while that due to the Russian strains may be as high as 90 per cent. In India Sen and Srinivasan (1937) recorded a mortality rate of 76 per cent, while Edwards (1930) observed a death rate of 25 per cent.

Antigenically different *G. annulata* strains have also been recognized. However, there appears to be an antigenic component common to all strains, and in addition an unknown number of different specific components. A partial cross-immunity exists between the Algerian and Palestinian strains (Sergent, Donatien, Parrot and Lestoquard, 1937, 1945; Adler and Ellenbogen, 1935, 1936) and also between the Algerian and Iranian strains (Sergent, Parrot, Lestoquard and Delpy, 1939; Sergent *et al.* 1945). Observations of Yakimoff *et al.* (1940) on the other hand, have shown that a complete cross-immunity exists between the Algerian and Russian strains even though the latter strain is more virulent.

According to Sergent, Donatien, Parrot and Lestoquard (1932) and Cordier, Menager and Delorme (1936) the serial passage of the " Kouba " strain was followed by a remarkable change in the behaviour of *G. annulata*. After a number of passages only Koch bodies appeared in the experimental animals, while the erythrocytic parasites could not be demonstrated in the peripheral blood. These investigators state that all attempts to infect ticks failed when this manifestation became evident. They draw the inference that the " Kouba " strain can be used safely for the immunization of cattle without the danger of creating reservoirs for the infection of ticks.

(*h*) *Isolation of pure strains.*—Sergent *et al.* (1945) have established that the two host tick, *Hyalomma mauretanicum* (= *H. detritum*) is only capable of transmitting *G. annulata* but not *G. mutans* and the other blood parasites of cattle. This host-specific relationship thus offers a reliable method for isolating pure strains of *G. annulata*.

## Transmission.

A. *Natural transmission.*

(*a*) *Biological transmission.*—Ticks responsible for the transmission of *G. annulata* are listed in the appended Table VIII. Six *Hyalomma* spp. are capable of transmitting the disease, and stage to stage transmission within the same generation has been proved to occur in all. Ray (1940-41, 1950) claims to have proved that *G. annulata* is transmitted transovarially through four generations by *H. savignyi*, while Kornienko and Shmyreva (1944) state that hereditary transmission takes place in *H. turkmeniense* (= *H. excavatum*). In this form of transmission transfer is effected by adult ticks. Delpy (1949) concludes from his experimental observations that there is no evidence of hereditary transmission of *G. annulata* by the mother tick to its progeny in *H. detritum, H. excavatum, H. rufipes glabrum* and *H. savignyi*. Daubney and Sami Said (1951) state that there is no transmission through the egg to the larval stage in the case of *H. excavatum.*

Attempts to transmit *G. annulata* with *Rhipicephalus appendiculatus* (Sergent, Donatien, Parrot, Lestoquard and Plantureux, 1927), *Rhipicephalus bursa* and *Rhipicephalus sanguineus* (Sergent, Donatien, Parrot and Lestoquard 1945); and *Anopheles maculipennis* var. *labranchiae* (Sergent, 1940, 1941) have failed.

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

(c) *Intra-uterine transmission.*—This form of transmission has been recorded by Sergent, Donatien, Parrot, Lestoquard, Plantureux and Rougebief (1924), Springholz-Schmidt (1937) and Magneville (1925). It is apparently of extremely rare occurrence as the infectious agent has so far been demonstrated only in two calf foetuses and in a seven-day old calf.

## B. *Artificial Transmission.*

Tartatowsky (1905) reports that the first successful artificial transmission of tropical gonderiosis was achieved by Dschunkowsky and Luhs in 1905. The failures of Dschunkowsky and Luhs (1904) in Transcaucasia, Ducloux (1905), in Tunis, and Bitter (1905) and Mason (1922) in Egypt were probably due to the fact that indigenous cattle, which had a previous immunity were used for the transmission experiments. Since then experience in all enzootic regions has shown that the infection is easily transmissible to susceptible cattle when either infective blood or organ suspensions are used as inoculum. Apparently it makes little difference whether the infective material is injected intravenously, subcutaneously or intraperitoneally; positive results are nearly always obtained. Sergent *et al.* (1945) transmitted the disease successfully to 83 per cent of their experimental animals in North Africa. Whether or not the remaining cattle developed an inapparent infection was not determined. The "Kouba" strain has been maintained without any difficulty through 223 serial passages. Yakimoff and Gousseff (1936) in Russia, and Ware (1933) and Sen and Srinivasan (1937) in India also succeeded in transferring the disease serially in cattle.

## *Epizootology.*

In common with other diseases which require an arthropod vector, tropical gonderiosis is characteristically a disease of place (Table VII). Areas in which the vectors (Table VIII) and susceptible ruminants (Table IX) occur must be regarded as potential tropical gonderiosis areas.

Cyclic variations in the seasonal incidence of tropical gonderiosis do occur. General experience is that no outbreaks occur in winter, while during summer and early autumm a large number of susceptible animals contract the disease. Sergent, Donatien, Parrot and Lestoquard (1931) state that in Algiers most cases occur from June to September. The density of the vectors and the presence of susceptible ruminants determine the enzooticity or degree of prevalence. Relapses from tropical gonderiosis can be expected to occur at any time of the year.

Environmental factors favourable for the propagation of the vectors are of considerable interest from the epizootological standpoint. *Hyalomma* spp. require a warm and a relatively humid climate and adequate shelter for their development. *H. mauretanicum* (Sergent, Donatien, Parrot and Lestoquard, 1931) and *H. excavatum* (Daubney and Sami Said, 1951) are prone to hide in crevices, and have often been found in stone walls of stables and kraals, where they remain dormant in winter. At the beginning of summer the eggs hatch and the engorged larvae and/or nymphae moult. When weather conditions are favourable they emerge from their hiding places and attack cattle and other animals.

Tropical gonderiosis is transmitted in all instances to cattle and other susceptible animals by ticks. Premune and affected animals serve as reservoirs for the infection of ticks. It is claimed by Ray (1940-41, 1950) and Kornienko and Shmyreva (1944) that hereditary transmission can take place in two of the

known vectors (*vide supra*). Consideration of this phenomenon suggests that the infection can maintain itself in nature for a considerable time in the complete absence of the susceptible mammalian host.

In enzootic areas, young calves contract the disease which renders them premune. This is reflected in the relatively low mortality in adult cattle, born and bred in tick infested areas, as compared with the high morbidity and mortality rate in stock introduced from tropical gonderiosis free areas. (Schern, Mavrides and Major, 1920; Ware, 1931; Miegeville, 1933; Adler and Ellenbogen, 1934; Grimpet, 1937; Sen and Srinivasan, 1937; Sergent *et al.*, 1945).

Tropical gonderiosis often occurs in association with babesioses and anaplasmosis. In these circumstances it is difficult to estimate the direct losses due to the former disease unless systematic smear examinations are made from all animals that die within an area. Although mention of such an association is made in the literature detailed records are not available.

Tropical gonderiosis is one of the most important stock diseases in North Africa, Southern Europe and Asia. Its presence has greatly interfered with the establishment of pure bred stock. The severe losses not only among calves but also of susceptible older animals introduced into the enzootic areas are well known to veterinarians and stock owners. Production of milk and beef could have been very much higher had it not been for this hazard.

### Pathogenicity.

Members of the family Bovidae susceptible to *G. annulata* are listed in Table IX. In addition to cattle, zebus, water buffaloes and the American bison are susceptible to natural tropical gonderiosis infection. The mortality rate in cattle is dependent upon the virulence of the strain. It has been estimated that the mortality varies from 35 to 40 per cent in Palestine (Adler and Ellenbogen, 1934), 20 to 40 per cent in Algiers (Donatien and Lestoquard, 1938), up to 90 per cent in the enzootic regions in Russia (Yakimoff and Goussef, 1936), up to 76 per cent in India (Sen and Srinivasan, 1937) and approximately 75 per cent in Bulgaria (Pavlov, 1942). Cordier, Menager and Delorme (1936) state that zebu cattle are more resistant than European breeds and cross-bred animals.

It has been reported from Palestine that calves are more resistant than adult stock (Adler and Ellenbogen, 1934). Delpy (1946) observed in Iran that calves from premune cows are more resistant than those from fully susceptible animals. A careful survey made by MacHattie in Iraq showed that 50 per cent of calves from dairy cows (Ayrshire crosses) died from tropical gonderiosis. Raghavachari, Shah and Ray (1945) estimated that 13 to 23 per cent of calves from indigenous cows in India succumbed to *G. annulata* infection.

Sergent *et al.* (1945) established that sheep are refractory. Neitz (1953) failed to transmit G. *annulata* to sheep with infective *H. excavatum* adult ticks which were obtained from Dr. A. Rafyi of Iran.

### 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 endothelial lining of blood vessels also becomes affected resulting in an oedema of the lungs, subcutaneous and intermuscular tissues. The parasitized erythrocytes liberate haemoglobin sometimes followed by haemoglobinuria. Haemoglobinaemia results in an increased production of bile and in the development of icterus.

## Symptoms.

Studies made on naturally and artificially infected cattle established that the severity of tropical gonderiosis usually depends upon the intensity and the duration of the parasitic attack. After exposure to tick infestation, the incubation period usually varies from 9 to 25 days with an average of 15 days. After an artificial infection the period varies from 12 to 30 days with an average of 17 days. In cases studied by Brumpt (1923, 1924) the period which elapsed between infection and appearance of an acute attack, varied from three-and-a-half to seven months. Sergent *et al.* (1945) observed that after administration of infective blood the period varied from one to six months.

Depending upon the virulence of the strain and the resistance of the animal, tropical gonderiosis may be classified according to its symptoms into five types: (1) the mild, (2) the peracute, (3) the acute, (4) the subacute and (5) the chronic form.

(1) *The mild form.*—This form is usually observed in cattle, particularly young calves, subjected to the immunization process with a relatively mild *G. annulata* strain. Frequently animals pass through the reaction without exhibiting any symptoms, and the presence of the infection may be entirely overlooked unless Koch bodies and endoglobular parasites are detected by blood examination. Sometimes there are an accompanying mild fever, inappetence, slight digestive disturbances, lachrymation and listlessness lasting a few days. On blood examination moderate anaemic changes can be observed in addition to the parasites.

(2) *The peracute form.*—This form has been observed in artificially and naturally infected animals. It is of fairly common occurrence, and is difficult to distinguish from the peracute forms of babesioses, anaplasmosis, anthrax, mineral and plant poisoning. The onset is sudden. The affected animal has a high temperature (106°—107° F) and shows listlessness, drooping ears, lowered head, lachrymation, serous nasal discharge, salivation, swelling of the superficial lymphatic glands, muscular tremours, sluggish gait, marked drop in milk production, accelerated pulse, dyspnoea, anorexia, constipation and also anaemia and icterus. Death occurs within 72 to 96 hours, and is preceded by hypothermia. Blood and lymphatic gland smears show a variable number of Koch bodies and up to 50 per cent of the erythrocytes may be parasitized.

(3) *The acute form.*—This is the usual type observed. The affected animal exhibits pronounced symptoms often terminating fatally. There is an elevation of the body temperature varying from 104° to 107° F. The fever is either continuous or irregularly intermittent, and persists for 5 to 20 days. Clinical symptoms usually appear a few days after the initial rise in temperature. Koch bodies may be demonstrable two days before the commencement of fever, and the endoglobular forms three to five days later. The animal shows inappetence, cessation of rumination, drooling from the mouth, serous nasal discharge, swelling of the superficial lymphatic glands, swelling of the eyelids,

lachrymation, accelerated pulse, general weakness, drop in milk production and rarely nervous symptoms. In the course of a few days marked anaemia develops. The red cell count drops from 7 to 3 million per c.mm. Cases of haemoglobinuria have been described by Sergent *et al.* (1924, 1945). Bilirubinaemia and bilirubinuria are always present. The conjunctiva in addition to the anaemia or icteric appearance, may show petechiae. At the beginning of the pyrexial period the faeces are firm but diarrhoea soon sets in, and when the disease runs a prolonged course, the evacuations are frequently mixed with blood and mucus. The animal becomes markedly emaciated and assumes a recumbent position. If at this stage the animal commences to feed and begins to show regeneration of the erythrocytes, recovery may take place. If regeneration of the erythrocytes does not take place, the anaemia becomes so severe, and dyspnoea so pronounced that death ensues 8 to 15 days after the onset of the disease. The red cell count in such cases may drop below one million red cells per c.mm.

(4) *The subacute form.*—This form is often encountered in animals suffering from a relatively mild strain of *G. annulata*. The symptoms resemble those of the acute form but are not so marked. The fever is usually irregularly intermittent and persists for as long as 10 to 15 days. Animals usually recover from this form but instances of abortion have been recorded.

(5) *The Chronic form.*—This form takes a more protracted course than either the acute or subacute type. The fever is irregularly intermittent. Inappetence, marked emaciation and a variable degree of anaemia and icterus are observed. Animals may recover after about four weeks but it may take two months and longer before the animals regain their former condition. In other instances the disease may suddenly assume an acute form, and terminate fatally in one or two days.

## Pathology.

The lesions vary according to the duration and the severity of the disease.

