

Anaplasmosis: A Disease of Cattle

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ANAPLASMOSIS is a serious blood disease of cattle, and it is slowly spreading to new areas. Much has been found out about the ways in which it is transmitted, but a preventive or a cure is still to be discovered by research.

ANAPLASMOSIS was probably introduced into the United States by the Spaniards at the same time that splenic or cattle tick fever entered this country (*13*).² The disease, which is not transmissible to man, chiefly affects cattle, although the antelope, black wildebeest, blesbok, buffalo, camel, deer, duiker, elk, goat, and sheep are reported to be susceptible.

Mature cattle are more subject to infection than young animals. Although calves doubtless acquire mild cases, they are seldom observed to be sick or dying in anaplasmosis-infected herds. Cattle of all breeds may acquire the disease. Some immunity appears to develop in herds in which anaplasmosis has long been prevalent, but a few authentic cases have been reported of a second attack after a year in cows that have recovered.

Anaplasmosis is generally considered to be a tropical or semi-tropical disease, but it also occurs in localities that have low winter temperatures. It has been reported from Africa, Argentina, Brazil and several other South American countries, Cochin-China, France, Italy, Java, the Philippine Islands, the Union of Soviet Socialist Republics, and the Island of Taiwan.

In the United States the malady is known to exist in 23 States—Alabama, Arizona, Arkansas, California, Colorado, Delaware, Flor-

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² Italic numbers in parentheses refer to Literature Cited, p. 586.

ida, Georgia, Idaho, Kansas, Louisiana, Maryland, Mississippi, Missouri, Montana, Nevada, North Carolina, Ohio, Oklahoma, Oregon, Texas, Virginia, and Wyoming. It may be present but unrecognized in other States. It is slowly spreading to new areas.

The incidence of anaplasmosis seems to be influenced by climatic conditions favorable to insects and ticks that carry the infection. In the United States natural outbreaks usually occur during the summer and fall. Occasionally cases develop in winter in the South. On the Pacific coast the infection, carried by ticks, is common in winter and spring. The disease occurs more frequently in timbered or brushy wet areas than on dry treeless plains, where most species of ticks, horseflies, and mosquitoes are less abundant. Excessive rainfall, floods, irrigation ditches, and marshes provide favorable conditions for the propagation of insects and hence opportunities for the transmission of the disease.

NATURE AND SYMPTOMS OF THE DISEASE

Anaplasmosis is an infectious disease caused by a minute parasite which invades the red blood cells and destroys a large number of them, so that the blood becomes pale and watery. Under natural conditions the microparasite is carried from infected to healthy animals by ticks, horseflies, mosquitoes, and probably other biting insects, as well as by unclean surgical procedures. (An account of the results of research on the transmission of the disease is given at the end of this article.)

In 1910 Theiler (25), of South Africa, described the parasites that cause anaplasmosis as minute, specklike, protozoan bodies in the red cells of the blood and named them *Anaplasma marginale* because of their position near the margin of the cells. Theiler also observed a variety, *A. centrale*, centrally located in the cells and less virulent than the marginal type.³

Red blood cells may contain one to three, or even more, microparasites (fig. 1). Early in the course of anaplasmosis only a few of the blood cells contain the parasites, but as the disease progresses the temperature rises, ranging from 103° to 107° F. (normal range 100.5° to 102.5°) and the number of red cells involved rapidly increases. Later the temperature drops to normal and may even be subnormal at the time of death. In convalescent cases, the number of parasites lessens, but red cells containing fine granular bodies called stipple cells appear. Cells of this type also occur in other forms of anemia.

