Clostridial Infections in Calves

Intestinal spores only prevented by vaccination.

by Heather Smith Thomas

Clostridial infections are bad news and almost always fatal. The good news is that most can be prevented by vaccination. The not-so-good news is that there are a few that cause gut infections in calves for which there are no vaccines.

Several serious livestock diseases are caused by this group of anaerobic bacteria called clostridia. When exposed to adverse conditions, such as heat or drying, these bacteria form a protective shell-like covering in a dormant stage called spores. The spores can remain viable a long time. Some live in soil and infect animals later when ingested with feed or when introduced into a wound.

Spores can also exist in a latent/dormant state within animals, then suddenly come to life and multiply, producing deadly toxins when conditions become favorable in an airless environment. Toxins of different types of clostridia vary in their effects and how they gain access to the bloodstream. These bacteria multiply in the absence of oxygen and release deadly toxins faster than the body can mount a defense, causing sudden death.

Veterinarian Steve Hendrick of Coaldale Veterinary Clinic, Coaldale, Alta., Canada, says most producers are accustomed to dealing with contagious diseases — where an animal gets sick and can pass the disease to another.

“This is not the case with clostridial diseases,” he says. “They are caused by spores that live happily in the environment, resistant to heat, cold and any other harsh environmental conditions. They can survive in a hot, dry desert or sub-zero winter weather.”

They can be found in the intestinal contents of normal animals and humans as part of the gastrointestinal (GI) tract flora, and they can be found in soil that contains manure. They cause disease when diet or management changes produce an environment more favorable for swift multiplication. The only way to protect cattle is by vaccination.

Seven- and eight-way vaccines combine protection against the most common clostridial diseases in cattle, such as blackleg, redwater, malignant edema, Black disease, enterotoxemia, and there is a separate vaccine for tetanus. There are some intestinal infections in calves, however, caused by types of Clostridium perfringens that are not included in these vaccines.

Blackleg

This acute and highly fatal disease is caused by C. chauvoei. The spores enter the body via the digestive tract, where they pass through the gut wall into the bloodstream and end up in muscles and other tissues. They may live in the gut, spleen and liver without causing problems, and they can lie dormant in muscles until conditions are perfect for multiplication.

“Then these bacteria produce deadly toxins if there is injury...”
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or bruising of the muscles, or any other condition that reduces oxygen level in the tissues,” says Hendrick. Muscle trauma associated with exertion, transport or herding/handling may trigger multiplication. In an unvaccinated herd, cattle of all ages are susceptible, but this disease appears most frequently in the fastest-growing calves.

Many cattle died of blackleg in earlier years; this was one of the first cattle vaccines developed. Today the only blackleg vaccine available is the combination that includes protection against some of the other clostridial diseases, as well.

In an unvaccinated animal, blackleg symptoms (depression, lameness, swelling of the upper leg) appear suddenly; you may just find the animal dead. Gas bubbles in the muscles can often be felt under the skin, cracking when you touch the skin.

“Treatment is usually futile unless begun at the first sign of symptoms,” says Hendrick. “Large doses of penicillin may save the animal.”

Prevention consists of vaccinating calves at 2 to 4 months of age, with a booster at weaning time. This two-dose schedule usually gives lifelong immunity against blackleg. Some of the other clostridial diseases, such as redwater and Black disease, can be a threat at any time during the life of the animal, so a combination vaccine is given annually. It may be given more often if redwater is a concern.

Malignant edema

Malignant edema affects cattle of all ages and is caused by *C. septicum*.

“These bacteria are found in feces and in soil wherever there are livestock populations,” says Hendrick. They gain entrance via deep wounds, contaminated surgical sites that are not adequately disinfected, or vaginal or uterine injuries postcalving.

Symptoms include loss of appetite, dullness, swelling around the wound and lower parts of the body, and high fever. The animal dies within 24 to 48 hours. Postmortem examination reveals dark, foul-smelling dead tissue under the skin.

Prevention is by vaccination.

Difficult difficile

*Clostridium difficile* causes enterotoxemia in foals, but it can also cause problems in calves, says Iowa State University’s J. Glenn Songer, citing his own research and that of others. The studies involved mostly young Holstein calves that had been fed pooled frozen colostrum before delivery to the feedyard.

“We found that a lot of these very young feedlot calves (1-6 weeks of age) get diarrhea and become emaciated. If they respond to treatment, it’s usually to something like tylosin, which tends to work for a while,” he explains, noting a tylosin resistance. “Soon the tylosin-resistant bug becomes the dominant one. Then you can’t treat with that anymore.”