(A) *Macroscopical lesions.*—In the peracute form the carcase does not show emaciation. However, in the acute, subacute and chronic forms emaciation is marked. The skin may show decubital wounds and a variable number of ticks. The visible mucous membranes are yellow in colour. The subcutaneous and intermuscular tissues are yellow and are usually infiltrated with a clear serous fluid, giving them a gelatinous appearance. The skeletal muscles are pale.

The myocard shows signs of muscular degeneration, and a variable number of petechiae and ecchymoses appear on the epicardium and endocardium. Hydrothorax, hydropericardium and ascites are not constantly present. The lungs are frequently oedematous and pinkish yellow in colour. The mucous membranes of the pharynx, larynx, frontal and maxillary sinuses, trachea and bronchi, and the visceral and parietal pleura are often spotted with petechiae. The liver is increased in size, very soft, friable and brownish yellow to lemon yellow in colour; parenchymatous degeneration is evident. The gall bladder is often markedly distended with dark green viscid bile. The spleen is enlarged and the pulpa soft; the Malpighian corpuscles are prominent. The superficial and internal

lymphatic glands are as a rule markedly swollen, and may show a variable degree of hyperaemia. The mediastinum and the capsula adiposa of the kidneys may contain a large amount of serous fluid. The kidneys are pale brownish yellow in colour, and show a variable number of haemorrhagic " infarcts " or alternatively " lymphomata ". Petechiae may be present in the cortex of the adrenal glands. The urinary bladder contains bile-stained or haemoglobin-stained urine; haemorrhages may be seen in the mucous membrane. Apart from hyperaemia and petechiae in the meninges, Barboni (1942) has observed cerebral haemorrhages in 2 per cent of affected animals.

The rumen and reticulum contain a relatively small amount of ingesta, while the contents of the omasum are firm and partially dehydrated. The abomasum usually shows characteristic ulcers which vary in size from $2 \cdot 0$ to $5 \cdot 0$ mm. They consist of a central necrotic area, surrounded by a haemorrhagic zone. Similar ulcers as well as irregularly disseminated red streaks or patches may be encountered along the entire length of the small and large intestine.

(B) *Microscopical lesions.*—Sergent *et al.* (1945) have given a brief summary of the lesions. Microscopically the blood shows degenerative and regenerative changes. Anisocytosis, punctate basophilia, polychromasia, Jolly bodies, normoblasts and an increased number of reticulocytes are regularly found. Koch bodies and a variable number of endoglobular parasites are present. The red cell count may be less than one million per c.mm. Congestion and haemorrhages are observed in all organs particularly the liver, spleen and lymphatic glands. Hyperplasia of the lymphoid tissue is observed in spleen, lymphatic glands, liver and kidneys.

## Diagnosis.

The clinical symptoms presented by animals suffering from tropical gonderiosis are such that they may be confused with those of babesioses and anaplasmosis. It is essential that blood and gland smears be examined for the presence of Koch bodies and endoglobular parasites before a diagnosis is made. It must, however, be remembered that during the early stage of the disease parasites may not be easily demonstrable.

The carrier state of tropical gonderiosis can be determined by subinoculating blood from suspected cases into susceptible cattle. Splenectomy of suspected *G. annulata* premune animals can also be used for diagnostic purposes. A relapse can be expected within three weeks after operation (Sergent *et al.*, 1945; Neitz and Jansen, 1956). However, a correct identification of the endoglobular parasites can only be made by applying the xenodiagnosis. This involves infesting the suspected premune animal with larvae or nymphae of any one of the known vectors (*Hyalomma* spp.) and feeding the succeeding stage on susceptible cattle. The diagnosis is thus dependent upon the nature of the ensuing reaction and the demonstration of the infectious agent.

## Treatment.

(A) *Specific treatment.*—There is no known drug that is entirely reliable for the treatment of tropical gonderiosis. The numerous reports of cures should be considered with full knowledge that spontaneous recovery is common. The procedures instituted during the last four decades involved the use of protozoacidal and bactericidal drugs. The chemotherapeutic agents and the results

claimed by different workers are listed in the appended Table X. Consideration of these results permits one to conclude that no really satisfactory chemotherapeutic agent has yet been found, unless the curative effects of the acridine derivatives, paludrin, lomidine and the 4-amino-quinoline preparations alone or in combination with the 8-amino-quinoline compounds should be fully confirmed. There is considerable difference of opinion among workers who have used the same or closely allied products. This becomes apparent when the results obtained with the different acridine derivatives are considered.

The reports show that in all instances treatment was commenced during the reaction period, and that the number of recoveries was used as a criterion for the efficacy of the drug. Such a procedure is of great practical importance for determining the curative value of the drug but it does not reveal all properties possessed by the agent, and thus precludes systematic chemotherapeutic studies. Sen and Srinivasan (1937) merely state that the 8-amino-quinoline compound, plasmoquin is of no value for the treatment of tropical gonderiosis. Cordier and Ounais (1946) report that the administration of quinacrine (an acridine derivative) in combination with rodoprequine (a homologue of pamaquin) has a curative effect on the disease, and that " shrivelling " of the parasites was noticed. No explanation was given which of the two drugs caused the degeneration of the endoglobular parasites, and no attempt was made to determine whether the affected parasites were still capable of developing in the intermediate host. Studies on East Coast fever (Neitz, 1950) have shown that pamaquin has a selective action on the haemotropic forms of *Th. parva,* and that such affected parasites are no longer capable of developing in the vector, *Rh. appendiculatus.* The course of East Coast fever in animals treated with pamaquin, on the other hand, did not differ from that in control animals. In a subsequent report Neitz (1951) showed that plasmoquin ($=$ pamaquin) also has a selective action on the erythrocytic stages of *G. annulata.* It thus becomes apparent that the degeneration of the *G. annulata* endoglobular parasites observed by Cordier and Ounais (1946) was due to rodoprequine.

It is obvious that the evaluation of chemotherapeutic agents for the treatment of tropical gonderiosis is a matter of great importance. The writer is of opinion that tests should be conducted on sporozoite-induced *G. annulata* infections and that treatment should be commenced 72 hours after tick infestation. This matter is stressed because it was found that in *Th. parva* infection aureomycin has no influence on the course of the disease when administered during the reaction period. Its schizonticidal properties, however, became evident when repeated treatment at arbitrary irregular intervals was initiated during the incubation period. The conclusion of Cordier and Ounais (1946) that aureomycin is not effective on tropical gonderiosis when administered during the reaction period is undoubtedly correct. However, the writer is of opinion that repeated treatment with this drug during the incubation period following a sporozoite-induced *G. annulata* infection may still reveal its schizonticidal properties as in the case of East Coast fever and Corridor disease.

(B) *Symptomatic treatment.*—This form of treatment is of great value. It is, however, of considerable importance that treatment should begin as soon as possible after the onset of symptoms. In practice, careful microscopic examination of blood smears should be conducted in order to exclude the possibility of intercurrent infections.

The animal should be protected against unfavourable weather conditions, and great care should be taken to keep the animal quiet and to prevent bodily exertion (Sergent *et al.* 1945). Careful nursing throughout the course of the disease is essential.

TABLE X.

*Drugs which have been tried in the treatment of tropical gonderiosis.*

| Drug. | Effect on the Course of the Disease. | References. |
|---|---|---|
| *Antimony compounds—* | | |
| Antimosan | + | Bogoroditski and Lavrent'ev 1935. |
| Antimosan | − | Sen and Srinivasan, 1937. |
| Antimosan | − | Sergent *et al.*, 1945. |
| Antimosan | ? | Freund, 1929. |
| Pentastib | − | Sergent *et al.*, 1945. |
| Tartar emetic | − | Sen and Srinivasan, 1937. |
| Tartar emetic | − | Sergent *et al.*, 1945. |
| Stibosan | ? | Freund, 1929. |
| *Arsenic compounds—* | | |
| Arsenobenzol | − | Sergent *et al.*, 1945. |
| Atoxyl | ? | Freund, 1929. |
| Myosalvarsan | − | Sen and Srinivasan, 1937. |
| Novarsenobillon | − | Sen and Srinvasan, 1937. |
| Sodium cacodylate | − | Sen and Srinivasan, 1937. |
| Stovarsol | − | Sergent *et al.*, 1945. |
| Sulfarsenol | − | Sen and Srinivasan, 1937. |
| *Antimony and Arsenic compounds—* | | |
| Atoxyl in combination with Antimosan or Stibosan | + | Freund, 1929. |
| *Mercury compounds—* | | |
| Mercuric Chloride | − | Sen and Srinivasan, 1937. |
| *Silver compounds—* | | |
| Flavargine | + | Baboshina, Ali Zade and Shmulevich, 1949. |
| Ichthargon | − | Sergent *et al.*, 1945. |
| Ichthargon | − | Olmetschenko, 1930. |
| Protargol | − | Olmetschenko, 1930. |
| *Acridine Derivatives—* | | |
| Acriflavine | − | Sen and Srinivasan, 1937. |
| Gonacrine | ? | Sen and Srinivasan, 1937. |
| Gonacrine | − | Sergent *et al.*, 1945. |
| Gonacrine | − | Cordier and Menager, 1933. |
| Gonacrine | + | Grimpet, 1937, 1938. |
| Gonacrine | + | Brun, 1949. |
| Trypaflavin | ? | Sen and Srinivasan, 1937. |
| Trypaflavin | + | Tüdzil, 1946. |
| Trypaflavin | + | Grimpet, 1952. |
| Atebrin | + | Sen and Srinivasan, 1937. |
| Quinacrine (= Atebrin) | + | Cordier and Ounais, 1946. |
| Quinacrine (= Atebrin) | + | Cardassis, 1956. |
| Quinacrine (= Atebrin) | − | Sergent, *et al.*, 1945. |

TABLE X (*continued*).

*Drugs which have been tried in the treatment of tropical gonderiosis.*

| Drug. | Effect on the Course of the Disease. | References. |
|---|:---:|---|
| *Acridine compounds and 8-aminoquinoline compounds—* | | |
| Atebrin and Plasmoquin............ | − | Sen and Srinivasan, 1937. |
| Quinacrine and Rodoprequine....... | + | Cordier and Ounais, 1946. |
| *4-Aminoquinoline compounds—* | | |
| Chloroquine sulphate.............. | − | Tzur, Zaga, Neuman and Senet, 1954. |
| Chloroquine sulphate.............. | + | Jore d' Arces, 1952. |
| Nivaquine........................ | + | Camou, Grimpet and Vercelotti, 1952. |
| Resochin......................... | + | Neitz, 1951. |
| *8-Aminoquinoline compounds—* | | |
| Plasmoquin....................... | − | Sen and Srinivasan, 1937. |
| Plasmoquin....................... | + E-stages | Neitz, 1951. |
| *4- and 8-Aminoquinoline compounds—* | | |
| Resochin* and Plasmoquin*.......... | + | Neitz, 1951. |
| *Quinoline derivatives—* | | |
| Acaprin.......................... | + | Cerruti, 1935. |
| Acaprin.......................... | + | Mlinac, 1942. |
| Acaprin.......................... | + | Pavlov, 1942. |
| Piroplasmin (= Acaprin)........... | − | Lavrent 'ev, 1938. |
| Piroplasmin (= Acaprin)........... | − | Yakimoff, Gusev and Melnikova, 1940. |
| Zothelone (= Acaprin)............. | − | Grimpet, 1937. |
| Zothelone (= Acaprin)............. | − | Sergent *et al.*, 1945. |
| *Alkaloids—* | | |
| Emetine.......................... | − | Sergent *et al.*, 1945. |
| Quinine.......................... | − | Sen and Srinivasan, 1937. |
| Quinine.......................... | − | Sergent *et al.*, 1945. |
| Quinine.......................... | − | Freund, 1929. |
| *Amidine compounds—* | | |
| Stilbamidine...................... | − | Adler and Tchernomoretz, 1943. |
| Lomidine......................... | − | Rampson, 1948, 1949. |
| Lomidine......................... | + | Grimpet, 1948. |
| Lomidine......................... | + | Brun, 1949. |
| *Antibiotics—* | | |
| Aureomycin....................... | − | Cordier and Ounais, 1954. |
| Penicillin........................ | + | Rabillon, 1951. |
| *Naphthalene derivatives—* | | |
| Bayer 205........................ | ? | Sen and Srinivasan, 1937. |
| Forneau 309 (= (Bayer 205)....... | − | Sergent *et al.*, 1945. |
| Piroblue......................... | − | Sen and Srinivasan, 1937. |
| Trypan blue...................... | − | Sergent *et al.*, 1945. |
| Trypan blue...................... | − | Freund, 1929. |
| Trypan blue...................... | − | Olmetschenko, 1930. |
| Trypan red....................... | − | Olmetschenko, 1930. |
| *Thiazin Dye—* | | |
| Methylene blue................... | − | Olmetschenko, 1930. |

TABLE X (*continued*).

