# Arthropod Borne Protozoan Infections
(Affecting Domesticated, Food-Producing Animals)

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## Introduction

The arthropod borne protozoan infections include a wide range of infectious agents which are generally hemotropic in nature and are transmitted by blood sucking insects such as ticks, flies, mosquitoes, etc. Transmission may occur mechanically by the transfer of infected blood from one animal to another by insects or improperly cleaned surgical instruments. More often, biological vectors are involved, in which the causative agent undergoes cyclical development in the invertebrate host, prior to producing infection in the susceptible vertebrate host.

These protozoan agents may be extra- or intra-erythrocytic in nature, but are generally characterized by febrile reactions in the acute phase, varying degrees of anemia, and carrier infections following recovery.

Anaplasmosis will be considered in this section because of the many similarities between this disease and the arthropod borne hemoprotozoan infections. Recent studies suggest that the causative agent, *Anaplasma marginale*, is more closely related to *Rickettsia* than *Protozoa*, although some question still exists as to its correct classification.

Included under this heading are diseases caused by *Theileria*, *Trypanosoma*, *Anaplasma*, *Babesia*, *Leucocytozoon*, and *Besnoitia*. A list of agents with the disease they produce, animal species affected, and the principal vectors is given in Table 1. East Coast Fever (*Theileria*) and trypanosomiasis (*Trypanosoma*) have been discussed in detail in separate chapters. Babesiosis and anaplasmosis, major animal disease problems throughout the world, are discussed in some detail in this chapter.

TABLE I

| ORGANISM | ANIMALS AFFECTED | MORPHOLOGY OF ORGANISM | VECTORS |
|---|---|---|---|
| BABESIA (babesiosis) | | Intraerythrocytic | Ticks |
| B. bigemina | Cattle | 4.5μ X 2.5μ (Large, round and piriform; acute angle) | Boophilus annulatus, B. calcaratus, B. decoloratus, B. microplus, Haemaphysalis punctata, Rhipicephalus appendiculatus, R. bursa, R. evertsi. |
| B. bovis, B. berbera, B. argentina | Cattle | 2.4μ X 1.5μ (Small and more rounded; obtuse angle) | B. annulatus, B. calcaratus, B. microplus, Ixodes persulcatus, I. ricinus, R. bursa. |
| B. divergens | Cattle | 1.5μ X 0.4μ (Small, narrow and marginal location; obtuse angle) | Dermacentor reticulatus, Haemaphysalis punctata, I. ricinus. |
| B. major | Cattle | 2.6μ X 1.5μ (Similar to B. bigemina but smaller) | B. calcaratus, Haemaphysalis punctata, I. ricinus, probably Boophilus ticks. |
| B. motasi | Sheep and Goats | 3.0μ X 2.0μ (Large, piriform; acute angle) | D. silvarum, Haemaphysalis punctata, R. bursa. |
| B. ovis | Sheep and Goats | 1.5μ X 1.0μ (Small and more rounded; obtuse angle) | I. persulcatus, R. bursa. |

Table I Cont'd.

| ORGANISM | ANIMALS AFFECTED | MORPHOLOGY OF ORGANISM | | VECTORS |
|---|---|---|---|---|
| B. caballi | Horses | 3.0μ X 2.0μ (Large, pear-shaped; acute angle) | | D. marginatus, D. nitens, D. pictus D. silvarum, Hyalomma dromedarii, H. excavatum, H. marginatum, H. scupense, R. bursa, R. sanguineus. |
| B. equi | Horses | 1.0 - 2.0μ (Small and rounded; Maltese Cross is characteristic) | | Dermacentor spp., H. dromedarii, H. excavatum, H. marginatum, H. plumbeum, H. uralense, R. bursa, R. evertsi, R. sanguineus. |
| B. trautmanni | Swine | 3.5μ X 2.0μ (Large, narrow and long; acute angle) | | R. sanguineus, Dermacentor spp. and Hyalomma spp. |
| B. perroncitoi | Swine | 0.7 - 2.0μ (Small and more rounded) | | Vectors unknown. |
| | | Erythrocytic Forms | Lymphocytic Forms | |
| THEILERIA (theileriosis) | | Intraerythrocytic and Intralymphocytic | | Ticks |
| T. parva | Cattle | 1.8 X 0.75μ (Rod forms more common; very pleomorphic) | 8.0μ in diameter | R. appendiculatus, R. ayrei, R. capensis, R. evertsi, R. jeanelli, R. neavei, R. simus, H. anatolicum, H. dromedarii, H. truncatum. |
| T. annulata (Egyptian Fever of Cattle, Tropical Theileriosis, Mediterranean Coast Fever) | Cattle | 0.5 - 1.5μ (Oval forms more common) | Similar to T. parva | Hyalomma spp. |

Table I Cont'd.

| ORGANISM | ANIMALS AFFECTED | MORPHOLOGY OF ORGANISM | | VECTORS |
|---|---|---|---|---|
| | | Erythrocytic Forms | Lymphocytic Forms | |
| *T. mutans* (Tzaneen Disease) | Cattle (Generally non-pathogenic) | 1.0 - 2.0μ diameter (Mostly round and oval) | Rarely seen but similar to *T. parva* | *B. annulatus*, *Haemaphysalis bispinosa*, *Rhipicephalus spp.* |
| *T. lawrencei* (Corridor Disease) | Cattle and African Buffalo | Similar to *T. parva* | | Principally *R. appendiculatus*, *Rhipicephalus spp.*, probably the same as *T. parva*. |
| *T. hirci* | Sheep and Goats | 0.6 - 2.0μ (Mostly round and oval) | 4.0 - 10.0μ in diameter | *R. bursa*, *R. evertsi*. |
| *T. ovis* | Sheep and Goats (Generally non-pathogenic) | Similar to *T. hirci* | | *Ornithodoros lahorensis*, others are probably involved. |
| *TRYPANOSOMA* (trypanosomiasis) | | Extraerythrocytic | | Flies |
| *T. theileri* | Cattle (Non-pathogenic) | 25 - 120μ in length | | *Tabanids*. |

Table I Cont'd.