“We don’t know very much about *C. difficile* in calves because we have not yet been able to reproduce this disease in calves, and all of these diseases are multi-factorial. Reproducing any kind of clostridial disease in the gut of a calf is a challenge ... We don’t actually know that *C. difficile* is causing a problem, but we are sure enough that we think it should be researched,” says Songer.

“Even though we have not been able to reproduce the disease by inoculating calves with these bacteria, we have purified the toxins of *C. difficile* and put them into ligated segments of small intestine and colon in certain calves. The result of a few hours’ incubation of toxin within those loops are lesions very similar to what we see in calves that are toxin-positive and have diarrhea in these calf ranches. So I think there’s a fair chance that *C. difficile* is a causative agent,” he says.

Black disease

Black disease is caused by *C. novyi* and is seen more often in feedlot cattle than in cow-calf herds. Spores may enter the body via wounds or by mouth, then localize in muscles.

As with blackleg, anything that damages cells and reduces oxygen in tissues near the spores enables them to multiply and produce toxins. Symptoms are similar to malignant edema, but usually the affected animal is found dead because the infection is so swiftly fatal.

Prevention requires two doses of vaccine.

Sudden death

*C. sordellii* is also highly fatal, and symptoms are rarely seen. The animal is simply found dead. This disease tends to affect feedlot animals rather than cows.

Route of transmission is thought to be ingestion. The bacteria can reside in muscle tissue (just like blackleg) and remain harmless until triggered into multiplying and producing toxins by anything that lowers oxygen content of the tissues. This toxin is more deadly than that produced by blackleg or malignant edema. Cattle of all ages are susceptible, but calves and young adult cattle are most often affected, unless vaccinated.
Redwater

Redwater (Bacillary hemoglobinuria) is caused by C. haemolyticum. Spores survive in the environment a long time. This disease occurs most often where snails in marshy areas serve as intermediate host to liver flukes. Damage from flukes enables the bacteria to proliferate in liver tissue, producing deadly toxins.

The disease is seen less commonly in cattle pastured in dry areas with no access to surface water inhabited by snails.

“Many people don’t realize redwater disease starts with liver damage. If you live in an area that has liver flukes, cattle are at risk for redwater,” explains Hendrick. “The difference between a seven-way and an eight-way vaccine is inclusion of redwater.”

After being ingested, the bacteria localize in the liver and remain dormant; they only begin rapid proliferation if the liver is damaged. Damage may be due to flukes, abscesses, chemicals, plant toxins, bacterial or viral infections.

“Toxins from multiplying bacteria destroy red blood cells. The breakdown of red cells releases hemoglobin pigment, which ends up in urine — hence the term redwater. The animal is dull, with high fever, bloody diarrhea, dark red foamy urine, blood-stained feces and anemia,” he says.

If treated in early stages, large doses of penicillin given every 12 hours may help, along with immediate treatment with antitoxin. Intravenous (IV) fluids may also help.

Prevention is by vaccination, though it may need to be repeated every four to six months if you live in an area with freshwater snails. Control of liver flukes can also help. This is one clostridial disease you probably won’t see in young calves, because they have not yet had time to suffer liver damage from flukes.

Tetanus

Although cattle are less susceptible to tetanus (caused by C. tetani) than most mammals, it sometimes occurs when bacteria enter via a deep wound or surgical incisions without appropriate disinfectant, or via the moist umbilical stump of a newborn calf or the raw area above the scrotum when bull calves are banded.

“These bacteria live in the intestine of many animals and are present in soil containing manure. After entering the body, the bacteria remain in the area where introduced and stay dormant until conditions are ideal. They multiply best in an airless environment such as deep puncture wounds with damaged tissue and poor blood supply (not much oxygen) at the site,” says Hendrick.

Powerful toxins are released when these bacteria multiply, attacking nerve tissue and eventually affecting the brain and spinal cord. The animal develops muscle spasms and incoordination. About 60% of affected cattle die. Treatment includes tranquilizers and penicillin to halt bacterial multiplication and toxin production. Supportive treatment (force-feeding or IV fluids) must be given until the animal can eat and drink again, and this may take several weeks.

Most cattle are not vaccinated against tetanus, but in some regions veterinarians may recommend vaccination.

Enterotoxemia

Sometimes a calf may develop a bacterial infection in the gut that proliferates rapidly and produces gas and toxins. If this condition is not treated quickly and reversed, toxins may get into the bloodstream and the calf may go into shock and die within a few hours. The calf may be fine in the evening and dead the next morning. In some instances, the calf may be dull and bloated (usually with the abomasum distended with gas, rather than the rumen), and might not die as quickly.

