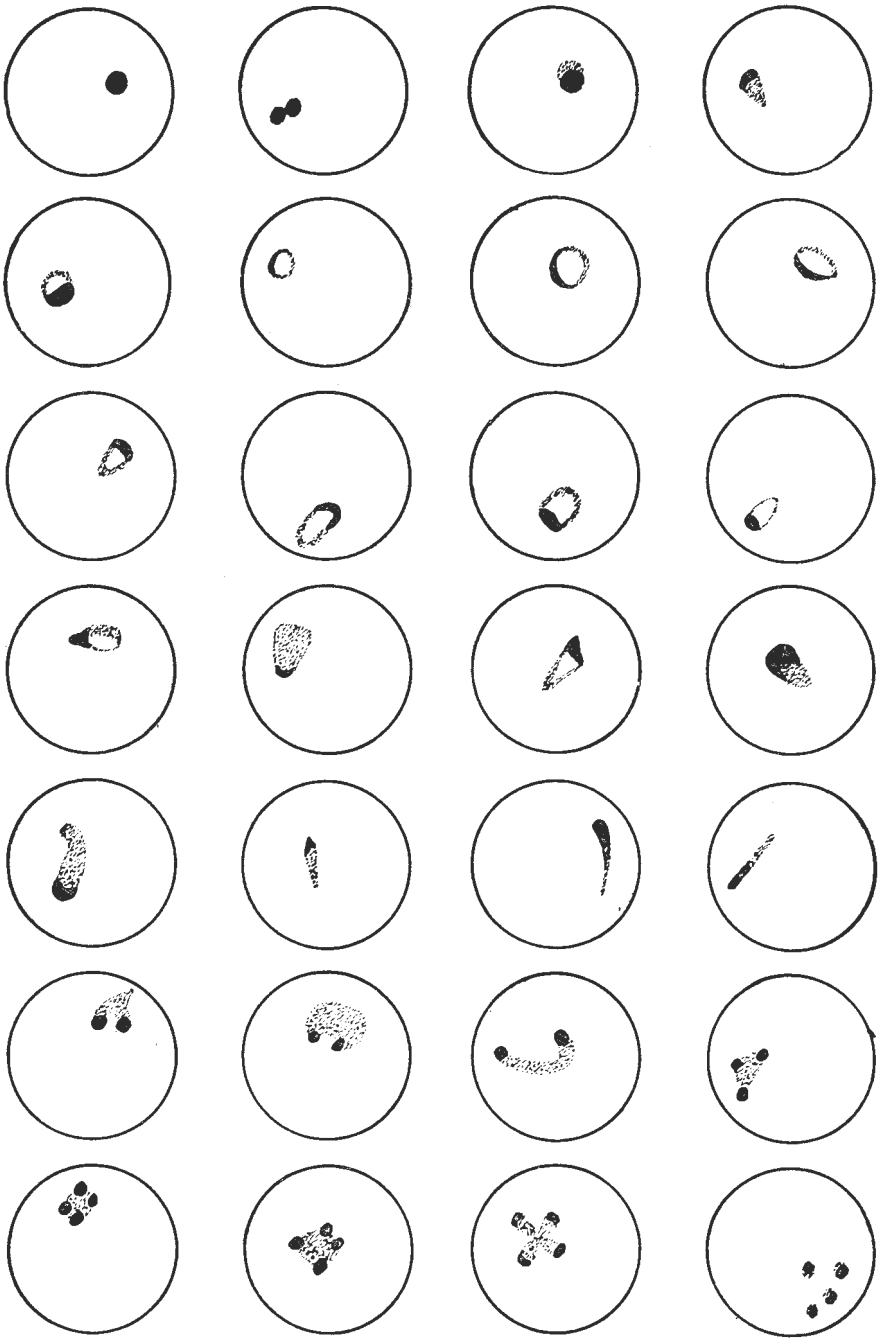


FIG. 5.—*Gonderia utis*.

*G. ovis* appeared in the peripheral blood. Rastegaieff (1935, 1936) also mentions that she successfully transmitted *G. ovis* to sheep and goats with the adult stage of *Ornithodoros lahorensis* Neum. The incubation period following the tick infestation was three days. In a subsequent report (Rastegaieff, 1937) claims to have transmitted the infection with the adults of *Dermacentor silvarum* Olenev. The incubation period was four days when the erythrocytic stages appeared in the peripheral circulation. At no time were Koch bodies detected in any of the experimental sheep and goats.

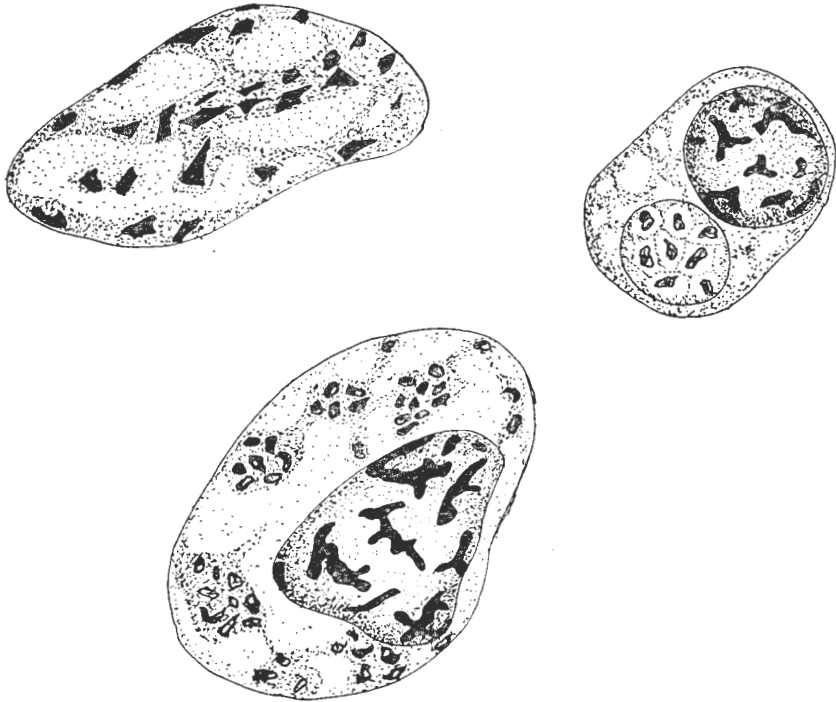


FIG. 6.—*Gonderia ovis*.

The extremely short duration of the incubation periods (3 to 4 days) obtained by Rastegaieff (*loc. cit.*) in her experiments casts some doubt whether the tick transmission of *G. ovis* was successful. This doubt is based on the fact that in the transmission of either *Theileria parva* or *Gonderia annulata* a period of about 3 days elapses after the attachment of ticks before sporozoites are liberated, and that under very favourable conditions at least another three days are required before schizonts can be demonstrated in the parotid lymphatic gland draining the site of tick infestation, namely the ear. Consideration of these facts suggests that Rastegaieff (*loc. cit.*) accidentally employed sheep and goats harbouring a latent infection of *G. ovis*.

Jansen an Neitz (1956) successfully transmitted *G. ovis* with the two-host tick, *Rhipicephalus evertsi* Neum. in South Africa. The parasite was acquired by the larval and nymphal stages, and transmitted by the ensuing adult females and/or males to six fully susceptible splenectomized sheep. Koch bodies were demonstrated in the swollen lymphatic glands 14 to 17 days after tick infestation,

while the erythrocytic stages appear in the peripheral circulations 3 to 9 days after the appearance of the schizonts. These observations are in accordance with the general scheme observed in the transmission of either *T. parva* or *G. annulata*.

(b) *Mechanical transmission*.—Blood-sucking insects have not been incriminated as vectors.

(c) *Intra-uterine transmission*.—This form of transmission has not yet been recorded in benign gonderiosis of sheep and goats.

#### B. *Artificial transmission*.

The erythrocytic stages of *G. ovis* can be readily transmitted by means of blood and organ emulsions by the intravenous and subcutaneous route. The appearance of schizonts in an artificially infected goat has so far been recorded only by Lestoquard (1929). A young goat employed by him received blood from a sheep which harboured schizonts and endoglobular parasites. The animal was thereupon splenectomized. After the operation a culture of Paratyphoid B was administered intravenously. Smear examination conducted subsequently revealed the presence of Koch bodies in the lymph glands and the erythrocytic stages of *G. ovis* in the peripheral blood.