*Drugs which have been tried in the treatment of tropical gonderiosis.*

| Drug. | Effect on the Course of the Disease. | References. |
|---|---|---|
| Paludrin............................ | + | Cardassis, 1956. |
| Todorit............................. | + | Yakimoff, Nezwetaieff and Yokowleff 1936. |
| Todorit............................. | — | Sen and Srinivasan, 1937. |
| *Treatment with a combination of drugs—* Piroplasmin, Flavacridine and Uro-tropin | — | Yakimoff, Gusev and Melnikova, 1940. |
| Acaprin, Flavargine and Lp2........ | + | Baboshina, Ali Zade and Shmulevich, 1949. |
| Acaprin, Novoplasmin and Proflavine | — | Baboshina, Ali Zade and Shmulevich, 1949. |
| Pulvis chirata, Sodium bicarbonate *per os* and Tartar emetic intravenously. | — | Ajwani and Subbarayuda, 1934. |
| *Glucose, Sodium chloride, distilled water, formalin and camphorated spirit | + | Laizet, 1943. |

— = Of no apparent value; + = Beneficial influence; ? = Questionable value.
* Treatment commenced during incubation period.

## Prognosis.

Prognosis should always be guarded. Not only is the mortality very high but financial losses experienced by stock owners as a result of emaciation, and decreased milk yield are also great. Several months may elapse before animals become fit for slaughter. Milk production may only become normal again after the next calving.

## Prophylaxis.

Prophylaxis of tropical gonderiosis is based upon (*a*) the elimination of the arthropod vectors, (*b*) quarantine measures, and (*c*) the immunization of cattle.

(*a*) *Elimination of arthropod vectors.*—The only effective weapon for destruction of ticks is regular systematic dipping or spraying, combined with careful hand-dressing. The dipping of cattle in arsenical dip ($0 \cdot 16$ per cent $As_2O_3$) at weekly intervals has been employed successfully in several of the countries in the enzootic regions of Africa, Europe and Asia. The dipping tanks are constructed according to the plans evolved in South Africa. Satisfactory results have been obtained in Algeria (Sergent, Donatien, Parrot and Lestoquard, 1945), India (Raghavachari, Shah and Ray, 1945), Southern Russia (Kurchatov and Markhevka, 1940), and Central Asia (Bogoroditskii, Bernadskaya and Lavrent'ev, 1935). Experience has shown that tropical gonderiosis areas are usually badly infested with various species of ticks, so that it may not be possible to keep animals entirely free from these ectoparasites. Moreover, it is known that ticks can maintain themselves in nature by feeding on animals other than cattle as

well as on wild animals. It should be remembered that recovered animals are carriers of the disease and thus serve as excellent reservoirs for the infection of the vectors. Complete eradication of infective ticks, therefore, is impossible. Systematic dipping, nevertheless, reduces the incidence of the disease as shown by Kurchatov and Markhevka (1940) and Sergent *et al*. (1945).

In addition to dipping rotational grazing is also carried out in enzootic regions. Prophylactic measures advocated by Markov (1944) consist of destruction of ticks on stock during movement from an infected to a clean area. Pastures not used by infected cattle during the preceding ten months are regarded as free from infection. Dipping must be persevered with at the new grazing sites. Tick-proof stables should also be provided with a channel guarding the entrance, filled with mineral oil. Hay for the animals should have been stored for at least six months. For the accommodation of imported cattle Bogoroditskii (1938) recommends the use of new byres or byres in which animals other than cattle had been kept within the period of the life-cycle of the vectors. The buildings should be so designed as to prevent access of ticks to cattle, and to exclude vectors from hiding in crevices. Lavrent'ev, Palovskaya and Baiteryako (1951) suggest that during winter cattle be housed in well-constructed and tick-free stables. At the beginning of summer cattle should be dipped before they are transferred on to clean pastures.

(*b*) *Quarantine Measures*.—The danger of introducing tropical gonderiosis into areas free from the disease is fully realized. Mlinac and Romic (1948) record the introduction of tropical gonderiosis into Djakova (Yugoslavia) by an ox-team, which formed part of a military convoy from Bosnia or Serbia, during the Second World War. The importation of stock from enzootic into clean areas should not be allowed until it has been established by biological tests and by the xenodiagnosis that they are not carriers.

(*c*) *Immunization*.—The attempts to immunize cattle against tropical gonderiosis by means of formalized blood derived from reacting animals failed (Sergent *et al*. 1924-1927). Systematic surveys during subsequent years revealed the existence of relatively mild *G. annulata* ( = *Theileria dispar*) strains in Algeria. Sergent, Donatien, Parrot and Lestoquard (1932) isolated the "Kouba" strain which caused a mortality in about 3 per cent of the artificially infected cattle. The strain was maintained by serial passages in cattle, and after 18 generations the schizonts failed to produce erythrocytic-invading forms which are concerned with setting up infection in ticks. The "St. Charles" strain also showed this phenomenon after 11 serial passages (Sergent *et al*., 1945).

The "Kouba" and other strains of low virulence have been used extensively for the immunization of cattle, particularly young calves in North Africa. The vaccine consists of blood collected in citrate from reacting animals at the time when Koch bodies are demonstrable in lymphatic gland, liver or blood smears. The infective blood is injected subcutaneously within 72 hours after issue. The dose is 5 to 10 c.c. Donatien and Lestoquard (1938) state that the results under field conditions proved to be very satisfactory. Miegeville (1933) immunized 23 imported cattle. Of these nine developed typical reactions and two died. Delpy (1949) injected a mild strain from Algeria into 48 animals. On exposure to tick infestation in Iran four of them developed tropical gonderiosis and died. Adler (1952) reports that in Israel 6,000 to 8,000 calves are immunized annually with a relatively mild Algerian strain, and that the mortality rate is low.

Consideration of these results shows that the immunization process developed by Sergent, Donatien, Parrot and Lestoquard (1932) is an effective prophylactic measure. It should, however, be remembered that tropical gonderiosis reactions can appear in immunized stock following natural infection with antigenically different strains. Spontaneous relapses should not be confused with reactions developing in artificially infected animals after an incubation period varying from two to seven months (Brumpt, 1923, 1924; Sergent *et al.*, 1945). Relapses can also be expected when immunized animals react to babesioses, anaplasmosis and foot and mouth disease.

## Immunity.

Naturally recovered animals develop a durable premunity. It may, however, wane within three years after recovery (Sturman, 1935). Natural reinfection in these circumstances is usually followed by a non-fatal disease. The erythrocytic stages of *G. annulata* persist throughout life. It has not yet been determined whether Koch bodies are retained for the same period. The artificial infection with the " virus fixe " strains (" Kouba " and " St. Charles " strains) results in a sterile immunity, while the transmission of the " virus latent " (strains isolated from naturally infected and reacting animals) is followed by premunition (Donatien and Lestoquard, 1930; Sergent *et al.*, 1945).

Antigenically identical as well as immunologically different strains have been isolated in some of the enzootic areas. There appears to be an antigenic component common to all strains, and in addition an unknown number of different specific components. Sergent, Donatien, Lestoquard and Delpy (1939) claim that better cross-immunity results are obtained when the original infection has been produced by bites of ticks. A variable degree of cross-immunity exists between the Algerian and the Palestinian and Iranian strains. A complete reciprocal immunity was established between one of the Algerian and a Russian strain (*vide supra*—Section Aetiology).

Splenectomy of cattle recovered from a natural infection is invariably followed by a relapse within three weeks after the operation (Donatien, and Lestoquard *loc. cit.;* Sergent *et al.*, 1945; Neitz and Jansen, 1956). No relapse follows after the splenectomy of animals which have recovered from " virus fixe " infections (Donatien and Lestoquard *loc. cit.*).

Premunity to *G. annulata* may be interrupted by babesioses, anaplasmosis (Sergent *et al.*, 1945) and by foot and mouth disease and the reaction following the administration of anthrax vaccine (Grimpet, 1937, 1937). Velu (1933) records spontaneous relapses due to *G. annulata* in the indigenous cattle in Egypt, India, Anatolia and Transcaucasia.

Animals which have recovered from a natural or an artificial *G. annulata* infection are fully susceptible to *Th. parva* (Sergent *et al.*, 1934; Neitz and Jansen, 1956) and *G. mutans* (Sergent *et al.*, 1945; Neitz and Jansen, 1952).

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## 3. GONDERIA MUTANS INFECTION.

### Definition.

Benign bovine gonderiosis is a tick-borne disease caused by *Gonderia mutans* (Theiler, 1906). It is characterised by pyrexia, anorexia, mild anaemia and swelling of the superficial lymphatic glands. Recovered animals develop a durable premunity.

### Synonyms.

Benign bovine Theileriosis, Tzaneen disease, Marico calf disease, Mild gallsickness; Milde Theileriose of Gonderiose van beeste, Tzaneensiekte, Mariko kalwersiekte, Milde galsiekte (Afrikaans); Milde Theileriose of Gonderiose van runderen (Netherlands); Pseudoküstenfieber, Milde Theileriose oder Gonderiose der Rinder (German); Theileriose ou Gonderiose a *Gonderia mutans* (French).

### History.

Endoglobular blood parasites, resembling those of *Theileria parva* (Theiler, 1904), were encountered by Theiler (1906) in South Africa. He named the microorganism *Piroplasma mutans*. Franca (1909) transferred *P. mutans* to the genus *Theileria* even though he was aware that schizonts had not been encountered in the life-cycle of this protozoon. He considered that the resemblance, that exists between the erythrocytic parasites of *P. mutans* and *Theileria parva*, was sufficient reason for changing the generic name. Franca's modification of the nomenclature was not generally accepted, and for this reason du Toit (1918) created the genus *Gonderia* into which he placed piroplasms, the erythrocytic stages of which resembled those of *Th. parva*, but in which no schizonts had been demonstrated. The genus *Gonderia* was retained in the family Babesidae Poche, 1913. Wenyon (1926) revised du Toit's classification and transferred *Gonderia mutans* to the genus *Babesia*. Subsequent studies on the life-cycle of *B. mutans*, however, showed that this organism could not be retained in the genus *Babesia*. The demonstration of schizonts (Koch bodies) in its life-cycle caused Theiler and Graf (1928) to retransfer *B. mutans* to the genus *Theileria*. Recently Neitz and Jansen (1956) once more revised the classification of the Theilerias. In order to

avoid unnecessary confusion in the nomenclature they redefined and reinstated the generic name *Gonderia* to include parasites which multiply by schizogony in the lymphocytes and by fission in the erythrocytes. This genus was placed in the family Gonderidae Neitz and Jansen, 1955. In the family Theileridae they retained a single genus and species, *Theileria parva,* which only multiplies by schizogony in the lymphocytes, and not by binary fission in the erythrocytes.

## *Distribution.*

The distribution of *G. mutans* is given in Table XI. (See also Map No. 3).

### TABLE XI.

| Continent. | Country. | Animal. | References. |
|---|---|---|---|
| Africa...... | Algeria.......... | Cattle....... | Sergent, 1921; Sergent and Esperandieu 1920. |
| | Angola.......... | Cattle....... | de Mello and Carbal, 1923. |
| | Belgian Congo... | Cattle....... | Broden and Rodhain, 1909; Rodhain, Pons van den Branden and Bequaert, 1913; Schwetz and Storck, 1930. |
| | Cameroon....... | Cattle....... | Springfeldt, 1909, 1911. |
| | Cyrenaica........ | Cattle....... | Brumpt, 1920. |
| | Dahomey........ | Cattle....... | Pécaux, 1912. |
| | Egypt........... | Cattle....... | Balfour, 1908; Dreyer, 1910; Littlewood, 1916. |
| | Eritrea.......... | Cattle....... | Martoglio, Stella and Carpano, 1911. |
| | French Congo.... | Cattle....... | Malbrant, 1938. |
| | French Sudan.... | Cattle....... | Curasson, 1928. |
| | Gold Coast...... | Cattle....... | MacFie, 1915; Beal, 1920; Stewart, 1940. |
| | Italian Somaliland | Cattle. ..... | Croveri, 1920. |
| | Ivory Coast...... | Cattle....... | Bouet, 1908. |
| | Kenya........... | Cattle....... | Whitworth, 1930. |
| | | African Buffalo | Walker, 1932. |
| | Madagascar...... | Cattle....... | Theiler, 1907. |
| | Mauritius........ | Cattle....... | Theiler, 1907. |
| | Morocco......... | Cattle....... | Balozet, 1920. |
| | Nigeria.......... | Dwarf and Hausa cattle | MacFie, 1914. |
| | | Cattle....... | Connal and Coghill, 1916; Henderson, 1931. |
| | Northern Rho-desia | Cattle....... | Turnbull, 1926; Smith, 1930. |
| | Nyasaland....... | Cattle....... | Garden, 1913, 1915. |
| | Portuguese East Africa | Cattle....... | de Abreu, 1954. |
| | Senegal.......... | Cattle....... | Leger and Bedier, 1923. |
| | Sierra Leone..... | Cattle....... | York and Blacklock, 1915. |
| | South Africa..... | Cattle....... | Theiler, 1906; Meyer, 1913. |
| | | African Buffalo | Neitz, 1940, 1956. |
| | Sudan........... | Cattle....... | Balfour, 1908; Littlewood, 1916. |
| | Tanganyika...... | Cattle....... | Lichtenheld, 1910; Ollwig and Manteufel, 1910. |
| | Tunis........... | Cattle....... | Brumpt, 1920; Ducloux and Cordier, 1931. |
| | Uganda.......... | Cattle....... | Brumpt, 1920; Mettam, 1933, 1936. |
| America.... | United States of America | Cattle....... | Splitter, 1950. |