This disease has been called overeating disease, purple gut, toxic gut and other names.

“The general term for this infection is enterotoxemia, which literally means toxemia from bacteria found in the intestine. This condition is fatal unless the animal is treated immediately,” says Hendrick.

The most common cause of enterotoxemia in calves is C. perfringens, one of the clostridia normally found in the GI tract of livestock and passed in feces. These bacteria rarely cause gut infections in adult animals since adults tend to have immunity, but they can cause fatal disease in calves when conditions within the gut enable them to proliferate rapidly.

There are five types of C. perfringens, which can affect calves of different ages and in different ways. The five types (A, B, C, D and E) are classified by the type of toxin they produce.

Type C causes acute and fatal intestinal infection in young calves unless they’ve been vaccinated. Enterotoxemia in calves is usually due to types C or D, with signs ranging from acute abdominal pain and diarrhea to convulsions, blindness, coma and swift death. Type C needs an abundant supply of carbohydrates (such as milk) before it can rapidly multiply and produce toxins.

“There is usually a slowdown or stoppage of intestinal movement, allowing toxins to accumulate and be absorbed by the gut,” says Hendrick. This slowdown can happen if a calf ingests a large amount of milk, such as loading up on milk after a period of not eating. This is why old-timers called it “overeating disease.” The large volume of milk takes more time to digest, creating the slowdown in gut contents that allows bacteria to proliferate.

Symptoms include severe gut pain that comes on suddenly. The calf kicks at its belly or throws itself on the ground. The affected calf is dull and may be slightly bloated with gas distention in intestines and abomasum generally rather than the rumen.

If not treated with antitoxin immediately, the calf will go into shock with convulsions and paddling of the feet and will soon die, often within 12 hours of the onset of symptoms. If the calf lives more than a few hours, it may develop diarrhea with blood in the feces. Postmortems show a purple-red section of small intestine and blood-tinged thick fluid in the gut.

Type D generally affects older calves, and the toxins may escape from the gut into the bloodstream without causing significant gut damage. These calves don’t show the massive bloody intestinal lesions.

Consider reasons for acute gut infection

“The calves that develop [acute gut infection] at about 2 months of age are often individuals that got separated from their mothers for an extended period for some reason, and then load up on milk when they get back together. The gut bulges with all the milk they’ve taken on,” says veterinarian J. Glenn Songer of Iowa State University. This creates a perfect environment for proliferation of toxin-forming bacteria. The stomach is full of milk, providing a rich source of nutrients for the bacteria, and the gut slows down because it’s so full.

Any C. perfringens that’s already present takes off like crazy in that nutrient-rich environment,” he explains. “It’s a wonder we don’t see this disease more often.”

Many things could precipitate this problem, such as calves being separated from their mothers at branding time, or when cattle are huddled together for shelter during a storm and the calves don’t nurse until the storm is over, or when the cow is bulling and being chased around for most of the day by the bull with her calf never having a chance to nurse until she goes out of heat. The calf might be off feed because of scours and then loads up on milk when he starts to feel better.

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With Type C, earliest problems may start in calves as young as a few days of age, but some calves may be 2 to 3 months old.

“They are generally healthy, fast-growing calves that suddenly develop acute gut pain — kicking their belly (and sometimes running frantically, trying to get away from the pain), throwing themselves to the ground and thrashing, like a colicky horse,” says Hendrick. They may stagger, legs buckling, and when you get them up, they stagger and collapse again, and lie there kicking.

On the other hand, a calf may suddenly become dull and bloated, not wanting to move. In either case, these calves were happy and healthy suckling Mama until the acute bacterial infection started creating toxins. When proliferating bacteria and their toxins start to damage the gut and it shuts down, this may cause a sudden buildup of gas in a certain area, and hence the acute pain. If the calf is not treated immediately, toxins go through the damaged gut wall into the bloodstream to create toxemia. Toxins throughout the body attack internal organs. The calf goes into shock and soon dies, unless this condition can be reversed.

In a typical scenario, the infection comes on so fast that the gut shuts down before the calf scours.

“Vaccinating cows ahead of calving with C. perfringens Type C and D toxoid (to produce antibodies which the calf obtains via colostrum) or vaccinating calves at birth or soon after helps reduce or eliminate incidence of this deadly kind of enterotoxemia,” says Hendrick.

