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EC73-1907 Internal Parasites of Cattle

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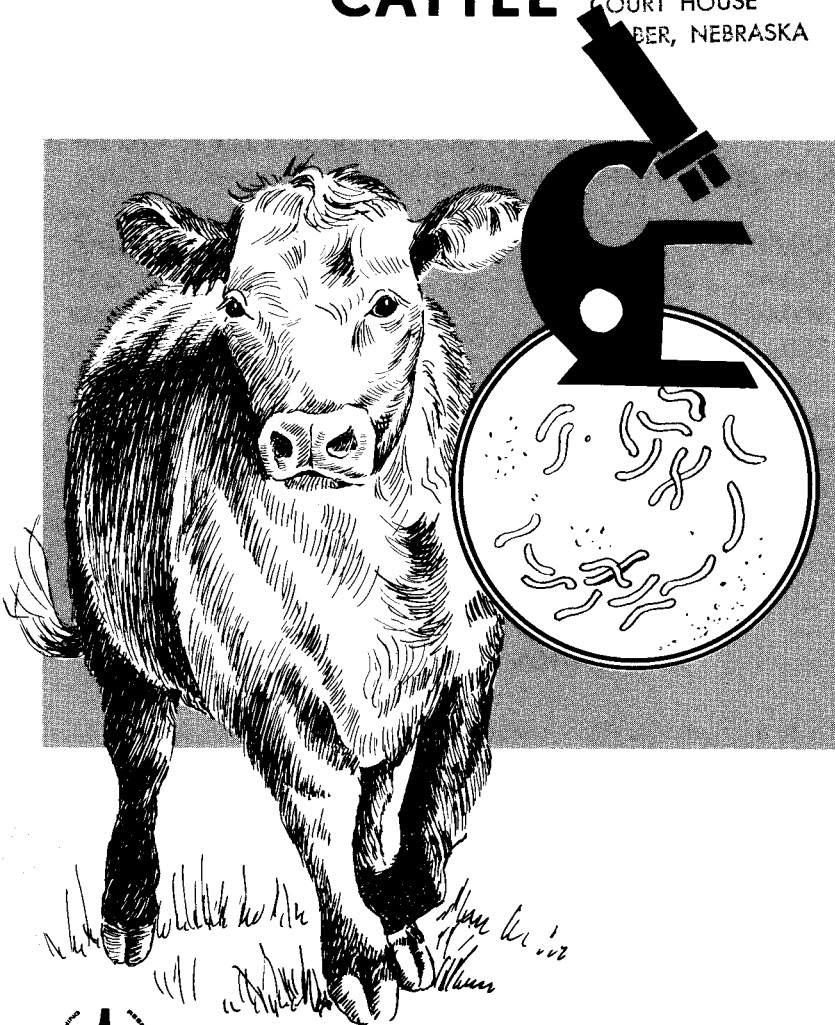
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INTERNAL PARASITES OF CATTLE

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INTERNAL PARASITES of cattle

by D. L. Ferguson
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INTRODUCTION

Internal parasites continue to be one of the major problems confronting cattle producers. In 1965, the United States Department of Agriculture estimated that internal parasites in cattle caused an average annual loss of \$161,678,000.

The internal parasites responsible for the annual loss are listed below.

Anaplasmosis	\$ 36,001,000
Coccidiosis	14,569,000
Worm Parasites	100,046,000
Liver Flukes	3,022,000
Trichomoniasis	8,040,000

In cattle the effects of internal parasites will be well camouflaged. However, additional feed will be required to reach market weight. Parasitologists have estimated that subclinical infections of internal parasites may reduce weight gains in cattle as much as 10 to 20 percent. Contrary to popular opinion, worms do grow best in healthy, well-fed cattle.

DIAGNOSIS OF WORMS

To determine if an animal is infected with worms, a fresh fecal sample is collected. A weighed amount of the fecal material is mixed with a salt or sugar solution that causes the worm eggs to float. The eggs are then collected on a glass slide and counted. This procedure shows the number of worm eggs per gram (EPG) of feces. The larger the number of adult female worms inside an animal, the greater the concentration of eggs in the feces. Accurate identification of the numerous species of worms is accomplished by microscopic examination of eggs and larvae.