TABLE XI (*continued*).

| Continent. | Country. | Animal. | References. |
|---|---|---|---|
| Asia........ | Annam.......... | Cattle and Water Buffaloes | Schein, 1908. |
| | | Splenec- tomized Water Buffaloes | Galliard and Cébe, 1941, 1949. |
| | China........... | Cattle....... | Martini, 1907. |
| | Daghestan....... | Cattle....... | Yakimoff and Kazansky, 1935. |
| | India........... | Cattle....... | Ware, 1931; Ramanarayanan, 1942. |
| | Indonesia........ | Cattle....... | de Does, 1906. |
| | Iran............. | Cattle....... | Delpy, 1937. |
| | Japan........... | Cattle....... | Miyajima and Shibayama, 1906. Ishii and Ishiware, 1948. |
| | Palestine......... | Cattle....... | Freud, 1929; Gilbert, 1924. |
| | Turkestan........ | Cattle....... | Kohl-Yakimoff, Yakimoff and Schokhor, 1913. |
| | Uzbekistan....... | Cattle....... | Lavrent'ev, 1935. |
| Australia... | Queensland...... | Cattle....... | Dodd, 1910. |
| Europe..... | Crimea.......... | Cattle....... | Yakimoff and Rastegaieff, 1927. |
| | Cyprus.......... | Cattle....... | Doyle, 1924. |
| | England......... | Cattle....... | Hignett, 1953. |
| | France.......... | Cattle....... | Sergent, Edm., Sergent, Et., and Lhéviter, 1919. |
| | Germany........ | Cattle....... | Miessner, 1931. |
| | Greece.......... | Cattle....... | Cardamitis, 1912; Cardissis, 1956. |
| | Italy............ | Cattle....... | Carpano, 1927; Dominici, 1935; Graf, 1932. |
| | Portugal......... | Cattle....... | Bettencourt, 1907. |
| | Roumania....... | Cattle....... | Metzianu, 1947. |
| | Russia.......... | Cattle....... | Yakimoff, Rastegaieff and Lewkowitsch, 1932. |
| | Sardinia......... | Cattle....... | Cerruti, 1934. |
| | Spain........... | Cattle....... | Garcia, 1934; Perez and Laguna, 1945. |

*Aetiology.*

*Gonderia mutans* (Theiler, 1906).

> Synonyms: *Piroplasma mutans* Theiler, 1906.
> *Babesia mutans* (Theiler, 1906).
> *Theileria mutans* (Theiler, 1906).
> *Theileria buffeli* Neveu-Lemaire, 1912.

(*a*) *Morphology.*—(i) Erythrocytic parasites:— In blood smears fixed with May-Grünwald and stained with Giemsa, *G. mutans* appears in the red blood cells as pear-shaped, comma-shaped, oval, round or anaplasma-like organisms. The pear-shaped forms are $0\cdot8$ micron in width and $2\cdot0$ microns in length; comma-shaped forms $0\cdot5$ micron in width and $2\cdot4$ microns in length; oval forms $0\cdot6$ micron in width and $1\cdot5$ microns in length; round forms $1\cdot0$ to $2\cdot0$ microns in diameter and anaplasma-like forms $0\cdot9$ to $1\cdot2$ 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.

THE WORLD

DISTRIBUTION OF

GONDERIA MUTANS

(W.O.NEITZ, 1956)

MAP No. 3.
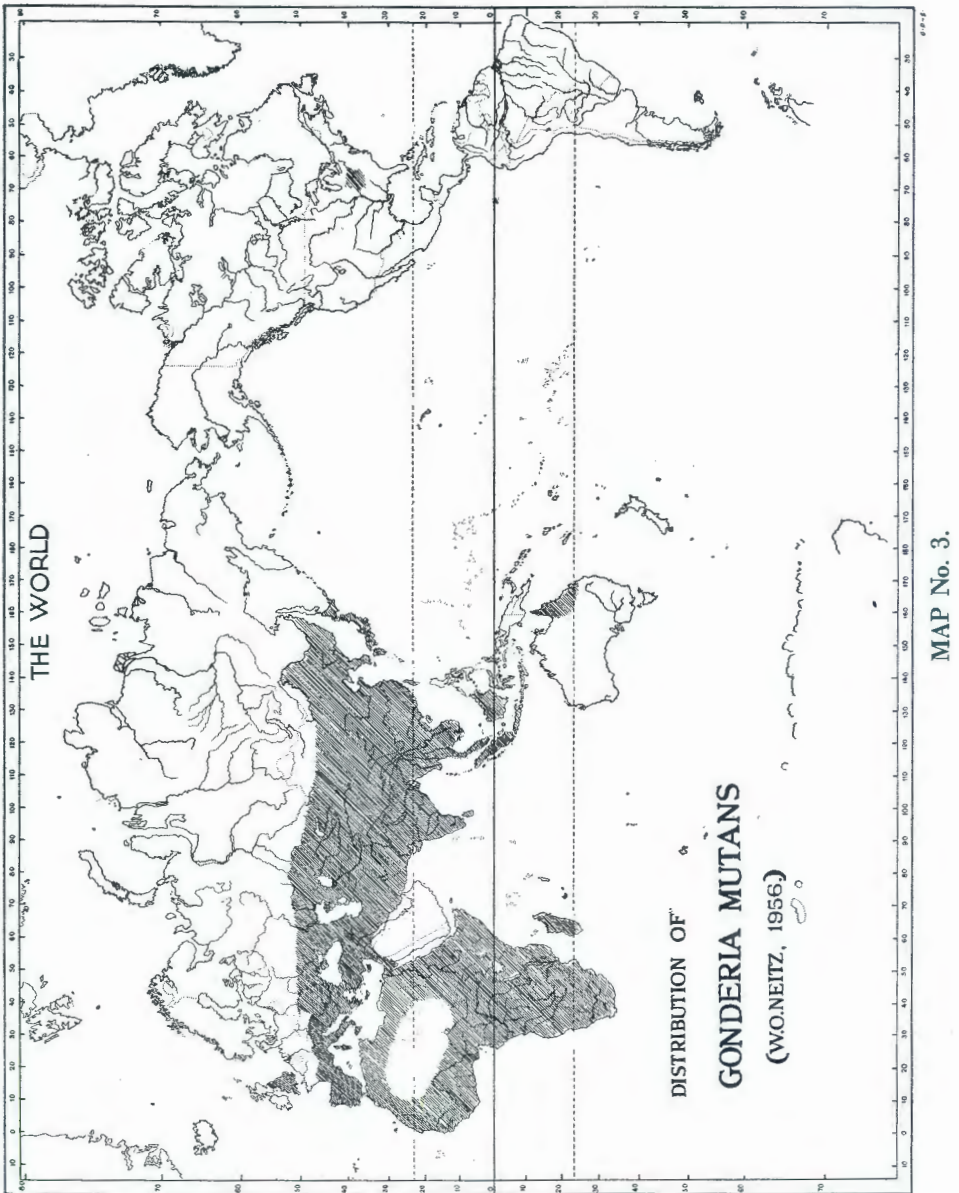
(ii) Histiotropic parasites:— In spleen and lymphatic gland smears fixed with May-Grünwald and stained with Giemsa, the schizonts (Koch bodies) appear as masses of blue staining cytoplasm containing one to eighty reddish purple dots varying from 1·0 to 2·0 microns in size. The Koch bodies vary in size from 1·0 to 10·0 microns and in some cases they may be up to 20·0 microns in diameter. The average size is 8·0 microns. They are seen either free or within the lymphocytes. Schizonts commonly referred to as agamonts (macroschizonts)

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are the type commonly seen, while gamonts (microschizonts) are not readily demonstrable. Although mature Koch bodies liberating merozoites have not yet been encountered, there is every reason to believe that this stage also occurs in the development of *G. mutans* otherwise the lymphocytes and erythrocytes would not become parasitized.

(*b*) *Multiplication.*—*G. mutans* 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 du Toit (1918) multiplication also occurs within the erythrocytes. Division into two, giving rise to two daughter cells (Sergent, Donatien, Parrot and Lestoquard, 1945), or alternatively into four takes place (du Toit, 1918) resulting in the cross forms, in which four minute pear-shaped individuals radiate from a central point.

(*c*) *Habitat.*—The erythrocytic stages of *G. mutans* can readily be demonstrated in blood smears for periods of up to four weeks after recovery. More than ten per cent of the erythrocytes may be parasitized. The host cells may harbour 1 to 6 micro-organisms. In premune animals it is usually difficult to demonstrate parasites. Splenectomy of such animals is followed by a relapse within three weeks after the operation. Smear examination reveals that more than 50 per cent of the red blood cells may become parasitized with one or more organisms. Schizonts can as a rule only be demonstrated in small numbers in spleen and lymphatic gland smears. In acute cases, however, they appear in fairly large numbers, and are demonstrable in blood and organ smears.

(*d*) *Life-cycle.*—When the ears of susceptible cattle are infested with *G. mutans* infective *Rhipicephalus appendiculatus* adult ticks, Koch bodies appear in the parotid lymphatic glands 9 to 22 days later. Two to three days later, schizonts can also be found in the other superficial lymphatic glands. They are demonstrable for periods varying from three to seven days. Erythrocytic stages appear in the peripheral blood 7 to 30 days after the first appearance of Koch bodies, and persist in the host throughout life.

The writer established in a series of experiments that the erythrocytic parasites can maintain themselves in the vertebrate host in the complete absence of the schizonts. This phenomenon was determined in the following way:— Blood from a calf harbouring a pure infection of *G. mutans* was injected into a fully susceptible splenectomized calf. Four weeks later parasites appeared in the peripheral circulation. Clean *Rh. appendiculatus* nymphae were allowed to feed on the ears of this animal. Four months later the ears of this calf were reinfested with the ensuing adults. After an incubation period of 12 days, this calf developed a thermal reaction which persisted for five days. Koch bodies could be demonstrated in the markedly swollen parotid and moderately swollen prescapular and precrural glands. The same results were obtained when the experiment was repeated on another two splenectomized calves. Consideration of these results permits the conclusion that the erythrocytic stage of *G. mutans* can maintain itself in the vertebrate host in the complete absence of schizonts, and that the latter stage confers the immunity.

No attempts have yet been made to study the development of *G. mutans* in the intermediate host.

TABLE XII.

The Biological Transmission of *Gonderia mutans*.

| Vector. | Country. | No. of hosts. | L. | N. | I. | E. | L. | N. | I. | References. |
|---|---|---|---|---|---|---|---|---|---|---|
| *Boophilus annulatus* (Say) | Germany with tick from the U.S.A. | 1 | ?X | ?X | ?X | | ?) | ?) | ?) | Miessner according to Reichenow (1935). |
| *Rhipicephalus appendiculatus* Neum. | South Africa | 3 | | X—) X—) X—) | X—) X—) X—) | | | | | Theiler (1907, 1909). De Kock, van Heerden, du Toit and Neitz (1937). Neitz and Jansen (1950). Neitz (1938). |
| *Rhipicephalus evertsi* Neum. | South Africa | 2 | X— | X— | X—) | | | | | Theiler (1907, 1909). |

TABLE XIII.

Domestic and Wild Members of the Family Bovidae Susceptible to *Gonderia mutans*.

| Host. Vernacular Name. | Host. Zoological Name. | Country. | Observations. | References. |
|---|---|---|---|---|
| Cattle (European, Asiatic and African breeds including the Zebu cattle) | *Bos* spp. | Africa, Asia, Australia, America Europe | Naturally and artificially infected | Theiler (1906, 1907, 1909); Knuth and du Toit (1921); Sergent *et al.* (1945). |
| Indian Water Buffalo | *Babalus bubalis* Linn. | Indo-China (Annam) | Naturally and artificially infected | Schein (1908); Galliard and Cébe (1941, 1949). |
| African Buffalo | *Syncerus caffer* Sparrman | Kenya / South Africa / Zululand (S. Africa) | Artificially infected / Artificially infected / Naturally infected | Walker (1932). Neitz (1940). Neitz (1956). |
| Sheep | *Ovis aries* Linn. (Only Koch bodies could be demonstrated in splenectomized sheep.) | South Africa | Infested with infective *Rh. appendiculatus* adults. | Neitz (1956). |
| Goat | *Capra hircus* Linn. (Only Koch bodies could be demonstrated in splenectomized goats.) | South Africa | Infested with infective *Rh. appendiculatus* adults. | Neitz (1956). |

(e) *Cultivation.*—No information is available on the cultivation. It may be possible to grow Koch bodies of *G. mutans* 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 four days at room temperature. Sergent *et al.* (1945) state that *G. mutans* remains viable for three weeks in citrated blood stored at 0° C.

(g) *Biological characteristics.*—The occurrence of immunologically different strains of *G. mutans* has not yet been established.

(h) *Isolation of pure strains of G. mutans.*—The writer has frequently observed that when clean *Rh. appendiculatus* nymphae feed on cattle harbouring a microscopic *G. mutans* infection and a submicroscopic *Babesia bigemina* and *Anaplasma* infection, the ensuing adults only transmit the former parasite. The isolation of a pure *G. mutans* strain by this procedure has greatly facilitated studies on benign gonderiosis.