Yet even with vaccination, some herds continue to have occasional problems. Veterinarians have speculated it must be a toxin-forming bacterial infection, but not C. perfringens Type C or D. Now we realize this problem can be caused by Type A or Type E, which are not included in eight-way clostridial vaccines.

Sometimes other bacteria are involved, such as C. difficile. The challenging thing with toxic gut infections in calves, regardless of the pathogen, is that it can kill within a matter of hours. Some producers are able to save these calves if they can find and treat them quickly with an oral antibiotic and castor oil — before the calf goes into shock — regardless of the exact cause of the infection.

Diagnosing a gut infection

The only way to determine which bacteria are affecting calves in a certain herd is to take samples for laboratory analysis. J. Glenn Songer of the Department of Veterinary Microbiology and Preventative Medicine at the College of Veterinary Medicine at Iowa State University says the most common cause, traditionally, was C. perfringens Type C.

“Type D, which causes infections we termed overeating disease in sheep (same as ‘pulpy kidney’), can also occur in calves. Today, with vaccination of cattle against types C and D, we see relatively few Type C and D infections in calves,” Songer says. “Yet, a surprising number of producers fail to vaccinate, so some infections, particularly Type C, do occur.” He notes that among isolates today, more than 90% are Type A, with the next most common being Type C at 4%-5%, and then Type D.

In calves with hemorrhagic enteritis and sudden death, Type E can also be a problem.

“This illustrates the need for diagnostic work if a person wants to find out how to prevent these infections,” Songer says.

“We must go farther than just postmortem examination. We need to do a bacteriologic culture, and once we get the ‘bugs’ cultured, we need to genotype them to know exactly what we are dealing with.”

Providing tips for what samples to submit for diagnostics, Songer says the ideal is a live affected animal that has not yet been treated.

“With clostridial enteric disease, calves are usually affected so quickly that they are often found comatose or near death before the rancher has a chance to treat them,” he says. “In calves with perfringens-associated enteritis that suffer hemorrhagic abomasitis and sudden death, we find about 10% of those deaths are from Type E infections.”

There are vaccines that include protection against Type E.

“Some producers use autogenous products created from cultures taken from their own cattle, but there is no data on how effective they are,” he explains. “There is a commercial vaccine, however, some of the electrolytes you might give to a beef calf have a lot more sugar or dextrose.” They slow down the rate of abomasal emptying, causing a back-up of GI tract contents.

“Anything that slows abomasal emptying may be a risk factor. The bacteria are probably already present in the stomach, so if they have more time to proliferate, you could end up with bloat,” says Smith. Risk factors include products with high sugar levels, sudden changes in the type of milk you are feeding a calf, etc.

Beef producers often call this condition sloppy gut, rather than bloat. When the calf moves around, you hear fluid sloshing.

“This may occur after a siege of bad weather when cattle were standing bunched up under a tree for shelter and the calves didn’t nurse for awhile. Then they are really hungry and load up on milk. They get a big full belly that empties more slowly, which gives bacteria more time to grow,” he says.

“As soon as the bacteria proliferate, the calf goes off feed and might kick at his belly. It can be very painful. In one study the researchers were able to reproduce the disease, but to do this they had to give calves milk replacer with cornstarch and extra glucose mixed with water — a very rich energy product. It probably provides nutrients for bacteria to grow, and then slows turnover of milk in the stomach,” he explains.

Vaccination can sometimes reduce incidence of disease if the problem is caused by C. perfringens Type C or D.

“But vaccine alone won’t halt a problem if the farm

Bloat or sloppy gut

There are several bacteria that may cause bloat in young calves. Geoff Smith, College of Veterinary Medicine at North Carolina State University, often sees abomasal bloat in dairy calves. “It also occurs in beef calves and may have a similar cause. It generally affects calves 1 to 3 weeks of age. We don’t know exactly what causes it.”

It is possibly due to excess fermentation of high-energy contents in the gastrointestinal (GI) tract, allowing gas-producing bacteria to proliferate. Clostridium perfringens, Sarcina ventriculi or Lactobacillus species may play a role. According to Smith, large amounts of fermentable carbohydrates present in the abomasum from milk, milk replacer or high-energy oral electrolyte solutions, along with fermentable enzymes produced by the bacteria, could lead to gas production and bloat. This process could be exacerbated by anything that slows movement of food through the tract.

These calves may or may not have diarrhea.