| ORGANISM | ANIMALS AFFECTED | MORPHOLOGY OF ORGANISM | VECTORS |
|---|---|---|---|
| *T. brucei* | Horses, camels, and dogs; cattle are relatively resistant | 15 - 35μ in length | Tsetse fly (*Glossina*). |
| *T. gambiense* | Man | Similar to *T. brucei* | Tsetse fly (*Glossina*). |
| *T. rhodesiense* | Man | Similar to *T. brucei* | Tsetse fly (*Glossina*). |
| *T. congolense* | Cattle | 9 - 18μ in length | Tsetse fly (*Glossina*). |
| *T. vivax* | Cattle, sheep and goats | 18 - 26μ in length | *Glossina*, *Tabanids* and other biting flies. |
| *T. evansi* (Surra) | Horses; but cattle are susceptible | 15 - 34μ in length | *Tabanidae* (horseflies) and other biting flies, also vampire bats. |
| *ANAPLASMA* (anaplasmosis, Gall Sickness) | | Intraerythrocytic | Ticks, mosquitoes and flies |
| *A. marginale* | Cattle | 0.3 - 1.0μ in diameter (Coccoid) | 20 species of ticks, 10 species of *Tabanus*; other flies and mosquitoes are potential vectors. |

Table I Cont'd.

| ORGANISM | ANIMALS AFFECTED | MORPHOLOGY OF ORGANISM | VECTORS |
|---|---|---|---|
| A. ovis | Sheep | 0.3 - 1.0μ (Coccoid) | Unknown - probably similar to A. marginale. |
| A. centrale | Cattle (Benign infection) | In size similar to A. marginale - distinguished by its central location in the erythrocyte | Thought to be similar to A. marginale. |
| LEUCOCYTOZOON (leucocytozoonosis) | | Intraerythrocytic | Flies or gnats |
| L. simondi | Domestic and wild ducks and geese | Gametocyte 14 - 15μ X 4.5 - 5.5μ | Simulium venustum, S. croxtoni, S. euryadminiculum, S. rugglesi. |
| L. smithi | Domestic and wild turkeys | Gametocyte 20 - 22μ X 6μ | S. occidentale, S. nigroparvum, and S. slossonae. |
| L. caulleryi | Domestic chickens | Gametocyte Round and about 15μ in diameter | Culicoides arakawe. |

# BABESIOSIS
## (Piroplasmosis, Texas Fever, Redwater, Tick Fever)

Definition: Babesiosis is a febrile, tick-borne disease of domestic and wild animals caused by protozoan parasites of the genus *Babesia* and characterized by extensive erythrocytic lysis leading to anemia, icterus, hemoglobinuria, and death.

Etiology: Babesiosis is associated with the appearance of the specific infecting *Protozoa* in the erythrocytes. Neitz (1956) recognized 17 distinct species of *Babesia* from various vertebrate hosts.

*Babesia bigemina*: This species causes one of the most important diseases of cattle in the tropics and subtropics. *Babesia bigemina* is a relatively large parasite; when ultrathin sections of infected erythrocytes were viewed by electronmicroscopy, the double membrane, nucleus, endoplasmic reticulum, and polar bodies were connected with a conoid structure which was located on the anterior part were found (Gonzalez *et al.*, 1971). The round forms are 2 to 3μ in diameter and the elongate ones 4 to 5μ long. In bovine erythrocytes, the parasites may assume various shapes, but the most characteristic is oval to pear-shaped. Paired *B. bigemina* usually diverge with an acute angle.

*Babesia bovis*: This species is the most important cause of European babesiosis. It is common in many regions of the world, but information on its true prevalence must await a decision as to whether *Babesia berbera* and *Babesia argentina* are synonymous. *Babesia bovis* is a small form measuring about 2.4 by 1.5μ (Davies *et al.*, 1958). The parasite usually appears to be on the periphery of the erythrocytes, with paired organisms diverging with an obtuse angle.

*Babesia divergens*: This species is smaller than *B. bovis*. The

organisms usually occur as paired, club-shaped organisms about 1.5 by 0.4μ; the angle of divergence of paired organisms is relatively large, so that they diverge more from each other than B. bovis; in addition, they tend to lie along the circumference of the host erythrocyte.

Babesia argentina: This species resembles, and may be synonymous with, B. bovis. Babesia argentina is more robust than B. bovis, the ring forms being commonly found. The paired forms are piriform with an obtuse angle, about 2.0 by 1.5μ, and usually lie slightly off center of the host erythrocyte.

Babesia major: This species also resembles B. bovis but is larger. The piriform, paired forms measure 2.6 by 1.5μ, and the round ones are 1.8μ in diameter. The parasites lie in the center of the host erythrocyte. It is distinguished morphologically from B. bovis by its elongated sub-piriform shape and by the fact that most paired forms meet at an angle less than 90°.

Babesia organisms are generally host-specific, and in addition to the bovine baberiosis the following parasites have been described in other species:

B. motasi, B. ovis, B. foliata, and B. taylori in sheep;

B. caballi and B. equi in horses;

B. trautmanni and B. perroncitoi in pigs;

B. canis and B. gibsoni in dogs;

B. felis in cats; and

B. rodhaini in mice and rats.

History: The classical investigations of Smith and Kilborne (1893) in the United States of America of the cause of Texas fever were the first to establish that a protozoan disease could be transmitted by an intermediate arthropod host; and their discovery that B. bigemina was carried by the

cattle tick, Boophilus annulatus, paved the way not only for the control of Texas fever, but also for the study of arthropod-borne diseases in general. Bovine babesiosis is of historical interest because it has now been virtually eliminated from the United States where it was once very prevalent. Its eradication was accomplished by eliminating the tick-vector, B. annulatus. Babesiosis is still present in many parts of the world.

Signs: Infections with B. bigemina and B. argentina are usually so similar that they can be distinguished only by microscopic examination of the blood. Moreover, mixed infections with these two parasites frequently occur. It is more convenient to deal with them together, emphasizing those differences characteristic of one or the other etiological agent. When bovine babesiosis is contracted from ticks, the incubation period is commonly two to three weeks; but when induced by the inoculation of infected blood, it is usually between five and 14 days, although it may be longer. There is a marked difference in susceptibility of individual cattle. Some cattle pass through an attack without showing any clinical signs; whereas, others become so severely affected that death may result. Calves may possess a natural resistance to the disease and show no clinical signs at all; yet, they may acquire an infection which protects them in later life. This resistance gradually diminishes with the age of the calf, so that by the time they are a year old they may be susceptible enough to suffer a severe attack. Well-bred bulls in good condition suffer severely, and the susceptibility of lactating cows and all pregnant and aged cows is high if they have never been previously exposed.