## Transmission.

### A. Natural transmission.

(a) *Biological transmission.*—Ticks responsible for the transmission of *G. mutans* are listed in the appended Table XII. In the case of the two *Rhipicephalus* spp. stage to stage transmission within the same generation occurs. Reichenow (1935) states that Miessner obtained *Boophilus annulatus* adult ticks from the United States of America, and succeeded in transmitting *G. mutans* with their progeny in Germany. Sergent, Donatien, Parrot and Lestoquard (1945) failed to transmit *G. mutans* with *Hyalomma mauretanicum* Senevet (= *Hyalomma detritum* Schulze) and *Rhipicephalus bursa* Canestrini and Fanzago, in Algiers.

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

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

### B. Artificial transmission.

The erythrocytic stages of *G. mutans* can be transmitted readily by means of infective blood and organ emulsions by the intravenous, subcutaneous and intraperitoneal route (Theiler, 1906; Theiler and Graf, 1928; de Kock *et al.,* 1937). Benign gonderiosis has been successfully transmitted by du Toit (1930). Blood from a calf suffering from anaplasmosis and benign gonderiosis was injected into two susceptible calves. Both animals contracted anaplasmosis, and also showed the erythrocytic stages of *G. mutans* in their blood. In the course of a severe anaplasmosis reaction a few Koch bodies were found in the lymph glands in one of the calves about four weeks after the injection.

## Epizootology.

The limited knowledge about the vectors of *G. mutans* makes it impossible to give a general account of the epizootology of benign bovine gonderiosis in the different countries listed in Table XI. It is nevertheless believed, that a discussion based on observations made in South Africa, where not only the vectors but also the incidence and distribution of *G. mutans* are known, will lead to a clearer understanding of the epizootology.

Du Toit (1931) is of opinion that practically 100 per cent of cattle maintained in Transvaal, Natal, Transkei and Eastern Cape Province, harbour a latent infection of *G. mutans*. In these enzootic areas young calves contract benign gonderiosis which renders them premune. This is reflected in the relatively low incidence of this disease in adult cattle born and bred in tick infested areas, as compared with the high morbidity rate in stock introduced from areas such as the Orange Free State and Western Cape Province, where benign gonderiosis occurs sporadically.

Benign gonderiosis often occurs in association with either paratyphoid, babesiosis, anaplasmosis and heartwater. In these circumstances it is difficult to estimate the direct losses due to benign gonderiosis unless post mortem examinations and systematic smear examinations are made from all animals that die within an area. Diesel, van Heerden, Daly, Canham, Williams, Nicol and Blomefield (1956) have collected such data from the above-mentioned enzootic areas, where stock-owners regularly submit smears from their dead cattle. They point out that the annual losses from tick-borne diseases remain constant from year to year, and that the mortality, possibly due to benign gonderiosis, is less than three per cent of that resulting from the other tick-borne diseases. Of the 250,000 spleen smears examined annually about 350 reveal Koch bodies, the schizonts of *G. mutans*. Consideration of these facts shows that the mortality due to benign gonderiosis is negligible.

The high incidence of the virulent form of benign gonderiosis (Tzaneen disease) described by de Kock *et al.* (1937) is attributable to several factors. The experimental animals were exposed to a massive tick infestation on the townlands of Tzaneen in the Northern Transvaal. Besides contracting a *G. mutans* infection, many of the cattle also developed either babesiosis, anaplasmosis or heartwater. Under the stress of the concurrent infections, *G. mutans* became activated, and this resulted in the development of lesions simulating those commonly seen in East Coast fever.

It is anticipated that the epizootology of benign gonderiosis in Northern and Southern Rhodesia, Nyasaland, Portuguese East Africa, Central and East Africa is similar to that of South Africa. This assumption is based on the fact that the two known vectors, *Rh. appendiculatus* and *Rh. evertsi,* also occur in these territories. A comprehensive account of the epizootology in the remaining countries listed in Table XI will only become possible when the vectors have been determined.

### *Pathogenicity.*

It has been determined that cattle (Theiler, 1906) the Indian water buffalo (Galliard and Cébe, 1941, 1942) and the African buffalo (Walker, 1932; Neitz, 1940, 1956) are susceptible to *G. mutans*. The mortality rate in cattle is less than one per cent. No deaths have yet been recorded in either of the two buffalo species (see Table XIII).

De Kock *et al.* (1937) reported that cattle and sheep developed a febrile reaction accompanied by swelling of the superficial lymphatic glands 12 to 18 days after infestation with adult *Rh. appendiculatus* drag-ticks, gathered from the townlands of Tzaneen in the Northern Transvaal. Examination of gland smears from cattle showed Koch bodies in relatively small numbers, while those from the sheep failed to reveal any schizonts. The cause of the reactions manifested by the sheep has been reinvestigated recently by the writer. *G. mutans*

infective *Rh. appendiculatus* adult ticks were allowed to feed on two splenectomized sheep and two splenectomized goats. These animals were known to be free from blood parasites. After an incubation period varying from 12 to 16 days a fever, which persisted for several days, developed in all the animals. Smears prepared from the swollen superficial lymphatic glands showed a small number of Koch bodies. Blood smears were examined daily for a period of eight weeks but the erythrocytic stages of *G. mutans* were never seen. It thus appears that sheep and goats are only susceptible to the sporozoites and schizonts of *G. mutans*. The inference is drawn that these animals are imperfect hosts, and that they cannot serve as reservoirs for the infection of ticks.

### *Pathogenesis.*

The symptoms and lesions following a natural infection suggests that *G. mutans* liberates a toxin.

### *Symptomatology.*

Studies made on naturally infected cattle in South Africa have shown that the disease is nearly always mild. However, an acute form of the disease can be expected in cattle from non-enzootic areas after exposure to a massive tick infestation in enzootic regions. In these circumstances other tick-borne diseases can serve as complicating factors, and are often responsible for the mortality (de Kock *et al.*, 1937). Observations on the infection in sheep and goats after tick infestation have so far only been made under laboratory conditions. These animals have always shown a mild form of the disease.

The incubation period following tick infestation varies from 14 to 22 days with an average period of 17 days. After an artificial infection the period usually varies from 28 to 42 days but it may be less than 10 days and as long as 73 days. As a rule only the erythrocytic stages of *G. mutans* are demonstrable. In a single instance du Toit (1930) observed Koch bodies in the glands of a calf four weeks after injection of infective blood. Sheep and goats are refractory to the erythrocytic stages of *G. mutans* (*vide supra*).

(i) *Mild form.*—Cattle, sheep and goats, infested on the ears with infective *Rh. appendiculatus* adults show swelling of the parotid lymphatic glands 8 to 15 days later. Hyperthermia (104-106° F) commences after about 7 days, and persists for 2 to 8 days. Two to three days after the initial rise in temperature inappetence and listlessness accompanied by swelling of the prescapular and precrural lymphatic glands are observed. Koch bodies are found fairly easily in the parotid lymphatic gland but usually only after a prolonged search in the remaining glands. In non-splenectomized cattle the erythrocytic stages of *G. mutans* are demonstrable in the peripheral blood 20 to 40 days after the initial rise in temperature, while in splenectomized animals they may be found as early as 7 days after the first appearance of Koch bodies in the lymphatic glands. When the fever subsides the lymphatic glands decrease in size. A mild anaemia may develop in cattle. Animals make a rapid recovery.

(ii) *Acute form.*—Exposure to a massive tick infestation, during summer and autumn, is followed three to four days later by an irregularly intermittent fever which persists for about 14 days. It then becomes continuous (105-107° F) for 7 to 15 days. In animals recovering

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from the disease, the fever again becomes irregularly intermittent for periods of up to 40 days. A striking feature is that within a few days after exposure the whole body is infested with a variety of ticks, particularly *Rh. appendiculatus*. The ears become markedly swollen, start suppurating and get fly-struck. The myiasis and necrosis may cause partial or complete sloughing of one or both ears. Affected animals show anorexia, drooling from the mouth, lachrymation, sometimes ophthalmia, listlessness, marked loss in condition, general weakness and often diarrhoea. Anaemia and icterus may be present. Smears prepared from the markedly swollen superficial lymphatic glands show a variable number of Koch bodies. Blood smears may reveal the presence of *G. mutans* and Koch bodies. The disease may terminate fatally in three to six weeks after exposure. However, the majority of cases recover but four to eight weeks elapse before animals regain their former condition.

The acute form of the disease is often complicated by other tick-borne diseases either during the reaction or after recovery. High mortality can be expected unless specific and symptomatic treatment of the concurrent infections is applied.

## Pathology.

The lesions vary according to the duration and severity of the disease.

A. *Macroscopical lesions.*

In the mild form of the disease swelling of the lymphatic glands and spleen and an anaemia are the only lesions that can be expected at autopsy.

Studies on the pathology of the acute form have been recorded by de Kock *et al.* (1937). Emaciation and anaemia sometimes accompanied by icterus are observed. The subcutaneous and intermuscular tissues may be infiltrated with clear serous fluid, giving them a gelatinous appearance.

The myocard is flabby, and a variable number of petechiae appear on the epicardium and endocardium. Hydrothorax and hydropericardium are not constantly present. The lungs may show slight hyperaemia and oedema. The visceral and parietal pleura may be spotted with a few petechiae. The liver is enlarged, soft, friable and brownish yellow in colour. The gallbladder may be markedly distended with dark green bile. The kidneys appear moderately hyperaemic and in very rare instances reveal isolated hyperplastic lymphoid nodules commonly referred to as " infarcts ". The spleen is moderately swollen; the Malpighian corpuscles are fairly prominent. The lymphatic glands are swollen and oedematous. Some of these, particularly the parotid and prescapular lymphatic glands, are hyperaemic and in addition show localized haemorrhages. The urinary bladder is usually distended with apparently normal or bile-stained urine.

The rumen contains a relatively small amount of ingesta, while the contents of the omasum are firm and partially dehydrated. The mucous membrane of the abomasum shows a variable number of localized ulcerative lesions. Irregularly distributed red patches may be seen in the intestinal mucosa. The contents may be mixed with bile. The colon and rectum contain firm or fluid faecal material and a variable amount of mucus.

B. *Microscopical lesions.*

De Kock *et al.* (1937) have given a brief account of the histopathological changes in the acute form of *G. mutans* infection. The main lesions are observed in the liver, kidney, spleen and lymphatic glands. Lymphoid hyperplasia may be evident in the periphery of the liver lobules, and around the blood vessels in the cortex of the kidneys. In some instances an increase in the number of cells of the lymphocytic series is seen in the adventitia of the larger blood vessels between the cortex and medulla.

The lymphoid tissue in the Malpighian corpuscles of the spleen is distended by a " homogeneous material " of unknown nature. It invades the lymphoid tissue and appears like a reticular network. It causes disorganisation of the lymph nodules, and reduction in the number of cells of the lymphocytic series. This " homogeneous material " may also extend into the pulpa of the spleen.

In the lymphatic glands there is a reduction in the number of lymph nodules. The demarcation between primary and secondary nodules is no longer clearly visible. The nodules may disappear completely and are then replaced by the same type of " homogeneous material " as observed in the spleen. This material may also extend into the adjacent tissues and finally occupies the whole gland. There is also a reduction in the number of small lymphocytes and an infiltration of neutrophiles.

### Diagnosis.

The clinical symptoms presented by cattle suffering from benign gonderiosis are such that they may be confused with those occurring in the early stages or in atypical forms of either East Coast fever, Corridor disease, Rhodesian malignant bovine gonderiosis and tropical gonderiosis. It is essential that blood and gland smears be examined for the presence of Koch bodies and endoglobular parasites. Their presence signifies that the animal is suffering from one or other of the above-mentioned diseases. Since the final diagnosis is dependent upon the epizootology, pathogenicity, symptomatology, pathology and the frequency with which Koch bodies and endoglobular parasites appear in blood and organ smears, the disease should be allowed to run its normal course.

Consideration of the distribution of benign gonderiosis permits the division of the enzootic regions listed in Table XI into three fairly well-defined zones. Zone No. 1 includes West Africa, Australia, England and the United States of America in which only *G. mutans* occurs; Zone No. 2 includes Central, East and Southern Africa in which *G. mutans* may occur in association with *Th. parva, G. bovis* and *G. lawrencei;* Zone No. 3 includes North Africa, Southern Europe and Asia in which *G. mutans* may occur in association with *G. annulata.*

The demonstration of Koch bodies and/or endoglobular parasites in cattle, born and bred in Zone No. 1, permits the diagnosis of benign gonderiosis. In Zones No. 2 and 3 the above-mentioned criteria should always be considered before a final diagnosis is made. Benign gonderiosis very rarely terminates fatally in contradistinction to East Coast fever, Corridor disease, Rhodesian malignant bovine gonderiosis and tropical gonderiosis where the mortality is high. Examination of blood and organ smears shows that Koch bodies occur in relatively small numbers in benign gonderiosis, *G. bovis* infection and Corridor disease. A differential diagnosis can nevertheless be made if the average

size of the Koch bodies is taken into account. The average diameter of Koch bodies of *G. mutans* is 8·0 microns, and that of those of *G. lawrencei* and *G. bovis* 5·0 microns. The demonstration of large numbers of Koch bodies and endoglobular parasites signifies that the affected animal reacted to either East Coast fever or tropical gonderiosis.