“Many have a big belly — both sides — rather than typical rumen bloat seen in older animals. The whole abdomen looks full, not just the left side,” says Smith. At necropsy most of these calves have distended abomasum, forestomach and abomasal edema, hemorrhage, and mucosal necrosis.

“We often find clostridial bacteria involved, but if you just give a calf these bacteria you can’t produce the disease. There have to be other factors involved as well,” he says, noting that one cause in dairy calves seems to be very high osmolality milk replacers or electrolyte solutions.

“Normally, milk is isotonic,” he explains, “but some milk replacers or
against Type A. Some people vaccinate calves within a couple of days of birth with this one. Vaccinating the dam ahead of calving may be more effective. Then you get toxin-neutralizing antibodies in the colostrum and that’s what you need to protect the calves.”

If cases are occurring in calves older than a month or two, the calves themselves should be vaccinated with a Type C product and a Type A product. Calves vaccinated at branding time, for instance, would be protected if given both products.

“I would be especially concerned about Type A if calves are still having problems at that age. You should never let one of these cases go by without submitting a sample — preferably the whole calf — to find out for sure which type you are dealing with,” says Songer.

If a rancher is diligently monitoring calves and finding cases soon enough to treat and save them, there wouldn’t be a dead calf to submit for diagnosis and bacterial typing, but Songer says a person could try sending a stool sample.

“This is better than nothing, but not much better. The colon is the primary habitat of perfringens in normal animals. The disease itself is associated with the abomasum and small intestine. You could have bacteria in the small intestine, and the fecal matter being generated there passes on through and gets mixed up with what’s normal in the colon, especially in older animals,” he says. This might confuse the diagnosis.

“The most likely bug, by far, that you’d isolate from the colon of a normal calf is Type A, since it’s there anyway. That doesn’t mean the Type A strains that you get if you culture a normal calf are the same ones that would produce disease. The ones that cause disease could be quite different. You may not be able to produce disease at all from the bacteria normally found in the gut,” he explains.

“This makes diagnosis more complicated; you may still not know what you are battling, but it’s probably worth a culture. If you find a Type A, this probably doesn’t tell you much, but if you get a Type C or an E, this might be an indication of what’s going on, farther up the pipe. If you don’t have a dead calf to send in, or one that can be euthanized, it may be difficult to tell which organism is causing the problem,” he says.

“If a young calf — 1 or 2 weeks old — dies or is moribund and you can get a good postmortem at a diagnostic lab, you could be able to find out what’s in the small intestine and get a better idea of what’s causing the infection.” If it’s not something included in the eight-way vaccine, you could try another product.

Management is inadequate. It’s like putting lipstick on a pig. It doesn’t address the underlying problem. Outbreaks are often due to some kind of management reason. In a dairy for instance, the neighbor down the road may be using the same milk replacer and he’s not having a problem,” says Smith.

Producers in the beef industry don’t have as much control over the calf’s environment.

To treat a calf with abomasal bloat, Smith says he rolls the calf onto its back, clips and scrubs the appropriate area of the belly, and sticks a long needle covered by a plastic catheter into the abdomen.

“I have someone squeeze on the calf’s belly while I stick the needle in,” he explains. “The catheter is like an IV catheter. I can pull the sharp needle back out as soon as I get the catheter in place, so the needle won’t puncture something it shouldn’t. While the catheter is in place, I have someone squeeze the belly, and I try to get as much gas out of the stomach as possible.”

Positioning the catheter on its back is important, Smith says.

“Some people try to stick the needle in with the animal standing, like we would in an adult cow that’s bloated, but this isn’t the rumen that’s full. If the calf is standing, as soon as you get a little gas out of the abomasum it drops off the needle and everything shifts.”

**Vaccination**

All cattle should be routinely vaccinated for clostridial diseases with an eight-way product that includes Types C and D, Songer recommends. Cows should be vaccinated before calving, and calves should be vaccinated at about a month of age. If some calves continue to have problems, you may need to consider using an additional product.

“Novartis has a good conditionally licensed Type A product,” Songer says, explaining that the license is conditional because researchers haven’t been able to produce the disease in calves to test the vaccine for efficacy. “They can’t get a fully licensed product because they have to be able to show that it works in the host animal.”

He says USDA allows companies to create a vaccine when there is a demonstrated need when it has been shown that the host animal challenge is all that stands in the way of vaccine development.

The Type A vaccine may be helpful in some instances.

“If a herd has a problem, I recommend

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making sure the cows are thoroughly vaccinated with a commercial product that contains Type C and also with this conditional product for Type A. The cost of losing one or two calves to this disease would more than pay for the vaccine,” he says. Type A vaccine is worth a try on ranches that still experience cases of enterotoxemia.