#### *Pathogenicity*.

Benign gonderiosis is transmissible from sheep to goats and *vice versa* (Lestoquard, 1926, 1929). No mortality due to this infection has ever been recorded in these animals.

#### *Pathogenesis*.

The swelling of the lymphatic glands draining the site of tick infestation, the proliferation of lymphocytes in these glands, and the development of a mild anaemia as observed in splenectomized sheep by Jansen and Neitz (1956) suggest that *G. ovis* liberates a toxin.

#### *Symptomatology*.

Benign ovine and caprine gonderiosis has not yet been recognized under natural conditions. Mild clinical symptoms have up to the present only been observed in splenectomized Merino sheep by Jansen and Neitz (1956).

The incubation period following a natural infection varies from 9 to 13 days. After an artificial infection the erythrocytic stages of *G. ovis* appear in the peripheral circulation of splenectomized and non-splenectomized sheep 21 to 30 days later.

The first signs of the disease are a gradual or sudden temperature elevation (105° to 107° F) accompanied by a moderate swelling of the parotid lymphatic glands. (The ears were used as the site for the tick infestation). The fever is either continuous and persists for 4 to 6 days or irregularly intermittent lasting 16 days. The swelling of the lymphatic glands persists for a week. Approximately 14 days after the commencement of fever a mild anaemia may be observed clinically. It persists for one to five weeks. The erythrocyte count may drop to as low as 3-4 million per c.mm. Sheep make a rapid recovery.

#### *Pathology*.

*Macroscopical lesions*.—A moderate swelling of the superficial lymphatic glands and an anaemia are apparently the only lesions that can be expected at autopsy.

*Microscopical lesions.*—Degenerative and regenerative anaemic changes, namely anisocytosis, punctate basophilia, polychromasia and Jolly bodies are observed in blood smears. A proliferation of the lymphocytes is observed in smears prepared from the swollen lymphatic glands. During the course of the febrile reaction Koch bodies are usually demonstrable in these smears after a prolonged examination.

#### *Diagnosis.*

Benign gonderiosis may be expected in any of the above-mentioned enzootic regions (Table XVII). The erythrocytic stages of *G. ovis* have frequently been encountered in blood smears, while schizonts in lymphatic gland smears have so far only been demonstrated in experimental animals after a prolonged search. In *G. ovis* enzootic areas, a diagnosis of the carrier state of benign gonderiosis is based upon the demonstration of the erythrocytic parasites. Difficulty in identifying the endoglobular parasites is experienced in countries where *G. ovis* and *G. hirci* occur enzootically. They are morphologically indistinguishable. The only reliable procedure in these circumstances would be the application of the xenodiagnosis and/or cross-immunity tests. Before these methods can be applied the vectors of the two parasites will have to be determined in the areas in which they occur enzootically.

#### *Treatment.*

Treatment is not necessary. It has been determined by the writer that the 8-aminoquinoline preparation, Pamaquin has a specific action on the erythrocytic stages of *G. ovis*. When three successive doses of 0.5 mg. per Kg. body weight are administered intravenously at 48 hourly intervals, the parasites disappear temporarily for a period of three weeks.

#### *Prognosis.*

The prognosis is always favourable.

#### *Prophylaxis.*

(a) *Elimination of arthropod vectors.*—This prophylactic measure is only necessary when experiments have to be conducted on benign gonderiosis. After shearing, pregnant ewes should be dipped. The dipping fluid should contain D.D.T. in a concentration of 0.1 to 0.2 per cent para-para D.D.T. plus 0.16 per cent  $As_2O_3$  (as sodium arsenite). After dipping the ewes should be transferred to a tick-free stable. Hay for the animals must be autoclaved to render it free from ticks. Before employing lambs, born under these conditions, for experiments it may even be necessary to splenectomize them, and to examine blood smears for a period of at least four weeks in order to exclude the possibility of an accidental intra-uterine transmission.

(b) *Quarantine measures and immunization.*—The benign nature of the disease caused by *G. ovis* does not warrant the application of these prophylactic measures.

#### *Immunity.*

Recovered sheep and goats develop a premunity. The erythrocytic stages of *G. ovis* are retained by animals for life. It needs to be determined whether schizonts are retained for the same period. Splenectomy of carriers is invariably followed by a relapse. The erythrocytic stages of the parasite appear in the peripheral circulation within three weeks after the operation (De Kock and Quinlan, 1926; Jansen and Neitz, 1956). The intravenous administration of Paratyphoid B cultures into splenectomized carriers activates the Koch bodies (Lestoquard, 1929).

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## THEILERIOSIS, GONDERIOSES AND CYTAUXZONOSIS IN WILD ANIMALS.

The susceptibility of the African buffalo to *Th. parva*, *G. mutans* and *G. lawrencei*, and that of the American bison to *G. annulata* has already been dealt with (*vide supra*). It is beyond the scope of this paper to consider the 46 *Theileria* spp. and the one *Gonderia* sp. described in the remaining wild animals in detail. These protozoa, their mammalian hosts, and their occurrence in either Africa, Asia, Australia, Europe or South America have been listed in the appended Table XVIII. In the majority of cases only the erythrocytic parasites were encountered. However, in the bushbuck, Bright's gazelle, Coke's hartebeest, eland, steenbuck, topi and the Australian spiny anteater the erythrocytic parasites as well as schizonts were encountered. Although the parasites were provisionally placed in either the genus *Theileria* or *Gonderia*, it is believed from the description of *Cytauxzoon sylvicaprae* Neitz and Thomas, 1948, a parasite of the duiker, which multiplies by schizogony in the histiocytes and by fission in the erythrocytes, that some of these protozoa may in reality be *Cytauxzoon* spp. Their generic status and host-relationship can only be established if the vertebrate life-cycle is studied. This will involve determining the arthropod vectors, and transmitting the infection to fully susceptible animals.

Observations in East Africa have shown that the *Theileria* sp. of the eland is pathogenic. Either sick or dead animals were encountered in the field. They were emaciated, and showed a tumor hepatitis, "infarcts" in the kidneys and diarrhoea.

During the course of zoological surveys made in the Union of South Africa it was found that two *Cytauxzoon* spp. are pathogenic for the duiker and the kudu. A brief description of the observations made are given below.

## 1. SYLVICAPRINE CYTAUXZONOSIS.

## CYTAUXZON SYLVICAPRAE INFECTION.

*Definition.*

Sylvicaprine cytauxzoonosis is an acute, in all probability an arthropod-borne disease, caused by *Cytauxzoon sylvicaprae* Neitz and Thomas, 1948. It is characterised by pyrexia, anorexia, listlessness, general weakness, staggering gait, loss in condition and coma before death.

*Synonyms.*

Cytauxzoonose van duikers (Afrikaans); Cytauxzoonose der Ducker (German); Cytauxzoonose a *Cytauxzoon sylvicaprae* (French).

*History.*

In March, 1943, a two-year-old duiker, which had been born and bred in captivity at the foot of the Magaliesberg near Pretoria, was brought to Onderstepoort for treatment. The animal had been sick for a fortnight. A few days before it was admitted it became very weak, and arrived in a semi-comatose state. It died 18 hours later. Microscopic examination of blood and organ smears revealed the presence of erythrocytic parasites resembling *G. mutans*, and a large number of schizonts in uni- and multinucleated histiocytes. This protozoon was named *Cytauxzoon sylvicaprae* by Neitz and Thomas (1948). A second case of cytauxzoonosis was encountered on the Roodekuil Estate near Warmbad in the Northern Transvaal. The duiker was found roaming aimlessly in the veld. It was caught and brought to Onderstepoort. It died on the way.

TABLE XVIII.  
The Occurrence of *Theileria* spp., *Gonderia* spp. and *Cytauxzoon* spp. in Wild Animals.