Difficulty in making a diagnosis is experienced on farms where rigorous tick control measures are practised, and where the demonstration of Koch bodies in an affected animal suggests that death or recovery may have resulted from an atypical form of either East Coast fever, *G. bovis* infection or Corridor disease (Zone No. 2), or tropical gonderiosis (Zone No. 3). These prophylactic measures usually prevent the spread of any one of these infections, and hence preclude confirmation of the tentative diagnosis. In these circumstances the farm should be placed under quarantine. Movements of cattle and/or buffaloes to and from the farm should be traced in order to determine whether an accidental introduction of any one of the above-mentioned diseases could have taken place. It should also be established whether or not any one of the suspected diseases had occurred previously on the farm. If such possibilties can be excluded a diagnosis of benign bovine gonderiosis is assured. The farm is then released from quarantine. Instances, however, do occur where the diagnosis, based on the history and epizootological observations, remains inconclusive. In these circumstances cross-immunity tests have to be resorted to before a final diagnosis can be made. This procedure has been applied in many instances in the Union of South Africa (Zone No. 2). It involves transferring recovered animals to the Onderstepoort Veterinary Institute where *Th. parva* and *G. lawrencei* infective *Rh. appendiculatus* nymphae and adults are maintained. The immunity is challenged with infective ticks, and in the event of animals reacting to either East Coast fever or Corridor disease, a diagnosis of benign bovine gonderiosis is made.

In applying cross-immunity tests for diagnostic reasons in Zone No. 3, it should be remembered that immunologically different strains of *G. annulata* occur in nature. It is, therefore, essential to employ infective ticks harbouring the regional strain of this protozoon, otherwise a faulty interpretation may be given to the results of such tests.

The carrier state of *G. mutans* can be determined by examining blood smears or subinoculating blood into susceptible cattle immune to redwater, gallsickness and heartwater. The latter procedure will involve examining blood smears of the recipient for periods of up to 75 days before a final diagnosis can be made.

## Treatment.

A. *Specific treatment.*

Treatment is as a rule not necessary. In the " acute form " of the disease it is self-evident that treatment of concurrent infections such as redwater, gall-sickness and heartwater may have to be undertaken, as these diseases often occur where cattle have been exposed to a massive tick infestation.

It has been determined that trypan blue (Stirling, 1927), Bayer 205 (Brumpt and Lavier, 1922), phenamidine isethionate (Randall and Laws, 1947), the quinoline preparations (Acaprin, Babesan, Pirevan and Piroparv), the acridine compounds (trypaflavin, acriflavin and Gonacrine) and the sulphonamides (uleron, sulphamezathine and solupyridine) have no specific action on the endoglobular parasites of *G. mutans* (Neitz, 1956). Ishii and Ishiqara (1948) claim that the quinoline derivative " Tropochin " is of value in the treatment of *G. mutans* infection in Japan.

The 8-aminoquinoline derivatives, Pamaquin and Plasmoquin (Neitz, 1951) and Pentaquin (Neitz, 1956) have a specific action on the erythrocytic stages of *G. mutans*. When four successive doses of 0·5 mg. per Kg. body weight are administered intravenously or intramuscularly at 48-hour intervals, the parasites disappear temporarily for about four weeks from the peripheral blood. Affected parasites are either contracted or devoid of their cytoplasm. The nuclear rests resemble *Anaplasma marginale* in appearance.

It needs to be determined whether the repeated administration of Aureomycin during the incubation period of a sporozoite-induced *G. mutans* infection will suppress the active development of schizonts as in the case of East Coast fever (Neitz, 1953).

B. *Symptomatic treatment.*

This form of treatment is necessary where a massive tick infestation is followed by suppuration, abscessation and myiasis of the ears and other parts of the body. The application of non-irritant insecticides and the treatment of wounds is of great value.

*Prognosis.*

The prognosis in uncomplicated cases of benign bovine gonderiosis is always favourable.

*Prophylaxis.*

(*a*) *Elimination of arthropod vectors.*—Methods employed for the destruction of ticks in East Coast fever enzootic areas (*vide supra*) are equally effective for the control of benign bovine gonderiosis. If calves are required for experimental work the following procedure should be adopted: Pregnant cows should be either dipped or sprayed and handdressed three times at weekly intervals with reliable acaricides, and then transferred to a tick-free stable. Hay for the animals must be autoclaved to render it free from ticks. Before employing calves, born and bred under these conditions, for experiments it may even be necessary to splenectomize them, and to examine blood smears, for a period of at least a month, to exclude the possibility of an accidental intra-uterine transmission of *G. mutans.*

(*b*) *Quarantine measure.*—This prophylactic measure is only applied in enzootic and potential enzootic East Coast fever areas where the demonstration of Koch bodies does not permit making a differential diagnosis of benign gonderiosis. The farm is placed under quarantine for a period of three months or longer. During this period the movements of cattle and buffaloes to and from the farm are traced. The farm is released from quarantine provided the history shows that the disease was in no way related to East Coast fever (*vide supra*—Diagnosis).

(*c*) *Immunization.*—No vaccine is available for the immunization of cattle against benign gonderiosis. It is, however, recommended that cattle from lightly tick-infested areas be immunized against heartwater, redwater and gallsickness before introducing them into the enzootic areas. This procedure will reduce the development of complicated cases of benign bovine gonderiosis.

## Immunity.

Naturally recovered animals develop a durable premunity. The erythrocytic stages of *G. mutans* persist throughout life (du Toit, 1930). It needs to be determined whether Koch bodies are retained for the same period. This question is raised because it has been established that the erythrocytic stages can multiply and maintain themselves in the vertebrate host in the complete absence of schizonts (*vide supra*—Aetiology). The infestation of animals, only harbouring a latent infection of endoglobular parasites, with infective *G. mutans* ticks is followed by a typical benign gonderiosis reaction. This permits the conclusion that there is no immunogenic relationship between the erythrocytic parasites and the schizonts, and that the latter stage is responsible for the immunity. The demonstration of erythrocytic parasites, therefore, merely signifies that the animal has been exposed to infective ticks, but it does not necessarily imply that a solid immunity is present.

Splenectomy of *G. mutans* carriers is invariably followed by a relapse within three weeks after the operation (de Kock and Quinlan, 1926; Quinlan, de Kock and Marais, 1935; Sergent *et al.*, 1945; Neitz and Jansen, 1956). Endoglobular parasites appear in large numbers, and more than 25 per cent of the red blood cells may be parasitized. De Kock *et al.* (1937) record a case where splenectomy of a naturally infected calf resulted in the reappearance of *G. mutans* in the peripheral blood and of Koch bodies in the lymphatic glands. Premunity of *G. mutans* may be interrupted by babesioses, anaplasmosis, heartwater, rinderpest and other infectious diseases.

Animals that have recovered from a natural *G. mutans* infection are fully susceptible to *Th. parva* (Theiler, 1913; Neitz, 1938), *G. annulata* (Sergent *et al.*, 1945; Neitz and Jansen, 1952) and *G. lawrencei* (Neitz, 1956).

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## 4. CORRIDOR DISEASE.

### GONDERIA LAWRENCEI INFECTION.

#### Definition.

Corridor disease is a highly fatal peracute, acute or subacute tick-borne disease of cattle caused by *Gonderia lawrencei* (Neitz, 1955). It is characterised by pyrexia, anorexia, malaise, a variable degree of lymphadenitis, general weakness, prostration and pronounced dyspnoea before death. Recovered animals develop a durable premunity.

#### Synonyms.

Buffalo disease, Malignant syncerine gonderiosis; Buffelsiekte, Korridorsiekte (Afrikaans); Büffelkrankheit (German); Gonderiose à *Gonderia lawrencei* (French).

## History.

East Coast fever was introduced into Southern Rhodesia by a consignment of cattle from Tanganyika in 1901. The cause of the high mortality in cattle that occurred subsequently at Umtali and Salisbury was investigated by Koch (1903). The demonstration of minute, rod-shaped, oval and round erythrocytic parasites in affected cattle, caused him to make a diagnosis of East Coast fever, a disease previously encountered by him in Tanganyika in 1897. Subsequent to the discovery of plasma bodies in affected cattle (Koch, 1905, 1906), the diagnosis of East Coast fever was based on the presence of Koch bodies and endoglobular parasites in blood, spleen and lymphatic gland smears. As time progressed it became apparent that schizonts (Theiler and Graf, 1928) also formed part of the life-cycle of *Gonderia mutans.* This important observation compelled veterinarians also to take the epizootology and symptomatology into account in order to differentiate between *Theileria parva* and *Gonderia mutans,* the only two protozoa of the order Leucosporidea known to occur in cattle in Southern Africa at the time.

Lawrence (1933) described two outbreaks of a serious mortality amongst cattle under ranching conditions in Southern Rhodesia. Investigations by him showed that besides babesiosis, anaplasmosis, spirochaetosis, sweating sickness and myiasis, a fairly large number of affected animals also harboured Koch bodies. At the time he regarded these as being a stage in the development of *G. mutans.*

The following year Lawrence (1934) reported " that on certain apparently well defined areas of the Nuanetsi Ranch (Nuanetsi district in Southern Rhodesia) there is a heavy annual mortality amongst cattle. The disease responsible for this mortality has never been satisfactorily determined. Its effects are noticeable, as a rule, only from the commencement of the rainy season until the first frosts. The Ranch Manager and his staff stated that if cattle are moved from the area in which the disease had 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 ". Lawrence (*loc. cit.*) confirmed the observations of the Ranch Manager when he transferred some of the animals to new grazing sites. Mortality continued at its former high level amongst animals which were left in the affected area but completely ceased within a short while in the moved stock, and no cases of the disease developed in any of the in-contact cattle.

Lawrence (*loc. cit.*) attributed the presence of the relatively large number of Koch bodies in smears to *G. mutans* (= *Th. mutans*), and suggested that, owing to the operation of factors, probably associated with the undiagnosed disease, the resistance to the usual latent infection of *G. mutans* breaks down, and allows this organism to develop rapidly in the lymphocytic tissues of the animal.

In South Africa a disease, with symptoms identical to those described in Southern Rhodesia, was encountered by Neitz, Adelaar and Kluge (1953) in the Corridor, a stretch of country 100 square miles in extent, lying between the Hluhluwe and the Umfolozi Game Reserves. The former reserve is inhabited by a large number of buffaloes (*Syncerus caffer* Sparrman) and various species of other antelopes. An outbreak of this disease followed when 585 head of cattle were introduced into the Corridor, and within four weeks 300 animals

died. The mortality ceased within a period of three weeks after removing the cattle to sections of the Corridor not frequented by buffaloes. Subsequently it was observed that when the cattle, which included several Corridor disease recovered animals, had been retransferred to the farms of origin, approximately 30 miles away from the site of the enzootic, the disease failed to establish itself. Subsequent investigations showed that *Rhipicephalus appendiculatus* is a vector (Neitz, Canham and Kluge, 1955), and that the buffalo can serve as a reservoir for the infection of ticks (Neitz, 1955). It also became evident from the studies on the biology, morphology and the life-cycle of this parasite that the infectious agent is distinct from either *Theileria parva* or *Gonderia mutans*. The protozoon was named *Gonderia lawrencei* (Neitz 1955).

## Distribution.

The occurrence of Corridor disease has so far been diagnosed with certainty only in Southern Africa.

*Southern Rhodesia.*—Corridor disease (= Buffalo disease) has been recorded on the Nuanetsi Ranch and Liebig's Ranch in the Nuanetsi district.

*Union of South Africa.*—Corridor disease has been encountered in the province of Natal by Neitz, Adelaar and Kluge (1953), and Neitz, Canham and Kluge (1955) in the Hluhluwe Game Reserve and in the Corridor, and by Kluge and Daly (1955) in the Umfolozi Game Reserve. In the Transvaal the disease has been diagnosed by Joubert and Williams (1955) in the Barberton district, and by Van der Merwe and Williams (1956) in the Letaba district on farms adjacent to the Kruger National Park. All these outbreaks were associated with the African buffalo.

The writer is of opinion that Corridor disease also occurs in the Kisenyi area of the Belgian Congo. This assumption is based on the fact that spleen, lymph gland and blood smears submitted by Bugyaki (1955) from an affected ox revealed schizonts in relatively small numbers and morphologically indistinguishable from those of *G. lawrencei*. *Rh. appendiculatus* nymphae that fed in the preceding stage on this animal failed to transmit the disease to fully susceptible animals at Kisenyi and at Onderstepoort. These observations are identical with those made by Neitz, Canham and Kluge (1955), who also failed to transmit Corridor disease to susceptible cattle with *Rh. appendiculatus* nymphae and adults that had engorged in the preceding stages on affected cattle. It is possible that Corridor disease also occurs in the areas of Nyasaland and East Africa inhabited by the African buffalo. (See also Map No. 4.)