In typical cases the heart action is rapid and pounding, with a pulse rate of 70 to 140 a minute (normal range 40 to 70). In severe cases the red-cell blood count usually drops from a normal of

³ Some workers claim that the causative agent is a type of filtrable virus notwithstanding the fact that filtered blood from sick animals fails to reproduce the disease. Dikmans (3) holds that the causative agent may be either a "reaction product," due to a virus, or a "parasite *sui generis*." Du Toit (4) says "It is difficult to ascertain why there has been so much reluctance to accept the parasitic theory of anaplasmosis. * * * If we take all the known facts about anaplasmosis into consideration, we come to the conclusion that the simplest and most natural view to take is that this disease is caused by *Anaplasma marginale* and that this organism probably belongs to the Protozoa."

about 5 to 7 million per cubic millimeter of blood to 2 million or less. In normal cattle the hemoglobin or blood-color test is approximately 80 (Tallquist scale), whereas in sick animals a color test of 20 to 30 is not uncommon.

In an advanced case the breathing is labored and difficult and the muzzle is dry. There are marked depression, tremors of the muscles, loss of appetite, and a great reduction in the milk flow. The skin, teats, udder, vagina, whites of the eyes, and all visible membranes become pale and yellow. Pica, or depraved appetite, evi-

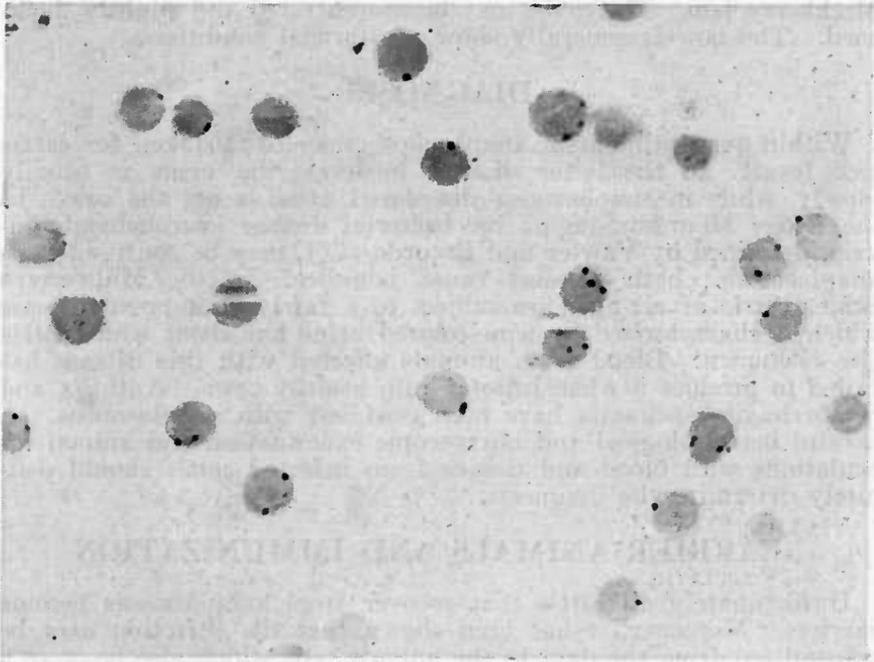


FIGURE 1.—Red blood cells from a field case of anaplasmosis. The black dots near the margins of the cells are *Anaplasma marginale*. $\times 1,200$.

denced by the eating of bones or dirt, is not uncommon. Sick animals may show brain symptoms and an inclination to fight.

Dribbling urination is common in anaplasmosis, but the urine is not bloody or wine-colored. Sick cattle are usually constipated, and abortion often occurs in advanced pregnancy. Death may follow within 1 or several days after the clinical symptoms of the disease appear. Recovery is usually slow.

There is a wide range in the death rate of affected herds. Sometimes from out of a considerable number infected in a herd only one cow may die, but the average death rate usually varies from 25 to 60 percent of the infected animals. When microscopic examination of the blood from a sick cow shows that 40 to 60 percent of the red cells contain marginal bodies, it is probable that the animal

will die; if the number of infected red cells is only 10 to 20 percent, it is more likely to recover.

On post mortem examination, cattle dead of anaplasmosis usually show a marked yellow discoloration of the skin, teats, and visible mucous membranes. Often the heart is flabby, and hemorrhagic blotches may appear on its surface. The blood is thin and watery, and the lungs are usually pale and filled with air bubbles. The liver is enlarged and jaundiced, and contents of the bile bladder are dark green, granular, and gelatinous in appearance. The spleen, or milt, is generally enlarged and soft, and in appearance resembles blackberry jam. The urine may be concentrated and slightly darkened. The bowels generally show a catarrhal condition.

