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DIARRHEA IN WEANED CALVES

It is not uncommon to wean calves and then experience an outbreak of respiratory disease. Likewise, sometimes the weaned calves will break with diarrhea and this can be more difficult to diagnose the cause. Diarrhea in recently weaned calves are commonly caused by BVD (Bovine Virus Diarrhea), mineral deficiencies (particularly copper or selenium), worms, and coccidiosis.

We have written about BVD often in the past and it is important for you to work with your veterinarian to optimize your vaccination program to prevent BVD and to make sure you are a low or zero risk of having BVD persistently infected cattle (BVD-PI) in your herd. Most of us deworm calves at weaning, thus heavy worm infestations that will cause diarrhea are not real common. However, if you do not deworm at weaning, worm infestation can cause diarrhea and other problems. Most of us know if our herds are deficient in copper or selenium. Recall, that neither copper nor selenium are transferred through the cow's milk and the calves can be "rock bottom" low in these nutrients at weaning and this can easily result in diarrhea and associated losses. The last item on the list of common causes of diarrhea in weaned calves is coccidiosis. The remainder of the column is devoted to this disease; however, it is important to make sure the other common problems listed above are not part of your problem.

Coccidiosis can cause significant economic losses in cattle. Industry experts estimate the losses at $100 million each year. Although most cattle are exposed to coccidia and infected, many of the infections are self-limiting and mild or asymptomatic. The parasites that cause this condition are members of the genus ***Eimeria***, and the most important of this genus for causing disease in cattle are ***Eimeria bovis*** and ***Eimeria zuernii***.

The life cycle of these parasites is complex. Single cell **oocysts (eggs)** are passed in the feces of cattle, are resistant to disinfectants, and can remain in the environment (particularly moist, shady areas) for long periods of time and maintain their infectivity. The oocysts sporulate and these sporulated oocysts are ingested by the host and the **sporozoites** are released in the intestine. Sporozoites enter the intestinal cells, form **trophozoites**, which in turn divide into many **merozoites**. These merozoites penetrate additional intestinal epithelial cells and form more **meronts**. Eventually, **macrogametes** and **microgametes** are formed which combine to produce the next generation of oocysts. When the oocysts are mature, they rupture the host cell and are released into the lumen of the intestine and pass out in the feces. The multiplication rate of these organisms is phenomenal as illustrated by the following:

1 oocyst
X 8 sporozoites
X 120,000 first generation merozoites
X 30 second generation merozoites
X 80% macrogametocytes
= 23,040,000 oocysts

The potential damage to the intestinal cells is obvious. It is estimated that as few as 50,000 infective oocysts ingested by a young susceptible calf can cause severe disease. The replication of the coccidia within the host's intestinal cells and the subsequent rupture of the cells is responsible for the disease and the clinical signs that develop. The severity of the disease is directly related to the dose of infective oocysts that are ingested. The more oocysts ingested, the more severe the subsequent disease. With light infections, the damage to the gut cells is minimal and because the cells in the gastrointestinal tract are replaced rapidly the damage is quickly repaired. In the case of heavy infections, about two weeks after the oocysts are ingested; most of the epithelial cells at the base of the intestinal glands are occupied by meronts or gametocytes. As these cells rupture, damage is severe and there is loss of blood into the feces. Also, fluid, electrolytes, and blood proteins (albumin) are lost.

Most animals infected with coccidia do not show signs of illness. This is due to the normally low dose and after a course of infection the animal is immune to that particular ***Eimeria*** species. However, this does not mean they are immune to all ***Eimeria*** species. Therefore, coccidiosis is primarily a disease of the young where there is crowding, stress, and/or nonimmune animals. Older cows certainly act as a reservoir and shed oocysts into the environment. Stress such as shipping, weaning, dietary changes, cattle shows or sales, and other problems can precipitate an outbreak of coccidiosis. Older cattle which are immune to their own endemic species of coccidia can become infected and/or ill when moved to a new herd and exposed to a different species.

The clinical signs of coccidiosis can include the following:

Diarrhea (bloody at times)
Straining (tenesmus)
Loss of appetite
Fever (slight)
Debility
Death (in severe cases)

Many cattle are affected and experience weight loss or decreased weight gains without showing obvious signs of illness and these cattle account for the majority of the economic losses.

Your veterinarian can diagnose coccidiosis on the basis of clinical signs, fecal oocysts examinations, and post mortem examination of dead animals (if that occurs). Once an accurate diagnosis is made there are a number of drugs useful in treatment or prevention. Some of the drugs that can be used for **treatment** include:

Amprolium	Corid®	4.5 mg/lb (10 mg/kg) daily for 5 days
Sulfaquinoxaline		1.2 mg/lb (2.7 mg/kg) daily for 3-5 days
Sulfamethazine		50 mg/lb (110 mg/kg) daily for 5 days

Some of these drugs and dosages may require a veterinarian’s prescription and extended withdrawal time, be sure to check with your veterinarian before treating animals. Other drugs can be very useful in helping to **prevent** coccidiosis and some of these are listed below:

Lasalocid	Bovatec®	0.45 mg/lb (1 mg/kg)per day, max.360 mg/day
Decoquinate	Deccox®	22.7 mg/100 lb. daily for 28 days
Monensin	Rumensin®	100 to 360 mg/head per day

Both lasalocid and monensin are polyether ionophores which are used to increase feed efficiency and weight gains; however, they also have effectiveness to prevent (but not for treatment of) coccidiosis. Monensin has a lower threshold for toxicity and cattle must be gradually introduced to it in their diet to prevent diarrhea, feed refusal, or toxicity. **Drugs useful for treatment are not necessarily useful for prevention and vice versa.** Drugs administered in feed or water may not be consumed by sick animals, so you must be aware of this in treating ill cattle and the most severely affected cattle will have to be treated individually.

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