Development of high fever, up to 41.5° C. rectal temperature, is sudden in onset. There is anorexia and rumen atony. Often, the first

sign is that the animal isolates itself from the herd, becomes uneasy, seeks shade, and may lie down. Cattle may stand with an arched back, have a roughened hair coat, and show evidence of dyspnea, and tachycardia. The mucous membranes are first injected and reddened, but as erythrocytic lysis occurs, the color changes to the pallor of anemia. Anemia is a contributory factor to the weakness and loss of condition seen in cattle that survive the acute phase of the disease. The anemia may be very severe with up to 75% or more of the erythrocytes being destroyed.

When cattle are driven, they move with difficulty and they may stagger because of posterior weakness. Constipation is present in the early stages. Cattle suffering a severe reaction have hemoglobinuria and icterus. A decrease in milk production, which occurs with the initial rise of temperature, is generally the first sign noted in lactating cows. The febrile stage of the disease usually lasts about a week, and the course of the disease is about three weeks. Pregnant cows may abort. Calves may be less severely affected, pyrexia not being as severe, and hemoglobinuria being absent.

Under field conditions, mortality usually occurs in the third week. With B. argentina infections, there is usually more rapid development of clinical signs, such as higher fever, greater weakness, and earlier death, than with B. bigemina. The mortality rate may reach 90%, particularly in the hot weather, but in colder weather or when the disease is mild, the death rate is much lower. The average death rate is stated to be 60% in fully susceptible cattle. Death may be accelerated by forced exertion, and a sudden decrease in body temperature usually presages early death. Infection with B. argentina causes a higher mortality than with B. bigemina. In Australia, 70-80% of cattle infected with B. argentina, and only 30% with B. bigemina, develop a reaction severe enough to need chemotherapy. In cattle which survive, the temperature gradually decreases to normal and the appetite is

regained, but recovery is very slow and may take weeks or even months. Involvement of the central nervous system is not an unusual complication of babesiosis due to B. argentina. Among the clinical signs are depression, ataxia, convulsions, opisthotonus, mania, and finally coma.

Incubation Time: According to Riek (1968), the incubation period is 5 to 15 days but depends to some degree on the species of parasites involved.

Postmortem Lesions: Cattle dying from babesiosis show postmortem lesions which are somewhat characteristic and vary with the acuteness and severity of the clinical signs. In cattle that have died after a few days of high fever, the lungs may be edematous and congested. The pericardial sac contains some sero-sanguineous fluid and there are subepicardial and subendocardial petechial hemorrhages. The liver is enlarged and icteric, and the gall bladder, which may show hemorrhages on the mucous surface, is distended with thick, dark green bile. The spleen is markedly enlarged with a dark pulpy consistency. The abomasal and intestinal mucosa may be icteric with patches of subserosal hemorrhages. The blood is thin and watery; the plasma may be red tinged due to hemoglobinemia. The kidneys are congested, especially with B. argentina infection, and the urinary bladder distended with dark reddish-brown urine. Jaundice is commonly distributed in the connective tissue. The lymph nodes are edematous and often have petechiation.

In cattle which have suffered a more prolonged illness, acute lesions are much less conspicuous except that subepicardial petechial hemorrhages may be present. The carcass is usually emaciated and icteric; the blood is thin and watery; the intermuscular fascia edematous; the liver yellowish-brown in color and the bile contains flakes of semi-solid material.

The kidneys are pale and often edematous, and the bladder contains normal urine. Although the spleen is enlarged, the pulp is firmer than in the acute babesiosis.

Microscopic Pathology: The microscopic lesions are characteristic of an acute intravascular hemolytic anemia. Multicentric hematopoietic hyperplasia and metaplasia often occur in the spleen and bone marrow. Hepatic centrilobular necrosis may occur as a consequence of the anemia. The lymph nodes are hemorrhagic and edematous with reticulo-endothelial and lymphoid hyperplasia being prominent. The lymph nodes, tonsil, and spleen often have hemosiderosis, erythrophagocytosis, and hyaline degeneration of arterioles. The terminal hepatic veins of the liver may be engorged with leukocytes. In cases of central nervous system involvement, there may be perivascular lymphocytic infiltration of the cerebral and cerebellar capillaries which are engorged with parasitized erythrocytes, especially *B. argentina*. Cerebral and cerebellar congestion and edema are common lesions.

The erythrocytic count often decreases to less than 1 million at the time of death. Erythroid hyperplasia of the bone occurs in the disease as is manifested by the presence of immature erythrocytes. Extreme poikilocytosis generally occurs. The myeloid:erythroid ratio of the bone marrow decreases in the acute phases.

Diagnosis:

In the Field: Fever, anemia, jaundice, and hemoglobinuria are suggestive clinical signs in cattle from an area where babesiosis is enzootic. If these are coupled with splenomegaly and the usual post-mortem lesions associated with erythrocytic destruction, the diagnosis of babesiosis is strengthened. However, a positive diagnosis of

babesiosis can be established by identification of the parasites in the blood, positive serological tests, and by transmission experiments.

Laboratory: Although the presence of B. bigemina can often be detected by the examination of thin blood films, B. argentina is often very difficult to find. However, Giemsa stained thick blood smears have been found to be a reliable and practical means for the detection of both species of Babesia. A wet film of blood, approximately 50μ thick, is made on slides, dried quickly at 65° C. for 45 minutes, and stained as soon as possible after drying with a 1:50 solution of Giemsa at pH 7.0-7.1.

Babesia argentina can be readily detected in capillaries of the brain by the brain-smear, biopsy technique, either at the abattoirs after death or from live cattle. Babesia appear as small, intraerythrocytic, paired, basophilic bodies. Clinically normal cattle with latent infections of B. argentina show the parasite in brain smears. The grey matter of the cerebral or cerebellar cortex is the tissue of choice when making smears. Babesia argentina was found to persist in the brain capillaries for 12 months in 50% of cattle tested. Babesia bigemina is found in brain tissue much less frequently than B. argentina. Biopsy specimens can be taken from the brains of cattle at three monthly intervals with a high degree of safety, although temporary nervous derangement may be observed.

The detection of the carrier state of bovine babesiosis has presented a particularly difficult problem because of the low parasitemias in a high percentage of carrier cattle. Fundamental knowledge concerning immunoserology of several Babesia spp. has led to the development of several diagnostic procedures for detection of Babesia antibodies. Todorovic et al. (1971) found that antigens originated from the infected parasites, as well as from plasma collected at the acute stages of infection, can be

employed in serological tests. The complement fixation reaction constituted one of the earliest tests, and in recent years considerable progress has been made in an improved complement fixation test which is an exceedingly valuable diagnostic tool, both in the field and in research.