PARASITE.		HOST.			References.	
Genus and Species.	Order and Family.	Zoological Name.	Vernacular Name.	History of Host.	Country.	References.
<i>Theileria celli</i> (Castellani and Chalmers, 1910). = <i>Babesia celli</i> Castellani and Chalmers, 1908	Primates Cercopitheciidae....	<i>Macaca sinica</i> (L.). = <i>Macacus pileatus</i> (L.).	Bonnet Monkey....	Healthy.....	Ceylon.....	Castellani and Chalmers, 1908, 1910.
<i>Theileria</i> sp.....	Tubulidentata Orycteropidae....	<i>Orycteropus afer</i> (Pall.).....	South African Ant-bear	Healthy.....	Zabaland.....	Neitz, 1931.
<i>Theileria rossica</i> Yakimoff and Saphronowitsch, 1917	Rodentia Muridae.....	?	Field Mouse.....	Healthy.....	Russia.....	Yakimoff and Saphronowitsch, 1917.
<i>Gonderia tachyglossi</i> (Priestly, 1915). = <i>Theileria tachyglossi</i> Priestly, 1915	Monotremata.....	<i>Tachygllossus aculeatus</i> (Shaw).....	Echidna Australian Spiny Ant-eater	Healthy and killed. Infested with <i>Apoanoma decrosum</i> (L. Koch)	Australia.....	Priestly, 1915.
<i>Theileria brimmiti</i> Leger and Mouzels, 1917. = <i>Gonderia brimmiti</i> (Leger and Mouzels, 1917).	Xenarthra Bragiopidae....	<i>Brachypus tridactylus</i> L.....	Three-toed Sloth....	Healthy.....	French Guiana.....	Leger and Mouzels, 1917.
<i>Nuttallia brasiliensis</i> (Regendanz and Kikuth, 1928). Reichenow (1953) believes that it is in reality a <i>Theileria</i> sp.	Marsupialia Didelphidae....	<i>Metachirus opossum</i> (L.). = <i>Metachirus quica</i> Temminck	Quica Opossum....	Healthy and splenectomized	South America (Petropolis)	Regendanz and Kikuth, 1928.
<i>Theileria</i> sp.....	Artiodactyla Cervidae.....	<i>Axis axis</i> (Erxleben). = <i>Cervus axis</i> Erxleben	Spotted Deer....	Healthy.....	India.....	Patton, 1910.
<i>Theileria daniae</i> (Bettencourt, and Franca and Borges, 1907). = <i>Theileria cervi</i> Bettencourt, Franca and Borges, 1907. = <i>Gonderia daniae</i> Bettencourt, Franca and Borges, 1907. = <i>Babesia daniae</i> (Bettencourt, Franca and Borges, 1907)	Artiodactyla Cervidae.....	<i>Dama dama</i> (L.). = <i>Cervus dama</i> L.	Fallow Deer....	Healthy.....	Portugal.....	Bettencourt, Franca and Borges, 1907.
<i>Theileria</i> sp.....	Artiodactyla Cervidae.....	<i>Muntiacus muntjak</i> Zimmermann. = <i>Cervulus muntjak</i> Brooke	Barking Deer....	Healthy.....	Annam Indo-China.....	Schein, 1923.
<i>Theileria tarandi rangiferis</i> (Kerzelli, 1909). = <i>Gonderia tarandi rangiferis</i> (Kerzelli, 1909). = <i>Protoplasma tarandi rangiferis</i> Kerzelli, 1909.	Artiodactyla Cervidae.....	<i>Rangifer tarandus</i> (L.).....	Reindeer....	Healthy. As many as 30 per cent harboured the parasite	Russia Archangelck.....	Kerzelli, 1909. Yakimoff and Mitzke-witsch, 1930.
<i>Theileria aristotelis</i> (Denier, 1907). = <i>Gonderia aristotelis</i> (Denier, 1907). = <i>Nuttallia aristotelis</i> (Denier, 1907).	Artiodactyla Cervidae.....	<i>Rusa unicolor</i> (Kerr). = <i>Cervus aristotelis</i> Cuvier	Sambar Deer....	Healthy.....	Annam Indo-China.....	Denier, 1907.
<i>Theileria</i> sp.....	Artiodactyla Cervidae.....	<i>Sika hortulorum</i> (Swinhoe). = <i>Cervus hortulorum</i> Swinhoe. = <i>Pseudaxis hortulorum</i> Swinhoe.	Pekin Deer. Dydowski's Deer	Healthy.....	East Siberia Ussuri Region	Springholz-Schmidt, 1937.

E. stages signify Erythrocytic Stages.

TABLE XVIII (continued)  
The Occurrence of *Theileria* spp., *Gondardia* spp. and *Cytauxzoon* spp. in Wild Animals (continued.)

PARASITE.		Host.				History of Host.	Country.	References.
Genus and Species.	Stages of life-cycle seen.	Order and Family.	Zoological Name.	Vernacular Name.				
<i>Theileria</i> sp.	E. stages.	Artiodactyla Cervidae.	<i>Sika nippon</i> (Temminck) = <i>Cervus sika</i> Temminck	Japanese Deer.	Healthy.	Japan.	Kondo, 1923.	
<i>Theileria</i> sp.	E. stages.	Suidae.	<i>Phacochoerus aethiopicus</i> Pallas.	Warthog.	Healthy.	Zululand.	Neitz, 1933.	
<i>Theileria camelensis</i> Yakimoff and Schokhor, 1917. = <i>Gondardia camelensis</i> (Yakimoff and Schokhor, 1917)	E. stages.	Camelidae.	<i>Camelus bactrianus</i> L.	Camel.	Healthy.	Russia, Turkestan. Abyssinia.	Yakimoff and Schokhor, 1917. Fruilent, 1938.	
<i>Theileria</i> sp.	E. stages.	Giraffidae.	<i>Okapia johnstoni</i> Sclater.	Okapi.	Healthy.	Belgian Congo.	Rodhain, 1936.	
<i>Theileria</i> sp.	E. stages.	Bovidae.	<i>Aepyceros melampus</i> (Lichtenstein).	Impala.	Healthy.	Kenya.	Ross, P. H., 1911.	
<i>Theileria</i> sp.	E. stages and Koch bodies.	Artiodactyla Bovidae.	<i>Aelaphus laelaphus</i> Gokiri (Günther) = <i>Bubalus caber</i> Günther	Coke's Hartbeest.	Healthy.	Kenya.	Ross, P. H., 1911.	
<i>Theileria</i> sp.	E. stages.	Artiodactyla Bovidae.	<i>Aelaphus baselaphus matschiet</i> (Schwarz)	Hartebeest.	Healthy.	Gold Coast.	Maefle, 1916. Simpson, 1918.	
<i>Gondardia annulata</i> (Dschunkowsky and Luhs, 1904). = <i>Theileria annulata</i> Dschunkowsky and Luhs, 1904	E. stages and Koch bodies.	Artiodactyla Bovidae.	<i>Bison bison</i> L.	American Bison.	Typical symptoms of Mediterranean Coast fever with fatal termination	Egypt, Cairo.	Carpano, 1937.	
<i>Theileria</i> sp.	E. stages.	Artiodactyla Bovidae.	<i>Cephalophus leucogaster</i> Gray.	Gaboon Duiker.	Healthy. Two animals harboured the parasite	Belgian Congo.	Schwetz, 1935.	
<i>Theileria</i> sp.	E. stages.	Artiodactyla Bovidae.	<i>Cephalophus aquatorietis melanotarsus</i> Gray	Duiker.	Healthy. 29 animals harboured the parasite	Belgian Congo.	Schwetz, 1935.	
<i>Theileria</i> sp.	E. stages.	Artiodactyla Bovidae.	<i>Cephalophus maxwelli</i> (H. Smith).	Maxwell's Duiker.	Healthy.	French West Africa.	Cursson, 1941.	
<i>Theileria</i> sp.	E. stages.	Artiodactyla Bovidae.	<i>Cephalophus nigrifrons</i> Gray.	Black-fronted Duiker.	Healthy.	Belgian Congo.	Schwetz, 1935.	
<i>Theileria</i> sp.	E. stages.	Artiodactyla Bovidae.	<i>Cephalophus rufilatus</i> Gray.	Red Flanked Duiker.	Healthy.	Gold Coast.	Simpson, 1918.	
<i>Theileria</i> sp.	E. stages.	Artiodactyla Bovidae.	<i>Cephalophus weynsi</i> Thomas.	Weyn's Duiker.	Healthy. Light animals harboured the parasite	Belgian Congo.	Schwetz, 1935.	
<i>Theileria</i> sp.	E. stages and Koch bodies.	Artiodactyla Bovidae.	<i>Domaliscus corriganii finela</i> (Matschie)	Tepi.	Healthy.	Uganda.	Hutchins, 1924. Montgomery, 1913.	
<i>Theileria stardi</i> Franca, 1913.	E. stages. Koch bodies described in 1917	Artiodactyla Bovidae.	<i>Gazella granti brighti</i> Thomas.	Bright's Gazelle.	Healthy.	Abyssinia.	Sturdy, 1912. Montgomery, 1913. Sturdy, 1917.	
<i>Theileria stardi</i> Franca, 1913.	E. stages.	Artiodactyla Bovidae.	<i>Gazella granti granti</i> Brooke.	Grant's Gazelle.	Healthy.	Abyssinia.	Sturdy, 1912. Franca, 1913. Montgomery, 1913. Carpano, 1913.	
<i>Theileria</i> sp.	E. stages.	Artiodactyla Bovidae.	<i>Gorgon taurinus taurinus</i> (Burch.)	Blue Wildebeest.	Healthy. Five animals harboured the parasite	Zululand.	Neitz, 1931.	
<i>Theileria</i> sp.	E. stages.	Artiodactyla Bovidae.	<i>Hippotragus equinus</i> (Desmarest).	Rouin Antelope.	Healthy.	Gambia.	Todd and Wolbach, 1912.	
<i>Theileria</i> sp.	E. stages.	Artiodactyla Bovidae.	<i>Hippotragus niger niger</i> (Harris).	Transvaal Antelope.	Healthy.	Transvaal.	Neitz, 1940.	