## Aetiology.

*Gonderia lawrencei* (Neitz, 1955).

Synonym :—*Theileria lawrencei* Neitz, 1955.

(a) *Morphology.*—(i) Erythrocytic parasites :— This stage has so far only been observed in buffalo calves. Morphologically the erythrocytic stages of *G. lawrencei* are indistinguishable from those of *G. mutans*. In blood smears fixed with May-Grünwald and stained with Giemsa, *G. lawrencei* appears in the red blood cells as pear-shaped, comma-shaped, oval or round 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 $1.8$ microns in length; oval forms $0.5$ micron in width and $1.0$ micron in length, and round forms $0.6$ to $2.0$ microns in diameter.
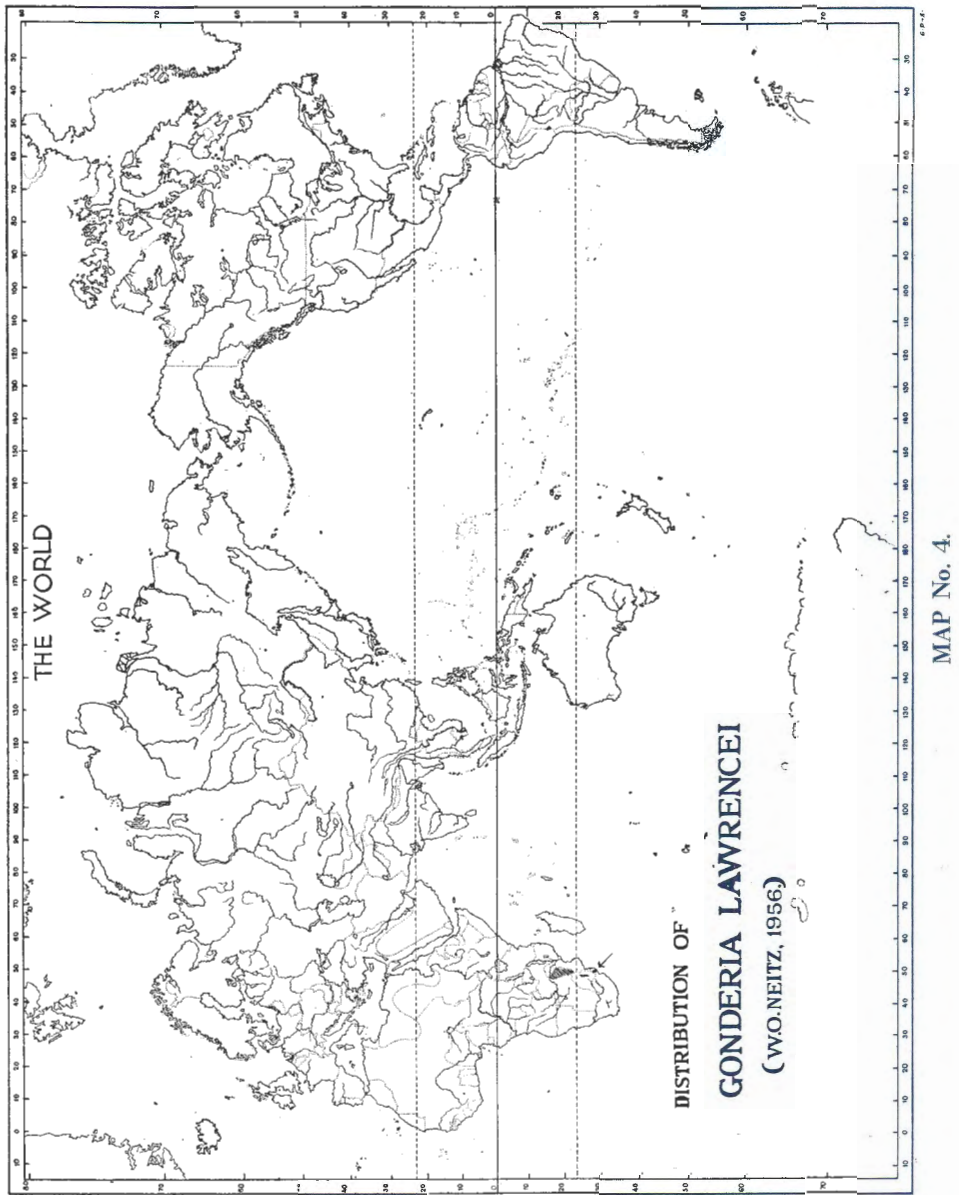
THE WORLD

DISTRIBUTION OF

GONDERIA LAWRENCEI

(W.O.NEITZ, 1956)

MAP No. 4.

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:—These forms have been observed in cattle by Lawrence (1934), Neitz, Adelaar and Kluge (1953) and Neitz, Canham and

### TABLE XIV.

#### The Biological Transmission of *Gonderia lawrencei*.

| Vector. | Country. | No. of Hosts. | L. | N. | I. | E. | L. | I. | N. | I. | References. |
|---|---|---|---|---|---|---|---|---|---|---|---|
| *Rhipicephalus appendiculatus* Neum. | Zululand. Onderstepoort. | 3 | X | X | | | | | X | | Neitz, Canham and Kluge (1955). Neitz (1955). Neitz (1955, 1956). |

### TABLE XV.

#### Domestic and Wild Members of the Family Bovidae Susceptible to *Gonderia lawrencei*.

| Host. | | Country. | Observations. | References. |
|---|---|---|---|---|
| Vernacular Name. | Zoological Name. | | | |
| Cattle. | *Bos taurus* Linn. | Southern Rhodesia. Zululand (Hluhluwe Game Reserve). Eastern Transvaal. | Naturally infected. Naturally infected. | Lawrence (1934). Neitz, Adelaar and Kluge (1953). Neitz, Canham and Kluge (1955). Joubert (1955). Van der Merwe (1956). |
| African Buffalo. | *Syncerus caffer* Sparrman. | Onderstepoort. Zululand. | Transmitted with infective ticks. Mild reaction. Two fatal cases in buffalo calves... | Neitz (1955, 1956). Kluge and Neitz (1956). |

Kluge (1955), and in two buffalo calves by Kluge and Neitz (1956). 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 $0 \cdot 5$ to $2 \cdot 0$ microns in size. The Koch bodies vary in size from $1 \cdot 0$ to $10 \cdot 0$ microns with an average of $5 \cdot 0$ microns. They are seen either free or within the lymphocytes. Two types of schizonts commonly referred to as agamonts (macro-schizonts) and gamonts (microschizonts) can be recognized. The latter type is not often seen. The common forms are the macroschizonts varying from $2 \cdot 0$ to $5 \cdot 0$ microns in diameter. When fully formed the macroschizonts liberate macromerozoites varying from $2 \cdot 0$ to $2 \cdot 5$ microns in diameter. The mature microschizonts liberate micromerozoites $0 \cdot 7$ to $1 \cdot 0$ 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.*—*G. lawrencei* 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 has 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 *G. lawrencei* have so far only been seen in two premune buffalo calves, and never in cattle (*vide infra*—Transmission). Less than one per cent of the erythrocytes are 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. The host cell may contain up to four schizonts.

(d) *Life-cycle.*—In the vertebrate host *G. lawrencei* 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.*—No attempts have yet been made to determine the action of these agents on *G. lawrencei*.

(f) *Biological characteristics.*—The occurrence of immunologically different strains of *G. lawrencei* has not yet been established. Cross-immunity tests, however, have shown that there is an immunological relationship between *G. lawrencei* and *Theileria parva* (*vide infra*—Immunity).

In the African buffalo sporozoite-induced infection results in *G. lawrencei* completing its life-cycle, and finally erythrocytic parasites appear which are capable of infecting ticks. In cattle, however, only the sporozoites derived from infective ticks and schizonts develop. Cattle can thus not serve as reservoirs for the infection of the vector.

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## Transmission.

A. *Natural transmission.*

(*a*) *Biological transmission.*—It has been established by Neitz, Canham and Kluge (1955) that adult *Rhipicephalus appendiculatus* drag ticks, gathered in the Corridor and the Hluhluwe Game Reserve, are capable of transmitting Corridor disease. In subsequent tests Neitz (1955) demonstrated that larvae and nymphae of *Rh. appendiculatus,* which had fed on a buffalo calf harbouring a latent infection of *G. lawrencei,* were able to transmit the disease in the ensuing stages (Table XIV). A single attempt to transmit the infection with *Rh. evertsi* adult ticks which had engorged in the preceding stages on the same buffalo failed.

Premune buffalo calves have been found to be excellent reservoirs for the infection of ticks with Corridor disease. Nymphae of *Rh. appendiculatus* which were allowed to feed on one of these animals thirteen months after its capture in the Hluhluwe Game Reserve in December 1954, proved to be infective. Infected nymphae remain infective for periods of up to three months, and infected adults for periods of up to seven months. (The end point has not been determined.

(*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.*

A single attempt to transmit Corridor disease to susceptible cattle by means of blood and organ suspensions from an affected animal failed (Neitz, Canham and Kluge, 1955). Trials to communicate the erythrocytic stage of *G. lawrencei* with blood of two premune buffaloes to two fully susceptible splenectomized calves were not successful. It thus becomes apparent that for biological transmission experiments of Corridor disease premune African buffalo calves should be available. (Older buffaloes are dangerous to handle.)

## Epizootology.

In common with other diseases which require an arthropod vector, Corridor disease is characteristically a disease of place. The density of infective ticks determines the incidence of the disease.

It has been proved beyond doubt by Neitz, Canham and Kluge (1955) and Neitz (1955) that the disease can be maintained in nature by premune buffaloes and the brown tick (*Rh. appendiculatus*). In these circumstances, the presence of Corridor disease becomes evident when cattle are introduced on to pastures frequented by buffaloes. Field observations in South Africa have shown that antelopes other than buffaloes do not serve as reservoirs for the infection of ticks. The appearance of Corridor disease in several bait animals used for tsetse fly surveys in the Umfolozi Game Reserve, and in an animal at the Mzimba Field Laboratory, was traced to small herds of buffaloes that had migrated from the Hluhluwe Game Reserve, Zululand (Kluge and Daly, 1955). Similarly it was found by Joubert and Williams (1955) and Van der Merwe and Williams (1956) that outbreaks of this disease in cattle followed when buffaloes from the Kruger National Park wandered on to adjacent farms. Kluge and Daly (1956)

have also shown that the exposure of 50 head of cattle in the Mkuzi Game Reserve (Zululand) inhabited by many species of antelopes other than the buffalo, was not followed by an outbreak of this infection. However, when these animals were transferred to a section of the Corridor in which buffaloes occurred, more than 50 per cent died from Corridor disease within four weeks after arrival. The remaining cattle were thereupon moved to pastures free from infective ticks and the mortality stopped within a short time.

Whether or not all areas, inhabited by buffaloes, are necessarily enzootic Corridor disease regions has not yet been determined. According to Diesel (1956) this disease has not yet been diagnosed in and around the Addo Forest (Eastern Cape Province) where cattle sometimes come in contact with the herds of the African buffalo.

In Southern Rhodesia, veterinarians and stock owners for many years have associated buffalo disease (Corridor disease) in cattle with the occurrence of the African buffalo in certain areas. However, Lawrence (1935-1953), Hooper-Sharpe (1936, 1938) and others have concluded from their epizootological observations that a form of " Theileriosis " is widely distributed in Southern Rhodesia, and that it occurs in cattle in the complete absence of the African buffalo.

It has recently been established by Lawrence and Neitz (1957) that *Rh. appendiculatus* adult ticks, which had fed as nymphae on an animal suffering from the Rhodesian form of " Theileriosis " ($=$ Rhodesian malignant bovine gonderiosis) transmitted this disease to an ox. This successful biological transmission is proof that the responsible protozoon is capable of completing its life-cycle in cattle, and that this parasite differs from *G. lawrencei* which is incapable of producing the erythrocytic stage in the bovine host (*vide supra*—Aetiology). A similar manifestation has been observed with *G. mutans*. This parasite is incapable of completing its vertebrate life-cycle in sheep and goats; only the sporozoites and schizonts of *G. mutans* develop in both these species but the erythrocytic parasites responsible for infecting ticks never appear (*vide supra*— Pathogenicity of *G. mutans*). The behaviour of *G. lawrencei* in cattle is very significant, and has a direct bearing on the epizootology of Corridor disease. It thus becomes apparent that this disease can only be maintained in nature by *G. lawrencei* premune buffaloes and the brown tick, *Rh. appendiculatus*. (It needs to be determined whether or not other species of ticks can also serve as vectors.)

Cyclical variations in the seasonal incidence of Corridor disease have been observed. In the enzootic regions of the Union of South Africa and Southern Rhodesia the highest incidence is during summer and early autumn. In the Corridor and Hluhluwe Game Reserve the disease has also been encountered in winter and in spring.

Corridor disease may occur in association with anaplasmosis, babesiosis, benign bovine gonderiosis, heartwater and sweating sickness. It has also been encountered within the enzootic East Coast fever areas. In these circumstances it is difficult to estimate the direct losses due to Corridor disease unless systematic smear examinations are made from all sick or dead animals within an area.