“To protect against clostridial infections (of whatever type) in the gut, you need humoral antibody that meets up with the toxins when the organism begins to proliferate and create inflammation. The sudden increase in toxins upsets the intestine/digestion and the resultant inflammation allows the antibody to come across from the circulatory system and neutralize the toxins, right there in the gut,” explains Songer.

A treatment to try

It sometimes seems impossible to prevent toxic gut infections, and some ranchers have learned to live with this situation by monitoring young calves and immediately treating any that are seen with symptoms of gut pain and/or bloat. If these calves are given an oral antibiotic such as liquid neomycin sulfate solution or some form of penicillin to combat the infection and castor oil to stimulate the shut-down gut to start moving again, affected calves quickly recover.

If a calf is already dull and going into shock, large amounts of intravenous (IV) fluids and treatment to reverse shock (in addition to antibiotic) are necessary. If the calf is already comatose, it’s usually too late. Checking calves often has enabled some ranchers to find and treat them in time.

“I am not a clinician, but I’ve had a lot of experience in the clinic and in the field, and I’ve talked with producers and practitioners who use a little lasalocid, given to the sick calf orally,” says Songer, referring to the ionophore often used for control of coccidiosis and some other types of infection. “You can’t use very much of this in a calf because it would be toxic, but this ionophore really knocks out C. perfringens. An oral penicillin like amoxicillin, or oral neomycin sulfate, would also work.”

If the antibiotic is in liquid form, he adds, it works better and faster in the gut than a pill or bolus.

The aftermath of gut infections

Usually the calves that survive enterotoxemia — with treatment halting infection and toxin production — go back to normal rather quickly, suckling and feeling good, but some show signs of gut discomfort a few days later. They go off feed, looking dull and unthrifty, grinding their teeth and refusing to suckle.

If the calf is not nursing or eating much, unless a person milks out the cow and force-feeds the calf, the cow dries up and the calf goes downhill from self-imposed starvation. Some of these calves have fluid buildup in the stomach. You hear a sloshing sound as they move.

The low-grade gut pain is similar to a calf with stomach ulcers. If you force-feed these calves and use medication to soothe the gut lining like mineral oil, kaolin-pectin, or Pepto Bismol, they heal within a few days or a week and start eating regularly again.

“The acute toxic infection can damage the gut lining, which would explain these kinds of signs,” says Songer. “Sometimes the problem can spill over into the rumen, but in most cases we find abomasal ulceration and necrotic lesions in the small intestine. There can be dramatic lesions in the lining,” he says.

Most of these calves never have diarrhea. The initial acute infection shuts down the gut so quickly the calves don’t scour. Afterward they may have some diarrhea but many tend to be constipated, passing small, firm balls of feces and sometimes mucus and what appears to be shed gut lining.

“In baby pigs we see constipation with C. difficile infections, but this may or may not be the pathogen involved with the calves’ infection. Whatever pathogen is causing inflammation may affect motility of the gut,” Songer adds.

If the calf is not nursing or eating much, his stools become firm and scanty. A little extra care, making sure calves get adequate food during their healing process, helps them get past this setback.

Braxy not limited to sheep

This acute infectious gastrointestinal (GI) tract disease in sheep is caused by Clostridium septicum, the same bacterium that commonly causes malignant edema in cattle, sheep and other domestic animals.

“It can also affect calves,” says veterinarian J. Glenn Songer of Iowa State University.

“Braxy has now become a general term for the disease in calves that develop abomasitis, or rumenitis, when they are a little older. The stomach of an affected animal will have lesions and tiny ulcers. In those ulcers we find lots of gram-positive rods. It’s common to find that these bacterial cultures yield isolates of C. septicum,” says Songer.

“There’s a high chance that C. perfringens Type A is the cause of most acute toxic gut infections, but C. septicum may also be a possibility. Vaccination with an eight-way clostridial product would give complete, or at least partial protection against this one, however. It is in the vaccine to prevent malignant edema. It might be that the formulation that prevents malignant edema may not be as good at increasing the antibodies needed to protect against braxy in the gut,” he says.

When a calf is necropsied and samples are sent to the lab, C. septicum is one more thing to look for.

PHOTO BY HEATHER SMITH THOMAS

If the antibiotic is in liquid form, he adds, it works better and faster in the gut than a pill or bolus.

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Editor’s Note: Heather Smith Thomas is a freelance writer and cattlewoman from Salmon, Idaho.