TABLE XVIII (continued).  
The Occurrence of *Theileria* spp., *Gonderia* spp. and *Cytauxzoon* spp. in Wild Animals (continued).

PARASITE.		HOST.				History of Host.	Country.	References.
Genus and Species.	Stages of life-cycle seen.	Order and Family.	Zoological Name.	Vernacular Name.				
<i>Theileria</i> sp.	E. stages.	Artiodactyla Bovidae.	<i>Kobus defassa albertensis</i> Matschie.	Waterbuck.	Healthy.	Uganda.	Mettam, 1932.	
<i>Theileria</i> sp.	E. stages.	Artiodactyla Bovidae.	<i>Kobus defassa</i> Rüppel, Schwetz and Collart (1929) incorrectly referred to this animal as <i>Cobus ellipsiprymnus</i> Ogilby. (Schwetz, 1935)	Waterbuck.	Healthy. Eight animals harboured the parasite.	Belgian Congo.	Rodhain, 1916. Schwetz and Collart, 1929.	
<i>Theileria</i> sp.	E. stages.	Artiodactyla Bovidae.	<i>Kobus ellipsiprymnus ellipsiprymnus</i> (Ogilby)	Waterbuck.	Healthy. 18 animals harboured the parasite.	Zululand.	Neitz, 1931; 1933.	
<i>Theileria</i> sp.	E. stages.	Artiodactyla Bovidae.	<i>Oryx beisa</i> (Rüppel).	Beisa.	Healthy.	Kenya.	Ross, P. H., 1911.	
<i>Theileria</i> sp.	E. stages.	Artiodactyla Bovidae.	<i>Oryx capensis</i> Thomas.	Fring-necked Oryx.	Healthy.	Kenya.	Ross, P. H., 1911.	
<i>Theileria</i> sp.	E. stages.	Artiodactyla Bovidae.	<i>Ourebia aurea</i> (Zimmermann).	Oribi.	Healthy.	Gold Coast.	Macfie, 1916.	
<i>Theileria</i> sp.	E. stages.	Artiodactyla Bovidae.	<i>Ourebia ourebi hastata</i> (Peters) = <i>Ourebia hastata</i> Peters	Oribi.	Healthy.	Belgian Congo.	Schwetz and Collart, 1929.	
<i>Theileria</i> sp.	E. stages Koch bodies seen in 1933	Artiodactyla Bovidae.	<i>Raphicerus campestris</i> (Thunb.).	Steenbuck.	Healthy.	Zululand.	Neitz, 1931. Kluge, 1953.	
<i>Theileria</i> sp.	E. stages.	Artiodactyla Bovidae.	<i>Redunca arundinum arundinum</i> (Bodd)	Reedbuck.	Healthy. 15 animals harboured the parasite.	Zululand.	Neitz, 1931.	
<i>Theileria</i> sp.	E. stages.	Artiodactyla Bovidae.	<i>Redunca fulvivulva</i> (Alz.).	Mountain Reedbuck	Healthy. Four animals harboured the parasite.	Zululand.	Neitz, 1931.	
<i>Cytauxzoon streptocerosi</i> Neitz and de Lange, 1956	E. stages and Koch bodies in histiocytes	Artiodactyla Bovidae.	<i>Streptoceros streptoceros streptoceros</i> (Pallas)	Kudu.	Sick, fever, anaemia and death.	Transvaal District	Neitz and de Lange, 1956.	
<i>Theileria</i> sp.	E. stages.	Artiodactyla Bovidae.	<i>Streptoceros streptoceros streptoceros</i> (Pallas)	Kudu.	Healthy. Five animals harboured the parasite.	Zululand.	Neitz, 1933. Neitz, 1952.	
<i>Cytauxzoon sylvicaprae</i> Neitz and Thomas, 1948	E. stages and Koch bodies in histiocytes	Artiodactyla Bovidae.	<i>Sylvicapra grimmia</i> (L.).	Duiker.	Sick, emaciated and died. Sick, staggering gait and died.	Transvaal District	Neitz and Thomas, 1948.	
<i>Theileria</i> sp.	E. stages.	Artiodactyla Bovidae.	<i>Sylvicapra grimmia</i> (L.) = <i>Sylvicapra grimmia grimmia</i> L.	Duiker.	Healthy. 33 animals harboured the parasite.	Zululand.	Neitz, 1956. Neitz, 1931; 1933.	
<i>Theileria</i> sp.	E. stages.	Artiodactyla Bovidae.	<i>Cephalophus grimmii</i> (L.). In all probability <i>Sylvicapra grimmia</i> (L.)	Duiker.	Healthy.	Angola.	Bettencourt, Franca and Borges, 1909. Bruce <i>et al.</i> , 1915.	
<i>Theileria</i> sp.	Koch bodies in spleen.	Artiodactyla Bovidae.	<i>Syncerus caffer</i> Sparrman.	African Buffalo.	Apparently healthy.	South Africa.	Theiler, 1924. Daubney, 1939.	
<i>Gonderia murans</i> (Theiler, 1906). = <i>Theileria mutans</i> (Theiler, 1906)	E. stages.	Artiodactyla Bovidae.	<i>Syncerus caffer</i> Sparrman.	African Buffalo.	Artificially infected. No reaction.	Kenya. South Africa.	Walker, 1932. Neitz, 1940.	

TABLE XVIII (continued).  
The Occurrence of *Theileria* spp., *Gondardia* spp. and *Cytosuxoon* spp. in Wild Animals (continued).

PARASITE.		HOST.			History of Host.	Country.	References.
Genus and Species.	Stages of life-cycle seen.	Order and Family.	Zoological Name.	Vernacular Name.			
<i>Theileria parva</i> (Theiler, 1904).....	E. stages.....	Artiodactyla Bovidae.....	<i>Syncerus caffer</i> Sparman.....	African Buffalo.....	Transmitted with infected ticks. Mild reaction.	Kenya.....	Lewis, 1943.
<i>Gondardia lawrencei</i> (Neitz, 1955). <i>Theileria lawrencei</i> Neitz, 1955	E. stages and Koch bodies	Artiodactyla Bovidae.....	<i>Syncerus caffer</i> Sparman.....	African Buffalo.....	Transmitted with infected ticks. Mild reaction. Two fatal cases in buffalo calves	Onderstepoort.....	Neitz, 1955, 1956. Kluge and Neitz, 1956.
<i>Theileria</i> sp.....	Only Koch bodies seen in kidney infarcts Koch bodies and E. stages in blood. Koch bodies in spleen and lymph glands	Artiodactyla Bovidae.....	<i>Immotragus</i> sp.....	Eland.....	Sick animal shot. Autopsy showed tumor hepatis and infarcts in kidney. Animal emaciated, sunken eyes, and marked diarrhoea. Dead eland	Tanganyika.....	Lichtenheld, 1941. Traurmann, 1941.
<i>Theileria</i> sp.....	E. stages.....	Artiodactyla Bovidae.....	<i>Taogelaphus scriptus</i> Sparman.....	Bushbuck.....	Healthy.....	Kenya..... French West Africa.....	Ross, P. H., 1941. Curasson, 1941.
<i>Theileria tragelaphi</i> Neitz, 1931.....	E. stages and Koch bodies in spleen and lymph glands	Artiodactyla Bovidae.....	<i>Taogelaphus scriptus</i> Sparman.....	Bushbuck.....	Healthy. 37 animals harboured E. stages, and of these two harboured Koch bodies	Transvaal..... Zululand.....	Theiler, 1931 Neitz, 1931, 1933.