Accurate diagnosis of gastrointestinal parasitism requires training and proper equipment. Clinical signs may be observed in animals which are heavily infected with larval worms. Examination of the feces of these animals might not detect infection since the immature worms have not produced eggs.

Necropsy (complete examination of the animal after death) is the most accurate method of diagnosis. For example, *Trichostrongylus axei* is a stomach worm of cattle that is found buried in the wall of the abomasum (fourth stomach). Because of its small size (1/8 to 1/4 inch in length), reddish brown color, and being buried in the mucosa of the stomach it is difficult to see at necropsy. The gross appearance of the stomach lining may give some clue to the presence of these worms. Microscopic tissue sections may have to be made to find the parasites.

The problems encountered in the diagnosis of parasitism and accompanying disease require the skill, judgment, and trained eye of a veterinarian. It is necessary that disease, nutrition, and parasitism be put in proper perspective so that the best treatment and management methods may be used to overcome the problem.

PREVENTION AND CONTROL OF WORMS

Treating cattle with an anthelmintic drug is generally recommended when fecal examinations indicate that a large number of worms are present. In mixed infections, counts of 300 worm eggs per gram of feces constitute a level for differentiating between clinical and subclinical parasitism. Using this as a guide, it is recommended that cattle with egg counts of 300 or more be treated.

Since moisture favors the development of free-living stages of infective larvae and dryness kills them, practices that reduce moisture on pasture will decrease parasitic contamination. Pastures should be well drained, watering or feeding troughs raised above the ground, and grazing should be kept to a minimum on the lush grass along the edges of ponds and streams.

In areas where cattle congregate, overgrazing should be avoided. Otherwise, the cattle will be forced to graze to the base of the roots of the plants where they may ingest large numbers of larvae.

Remove manure from the barn often and compost it or spread it on the ground where cattle do not graze. Larvae are protected by the moisture in dung pads and they may survive for long periods.

Feed an ample, well-balanced ration to help increase the resistance of the cattle. Make certain that adequate minerals are supplied. If possible, feed the cattle in drylot. Transmission of parasites is at a minimum on drylot.

TYPES OF INTERNAL PARASITES

Three common stomach worms are found in Nebraska cattle: The large stomach, barberpole or wire worm—*Haemonchus*, medium or brown stomach worm—*Ostertagia*, and small stomach or bankrupt worm—*Trichostrongylus*.

***Haemonchus*—Barberpole or Wire Worm**

Adult female worms measure $\frac{1}{2}$ to 1 inch in length. Males are $\frac{1}{4}$ to $\frac{1}{2}$ inch long. The common name arises from the fact that in the femal worm the white ovaries are twisted around the intestine.

Life Cycle

A mature female stomach worm will lay about 10,000 eggs per day. These eggs are deposited within the manure on pasture. Under favorable conditions of temperature and moisture, larvae will hatch from the eggs and become infective within five days.

The infective larvae are swallowed by cattle with the pasture foliage and burrow into the lining of the fourth stomach (abomasum) to develop. When the larvae emerge from the lining of the stomach, they attach to the stomach wall and begin to suck blood. It takes about 21 days for female worms to reach maturity and start laying eggs which are deposited on the pasture to begin the cycle again.