DIAGNOSIS

Within quarantine areas anaplasmosis may be mistaken for cattle tick fever. In the latter disease, however, the urine is usually bloody, while in anaplasmosis discolored urine is not the rule. In the Rocky Mountain region the bacterial disease icterohemoglobinuria, described by Vawter and Records (27), may be confused with anaplasmosis; both diseases cause jaundice. In the Midwestern States cattle of all ages are subject to a fairly widespread disease which is characterized by wine-colored urine but about which little else is known. Blood from animals affected with this disease has failed to produce it when injected into healthy cows. Anthrax and hemorrhagic septicemia have been confused with anaplasmosis. A careful bacteriological and microscopic examination and animal inoculations with blood and tissues from infected cattle should definitely determine the diagnosis.

CARRIER ANIMALS AND IMMUNIZATION

Unfortunately all cattle that recover from anaplasmosis become carriers. Moreover, it has been shown that the infection may be transmitted from the dam to the unborn calf, which also becomes a carrier. At the Beltsville, Md., station the Bureau of Animal Industry has a cow that is still a proved carrier 13 years after recovery from anaplasmosis. When introduced into clean herds, such animals serve as sources of infection, particularly if ticks or other vectors are present.

In areas where the disease exists only in scattered herds it is prudent to fatten all animals that recover and ship them for slaughter, thus reducing the sources of new infection. In areas where the infection is widespread, involving many herds, the slaughter plan is not altogether practical. The sick animals are treated, and those that recover are kept in the herd. The owner should try, however, in every possible way, to prevent the spread of the disease by mechanical or other means.

In some foreign countries, a procedure known as premunition is used to make cattle resistant to anaplasmosis. A small dose of blood

from a known carrier is inoculated into the susceptible animal, preferably at an early age (under 6 months) since calves do not react so severely as adult cattle. The vaccinated animals become immune but also become carriers, so that immunization by such vaccination is not recommended in the United States. A number of vaccines prepared by methods designed to destroy the infective agent have been used experimentally, but none have proved successful.

TREATMENT

The treatment of anaplasmosis is largely a veterinary problem, but the owner can give valuable assistance. Sick animals should be kept in the shade and given plenty of clean water, a little appetizing green feed, and protection against flies and mosquitoes. Good nursing is vital. Unnecessary or rough handling of sick cattle, especially range animals, may cause or hasten their death. In drenching cattle with salts, linseed oil, or other drugs, care should be taken to prevent the liquid from entering the lungs. Since cattle sick with anaplasmosis are usually constipated, mild saline purges are indicated. Animals that drink a great deal of water freely often recover; therefore, if affected animals do not drink water copiously, large quantities should be given by means of a stomach tube. When weakness develops, veterinarians often use hypodermic injections of camphor or strychnin.

Many drugs are used for the treatment of anaplasmosis. Among these, sodium cacodylate has its advocates, but its value has not been proved by controlled experiments. Injecting carrier cattle with a 10-percent solution of this drug in doses of 30 and 40 grains per 100 pounds of body weight was ineffective in freeing the animals of the microparasites (26). A modified sodium cacodylate treatment suggested by Boynton (2) consists in the intravenous injection of 1 liter of 5-percent dextrose in distilled water, to which has been added sufficient sodium cacodylate solution to make a dosage of 25 to 30 grains per 100 pounds of body weight. The cacodylate solution consisted of 4.5 grains of the drug per cubic centimeter of water. The required number of cubic centimeters of this solution was added immediately before injection.

Encouraging results from the intravenous injection of 2- to 4-percent mercurochrome have been reported by Dykstra and other veterinarians (5). A desirable mild purging action follows the use of this drug.