Complement-fixing antibodies develop in the sera of cattle within seven to 21 days post-infection, and persist through all stages of infection from the commencement of a detectable parasitemia to a time when they are too few in number to be detected in thick blood smears. A positive result is a reliable indication of a current babesial infection, even though the parasites are too few to be found in thick films. On the other hand, a negative result cannot be reliably interpreted as freedom from infection.

Fluorescent microscopy is proving valuable in the study of babesiosis, and the staining of Babesia by fluorochrome acridine orange has been reported. This substance stains only nuclear material, thus, making it suitable for demonstrating parasites in red blood cells. Formalin preserved blood can be kept for three months, and smears from it stained with acridine orange and examined with the fluorescent microscope show Babesia as brilliant reddish objects in blue fluorescent erythrocytes, easily seen under low power.

In addition, the gel-precipitation, fluorescent antibody, direct and indirect hemagglutination techniques have been applied for detection of specific Babesia antibodies, utilizing antigens from the parasitized erythrocytes as well as plasma collected from animals with acute infection.

Differential: Other conditions which should be differentiated in cattle are: anaplasmosis, trypanosomiasis, theileriasis, leptospirosis, eperythrozoonosis, hemobartonellosis, post-parturient hemoglobinuria, cold water hemoglobinuria, onion toxicity, and bacillary hemoglobinuria. In cerebral babesiosis, the signs may be confused with those of rabies.

Prognosis: Babesiosis in susceptible cattle is frequently severe with mortality approaching 60 to 90%. The prognosis of cattle with hemoglobinuria is very poor. The prognosis may be more favorable in indigenous cattle where mortality may be much lower.

Epizootiology:

Geographic Distribution: Babesiosis has often been categorized as a tropical malady; however, reports have originated from temperate and frigid sections of the world. Due to a variety of species which are susceptible to infection, there is a constant danger that even unaffected areas may be invaded by these hemoprotozoan parasites.

*Babesia bigemina* has a worldwide distribution in cattle. The geographic distribution of this species is through Central America, South America, Europe, Africa, Australia, and Asia.

*Babesia bovis*: This is a European species, but it has been reported from Africa, Asia, and the East Indies. It occurs mainly outside the tropical zones. The geographic distribution of this species is in south and central Europe, Russia, Africa, and Mediterranean coasts. This is the most important case of European babesiosis. It is common in many regions, but information on its true prevalence must await new surveys in the light of the recent recognition that *B. divergens* is a separate species and also a decision as to whether *B. berbera* and *B. argentina* are synonymous.

*Babesia argentina*: This species is widely distributed throughout the tropics, mainly through South America, Central America, Mexico, and Australia. *B. argentina* may be synonymous with *B. bovis*.

*Babesia divergens*: The geographic distribution of this species is in Balkan states, Austria, and England.

*Babesia major*: This species has been reported from North, South, and West Africa, South America, and from the Baltic region, Russia, and England.

Babesia berbera: This species has been reported from North Africa, Russia, and northern Europe and may be synonymous with B. argentina.

Transmission: In North America, the major transmitting agent in the past was B. annulatus, known locally as the blue tick, the Texas fever tick, the fever tick, or the southern cattle tick. In a very limited area of southern Florida, the transmitting agent was Boophilus microplus, also known as the tropical fever tick. This species has a wider host relationship than B. annulatus.

In other parts of the world, other species of Boophilus are responsible for transmitting Babesia. In southern Europe, it is Boophilus calcaratus; in central Africa, Boophilus decoloratus; and in South and Central America, Mexico, Australia, Brazil, Venezuela, and some of the Caribbean Islands, the transmitting agent is B. microplus. Very little is known about the life history of B. bovis, although a number of vectors have been suggested, e.g. Ixodes, Boophilus, and Haemaphysalis. The main vector is probably Ixodes ricinus.

Early investigations in Australia showed that B. bigemina could be transmitted by nymphs and adult ticks and B. argentina by larvae of the female B. microplus which had engorged on infected cattle. Haemaphysalis bispinosa, Haemaphysalis bancrofti, Amblyomma triguttatum, or Ixodes holocyclus are found on cattle in Australia, but are not capable of transmitting babesiosis.

Babesia argentina: It is transmitted by species of B. microplus and possibly by Rhipicephalus bursa. Babesia major: The vector in Russia is B. calcaratus. Babesia berbera: The vectors are B. calcaratus and R. bursa. Transmission occurs through stage-to-stage. Babesia divergens: It is transmitted by species of I. ricinus and Haemaphysalis cinnabarina punctata by transstadial and transovarial stages.

Hosts: Babesia parasites are generally host-specific; however, Neitiz (1956) discussed the susceptibility of various animals to Babesia infections and concluded that Babesia parasites are by no means as host-specific as formerly believed. Babesia bigemina are found in cattle and water buffalo, B. argentina and B. berbera in cattle, and B. bovis and B. divergens in cattle.

Control and Eradication:

Preventive Measures: As Babesia are transmitted by ticks, prevention depends on the elimination of the tick vector. This is frequently exceedingly difficult depending upon the particular species of ticks involved, but it may be accomplished by regular and prolonged dipping at two-week intervals. This is possible in other areas only through the complete absence of the animal host for sufficient time to permit the ticks to die out. In the United States, the disease has been practically eliminated by systematic dipping of all cattle to destroy the principal vectors, B. annulatus and B. microplus.

Cattle which survive an attack of babesiosis become carriers, and as long as the infection persists, the animals are resistant to reinfection. In enzootic areas, the animals are constantly reinfected as long as they are subject to infestation with infected ticks. In the absence of reinfection, the Babesia gradually disappear, and the cattle again become susceptible to the disease. Immunity to babesiosis, in the absence of reinfection, persists for a varying time up to about one year. Young calves from immune dams are protected up to two months against babesiosis by antibodies received from their mothers through colostrum. The immunity which accompanies infection with babesiosis forms the basis of the programs of protective inoculation which are carried out in the countries where babesiosis is enzootic.

Treatment: Successful treatment of babesiosis depends on early diagnosis and the prompt administration of effective drugs. There is little likelihood of success if treatment is delayed until the animal has been weakened by fever and anemia. A number of drugs have been used to treat this disease in the different species of animals with varying results.

Trypan Blue - This was one of the first drugs used to control babesiosis. Trypan blue, the sodium salt of ditolyl diazo-bis-8-amino-1-naphthol-3,6-disulfonic acid, is best used as a 1% solution at the rate of 50 to 100 ml. injected intravenously. Even though the drug is inexpensive, there are disadvantages to its use, and more recently developed drugs are generally preferred.