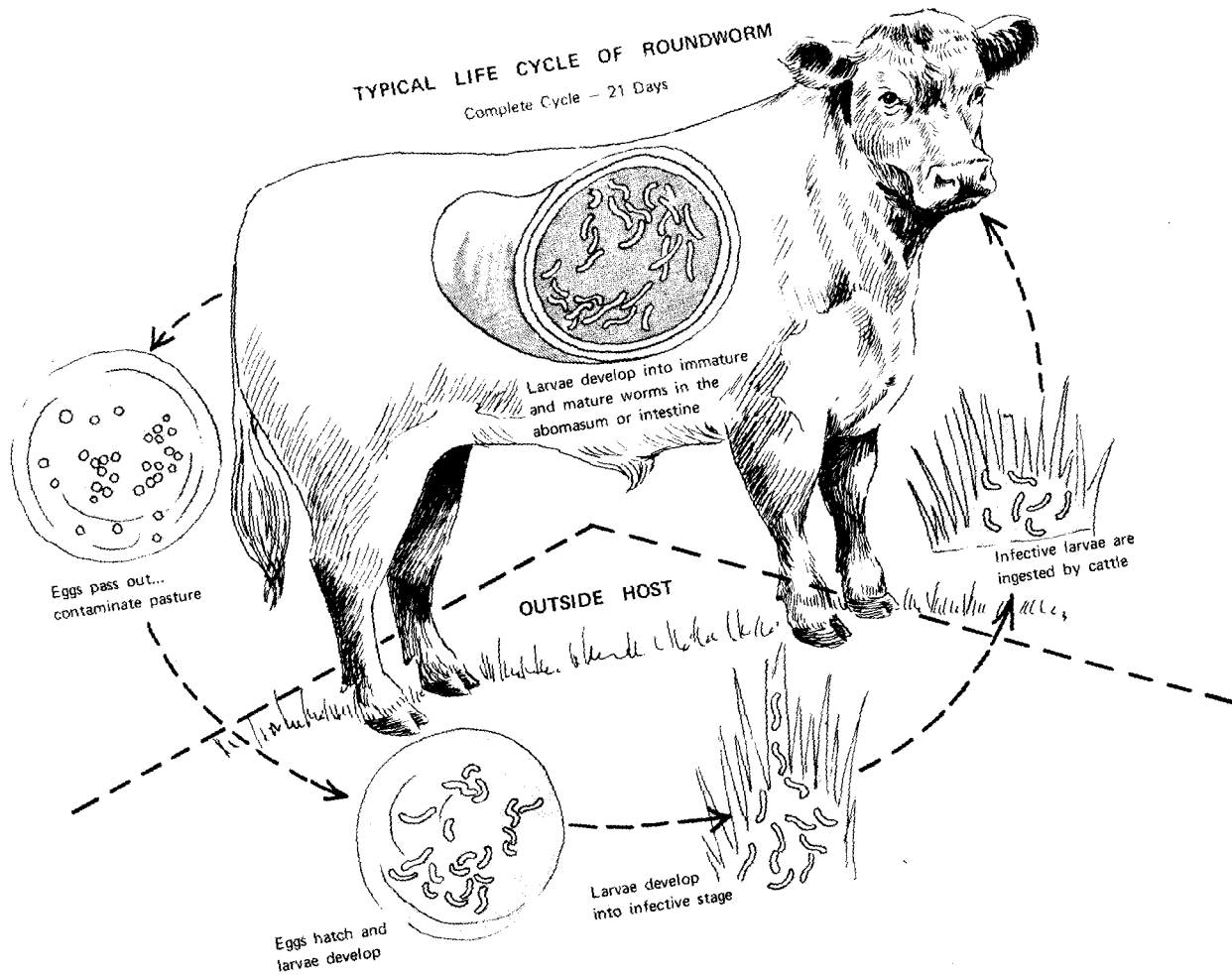
If weather conditions are dry and cold, worm eggs may remain dormant for weeks, with the subsequent emergence of large numbers of infective larvae when conditions become favorable (warm and wet).

Clinical Signs

Both young and mature cattle are affected by stomach worms. Since the worms live on blood, anemia may become a problem. Anemia is indicated by paleness of membranes or lining of the

TYPICAL LIFE CYCLE OF ROUNDWORM

Complete Cycle - 21 Days



mouth. Loss of appetite, loss of weight, rough hair coat, and listlessness frequently accompany anemia.

Larval worms are very active and heavy infections can cause digestive disturbances. There may be a mild diarrhea, with intermittent periods of constipation. "Bottle jaw" or swelling under the lower jaw is frequently a sign of infection.

***Ostertagia*—Medium or Brown Stomach Worm**

These worms are smaller than the large stomach worm. Adult females measure about ½ inch in length and males are slightly smaller.