*Distribution.*

Cytauxzoonosis has so far been diagnosed in duikers in the Pretoria and Waterberg districts of the Transvaal. Erythrocytic parasites indistinguishable from those of *C. sylvicaprae* have been encountered in duikers by Borges (1909) in Angola, by Bruce (1915) in Nyasaland and by Neitz (1931, 1933) in Zululand. It is thus possible that the distribution of sylvicaprine cytauxzoonosis is much wider than is known at present.

*Aetiology.*

*Cytauxzoon sylvicaprae* Neitz and Thomas, 1948.

(a) *Morphology.*—(i) Erythrocytic parasites:— This stage has so far been encountered in two duikers. Morphologically the parasites resemble *G. mutans*. In blood smears fixed with May-Grünwald and stained with Giemsa, *C. sylvicaprae* appears in the red blood cells as oval, comma-shaped or round organisms. The oval forms are 0·8 to 1·0 microns in width and 1·5 to 2·0 microns in length; comma-shaped forms 0·5 to 0·8 micron broad and 2·0 to 2·5 microns long, and the round forms a diameter of 2·0 to 2·5 microns.

The cytoplasm stains light blue. The nucleus appears as a deeply-stained, minute, reddish purple granule situated at the wider end of the oval or comma-shaped forms, and on the margin of the round parasites. When reproduction takes place the nucleus divides into two and finally into four granules.

(ii) Histiotropic parasites:— In organ smears fixed with May-Grünwald and stained with Giemsa, the extracellular schizonts appear as masses of blue cytoplasm containing a varying number of red-staining chromatin granules. Two types of plasma bodies can be distinguished. The immature schizonts are usually round, and have a diameter of 5·0 to 25·0 microns with an average of 10·0 microns. The smallest schizont is a round body of about 5·0 microns in diameter and contains 1 to 5 nuclei. The schizont increases in size while the nuclei multiply by repeated division until 20 to 30 and sometimes even more are present. The chromatin granules appear as irregularly shaped bodies varying from 1·0 to 3·0 microns in size. The granules divide giving rise to as many as 100 and more small (0·5 to 0·75 micron) circular bodies which are evenly distributed in the cytoplasm. The plasma bodies harbouring these small nuclei are the mature schizonts, and in them one may notice that the cytoplasm arranges itself around the nuclei, and finally there are budded off a number of merozoites leaving a mass of residual cytoplasm. The fully mature schizont ruptures and liberates merozoites. The round forms vary from 0·9 to 2·5 microns in diameter, while the oval forms are 0·6 to 1·0 micron broad and 1·6 to 2·5 microns long.

The intracellular schizonts are similar to the extracellular forms. Their situation in the cytoplasm of the host cell, however, partially protects them against the mechanical pressure in the process of making the smear with the result that they are smaller. The size varies from 5·0 to 15·0 microns in diameter with an average of 10·0 microns.

(b) *Multiplication.*—*C. sylvicaprae* multiplies by schizogony. When schizonts are fully formed they break up into merozoites which either remain in the host cell or enter other histiocytes to grow and reproduce by schizogony again, or they penetrate the erythrocytes in which they are seen in ordinary blood films. Multiplication also occurs within the erythrocytes. Division into four takes place resulting in cross-forms, in which four minute pearshaped individuals radiate from a central point.

(c) *Habitat*.—Approximately five per cent of the erythrocytes are parasitized. The host cell may harbour one to four parasites. Schizonts parasitize histiocytes which are either uni- or multi-nucleated. Parasitized cells in various stages of development are seen in smears prepared from the spleen, liver, lungs, kidneys and adrenals. The uni-nucleated histiocyte may harbour as many as five schizonts. Some very young schizonts only harbouring one nucleus may be observed in this type of host cell. An infected multi-nucleated cell with eight nuclei can harbour 50 and one with 17 nuclei as many as 200 schizonts. Uni-nucleated parasitized histiocytes vary from 15·0 to 20·0 microns in diameter. Multi-nucleated host cells with a round shape vary from 25·0 to 40·0 microns in diameter, while oval forms vary from 15·0 to 30·0 microns in width and 20·0 to 40·0 microns in length. Parasitized aggregations are very large varying from 50·0 to 100·0 microns in width and from 60·0 to 600·0 microns in length. The latter may harbour as many as 500 schizonts. Undisturbed round multi-nucleated host cells of 80·0 microns in diameter can harbour several thousand merozoites and several residual bodies. In these circumstances the nuclei of the host cell show a variable degree of degeneration.

(d) *Life-cycle*.—In the vertebrate host *C. sylvicaprae* multiplies by schizogony in the histiocytes. The forms in the erythrocytes reproduce by division into four daughter individuals. The final stage of the parasite is possibly a gametocyte or a gamete.

#### *Transmission.*

##### A. *Natural transmission.*

(a) *Biological transmission.* It is believed that transmission is brought about by an arthropod, probably a tick.

##### B. *Artificial transmission.*

As no susceptible duikers were available no attempts were made to establish whether or not this form of transmission is possible.

#### *Epizootology.*

Up to the present only two sporadic cases of sylvicaprine cytauxzoonosis with a fatal termination have been encountered in South Africa. The one occurred in a duiker kept in captivity and the second in an animal in nature. To what extent this infection occurs in duikers is as yet unknown. Erythrocytic parasites resembling *C. sylvicaprae* have been found in duikers belonging to the genera *Sylvicapra* and *Cephalophus* (Table XVIII) in South, Central and West Africa so that this disease may be more prevalent than what is known. Its apparently very rare occurrence may possibly be attributed to the fact that affected animals readily become the prey of jackals and other carnivores, and thus its normal incidence remains obscure.

#### *Pathogenicity.*

So far only the duiker (*Sylvicapra grimmia*) has been found to be susceptible. Whether or not other duikers belonging to the genera *Cephalophus*, *Guevei* and *Sylvicapra* are susceptible needs to be determined.

Sheep proved to be refractory to the intravenous injection of blood from affected duikers.



*Pathogenesis.*

The pathogenesis of this disease is unique in that there is no reference in the literature to any extensive and generalized endarteritis and endophlebitis with such a wide-spread interference of the blood flow caused by a protozoon parasite as in the case described by Neitz and Thomas (1948). This phenomenon was not evident in the second affected duiker encountered eleven years later.

*Symptomatology.*

The duration of the incubation period following either a natural or an artificial infection is unknown. In the duiker which had been maintained in captivity the course of the disease was approximately 15 days.

Information on the symptoms of the disease is limited to the observations made on two affected duikers. Sick animals show fever, inappetence, listlessness, general weakness, loss in condition, staggering gait, a variable degree of congestion of the visible mucous membranes, slow and laboured breathing and a slow and weak pulse. The eyelids and eyes show a peculiar trembling and twitching often associated with heartwater. Before death animals become very weak, lie in a recumbent position, and are disinclined to rise. The pulse is hardly perceptible, the temperature becomes subnormal, and death due to exhaustion supervenes.

*Pathology.*

The lesions of sylvicaprine cytauxzoonosis vary according to the degree of endarteritis and endophlebitis present. The congestion of the organs was a prominent feature in the first affected duiker which showed extensive lesions in the circulatory system (Neitz and Thomas, 1948) and less pronounced in the second animal in which the arteries and veins appeared not to be affected (Schulz, 1957).