According to Koger (10) large quantities of whole citrated blood from healthy bovines injected intravenously seem to reduce the mortality, especially if given early in the course of the disease. (Citrated blood is blood drawn from a healthy animal into a sodium citrate solution which prevents coagulation and thus facilitates its injection.)

Continued efforts are being made to perfect some specific remedy with which to control and treat anaplasmosis.

DETAILS CONCERNING THE TRANSMISSION OF ANAPLASMOSIS

MECHANICAL SPREAD BY UNCLEAN SURGERY

During 15 years of field and laboratory experience with anaplasmosis in various parts of the United States, the writer (23) has learned of and diagnosed many cases of the disease in herds harboring carriers. The cases followed dehorning or other surgical procedures in which sanitary precautions were not observed.

In temperate climates dehorning and tipping (6, 12) are usually done during the cool months to escape screwworm or other fly infestations. Even then, many cases of anaplasmosis have developed following these operations, probably because appropriate precautions were not taken to disinfect instruments between operations.

That contaminated instruments and hypodermic needles can transmit anaplasmosis is supported by experiments by Rees (16), who spread the disease by lancet pricks. Sanborn, Stiles, and Moe (29) reproduced infection by injecting 0.025 cubic centimeter of blood from an acute case intradermically into a healthy cow. Anaplasmosis may be transmitted from carrier animals by unclean needles used in vaccination or in drawing blood, by nose tongs, and by castration, slitting of the ears, or other surgical procedures.

It is essential that sterile instruments be used for every animal to be bled, injected, or operated upon. A sufficient number of bleeding needles should be provided, and they should be kept clean and sanitary. Used needles, saws, and other instruments should first be washed in cold water to remove blood and then sterilized by boiling for several minutes in water containing 2 percent of washing soda. When facilities for boiling are not available, the cleaned instruments and needles may be immersed for 15 minutes or longer in a 2-percent lye solution, which must be freshly prepared each day and kept covered, as it deteriorates upon exposure to the air. After being removed from the lye solution, the needles may be dipped in a 2-percent compound solution of cresol, 2-percent formalin, or 60- to 70-percent alcohol.

NATURAL MEANS OF TRANSMISSION

Investigators working in various parts of the world have shown that anaplasmosis can be experimentally transmitted from diseased to healthy cattle by ticks, horseflies, and mosquitoes. Additional research will doubtless reveal other vectors.

Several research workers have shown that the following 18 species of ticks are capable of transmitting anaplasmosis experimentally:

SCIENTIFIC NAME	COMMON NAME
<i>Boophilus annulatus</i>	Cattle fever tick
<i>B. decoloratus</i>	Blue tick
<i>B. microplus</i>	None
<i>Dermacentor albipictus</i>	Winter tick
<i>D. andersoni</i>	Rocky Mountain spotted fever tick
<i>D. occidentalis</i>	Western dog tick
<i>D. variabilis</i>	American dog tick
<i>Hyalomma lusitanicum</i>	None
<i>H. aegyptium</i>	Bont-leg tick
<i>Ixodes ricinus</i>	Castor-bean tick
<i>I. scapularis</i>	Common shoulder tick
<i>Ornithodoros lahorensis</i>	None
<i>Rhipicephalus appendiculatus</i>	Common brown tick
<i>R. bursa</i>	Common brown tick
<i>R. evertsi</i>	Red-legged tick or red tick
<i>R. sanguineus</i>	Brown dog tick
<i>R. simus</i>	Black pitted tick
<i>Argas persicus</i> ⁴	Chicken tick

⁴ D. E. Howell and associates, Oklahoma Agricultural Experiment Station, unpublished data.

In his original work (1910), Theiler (25) showed that *Boophilus decoloratus* not only transmitted the disease called red water in South Africa but also anaplasmosis, and that *Rhipicephalus simus* spread the same disease. In 1930 Rees (15) first experimentally incriminated *R. sanguineus* in this country as a carrier of anaplasmosis, and he later transmitted the disease in the same manner with *Dermacentor andersoni*, *D. variabilis* and *Ixodes scapularis* (17).