Life Cycle

The life cycle of *Ostertagia*, medium stomach worm, is similar to that of *Haemonchus*, the large stomach worm. Adult female worms in the abomasum produce eggs which are deposited within the manure on pasture. These eggs develop into infective 3rd stage larvae in 7 to 9 days. After ingestion by cattle these larvae molt to the 4th stage in 3 to 8 days. The 4th stage larvae then enter into the glandular areas of the abomasum where they produce small, cystic nodules in the mucosa. Adult worms emerge into the abomasal lumen to suck blood and start producing eggs 21 to 23 days after infection.

When infective larvae enter the mucosa of the abomasum, there may be a histotropic phase in the life cycle in which the immature worms remain in the mucosa for several months. Most of these stages are found in the gastric glands of the fundus of the abomasum. Removal of adult *Ostertagia* by anthelmintic treatment allows these larvae present in developmental arrest to begin development again and reach the adult stage.

Clinical Signs

Ostertagia infections resemble those produced by *Haemonchus*, with one exception—*Ostertagia* produces a severe, persistent diarrhea.

***Trichostrongylus*—Small Stomach or Bankrupt Worm**

Adult worms are reddish brown and measure from 1/8 to 1/4

inch in length. The life cycle is similar to that of the large and medium stomach worms.

Life Cycle

Female worms in the abomasum produce relatively small numbers of eggs which are passed in the feces and hatch on pasture in a day or more, depending on the temperature; oxygen and moisture are necessary for development and hatching. The larvae develop to the infective 3rd stage in 3 days to 2 weeks depending upon temperature and other conditions.

The 3rd stage larvae migrate out of the feces, get onto the vegetation or soil, and survive on their stored food materials without eating for days or months depending upon the environmental conditions.

After ingestion the larvae enter the mucosa of the abomasum, molt to the 4th stage in about 4 days, and reach the young adult stage in about 10 days.

Clinical Signs

Like the medium stomach worm, *Trichostrongylus* produces a profuse, watery diarrhea. The lining of the abomasum in infected cattle is often greatly congested or reddened and may be entirely or partially covered with a stringy exudate.

Treatment of Stomach Worms

Phenothiazine will effectively remove adult *Haemonchus*, but is less effective in removing *Trichostrongylus* and least effective against *Ostertagia*. It is relatively ineffective against immature stages of all three stomach worms. Toxic reactions following phenothiazine treatment are unusual in ruminants, but keratitis may result from photosensitization of the cornea. To avoid this reaction, treatment should be given in the afternoon and the animals kept in the shade for the following day.

Particle size and purity significantly affect the anthelmintic efficacy of phenothiazine. When the drug is finely ground and purity improved, the efficacy is increased. Phenothiazine acts primarily against adult worms and is relatively ineffective against immature stages. Therefore, treatments should be repeated at 2 to 3 week

intervals because of the continuous emergence of larvae from the tissues and the continued ingestion of larvae from the pasture.

While a consistent curative effect has not been demonstrated against certain species of worms, the administration of small doses of phenothiazine in the feed has a marked effect on the egg output and the larval development of these species. The reduction in pasture contamination that is likely to ensue is of obvious benefit in the control of parasitism.

Thiabendazole is effective against adults of all three species of stomach worms in cattle and has greater activity against the immature stages than phenothiazine. Thiabendazole is well tolerated by cattle and can be administered orally or mixed with the feed. In addition to its wide therapeutic index, toxic effects have not been reported.

Tetramisole has recently been developed for use in cattle. It is a broad spectrum anthelmintic drug that has activity against the three stomach worms. In addition, it is extremely effective against cattle lungworms. Note: Lungworms have not been reported in Nebraska cattle.

Several organo-phosphorus compounds have been used with success in cattle. These include: Coumaphos (CoRal, Asuntol), Neguvon (Trichlorfon), Ruelene, Ronnel, and Haloxon.

Intestinal Worms

Three species of *Cooperia* occur in the small intestine and rarely in the abomasum of cattle: *Cooperia oncophora*, *Cooperia pectinata*, and *Cooperia punctata*. These small, reddish worms are difficult to see without a microscope. The adult worms measure 2/8 to 3/8 inch in length.

Life Cycle

The life cycle for Cooperids is similar to that of the large stomach worm, *Haemonchus*. Eggs passed in the feces develop into infective 3rd stage larvae in 7 to 9 days. After ingestion by cattle these larvae pass to the small intestine, molt to the 4th stage in about 4 days, and to the adult stage by the 8th day. *Cooperia* infections in cattle may last up to 9 months or longer.