Quinuronium Derivatives - This drug is sold under the proprietary names such as Acaprin, Babesan, Pirevan, Piroplasmin and is essentially 6,6'-di(N-methyl-quinolyl) urea dimethosulfate. It has been reported to be highly effective against the large *Babesia spp.* and of some value, also, against the smaller piroplasms, *B. bovis* and *B. argentina*. In cattle, the drug is injected subcutaneously at the rate of 1 ml. per 100 pound body weight, with a maximum of 6 ml.

Acridine Derivatives - The acridine derivatives, sold under such trade names as Acriflavin, Gonacrine, Flavin, and Euflain, are a mixture of 2,8-diamino-10-methylacridinium chloride with a small amount of the 2,8-diamino salt. An intravenous injection in cattle with 20 ml. of a 5% solution has given good results against both *B. bigemina* and *B. argentina*.

Diamidine Derivatives - Phenamidine, composed of 4,4'-diamidinodiphenyl ether, was first reported to be of value against canine babesiosis and subsequently against *B. bigemina*. It is tolerated in cattle up to a level of 22.5 mg. per kg. injected intravenously, but it has been found

toxic at 30 mg. per kg. when used as a 40% solution. Although some workers disagree concerning its efficiency, Berenil, also Ganaseg, (4,4'-diamidino diaxoaminobenzene diaceturate) has been recommended for use against a number of species in different animals. It is now used extensively in the treatment of B. bigemina and B. argentina at 2 to 3 mg. per kg. intramuscularly. First introduced in 1960, Diampron (3,3'-diamidinocarbanilide diesthionate) has been used successfully in the treatment of B. divergens, B. berbera, and B. bigemina infections in cattle when administered subcutaneously or intramuscularly and at dose rates of 5 to 10 mg. per kg.

Other Drugs - Quinine, ichthargan, neoarsphenamine, and aureomycin have been used to treat babesial infections with varying results; however, they do not appear to be of much value when compared with the more recent compounds. Imidocarb (4A65) (3,3'-bis-(2-imidazolin-2-YL)-carbanilide dihydrochloride or diproprionate-Burroughs Wellcome Lab.) has been demonstrated to have prolonged prophylactic value and curative action against B. bigemina and B. argentina at 2 to 5 mg. per kg. subcutaneously or intramuscularly.

Sanitation and Disinfection: Sanitation and disinfection, aside from that involved in control and elimination of tick-vector, do not contribute to an abatement of the disease incidence in enzootic areas.

Immunization: Immunization (co-infectious immunity or premunition) against babesiosis is performed by inoculating cattle with Babesia spp. with and without specific chemotherapy. Cattle having recovered from a premunizing parasitemia will have a high degree of immunity for the duration of the carrier infection which usually lasts 4 to 8 months in the absence of re-exposure. The continued presence of infected ticks serves to boost the immunity established by inoculation. The standard premunizing

inoculum usually consists of $1X10^6$ to $1X10^8$ Babesia parasites contained in 2 ml. of blood. Because of biological variation the premunizing reaction often cannot be predicted. More severe reactions will occur in cattle that have had little or no previous exposure to infected ticks. For practical purposes, the period of close supervision should be from 7 to 14 days after premunization. The number of cattle inoculated should be restricted to a number that can be supervised comfortably. On farms with small paddocks and good handling facilities, up to 300 can be inoculated. If fully susceptible cattle are inoculated in large numbers without aftercare, losses of 1 to 5% may occur. It is good practice to inoculate and dip at the same time. Pregnant cows may abort after severe premunizing reactions.

Treatment following premunization is indicated when sickness is apparent or when the morning temperature is 41.0° C. or more. Daily temperatures should be taken during the time when the reaction is expected. Losses are greatly diminished when treatment is promptly administered as soon as the temperature increases because the onset of fever usually precedes clinical signs of infection. Cattle treated promptly often suffer no ill-effects; whereas, cattle treated when they appear sick may take days or weeks to recover.

Cattle premunized with either B. argentina or B. bigemina are protected more thoroughly from a homologous infection. Some cross Babesia spp. protection does occur, but is not complete. While unlikely, there are instances where antigenic variations occur within a single Babesia spp. so that the immunologic barrier may not be complete even against a homologous Babesia spp.

In the absence of a practical eradication program, premunization is a useful tool to prevent serious cattle losses but has the drawback of perpetuating infection.

# ANAPLASMOSIS
## (Gall Sickness)

Definition: Anaplasmosis is an acute or chronic, infectious, non-contagious disease of cattle. Acute infections are characterized by anemia, fever, and icterus. It is caused by the micro-organism, *Anaplasma marginale*, which is found in the erythrocyte and is transmitted by a variety of blood sucking arthropods. Chronic, or carrier infections, may persist for years without producing evidence of clinical disease, but during this time serve as a reservoir of infection from which arthropod vectors may transmit the causative organism to susceptible cattle.

Etiology: *Anaplasma marginale* was first thought to be a *Protozoa*, largely on the basis of its association with *Babesia*, and a disease syndrome similar to that produced by other hemoprotozoan agents. In the past, it has also been considered to be a viral agent, but currently the most acceptable theory is that it is more closely related to the *Rickettsia*, even though a degree of uncertainty still exists.

The intraerythrocytic *Anaplasma* body, measuring 0.3 to 1.0μ in diameter, is a small, dark blue or purplish coccoid body (using a Giemsa stain), generally located on the margin of the erythrocyte.

Larger *Anaplasma* bodies have been shown to be composed of several smaller units which are considered to be initial bodies. Reproduction of the organism is considered to be by binary fision. In the acute phase of infection, it is not uncommon to see parasitemias of 30-40% or higher with more than one body per erythrocyte. The parasitemia may drop rapidly after reaching a peak and is extremely difficult to demonstrate in carrier animals.

In addition to the typical coccoid forms including initial bodies, some workers have noted projections and ring-like forms. The projections are most

often seen with vital stains or by electron and phase microscopy. There is some uncertainty as to whether or not forms showing projections are A. marginale. Kreier and Ristic (1963) suggest that these atypical forms, while related to A. marginale, are antigenically different and have designated them Paranaplasma caudata.

History: Smith and Kilborne in 1893 described a small, deep staining, coccoid body on the erythrocyte margin which they considered a phase in the life cycle of Babesia bigemina. In 1910, Theiler recognized these marginal bodies as the causative agent of an entirely separate disease entity which he entitled anaplasmosis. He considered this organism to be a nucleus devoid of protoplasma, hence, the name Anaplasma. Theiler recognized a second disease entity caused by a centrally located, coccoid body, resembling A. marginale except for its central location in the erythrocyte, which he named A. marginale (variety centrale), now known and recognized as A. centrale. Anaplasma centrale is not an important animal pathogen due to the mild response associated with such infections, and has been widely used in South Africa as a premunizing vaccine against A. marginale.