*A. Macroscopical lesions.*

The first duiker showed oedema of the lungs. The liver was enlarged, greyish-yellow in colour with deep red mottling in the substance and under the capsule; the bile ducts were patent and apparently normal (duikers have no gall bladder). The spleen was enlarged, the edges rounded, the capsule tense, the pulpa soft and dark bluish-black in colour; the Malpighian corpuscles enlarged. The kidneys had a purplish colour throughout, and the zones were very indistinct. The wall of the abomasum including the mucous membrane was swollen and dark red in colour. The whole of the intestine was empty, but the mucosa and wall were swollen and had a deep red colour, so much so that a diagnosis of an acute gastro-enteritis was made at the time. It will be seen below that these changes were due to a passive engorgement of the splanchnic organs, and not primarily to an inflammatory process.

The second duiker showed oedema of the lungs, subepi- and subendocardial petechiae hydropericardium, hydrothorax, ascites, tumor splenis with enlarged Malpighian corpuscles, tumor hepatis, multiple, localized and circumscribed superficial erosions in the mucous membrane of the abomasum, and a moderate hyperaemia of the mucosa of the small intestine.

*B. Microscopical lesions.*

A detailed description of the histopathological changes has been given by Thomas and Neitz (1948). The most obvious and striking feature in the organs was the presence in the blood vessels of large multi-nucleated syncytial masses or aggregations of protoplasm. In general they were attached to the inner wall of the vessel either by a broad base or by narrow stalks or even by thin strands. Most of them were elongated, and tailed off within the vessel lumen to a rounded or a pointed free end, floating in the blood stream. They were found mostly in the arteries, in the pulmonary arteries, in the portal veins, and to a lesser extent in the systemic veins and capillaries. Measurement on the fixed, cut and stained preparations showed that the smaller bodies were about 20·0 to 40·0 microns in diameter. The majority were elongated and finger-like, varying in width from 20·0 to 200·0 microns and in length from 200·0 to 660·0 microns. In arteries with lumina under 1·0 mm. in diameter anything from one to fifty of the bodies could be counted in the plane of the cross-section of a single artery. The structure of these syncytial bodies was variable, a fact interpreted as due to different stages in the development of the parasite harboured.

The liver showed that the portal veins were markedly distended, and crammed nearly full with the syncytial conglomerates. Their intima was greatly damaged and partial thrombosis frequent. There was also a fairly extensive blocking of the liver sinusoids by such structures. The central vein did not appear to be affected. Patchy stasis of blood accompanied by localized cyanotic atrophy of hepatic cells was evident. There was also irregular sublobular necrosis of a mild degree accompanied by karyolysis. The remaining hepatic cells themselves were not normal, but showed vacuolization, pressure and displacement by the impacted syncytials. In Glisson's capsule there was infiltration or proliferation of round cells, mostly histiocytes.

Kidneys showed a fair amount of albuminous fluid in the glomeruli and tubules. The arteries were dilated and contained developing and full-grown syncytial bodies. The glomerular and other capillaries were engorged with blood, but showed very few impacted parasitized masses. A few necrosed areas could be seen in the cortex, and cellular infiltration round the main blood vessels.

There was a distinct hyperaemia and oedema of the lungs. The alveoli contained a variable amount of serous fluid. The larger blood vessels (pulmonary arteries) contained many syncytial masses. The peribronchial and periarterial lung tissue was partly consolidated owing to the oedema sometimes mixed with fibrin. There was obstruction of the vascular lumen with or without thrombosis, capillary embolism or perivascular development of syncytials and cellular infiltration. The alveolar capillaries in general were patent, but filled with blood, embolism being infrequent.

The spleen showed blood stasis. There were a few giant multinucleated masses in the pulpa, but many more appeared in the arteries along the trabeculae or even in some instances impacted in the pencil artery of the Malpighian corpuscle. Swelling of the arterial wall with damage and developmental stages of syncytials were also present.

Blood vessels of the myocardium in general, but especially the muscular capillaries were remarkably free from embolism, though greatly engorged with blood. Only a few syncytials could be seen in the larger veins and arteries. In the latter there was also swelling, and damage accompanying developmental stages. In parts there was a faint diffuse necrobiosis of the muscle.

The arteries and arterioles of the pia mater in general were distended and usually contained a few syncytial bodies. The capillaries were engorged but usually patent. Many, however, showed emboli, some of which were accompanied by infarction. Haemorrhage was present either along the course of the blood vessel or more diffusely into the surrounding cerebral tissue. Necrobiosis was most evident in the midbrain region. The vessels of the choroid plexus contained numerous syncytials.

In the abomasal and intestinal wall the arteries and arterioles were much distended and contained great numbers of syncytial masses. However, there were very few in the veins and capillaries, whose lumina were greatly distended with blood. There was very little embolism. Swelling and vacuolization of artery walls were frequently seen.

#### *Diagnosis.*

Although a tentative diagnosis of sylvicaprine cytauxzoonosis can be made by considering the clinical symptoms and lesions at autopsy, a definite diagnosis depends upon demonstrating the erythrocytic stages in blood smears and schizonts in spleen, lymphatic gland, liver, lung, kidney and adrenal smears. They are either free or occur within uni- or multi-nucleated histiocytes. They may be found readily or alternatively only demonstrable after a prolonged search.

#### *Treatment.*

No opportunity has presented itself to treat affected duikers. It needs to be determined whether or not either aureomycin or terramycin in combination with pamaquin will be effective, as in the case of East Coast fever (Neitz, 1956), if treatment is commenced during the initial stages of the disease.

#### *Prognosis.*

Information on the morbidity and mortality rate in sylvicaprine cytauxzoonosis is very limited. Observations on this disease are confined to two animals both of which died.

#### *Prophylaxis.*

It is self-evident that prophylactic measures cannot be applied in nature. Up to the present sylvicaprine cytauxzoonosis has only been encountered once in an animal kept in captivity. It thus appears from observations made in many zoological gardens in Africa, that the prevailing hygienic measures are not conducive to the spread of the disease, or alternatively that the duikers maintained in these circumstances are either all immune or fully susceptible.

#### *Immunity.*

No experimental information is available on the nature of the immunity in sylvicaprine cytauxzoonosis. The demonstration of erythrocytic parasites indistinguishable from those of *C. sylvicaprae* in several duikers in Angola (Borges, 1906), Nyasaland (Bruce *et al.*, 1915) and Zululand (Neitz, 1931, 1933) suggests that recovered animals develop a premunity.

2. STREPSICEROSINE CYTAUXZONOSIS.  
CYTAUXZON STREPSICEROSI INFECTION.

*Definition.*

Strepsicerosine cytauxzoonosis is an acute, in all probability an arthropod-borne disease, caused by *Cytauxzoon strepsicerosi* Neitz and De Lange, 1956. It is characterised by pyrexia, anorexia, listlessness, general weakness, anaemia, icterus, staggering gait, loss in condition and coma before death.

*Synonyms.*

Cytauxzoonose van koedoes (Afrikaans); Cytauxzoonose des Kudus (German); Catauxzoonose a *Cytauxzoon strepsicerosi* (French).

*History.*

A blood smear from a sick kudu bull calf, approximately five months old, was submitted by Mr. J. J. Booyens, Principal of the Rust-De-Winter school, Waterberg district to Onderstepoort on 20.9.51. Microscopic examination revealed the presence of numerous erythrocytic parasites resembling *G. mutans*. The animal had been procured four weeks previously from the farm Vischgat in the vicinity of Vaalwater, Waterberg district, Northern Transvaal. It was maintained together with a kudu heifer calf in a well-fenced paddock near the school. Both animals adapted themselves well to their new surroundings, and were very docile. Approximately 21 days after arrival the bull calf became listless, and on taking its temperature a week later it was found to be 106° F. The animal was admitted to Onderstepoort on the 21.9.51 in a very weak condition, and died nine days later. Examination of the prescapular lymphatic glands showed the presence of a few syncytial masses harbouring schizonts. These syncytial aggregations resembled those observed previously in a duiker that harboured *Cytauxzoon sylvicaprae* (*vide supra*).

*Distribution.*

So far only one case of strepsicerosine cytauxzoonosis has been encountered in the Waterberg district, Transvaal. Erythrocytic parasites indistinguishable from those of *C. strepsicerosi* have been encountered in kudus in Zululand (Neitz, 1933) and Transvaal (Neitz, 1952). It is thus possible that this form of cytauxzoonosis is more widely distributed than is realized at present.