Clinical Signs

In heavy infections with *Cooperia* sp., there is a profuse diarrhea, loss of appetite, dehydration, and progressive emaciation. There is no evidence of anemia. These worms apparently do not suck blood. Necropsy shows a severe congestion of the intestine.

Treatment of Cooperia

Piperazine, thiabendazole, tetramisole, coumaphos, and ruelene are effective in removing adult worms. Continuous low level administration of phenothiazine aids in preventing larval development if sufficient drug is ingested.

***Nematodirus*—Thread-necked Intestinal Worm**

Nematodirus is a common parasite of young calves and occurs in the small intestine. Adult females measure 3/4 to 1 inch in length and males are 1/2 to 3/4 inch long.

Life Cycle

After being deposited on pasture within the manure, worm eggs develop slowly, the infective 3rd stage being reached within the egg shell in 2 to 4 weeks. Larvae may remain inside the eggs for a long time before hatching and may pass the winter in this condition. Calves become infected by ingesting the larvae while grazing.

Within the small intestine, mature female worms start laying eggs about 21 days after infection. Relatively small numbers of eggs are produced by these worms.

Clinical Signs

Severe infection may cause diarrhea, loss of appetite and weakness. These signs usually develop during the third or fourth week of infection. Resistance to reinfection develops rapidly and the adult worms are mostly eliminated within 2 to 3 months. *Nematodirus* is apparently not a blood sucker.

Treatment of Nematodirus

Thiabendazole and tetramisole are effective against the adults.

Oesophagostomum—Nodular Worm

Nodular worms live in the small and large intestine of cattle. Adult worms measure 3/8 to 5/8 inch in length.

Life Cycle

Eggs are passed in the feces and infective larvae develop in 5 to 6 days. Cattle ingest the larvae in grazing. They enter the walls of the small and large intestine and molt to the 4th stage in 5 to 7 days. The larvae return to the intestinal lumen 7 to 14 days after infection, and molt to the adult stage in the large intestine in 17 to 22 days. Eggs appear in the feces 32 to 42 days after the infective larvae have been ingested by the cattle.

Clinical Signs

In severe infections, a rise in body temperature may appear 4 to 10 days after ingestion of infective larvae. Severe diarrhea, followed by loss of appetite, emaciation, and general weakness can develop 7 to 17 days after infection. As a result of larval damage, small nodules are produced in the wall of the small and large intestine. At first, these are only slightly raised areas, but later the nodules increase in size and are often filled with pus.

Treatment of Nodular Worms

Phenothiazine, piperazine, ruelene, tetramisole, trichlorfon, and thiabendazole are all effective against the adults; no treatment is available which will destroy the larvae in the nodules. Feeding low level phenothiazine has been recommended to prevent egg production by the female worms.

Tapeworms

The common tapeworms of cattle are *Moniezia benedeni* and *Moniezia expansa*. The life cycle includes a small soil mite as an intermediate host. Tapeworm eggs are ingested by the free-living mites which live in the soil and on grass. The young tapeworms develop for 6 to 16 weeks inside the body of the mite. Cattle are infected by ingestion of infected mites. Adult tapeworms develop in about 35 to 40 days and frequently reach a length of 10 to 12 feet.

Clinical Signs

Moniezia is usually considered nonpathogenic in cattle. However, extreme weakness, marked anemia, and a profuse diarrhea have been reported in heavy infections.

Treatment of Tapeworms

Lead arsenate appears to be the most effective drug currently used against tapeworms in cattle. Niclosamide is also effective. *Caution:* Wormers containing lead must be properly used. Lead is a poison which may cause chronic illness or death. Study the directions and use only as indicated.

Bovine Coccidiosis

Coccidiosis is caused by microscopic, one-celled parasites of the genus *Eimeria*. Fifteen species of *Eimeria* have been reported in cattle. Only three of these, *Eimeria zurnii*, *Eimeria bovis*, and *Eimeria ellipsoidalis* are regularly associated with clinical infections in the field.