Signs: The clinical characteristics of anaplasmosis are influenced by the host's age, virulence of the particular Anaplasma isolate, and to some extent, by the size and route of the infecting exposure. Calves less than a year of age are generally more resistant and in the absence of blood examinations, infections often go unnoticed. Recognizing considerable animal variation, increased age is generally associated with progressively more severe infection up to 3 or 4 years of age. Acute infections may occur in cattle 2 to 3 years of age, but severe infections are more common in older animals. Peracute, highly fatal infections commonly occur in splenectomized calves.

Infection is associated with a rise in body temperature, followed by progressive anemia associated with Anaplasma parasitemia, and may terminate as a fatal hypoxia. Most clinical signs are associated with the effects of anemia and erythrocytic destruction, and are usually characterized by pale mucous membranes, jaundice, weakness, and rapid breathing especially after exercise. Loss of weight and general condition is common.

Cattle with packed cell volumes (PCV) as low as 14-18% may show few, if any, signs of infection if viewed in a pasture under resting conditions. Frequently, when clinical signs are sufficiently severe to be noticed, the animal may be near death.

Incubation Time: This is extremely variable and depends largely on the size and route of the infecting inocula. Under field conditions, it will generally range from 16 to 40 days. Under laboratory conditions, using massive inoculations of infected blood, the incubation time can be shortened to as little as 2 to 3 days, and by using very small inoculums increased to as long as 60 to 80 days.

Pathologic Change:

Pathogenesis: Based on a recognizable parasitemia of 1% or over, the course of acute infection, while variable, may extend 4 to 14 days. Clinically, the course of infection may appear to be only a few days due to the lack of visible signs, which are dependent on a high parasitemia and a low PCV. When death does not occur, a rapid disappearance of the A. marginale parasitemia usually occurs, but recovery of condition and outward appearance may be more protracted. A persistent carrier infection usually occurs following recovery. Relapses may occur but they are generally less severe and usually will not cause signs of clinical illness.

The anemia of Anaplasma appears to be the result of an accelerated

removal of erythrocytes by macro-phages of the reticuloendothelial system, involving an auto immune response. The bile pigments resulting from this destruction are transported and excreted as urobilinogen by normal routes but at an accelerated rate. Hemoglobinuria does not occur.

The erythroid elements in the bone marrow of calves infected with anaplasmosis do not change significantly until the anemia becomes severe, at which time they become more prominent. The myeloid:erythroid ratios of the bone marrow decreases as the disease progresses. A rapid influx of immature erythrocytes in the circulating blood is generally favorable. Death may occur if the infection overwhelms the animal before the erythropoietic system can respond.

Postmortem Lesions: In acute anaplasmosis, there is an acute anemia which is characterized by pallor and icterus. The spleen is usually enlarged; the liver is enlarged with rounded edges, with evidence of icterus common. The gall bladder may contain dark and viscous bile. The epicardium may show evidence of petechial hemorrhages. Petechiation and edema of lymph nodes are common findings. There are no characteristic pathologic changes attributable to *A. marginale* in chronic carrier infections.

Microlesions: Depending on the length of infection before death, varying degrees of hyperplasia of the bone marrow is noted, along with extra medullary hemopoiesis in the spleen and liver. Marked reticuloendothelial and lymphoid hyperplasia occur in the lymph nodes, spleen, and tonsils. Perivascualr lymphatic infiltration occurs in many organs, e.g. liver, kidney, and lungs.

Diagnosis:

In the Field: Acute cases of anaplasmosis are characterized by pale mucous membranes, rapid breath, weakness, muscle tremor, a yellow tinge on

mucous membranes, and the availability of vectors. A firm diagnosis of anaplasmosis requires laboratory confirmation, based on finding Anaplasma bodies on Giemsa-stained blood smears or by a positive response to available serologic tests. There are usually no recognizable signs of chronic anaplasmosis, and diagnosis of this type infection is dependent on serologic means.

A number of serologic tests are available for the diagnosis of anaplasmosis. The oldest and still a standard is the complement-fixation test (CF). A capillary-tube agglutination test is available and in recent years a card agglutination test has been developed. Both of these procedures compare favorably with the CF test, all showing a high degree of correlation. The card agglutination test was developed primarily for field use and can be conducted on the farm within minutes of taking the blood sample. It can be conducted with serum or non-inactivated plasma and a stained antigen, which when combined shows specific agglutination when infection is present.

Differential: In tropical areas, anaplasmosis may be confused with babesiosis, leptospirosis, trypanosomiasis, theileriosis, or other hemotropic infections which may result in an anemic syndrome. It is not uncommon for cattle in tropical zones to be carrying two or more blood parasitic infections including anaplasmosis. Babesia parasitemias are generally more difficult to demonstrate than Anaplasma, but when present are distinctive. Hemoglobinuria occurs with babesiosis and leptospirosis, but is not seen with anaplasmosis. Elevated temperatures are a more consistent feature of babesiosis, even though a similar response is possible with anaplasmosis. The incubation time is considerably shorter in babesiosis. Anemia, while occurring in trypanosomiasis and theileriosis, is generally not as severe as in anaplasmosis and babesiosis. Young animals are more

resistant to anaplasmosis; whereas, age is not so much a factor with babesiosis, and calves are more severely affected with leptospirosis than are older animals. Identification of the hemo-parasite on Giemsa-stained blood smears or a positive serologic test response is essential for definite diagnosis.

Prognosis: In cattle over 3 years of age, the prognosis should be guarded; whereas, in calves and yearlings, recovery is more frequent. Non-apparent carrier infections may persist for the life of the animal following recovery.

Epizootiology:

Geographic Distribution: Anaplasmosis is present in most tropical and sub-tropical areas of the world and is also found in many temperate zones of North America. The distribution is generally limited to those areas having adequate vectors, but the wide variety of transmitting agents results in extensive distribution.

A recent survey in Colombia in South America showed a marked correlation between infection and altitude. Altitude influenced mean temperatures, hence, the presence or absence of vectors, to the extent that at elevations of 2600 meters or more anaplasmosis was not encountered in mature cattle even in tropical latitudes.