In Du Toit's article on anaplasmosis (4), presented at the Twelfth International Veterinary Congress in 1934, he mentioned that several species of ticks had been reported by different workers as transmitting this disease. Rozeboom, Stiles, and Moe (18) report transmission by *Dermacentor andersoni*, and Stiles (24) records 17 ticks as vectors. From California, Boynton, Herms, Howell, and Woods (1) reported in 1936 the successful transmission of anaplasmosis by *D. andersoni*, confirming the work of Rees. They also reported



FIGURE 2.—A glass funnel is used to permit horseflies to feed on cattle in experiments on the transmission of anaplasmosis at the Oklahoma Agricultural Experiment Station.

for the first time a new species, *D. albipictus*, and a definite biological transmission of anaplasmosis by *D. occidentalis*. The disease was transmitted through both male and female ticks naturally infected with *D. andersoni* in 1938 by Sanborn, Stiles, and Moe (20) and in 1940 hereditary transmission with the female of the species was accomplished by Howell, Stiles, and Moe (8).

Horseflies also have been proved to be mechanical carriers of anaplasmosis. In many localities where the disease exists, large numbers of horseflies (tabanids) abound and home-made traps and commercial sprays are often employed to control them. In discussing these conditions, Sanborn et al. (19) state: "A study of field conditions, the prevalence of Tabanids, their occurrence, habits and distribution has been found to correspond closely to that of anaplasmosis. This is particularly true in swampy regions near ponds, inundated areas along rivers, lowlands of creeks, and in wooded areas. These are the places where horseflies breed and develop, and where the greatest annoyance to livestock occurs."

The first successful transmission of anaplasmosis by horseflies was accomplished by Sanborn, Stiles, Moe, and Orr (21). Subsequently their findings were corroborated and summarized by Howell, Sanborn, Rozeboom, Stiles, and Moe (7), who showed that seven species of horseflies can transmit the disease experimentally: *Tabanus sulcifrons*, *T. abactor*, *T. venustus*, *T. equalis*, *T. erythraeus*, *T. americanus*, and *T. oklahomensis*.

In part the summary referred to states: "Fewer bites are required if the infecting animal is in the acute stage of the disease than if it is a carrier. Bites obtained five or more minutes after the infective feed were not able to transfer anaplasmosis to healthy animals." These findings may partly explain the sporadic nature of the disease and the apparent slowness of its spread.

In Florida in 1933 Sanders (22) incriminated *Tabanus fumipennis* and *Stomoxys calcitrans* as vectors, and Morris, Martin, and Oglesby (14), working in Louisiana, produced the disease from bites of *Tabanus atratus*.

Experiments conducted by Lotze and Yiengst (11) during the summer of 1941 at the Beltsville Research Center indicated conclusively that after feeding on an infected bull *Tabanus sulcifrons* transmitted anaplasmosis to three healthy cows within a period of 24 to 28 days. These large biting flies were of one of the seven species used by Sanborn et al. (fig. 2.)

Since mosquitoes are known vectors of certain diseases, including malaria, yellow fever, fowl pox, and equine sleeping sickness, these insects also were tested for their ability to transmit anaplasmosis.

Mosquitoes are often present during the anaplasmosis season in large numbers and cause considerable annoyance to cattle. The genus *Psorophora* often breeds in transient pools where cattle graze, and since they are vicious biters, they were suspected of transmitting the disease. That they do so was proved by experimental feedings.

During the summer of 1940 Howell, Stiles, and Moe (9) were the first workers to demonstrate the transmission of anaplasmosis by mosquitoes. In one instance 1,525 mosquitoes, *Psorophora columbiae* and *P. ciliata*, were fed on a cow with a clinical case of anaplasmosis and then allowed to feed on a healthy cow, which developed a typical case of anaplasmosis after 38 days. A second experiment, using three species of mosquitoes, *P. ciliata*, *P. columbiae*, and *Aedes aegypti*, resulted in a positive case of anaplasmosis 58 days after the first feeding, or 38 days after the last of 241 feedings.

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