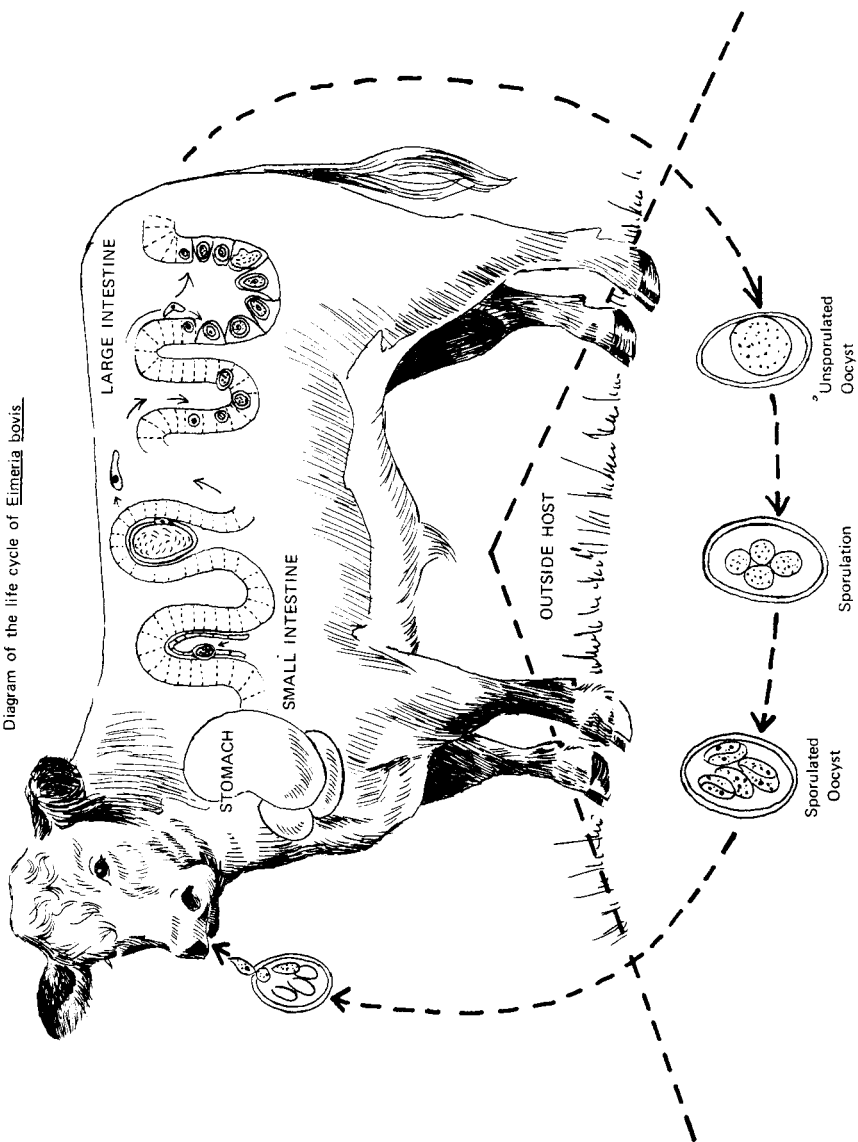
Coccidiosis usually affects calves between 3 weeks to 6 months of age when they are placed in lots contaminated by older cattle or other infected calves. Occasionally, mature cattle are infected when they are brought in from large pastures and crowded into small feedlots or barns. It is extremely rare to find cattle manure entirely free of these parasites.

Life Cycle

The stage found in the feces is the oocyst. It is covered by a protective shell, resistant to physical, chemical, and bacterial action. Oocysts freshly discharged in the feces must undergo a developmental process called sporulation, before they become infective to other animals. This process, occurring outside of the animal, requires 2 to 3 days and results in the formation within each oocyst of eight infective bodies called sporozoites.

When a susceptible calf swallows the infective oocysts, the sporozoites are released, penetrate the cells lining the intestine, and begin to divide into many intermediate stages. The stages continue to divide, and each division produces parasitic stages that cause damage to the host cells in which each lives.

Diagram of the life cycle of Elmeria bovis.



Male and female parasites unite to produce the oocyst which is then passed out of the animal's body in the feces. The life cycle is started again after sporulation occurs and the oocyst is swallowed by a susceptible calf.