*Aetiology.*

*Cytauxzoon strepsicerosi* Neitz and de Lange, 1956.

(a) *Morphology.*—(i) Erythrocytic parasites:— This stage has so far been encountered in a single kudu. Morphologically the endoglobular parasites resemble *C. sylvicaprae*. In stained smears the parasites of the kudu appear in the red blood cells as oval, comma-shaped or round organisms. The oval forms are 0·8 to 1·2 microns in width and 1·5 to 2·0 microns in length; comma-shaped forms 0·5 to 0·9 microns broad and 2·0 to 2·5 microns long, and the round forms have a diameter of 2·0 to 2·5 microns.

The cytoplasm stains light blue. The nucleus appears as a deeply stained minute reddish-purple granule situated at the wider end of the oval or comma-shaped forms, and on the margin of the round parasites. When reproduction takes place the nucleus divides into two and finally into four granules. (See Fig. 7.)

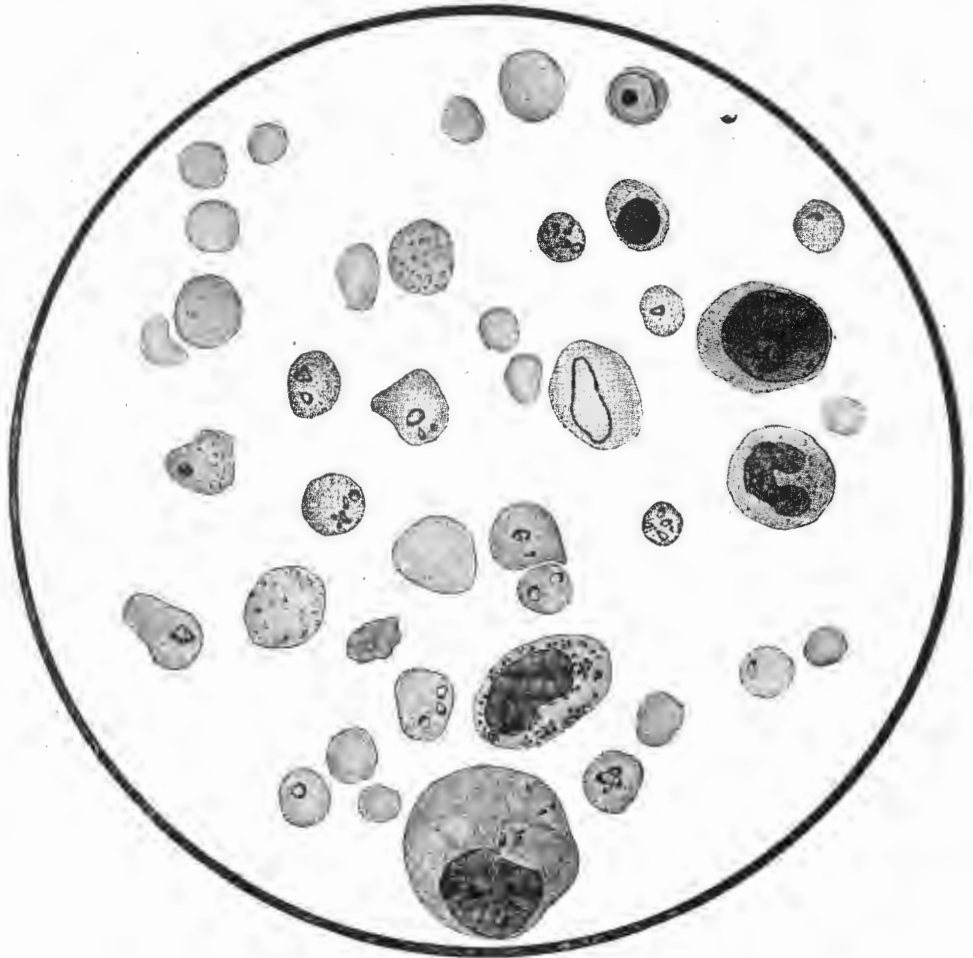


FIG. 7.—*Cytospora strepsicerosi* Neitz and de Lange 1956, of the kudu [*Strepsiceros strepsiceros* (Pallas)].

(ii) Histiotropic parasites:— The extracellular and intracellular schizonts of the kudu are morphologically indistinguishable from those of *C. sylvicaprae*. Two types of schizonts, namely immature and mature stages can be recognized. The former type is usually round and has a diameter of 5.0 to 25.0 microns with an average of 10.0 microns. The smallest schizont is a round body of about 5.0 microns in diameter, and contains 1 to 5 nuclei. The schizont increases in size, while the nuclei multiply by repeated division until 20 to 30 and sometimes even more are present. The chromatin granules appear as irregularly shaped bodies, varying from 1.0 to 3.0 microns in size. The granules divide

TIEILERIOSIS, GONDERIOSES AND CYTAUXZONOSSES.

giving rise to as many as 100 and more small (0.5 to 0.75 micron) circular bodies which are evenly distributed in the cytoplasm. The plasma bodies harbouring these small nuclei are the mature schizonts which liberate numerous merozoites. The round forms vary from 0.8 to 2.5 microns in diameter, while the oval forms are 0.6 to 1.0 micron broad and 2.5 microns long.

The intracellular schizonts are similar to the extracellular forms but are somewhat smaller. The size varies from 5.0 to 15.0 microns in diameter with an average of 10.0 microns.

(b) *Multiplication.*—*C. strepsicerosi* multiplies by schizogony. When schizonts are fully formed they break up into merozoites which either remain in the host cell or enter other histiocytes to grow and reproduce by schizogony again, or they penetrate the erythrocytes. Multiplication also occurs within the red blood cells. Division into four takes place resulting in cross-forms, in which four minute pear-shaped individuals radiate from a central point.

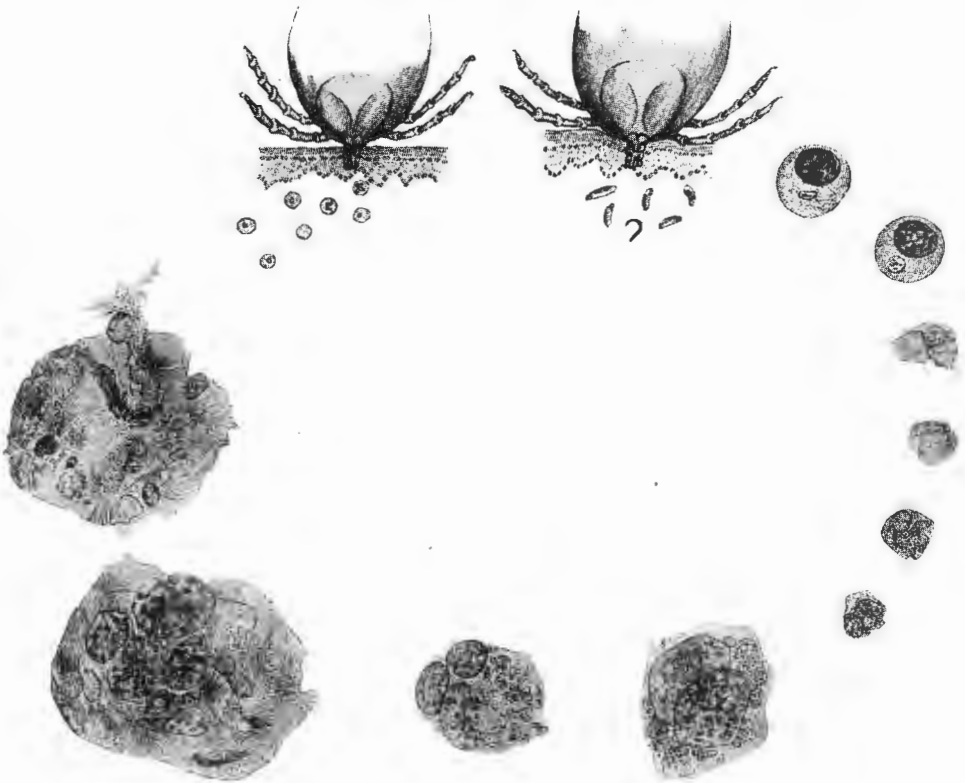


FIG. 8.—*Cytauxzoon strepsicerosi* Neitz and de Lange 1956, of the kudu [*Strepsiceros strepsiceros* (Pallas)].