Transmission: A large variety of arthropods may serve as transmitting agents. These vectors in some instances are true biological carriers, and in others transmission occurs by mechanical means. Any transfer of infected blood to susceptible cattle, even in minute amounts, can produce infection.

There are at least 20 species of ticks that have been shown to transmit infection, including *Boophilus*, *Hyalomma*, *Rhipicephalus*, *Dermacentor*, *Ixodes*, and *Haemaphysalis* ticks; and at least 10 species of biting flies

(mostly Tabanids) have been shown capable of transmitting anaplasmosis. It is possible that other biting insects may be involved in transmission. It has been suggested by Roberts (1968) that Hippelates flies (eye gnats) may play a role in anaplasmosis transmission.

In the United States, the biting insects are thought to be the principle vectors in the Gulf coast and southeastern states; Dermacentor andersoni is probably the most important vector in the intermountain areas; Dermacentor occidentalis is a major vector in the Pacific coast areas of California. In tropical South America, the Boophilus ticks apparently play a major role in transmission. In Africa and Asia, the Boophilus and Rhipicephalus ticks are important vectors.

It is reported that transmission of anaplasmosis by biting flies will occur only within a few minutes after a blood meal has been obtained from an infected animal. The situation is much different with ticks, as the organism can maintain itself in this host for an extended period of time. Transstadial and transovarial transmission occur with tick vectors.

Hosts: The principle host for anaplasmosis is cattle. Sheep, goats, and white-tailed deer (Odocoileus virginianus) can be infected artificially, but it is doubtful if these animals play a major role in the maintenance of infection in a given area. Black-tailed deer (Odocoileus hemionus columbianus) are carriers of anaplasmosis in nature and act as a reservoir of infection to cattle in California. The mule deer is also naturally infected, but its role in the epidemiology of anaplasmosis is not fully known.

Anaplasma marginale has been isolated from elk, buffalo, and several African antelope. It is not recognized as an economically important disease in animals other than the bovine.

Control and Eradication:

Preventive Measures: Insect control and the segregation or elimination of infected animals will prevent transmission and effectively limit the infectious agent, thus, reducing animal losses. Serologic tests are useful in identifying carrier animals. These measures are not successful in those areas of tick vectors and where reservoirs of infection are present in wild animals. In these areas a practical plan for eradication has yet to be developed.

Treatment: In acute anaplasmosis, success is frequently dependent on early recognition of infection and prompt treatment. The tetracycline compounds given early in the course of infection have been shown to be effective when administered intravenously or intramuscularly at a rate of 3 to 5 mg. per pound of body weight. Good nursing and supportive treatment, including blood transfusions, are helpful. In recent years, two additional chemotherapeutic agents have been recognized as being effective against anaplasmosis, Gloxazone* (alpha-ethoxyethylglyoxal dithiosemicarbazone) and Imidocarb* (3,3'-Bis(2-imidazolin-2-YL)-carbanilide dihydrochloride). Gloxazone is effective at 2 to 5 mg. per kg. intravenously, and Imidocarb at 5 mg. per kg. intramuscularly or subcutaneously.

In recent years, the use of treatment to eliminate carrier infections has become an integral part of control and eradication programs. Tetracyclines, administered orally at the rate of 5 mg. per pound daily for 60 days, has been reported effective in eliminating carrier infections. Carrier infections are also reportedly eliminated by 10 to 15 daily I.M. or I.V. injections of 5 mg. per pound tetracycline. If carriers in a herd are recognized by serologic means and treated, infection can, theoretically, be

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*Burroughs Wellcome and Co., Inc., Raleigh, North Carolina.

eradicated with no further hazard of transmission within the herd. This test and treatment approach has been successfully employed in a few instances.

Experimental evidence is available to suggest that Gloxazone given with oxytetracycline is more effective than either drug alone in eliminating carrier infections. Imidocarb in combination with Gloxazone, or administered alone, has been effective in eliminating carrier infections. Roby (1972) has reported the successful elimination of carrier infections by the subcutaneous or intramuscular injection of 5 mg. per kg. Imidocarb given 2 times at a 14-day interval.

Control measures based on diagnosis and treatment are particularly effective in areas where the principal vectors are biting flies and where transmission is primarily mechanical. Where biological vectors occur, such as the Dermacentor, Boophilus, and Rhipicephalus ticks, and where there are known wildlife reservoirs, the problems of control and/or eradication are greatly compounded.

Immunization: Soon after the identification of A. marginale by Theiler in 1910, he discovered a second agent, now referred to as A. centrale, which produces a mild infection and a high degree of protection against virulent A. marginale. This organism is still being used successfully in Africa and Asia as an immunizing agent against anaplasmosis. It has not been used in North America for this purpose.

A partial immunity has been produced, using killed vaccines which consist primarily of Anaplasma bodies derived from infected erythrocytes added to oil adjuvants. A commercially available vaccine, using two subcutaneous injections 4 to 6 weeks apart, claims to reduce the severity of infection and prevent death loss due to anaplasmosis. There is recent

evidence to suggest that the use of this vaccine is occasionally associated with a hemolytic anemia affecting calves born to dams previously vaccinated.

An attenuated live vaccine has been developed which, like A. centrale, produces a mild infection, but has the advantage of greater antigenic similarity to A. marginale from which it was derived. Cattle immunized with this agent are protected from challenge with a virulent A. marginale isolate of common origin.

Experimental evidence is available which suggests the presence of antigenic differences in various Anaplasma isolates. During experiments in South America, both the killed adjuvant vaccine and the live attenuated vaccine were overwhelmed by natural exposure to the local Anaplasma. This may have been due to antigenic variants, to the size of the challenge infection, or factors presently unknown.

Both A. centrale and the attenuated A. marginale depend on premunition for their protective potential. Premunition can also be induced by virulent A. marginale. Calves in enzootic zones exposed to vectors carrying Anaplasma frequently become infected early in life and because of their natural resistance may recover without apparent illness. Such infections may persist for life, resulting in an Anaplasma resistant animal. Older cattle may become premunized with virulent A. marginale, but in the absence of treatment to reduce the severity of infection, this procedure is hazardous. Cattle premunized with virulent Anaplasma show the greatest resistance to later field challenge, and for this reason therapeutic procedures aimed at reducing the severity of infection without eliminating infection are useful.

## LEUCOCYTOZOONOSIS

Protozoan parasites belonging to the genus Leucocytozoon (L. smithi, L. simondi, L. caullery) are members of the hemosporidia and occur in the blood of many species of wild and domestic birds. Heavy losses of wild ducks, turkeys, and grouse have been attributed to these organisms.