Signs of Coccidiosis

Typical signs of coccidiosis are diarrhea, rough coat, loss of appetite and weight, weakness, and a general emaciation. The general weakness may cause the calf to defecate without rising, thus soiling its tail and hindquarters. In more severe cases, the feces may contain blood, mucus, and stringy masses of tissue. Straining may be observed in the more advanced cases.

Diagnosis

Diagnosis of clinical coccidiosis can be made by finding large numbers of the characteristic oocysts of *Eimeria* on microscopic examination of feces. Identification can best be made by observations on freshly discharged oocysts. Usually, diarrhea precedes the heavy discharge of oocysts by a day or two and the diarrhea may continue after the oocyst discharge has returned to low levels. Therefore, it is not always possible to confirm a clinical diagnosis of coccidiosis by finding oocysts in the feces.

Certain coccidia may cause severe illness and even death before any oocysts have been produced. This occurs quite commonly with *Eimeria zurnii* in calves. Consequently, failure to find oocysts in the feces in a diarrheal disease does not necessarily mean the disease is not coccidiosis.

The most accurate way to diagnose coccidiosis is to examine material at necropsy from suspicious areas in the small intestine or colon under a microscope. To do this, scrapings of these areas are mixed on a glass slide with physiological saline solution and examined for oocysts and other life cycle stages.

Treatment and Control

Treatment of bovine coccidiosis is difficult because clinical signs of the disease do not become noticeable until the disease is advanced. The first signs in coccidiosis caused by *Eimeria bovis*, usually occur about 17 to 18 days after ingestion of oocysts. At this time, the

portion of the life cycle within the host has been nearly or entirely completed, and much of the invasion of intestinal mucosa has already occurred. Thus, treatment administered at this time can at best result in a lessening of the signs of coccidiosis. However, if drugs are given at an earlier stage of the disease, the clinical signs of infection can be largely or entirely prevented.

Sulfonamides remain the drugs of choice in treatment of coccidiosis. While most clinical infections are self-limiting and will subside within a few days without medication, sulfonamide therapy, if started early, will reduce oocyst production, hemorrhage and diarrhea, shorten the course of chronic disease and lower the mortality, and reduce the chances for secondary infection. Sulfaquinoxaline and sulfamethazine have given excellent clinical results in beef and dairy calves.

Of the newer drugs, amprolium, which has been highly effective in avian coccidiosis, has recently been found to be of value in controlling coccidiosis in cattle. Amprolium is an antagonist of thiamine, one of the essential vitamins, and thus interferes with the metabolism of the parasites. Therefore, treatment could be started in all members of a group of cattle at the time coccidiosis is first diagnosed in one or more individuals in the group, and could be continued as long as necessary to provide control.

The findings that this drug is effective in preventing coccidiosis in cattle indicates that it may be useful for this purpose in special situations in which the disease frequently occurs.

As in many diseases, it is easier to prevent coccidiosis than it is to treat it. Because several days are required for sporulation, the oocyst stage is the weakest link in the life cycle of the parasite. Separating a cow and calf from a contaminated lot interrupts the life cycle and helps control the disease.

Bovine Trichomoniasis

Trichomoniasis is caused by microscopic, one-celled parasites of the genus—*Tritrichomonas*. It occurs in both dairy and beef cattle. Losses result from reduced milk production, reduced calf crop, interference with breeding programs, and the necessity of treating or slaughtering infected bulls and some infected females.

The most important species of trichomonad in cattle is *Tritrichomonas foetus*. This small parasite lives in the reproductive system and causes temporary infertility, abortions, or chronic infection of the uterus.

Life Cycle

Trichomonads have a simple life cycle. They multiply by longitudinal binary fission. This occurs when new individuals are derived from one "parent", and no special reproductive structures are involved. An individual trichomonad divides into two halves, after which each half grows to the original form.

Clinical Signs

Genital trichomoniasis is a chronic disease in bulls, where the organism is found in the sheath and on the surface of the penis. The reproductive ability of the bull is not impaired by the infection, except that the trichomonads are deposited in the vagina of the female along with the semen. These trichomonads multiply in the vagina and the uterus, resulting in death of the embryo or fetus.