The veterinary authorities in Southern Rhodesia and South Africa have collected such data from potential East Coast fever areas, where stock owners regularly submit smears from all dead cattle. They point out that the annual losses from Corridor disease may be great unless prophylactic measures such as dipping, and removal of stock from infected pastures are practised. Prophylaxis against East Coast fever has undoubtedly been of enormous value in controlling Corridor disease.

### Pathogenicity.

Cattle and the African buffalo are both susceptible to Corridor disease (Table XV). The African buffalo is a " perfect host " because *G. lawrencei* can complete its vertebrate life-cycle in this animal. The ox on the other hand is an " imperfect host " since this protozoon is incapable of completing its mammalian life-cycle in this member of the family Bovidae (*vide supra*—Aetiology).

Observations made in Southern Rhodesia, and in the Union of South Africa have shown that calves are as susceptible as adult cattle. The mortality rate is approximately 80 per cent (Neitz, Canham and Kluge, 1955). Buffaloes are highly resistant. Deaths have so far only been recorded in two buffalo calves approximately two months old. When captured in the Hluhluwe Game Reserve they were found to be grossly infested with various species of ticks. They appeared to adapt themselves to their new surroundings but soon showed clinical symptoms of Corridor disease, and died within three weeks after having been caught. Lesions at autopsy resembled those seen in the fatal form of the disease in cattle (Kluge and Neitz, 1956). An extremely mild form of the disease, on the other hand, was observed by the writer in a fully susceptible year-old buffalo calf after infesting its ears with infective *Rh. appendiculatus* adults. A moderate swelling of the parotid lymphatic glands was noticed 19 days later. Koch bodies could be demonstrated on this day but not on subsequent days.

Attempts by the writer to transmit Corridor disease to sheep by means of infective *Rh. appendiculatus* adult ticks failed. The exposure of 20 sheep, derived from non-enzootic areas, in the Hluhluwe Game Reserve was not followed by the development of this disease in any of the animals (Kluge, Canham and Neitz, 1956).

### Pathogenesis.

The lesions present in the lungs, spleen, kidneys, myocardium, skeletal muscles, 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 a marked oedema of the lungs, and a variable degree of oedema of the subcutaneous tissues, and hydrothorax, hydropericardium and ascites.

### Symptomatology.

Corridor disease may be classified according to its symptoms into four types:—

(1) the peracute, (2) the acute, (3) the subacute and (4) the mild form.

After exposure to infective ticks the incubation period varies from 9 to 20 days with an average of 12 days.

(1) *The peracute form.*—This form is by no means uncommon, and is difficult to distinguish from the acute form of heartwater. The onset of the disease is sudden. The affected animal usually has a high temperature (105-106° F) but sometimes it does not exceed 104° F

throughout the course of the disease. The fever persists for three or four days and is accompanied by listlessness, drooping ears, lowered head, lachrymation, sometimes keratitis, swelling of the superficial lymphatic glands, muscular tremors, sluggish gait, marked drop in milk production, accelerated pulse and respiration and anorexia. The visible mucous membranes may be injected. Death occurs within three to four days after the initial rise in temperature. Before death the respiration is markedly accelerated, and the temperature becomes subnormal. The animal collapses. A large amount of froth exudes from the nostrils, and paroxysmal convulsions appear before death. Smears prepared from the blood and lymphatic glands during the course of the disease reveal a relatively small number of Koch bodies.

(2) *The acute form.*—This form is commonly encountered. The first indications of the disease is an elevated temperature (104-107° F) and the swelling of the parotid lymphatic glands. The fever may be continuous for five to fifteen days or alternatively the temperature returns to normal after five to ten days, and rises again after one to two days to 106° F, and remains at this level for a period of five to eight days. In cases where the disease persists for periods of up to 21 days, the primary febrile reaction is followed by an irregularly intermittent fever. The temperature becomes subnormal shortly before death. Clinical symptoms usually appear a few days after the initial rise in temperature. Koch bodies may be demonstrable in the parotid lymphatic glands one or two days before the commencement of fever. The animal shows inappetence, cessation of rumination, serous nasal discharge, lachrymation, keratitis, photophobia, drooping ears, sometimes swelling of the eyelids, ears, face and jowl region, swelling of the superficial lymphatic glands, muscular tremors, groaning, grinding of the teeth, swaying gait, partial or complete cessation of milk production and loss in condition. At the beginning of the pyrexial period the faeces are firm but diarrhoea often sets in four to six days later. The evacuations are frequently mixed with blood and mucus. The animal becomes markedly emaciated and is inclined to lie down, and when forced to rise it may cough. The apparent paresis of the hindquarters, which is sometimes seen, is due to degeneration of the adductor, semimembranosus, semitendinosus and gracilis muscles. 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 Corridor disease, particularly when it persists for longer than ten days, relapses due to *Babesia bigemina* and *Anaplasma marginale* appear. The symptoms of redwater and gallsickness may obscure the typical clinical manifestations of Corridor disease.

(3) *The subacute form.*—This form is encountered in about ten per cent of affected animals. 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 found in the lymphatic gland and blood smears. Cattle usually recover from this form but it may take several weeks before they regain their former condition.

(4) *The mild form.*—This form is of rare occurrence, and may be mistaken for benign bovine gonderiosis (*vide supra*). The symptoms are a relatively mild fever, which persists for three to five days, listlessness and a moderate swelling of the superficial lymphatic glands. This form has been observed in fully susceptible animals as well as in partially immune East Coast fever cattle after infestation with infective *G. lawrencei* ticks. In these cases Koch bodies are usually only demonstrable if lymphatic gland smears are prepared during the course of the febrile reaction.

## Pathology.

The lesions of Corridor disease are fairly uniform but may vary somewhat according to the duration and the severity of the disease. The pronounced oedema of the lungs regularly seen in the peracute form and in the acute form of short duration, may not be present to the same degree when the disease has a protracted course.

### A. *Macroscopical lesions.*

In the peracute form the carcase does not show emaciation. Oedema, hyperaemia and emphysema of the lungs are pronounced. Hydrothorax, hydropericardium and ascites are usually present. Serous infiltration of the subcutaneous and intermuscular tissues, mediastinum and the capsula adiposa of the kidneys is marked. The liver and kidneys are congested, and the spleen may be enlarged. The mucous membrane of the abomasum shows a variable degree of oedema, superficial erosions, however, are not as a rule evident. Petechiae may be present in the serous membranes, myocardium and in the mucous membrane of the bronchi and bronchioli. The superficial and internal lymphatic glands show a variable degree of swelling and hyperaemia.

The lesions present in the acute form with a relatively short course resemble those described in the peracute form very closely. In the acute form with a protracted course and in the subacute form, the carcase is usually emaciated. The skin may show decubital wounds. The perineal region and tail 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 may be seen in the abductor, semimembranosus, semitendinosus and gracilis muscles, and sometimes also in the trapezius and latissimus dorsi muscles.

The myocardium is flabby and a variable number of petechiae appear in the epicardium and endocardium. Hydrothorax and hydropericardium are not constantly present. The visceral and parietal pleura may be spotted with petechiae. The lungs show a variable degree of oedema and hyperaemia. The mucous membranes of the bronchi and bronchioli usually show a large number of petechiae. The liver may be increased in size, friable and brownish-yellow in colour; degenerative changes are evident. The gall bladder may be markedly distended with dark green viscid bile. The spleen is usually 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 brown in colour; haemorrhagic " infarcts " or greyish-white lymphomata are rarely seen in the cortex, and when present they are not so frequent and prominent as in typical cases of East Coast fever. The urinary bladder is usually markedly distended with clear yellow urine; haemorrhages may be seen 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 are firm and partially dehydrated. Superficial erosions are not always present in the mucous membrane of the abomasum. Similar erosions as well as irregularly disseminated red streaks or patches may be encountered along the entire length of the intestinal tract. Peyer's patches may be swollen. The contents of the small and large intestine have a distinct yellow tinge.

## B. *Microscopical lesions.*

The histopathological lesions consist of alveolar and interstitial oedema in the lungs, nephrosis, slight fatty degeneration of the liver, myocarditis, perivascular cuffing in the cerebrum and an ulcerative abomasitis. Severe muscular necrosis of various muscles may be present (Schulz, 1955).

### *Diagnosis.*

Epizootologically, *G. lawrencei* 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 Corridor disease and other diseases likely to be confused therewith, the occurrence of premune African buffaloes, and the origin of the sick animals. These considerations should guide the investigational procedures.

Although a tentative diagnosis of Corridor disease can be made by considering the epizootology, the clinical symptoms and lesions at autopsy, a definite diagnosis depends upon demonstrating the schizonts of *G. lawrencei* in blood and organ smears. The Koch bodies of *G. lawrencei* vary from $1 \cdot 0$ to $10 \cdot 0$ microns with an average diameter of $5 \cdot 0$ microns, and are morphologically indistinguishable from the schizonts of the *Gonderia* species responsible for the Rhodesian form of "Theileriosis". The Koch bodies of *Th. parva* and *G. mutans* vary from $1 \cdot 0$ to $15 \cdot 0$ microns with an average diameter of $8 \cdot 0$ microns. In Corridor disease and in the Rhodesian form of "Theileriosis" approximately five per cent of lymphocytes are parasitized with Koch bodies but mature microschizonts (gamonts) liberating merozoites are encountered only on rare occasions. The percentage of infected lymphocytes in benign bovine gonderiosis may be the same as in Corridor disease 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 Corridor disease (malignant syncerine gonderiosis) and Rhodesian "Theileriosis" (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* premune buffaloes occur in that region; in their complete absence a diagnosis of Rhodesian "Theileriosis" is justified. Should both diseases be suspected to occur in an area inhabited by buffaloes, the causal agents can be differentiated by making a xenodiagnosis (*vide infra*—Diagnosis of Rhodesian malignant bovine gonderiosis).

The *Gonderia* endoglobular parasites often encountered in cattle suffering from Corridor disease are, in the opinion of the writer, those of *G. mutans*, a parasite harboured by practically all cattle in the *Rh. appendiculatus* infested regions in Eastern and Southern Africa. This conclusion is based on the fact that Neitz, Canham and Kluge (1955) failed repeatedly to transmit *G. lawrencei* with *Rh. appendiculatus* adult ticks, which, in their preceding stage, had fed on

Corridor disease reacting animals harbouring a microscopic infection of *Gonderia* endoglobular parasites. These ticks transmitted benign bovine gonderiosis but not Corridor disease. Moreover, these investigators were not able to demonstrate endoglobular parasites in three fully susceptible calves during the course of a Corridor disease reaction which terminated fatally on either the eighth, tenth or fifteenth day after initial rise in temperature.

For the identification of *Gonderia* endoglobular parasites harboured by an animal, it is necessary to apply the xenodiagnosis. This diagnostic method was applied by the writer on a young buffalo calf, from the Hluhluwe Game Reserve, which harboured blood parasites morphologically indistinguishable from *G. mutans*. This calf was infested with clean *Rh. appendiculatus* larvae and nymphae. After moulting a large number (250 to 400) of the ensuing nymphae was allowed to feed on four calves raised under tick-free conditions. Two of them developed typical Corridor disease and died, while the remaining two contracted benign bovine gonderiosis and recovered. The tentative diagnosis, made in the latter two calves, was confirmed by challenging the immunity of one with Corridor disease infective ticks, and the other with East Coast fever infective ticks. Both animals reacted and died. (There is a very close immunogenic relationship between *G. lawrencei* and *Th. parva* so that ticks infected with either species can be employed for immunity tests.) The ensuing adult ticks, on the other hand, provoked a fatal form of Corridor disease in two calves. These results justified the conclusion that the buffalo calf harboured a mixed infection of *G. lawrencei* and *G. mutans*.

Consideration of these observations suggests that further work be conducted to determine the fate of either *G. lawrencei* or *G. mutans* within the vector when both parasites are ingested at the same time. It would appear that only one of the two parasites survives, and that this results in a pure infection within the vector. It is also remarkable that, when nymphae were picked out at random from a batch of ticks, as was the case in these experiments, they transmitted a pure infection of benign bovine gonderiosis to some calves, and Corridor disease to other animals. It needs to be determined whether or not in these circumstances infected ticks can harbour a pure *G. lawrencei* infection.

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*Treatment.*

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A. *Specific treatment.*

Chemotherapeutic studies based on those described in East Coast fever (Neitz, 1953) have shown that aureomycin is of value provided treatment is commenced during the incubation period. So far it has only been possible to conduct tests on a few animals at Onderstepoort.

For these tests six head of cattle were used of which three served as untreated controls. In each case the disease was induced by feeding *G. lawrencei* infective *Rh. appendiculatus* adult ticks on the ears of the animals. Treatment was commenced 72 hours later by the injection of aureomycin in a dose of 10 mgm. per Kgm. bodyweight. The same dose was repeated seven times at 48-hour intervals until a decrease in the number of schizonts became evident. In each case from this stage the number of schizonts continued to decrease rapidly until they could no longer be found after prolonged search. Apart from a mild febrile reaction and swelling of the superficial lymphatic glands the animals showed no clinical symptoms of the disease. After complete recovery it was shown that a high degree of cross-immunity existed when *Th. parva* infective *Rh. appendiculatus* nymphae were used for the challenge. One animal proved to be