The causative organism undergoes schizogony in the endothelial and parenchymatous cells of the liver, heart, and kidney of the infected host. Gametocytes occur in the circulating blood and the infected erythrocytes become grossly distorted, being elongated with pointed ends and may assume a spherical form. No pigment is produced. The vectors are gnats or black flies (Simulium). A sexual cycle occurs in the vectors.

Young birds more often show acute signs of infection. These consist of anorexia, emaciation, debility, incoordination, and sometimes evidence of central nervous system involvement. Death may occur in 2 or 3 days. Infections are generally less acute in older birds. Birds that recover generally remain as carriers.

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# BESNOITIOSIS
(Globidiosis)

Besnoitia besnoiti is usually transmitted by ingestion, but it has been reported to have been mechanically transmitted by Glossina palpalis. The parasite is known to occur in South Africa, Congo, Sudan, Angola, southern France, and Portugal. Cattle are primarily infected. The parasites measuring 5μ - 9μ X 2μ - 5μ are found in the dermis, subcutaneous tissues, and fascia and in the laryngeal, nasal, and other mucosae. Cysts may develop measuring up to 600μ in size in cattle.

As the disease progresses, hair falls out, the skin thickens and may crack with exudation of blood. Mortality is usually less than 10%, but the animals lose condition, pregnant cows may abort, bulls may become sterile, and hides become valueless. Premunition follows infection. At present there appears to be no specific treatment.

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## APPENDIX 2
## (Babesiosis)

Microbiological Procedure:

*Babesia* parasites from blood or internal organs (spleen, liver, brain capillaries, lymph nodes). Giemsa staining technique.

Histological Examination:

Multicentric hematopoietic hyperplasia and metaplasia, hepatic centrilobular necrosis. Lesions of intravascular hemolytic anemia. Lymphoid hyperplasia, hemosiderosis, erythrophagocytosis, hyaline degeneration, decrease of myeloid:erythroid ratio, perivascular lymphocytic infiltration, cerebral and cerebellar congestion and edema. Erythroid hyperplasia and parasites in erythrocytes.

Serologic Tests:

Complement fixation, agar gel precipitation, capillary agglutination, direct and indirect hemagglutination, direct and indirect fluorescent antibody and agglutination tests.

Animal Inoculation:

Laboratory animals are refractory to infection with bovine *Babesia spp*. Cattle inoculation with blood, tissue of infected animal is successful for inducing artificial infection.

APPENDIX 5
(Babesiosis)

Description:

Febrile, tick-borne disease characterized by anemia, icterus, and hemoglobinuria.

Cause and Distribution:

Babesiosis has a worldwide distribution related to the presence of *Boophilus spp.* of ticks in warm climates and to *Ixodes spp.* in temperate climates.

Host:

Domestic and wild animals naturally susceptible to *Babesia spp.*

Signs:

Fever, severe hemolytic anemia, icterus, hemoglobinuria, weakness, weight loss, and poor condition followed by death.

Pathology:

Severe emaciation. Blood thin and watery. Spleen, liver, lymph nodes enlarged and edematous. Multicentric hematopoietic hyperplasia and metaplasia of bone marrow. Perivascular lymphocytic infiltration of the cerebral capillaries.

Diagnosis:

Clinical signs and pathology. Presence of *Babesia spp.* in blood smears or demonstration of complement fixing, precipitating, or agglutinating antibodies in sera of tested animals. Transfer of blood to a susceptible splenectomized animal.

Incubation Time:

From 5 to 15 days, but depends on the *Babesia spp.* involved.

Mode of Transmission:

Under natural conditions transmission is carried out by ticks of the *Genera Boophilus*, *Ixodes*, and *Rhipicephalus*.

Period of Communicability:

Cyclical in tick to tick; transstadial in larvae, nymph, and adults. Transovarial or hereditary from ova to larvae of ticks. Cattle may remain carriers for a period of years.

Control Measures:

Control of ticks. Premunition - coinfectious immunity with *Babesia* infective blood. Vaccination - sterile immunity with killed *Babesia* parasites. Chemotherapy with premunition - *Babesia spp.* infective blood and drug therapy a week later. Chemoprophylaxis.

## APPENDIX 2
## (Anaplasmosis)

Microbiological:

*Anaplasma marginale* bodies in Giemsa-stained blood smears.

Histological Examination:

Hyperplasia of bone marrow, evidence of extra medullary hemopoiesis in spleen and liver, and reticulocytosis. *Anaplasma* parasitemias in acute cases.

Serologic Tests:

Complement fixation, capillary-tube agglutination, and card agglutination tests commonly used for diagnosis. Other tests have been described.

Animal Inoculation:

The inoculation of susceptible cattle with blood from acute or chronic infections readily produces infection. Sheep and deer can also be infected experimentally. Laboratory animals are refractory to infection.

## APPENDIX 5
## (Anaplasmosis)

Description:

An acute or chronic infectious blood disease of cattle characterized by a severe hemolytic anemia in acute cases, and transmitted in nature by a variety of blood sucking arthropods.

Cause and Distribution:

*Anaplasma marginale*. North and South America, Asia, Africa, Australia, and southern Europe. Presence of the disease is largely dependent on the availability of insect vectors capable of transmission.

Host:

Primarily cattle. Deer, elk, buffalo, sheep, and goats can be infected.

Signs:

A severe hemolytic anemia with the resultant shortness of breath, weakness, pale mucous membranes, icterus, and fever. Signs of infection are more severe in older animals. When death does not occur, weight loss and poor condition follow infection.

Pathology:

Low erythrocytic counts, low hemoglobin, and low packed cell volumes. Pallor, icterus, enlarged spleen and liver and other lesions related to the anemia. Evidence of bone marrow hyperplasia.

Diagnosis:

A febrile disease with evidence of acute anemia in an area of arthropod vectors is suggestive of anaplasmosis. Presence of *Anaplasma* bodies in blood smears or a positive serologic test for anaplasmosis is diagnostic.

Incubation Time:

Highly variable but under natural conditions it usually is 21-49 days.

Mode of Transmission:

A wide variety of arthropods. Biting flies and ticks are the most important vectors. Flies are mechanical vectors; ticks may be mechanical or biological vectors with transmission being transstadial or transovarial.

Period of Communicability:

Following recovery of acute anaplasmosis, cattle may remain carriers for a period of years and possibly the life of the animal.

Control Measures:

Vector control, identification of carrier animals with their removal from the herd, or by treatment to eliminate infection. Vaccines are present to reduce the severity of acute infection, but they allow the development of chronic carriers.