Early abortion is characteristic of infection. Abortion usually occurs 1 to 16 weeks after breeding. The fetus is often so small that it is not observed by the owner, and he does not realize that abortion has occurred, believing that the animal failed to conceive and that its heat periods are irregular.

If the fetus dies at a later stage of pregnancy (3-4 months), the fetus may be retained, or the uterus may be filled with pus (pyometra). Sometimes, permanent infertility may result from trichomoniasis, but the infection usually disappears spontaneously, and fertility returns after a few months.

A heifer or cow that has recovered from an infection may be resistant to a second infection. If she is bred to an infected bull, she may not become infected or may develop only a slight infection that will not interfere with pregnancy. This resistance, however, varies considerably in degree and duration among different females.

Symptoms in cows or heifers include a slight reddening and swelling of the vulva and the walls of the vagina. The discharge from the vagina is somewhat greater in quantity and more watery than usual. Some degree of irregularity in heat periods may occur.

Transmission

Transmission is nearly always by breeding. Heifers and cows become infected by being bred to an infected bull. Bulls become infected by breeding infected females. Herds usually become infected by introduction of infected females or bulls into the herd.

Diagnosis

The presence of trichomoniasis in a herd is most easily detected by examination of samples from the bull.

The bull to be examined should be withheld from service for one week, since breeding reduces the number of organisms present. Bulls that contaminate the prepuce by mounting other bulls should be isolated until collection of a clean sample is possible.

Three sampling techniques are usually employed for examining bulls:

1. A cotton gauze sponge soaked in physiological saline is passed to the fornix of the prepuce. This region is swabbed with a back-and-forth motion and then several drops of the fluid are squeezed onto a microscopic slide and examined.

2. In the pipette method for bulls, preputial fluid is collected from the deep portion of the prepuce by means of a 20-inch plastic pipette equipped with a 4 oz. rubber bulb. The material is placed on a slide and examined with a microscope.

3. The douche method for bulls consists of introducing 100-125 ml. of physiological saline into the prepuce, massaging thoroughly the glans penis through the sheath (about 100 vigorous strokes), and then recovering the fluid. The sample is centrifuged for 10 minutes at 2,000 rpm, a drop of the sediment is placed on the slide, covered with a cover glass, and examined under low power on a microscope. Occasionally the entire cover glass must be examined before trichomonads are found. Upon finding the parasite, it is necessary to switch to high power or oil objectives on the microscope to make a positive identification. If trichomonads are not found, a part of the material is inoculated into culture medium, in which trichomonads may multiply sufficiently to be found easily under the microscope after 3 days of incubation at 102.2°F. Addition of penicillin and streptomycin to the medium inhibits the growth of bacteria, making conditions more favorable for trichomonads.

Diagnosis in the bull may also be made by breeding several virgin heifers and examining the vaginal fluid 12 to 19 days after service.

Heifers or cows are examined by collecting a sample of fluid from the vagina with the aid of a pipette and bulb similar to that used for the bull, but with a sideward opening at the end opposite the bulb. Sterile saline solution is injected into the forward region of the vagina; then fluid from this region is sucked into the pipette. The fluid collected is examined for trichomonads in a way similar to that used for the samples from bulls.

Treatment

Infected bulls may be successfully treated by applying medicated salve to the penis and mucous membrane of the prepuce. During the treatment the bull should be under the influence of a tranquilizer or a local anesthetic should be applied to the genital region. Treatment should be repeated in 10 to 14 days. Since trichomoniasis is ordinarily self-limiting in females, treatment is usually unnecessary.

Diagnostic tests to confirm the success of treatment of bulls should be started two months after treatment and conducted at regular intervals thereafter.

Control

Control of bovine trichomoniasis depends on proper herd management. Infected bulls should be treated. Infected cows should be given a breeding rest, and should then be bred by artificial insemination to avoid infecting clean bulls.

Proper herd management of bulls used for artificial insemination is especially important, since they may spread the infection widely. They should be examined for *Tritrichomonas foetus* before purchase and the herds from which they originated should be studied at the same time. In addition, they should be examined repeatedly while in use.