(c) *Habitat.*—Approximately 70 per cent of the red blood cells are parasitized. The host cell may harbour one to twelve parasites. Schizonts parasitize histiocytes which are either uni- or multi-nucleated. Parasitized cells in various stages of development are seen in smears prepared from the spleen, liver and lymphatic glands. The uni-nucleated histiocytes may harbour up to five schizonts

which may contain one or more chromatin granules. An infected multi-nucleated cell with five nuclei can harbour 30, and one with 15 nuclei as many as 150 schizonts. Uni-nucleated parasitized histiocytes vary from 15·0 to 20·0 microns in diameter. Multi-nucleated host cells with a round shape vary from 25·0 to 40·0 microns in diameter, while oval forms vary from 12·0 to 24·0 microns in width and 20·0 to 50·0 microns in length. Parasitized aggregations, as seen in organ smears, are very large and vary from 40·0 to 80·0 microns in width, and from 50·0 to 200·0 microns in length. The latter may harbour as many as 200 schizonts. Undisturbed round multinucleated host cells of 80·0 microns in diameter can harbour several thousand merozoites and several residual bodies. In these circumstances the nuclei of the host cell show a variable degree of degeneration.

(d) *Life-cycle*.—In the vertebrate host *C. strepsicerosi* multiplies by schizogony in the histiocytes. The forms in the erythrocytes reproduce by division into four daughter individuals. The final stage of the parasite is possibly a gametocyte or a gamete (See Fig. 8).

(e) *Taxonomy*.—It has become apparent from the description of the protozoon responsible for strepsicerosine cytauxzoonosis that it is morphologically indistinguishable from *Cytauxzoon sylvicaprae*. Observations made on other members of the family Gonderidae show that they are stenoxeous parasites. In view of this, it is proposed to name the protozoon of the kudu [*Strepsiceros strepsiceros strepsiceros* (Pallas)], *Cytauxzoon strepsicerosi* nov. spec. Neitz and De Lange, 1956. The disease produced by this protozoon differs from the observations made on two cases of sylvicaprine cytauxzoonosis in that the anaemia was very pronounced in the kudu (*vide infra*). It needs to be determined whether or not the anaemia is a constant feature of strepsicerosine cytauxzoonosis.

#### *Transmission.*

##### *A. Natural Transmission.*

*Biological transmission*.—It is believed that transmission is effected by an arthropod, probably a tick.

##### *B. Artificial transmission.*

The lack of susceptible kudus at the time when the disease was recognized made it impossible to establish whether this form of transmission is possible.

#### *Epizootology.*

Up to the present only a single case of strepsicerosine cytauxzoonosis with a fatal termination has been encountered in South Africa. Since this animal became sick three weeks after its admission to the zoological garden of the Rust-De-Winter school, it is assumed that the animal became infected before it was procured. The kudu heifer associated with it had been acquired several months previously. This heifer could have served as a reservoir for the infection of the vector but microscopical examination of blood smears, prepared at the time, failed to reveal any parasites. The source of infection is, therefore, obscure.

To what extent cytauxzoonosis occurs in kudus is as yet unknown. Erythrocytic parasites resembling those of *C. strepsicerosi* have been found in several kudus in South Africa so that this disease may be more prevalent than is known. Its apparent absence in regions where kudus are common game animals may possibly be attributed to the fact that affected animals readily become the prey of carnivores, and hence its normal incidence remains obscure.

*Pathogenicity.*

So far only the kudu has been found to be susceptible. Whether or not other members of the sub-family Tragelaphinae are susceptible needs to be determined.

Sheep and calves proved to be refractory to the intravenous injection of blood from the affected kudu.

*Pathogenesis.*

The erythrocytic stage of *C. strepsicerosi* exerts its pathogenic action by penetrating and destroying the erythrocytes. The histiotropic parasites activate the histiocytes, the nuclei of which multiply repeatedly while the cytoplasm grows at the same time to accommodate them. These processes give rise to fever which is accompanied by a progressive anaemia. The liberated haemoglobin is transformed in the liver to bile pigments which become excreted. Malan (1951) who conducted the chemical analysis of the pigments in the blood established that besides the bile pigments there occurred a yellow pigment which was not carotin.

*Symptomatology.*

The affected animal showed fever (106° F) for two days after admission. Thereupon it became normal for a period of eight days when death supervened. It did not feed but drank milk and water readily until the day before it died.

The kudu showed general weakness, loss in condition, swelling of the superficial lymphatic glands, pallor and slight icterus of the visible mucous membranes, slow and laboured breathing, and a slow and weak pulse. Three days before death the flank fold and hind limbs became swollen. The animal was disinclined to move, and lay in a costo-abdominal position. It died from exhaustion.

At the time when the kudu was admitted it showed a blood count of 2.48 million erythrocytes and 7,200 leucocytes per c.mm. During the ensuing days the anaemia became more pronounced. A day before death the blood count was 1.31 million erythrocytes and 18,000 leucocytes per c.mm. Blood smears showed anisocytosis, punctate basophilia, polychromasia, Jolly bodies and normoblasts, and a 70 per cent infection of the erythrocytes.

*Pathology.*

*A. Macroscopical lesions.*

The carcass showed a marked generalized anaemia, hydropericardium, pronounced oedema of the lungs, degeneration of the liver with circumscribed red foci, moderate tumor splenis, swelling of the lymphatic glands, moderate hyperaemia of the kidneys and adrenals, and marked subcutaneous oedema of the flank fold and hind limbs.

*B. Microscopical lesions.*

De Lange (1956) described the following histo-pathological lesions: The myocardium was normal but the skeletal muscles showed engorgement of the blood vessels. In some areas of the lung the endothelial cells contained haemosiderin pigment granules; the bronchioli contained sero-fibrinous masses and catarrhal cells; the pulmonary arterioles and alveolar capillaries were engorged.



The liver showed local areas of blood stasis and degeneration of the liver cells with pycnosis and vacuolization; Glisson's capsule exhibited round cell infiltration; the portal veins were engorged. The renal tubules contained albuminous fluid; the arteries of the cortex and medulla were engorged. The adrenals showed foci of extensive haemorrhages at the junction between the cortex and medulla; the cortical blood vessels were engorged. In the spleen the endothelial cells contained masses of haemosiderin pigment granules. The lymphatic glands showed oedema and blood stasis, and large numbers of polymorphic leucocytes. The blood vessels of the choroid plexus showed congestion. No lesions were observed in the alimentary tract. No syncytial bodies harbouring schizonts were seen in any of the organs even though they were found by the writer in the living animal in smears prepared from the prescapular lymphatic glands, liver and spleen.

#### *Diagnosis.*

Although a tentative diagnosis of strepsicerosine cytauxzoonosis can be made by considering the clinical symptoms and lesions at autopsy, a definite diagnosis depends upon demonstrating the erythrocytic stages in blood smears, and schizonts in prescapular lymphatic gland, spleen and liver smears. It is of interest that the syncytial cells harbouring schizonts were found in the above-mentioned organ smears prepared from the living animal, but that they were not detectable in either the smears or sections of organs prepared after the death of the affected kudu.

#### *Treatment.*

No attempt was made to treat the affected kudu. It needs to be determined whether either aureomycin or terramycin in combination with pamaquin will be effective as in the case of East Coast fever (Neitz, 1956) if treatment is commenced during the initial stages of cytauxzoonosis.

#### *Prognosis.*

Since only a single fatal case of strepsicerosine cytauxzoonosis has so far been encountered no opinion can be expressed about the mortality rate.

#### *Prophylaxis.*

Prophylactic measures cannot be applied in nature. From the observations made in many zoological gardens in Africa, it appears that either the prevailing hygienic measures do not favour the spread of the disease, or alternatively that the kudus had acquired an immunity before they had been captured.

#### *Immunity.*

No experimental information is available on the nature of the immunity in strepsicerosine cytauxzoonosis. The demonstration of erythrocytic parasites indistinguishable from those of *C. strepsicerosi* in several kudus in Zululand and Transvaal (Neitz, 1931, 1952) suggests that recovered animals develop a premunity.

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