# recognizing and treating
# PREGNANCY TOXEMIA

*by Dr. Ken Brown, DVM*

Pregnancy Toxemia is a serious disease and should be treated by a veterinarian when possible.

Breeding season is over and you have but 4 weeks to go before kidding! You watch your herd carefully and check them daily for any signs of illness of trouble that can be prevented. But underneath a seemingly normal looking pregnant herd can run a silent problem waiting to erupt: pregnancy toxemia and the risk of losing both doe and future babies.

Pregnancy toxemia is most common in does carrying multiple fetuses and usually seen in late pregnancy, the last 4 to 6 weeks. During these last weeks, energy requirements rise dramatically with does pregnant with twins or triplets needing as much as 1.5 to 1.8 times more energy and protein than maintenance requirements. They are, literally, almost eating for three (or four). Each fetus can require 30-40 grams of glucose per day in late gestation to support their growth and is a significant portion of what the doe can produce and is provided to the fetus before the doe. The liver is responsible for meeting energy demands and mobilized fat stores can easily overwhelm the liver's capacity and result in hepatic lipidosis with a subsequent impairment of liver glucose production function.

The risk factors for developing pregnancy toxemia include: multiple fetuses, poor energy quality or amount in diet, environmental factors (heat or cold), obesity and general body condition, parasite burden (robbing nutrients), and the actual genetics of having multiple fetuses.

The primary cause, however, is inadequate nutrition usually due to insufficient energy density of the ration and a decreased rumen capacity as a result of fetal growth. Does that are too thin or obese (BCS $\leq$2.0 or $\geq$4.0) and with more than 1 fetus (common in goats) are at most risk of developing pregnancy toxemia. Some goats are subclinical and have other problems that can curtail proper nutrient absorption (parasites, etc.) and can quickly shift from a mild ketosis to clinical pregnancy toxemia if feed intake is curtailed by an event like stress (adverse weather, transport, handling) or another disease (e.g., pneumonia).

Several signs can indicate a problem with pregnancy toxemia, including small fecal pellets (poor diet intake), "off" or slowing in eating, reluctance to rise, isolation from the herd, dehydration, infrequent urination, teeth grinding, becoming sluggish, stiff or staggering gait, swelling of the lower limbs. The more the disease progresses, the more signs become evident which can be signs of ketoacidosis (needing daily treatment) and more obvious neurological signs related to the lack of glucose to the brain such as tremors, laying down more often, apparent blindness, staggering gait (ataxia), aimless wandering, stargazing, and eventual recumbency. Not all signs are present at every stage of the disease. When the disease is progressed sufficiently, the fetuses will die and release toxins into the bloodstream and the doe will then die from endotoxic shock.

Besides the physical signs listed above, the increased ketone bodies in the blood (often giving a "sweet" odor to the breath) and urine can be checked to help determine if the problem is ketosis (pregnancy toxemia), a thiamine deficiency, or a generalized anemia or hypoglycemia. It is easy to check the urine with ketone test strips purchased at any pharmacy over-the-counter.

Because energy intake is low and the fetuses are demanding more energy (glucose) as they grow, a hypoglycemia develops. The hypoglycemia will mimic many diseases and, as noted above, an early sign can be a simple "off" behavior, slight stagger to the gait or a reluctance to rise. It is during late geastation when the majority of fetal growth occurs (80% of it) and the energy demands of the fetus is very high in glucose thereby reducing glucose available for other parts of the body such as the GI tract (why they go off feed), skeletal muscles (why they stop wanting to rise), and even the brain (seen as neurological signs).

When calorie intake is low, the doe will start to mobilize energy from other sources, the first being fat (adipose tissue) and why obese does are more prone to pregnancy toxemia. The fat is metabolized by the liver, but is limited and the effort to metabolize the quantity of fat needed to meet glucose demands results in ketone bodies being released into the blood. It is at this point ketoacidosis occurs and signs begin to occur such as depression, or a general dullness, recumbency, rigid arching of the neck and back, and eventually death.

If the doe survives the pregnancy toxemia episode, dystocia and lactational ketosis (insufficient energy intake for milk production demand) are potential problems and the prudent herd tender will be monitoring for both of them.

**Treatment (based on a 100 pound doe size)**

Advanced cases of pregnancy toxemia are difficult to treat and are frequently unrewarding. If a goat is already comatose, heroic efforts are rarely useful and treatment should focus on the rest of the herd and preventing further advanced cases.

If not comatose, aggressive therapy should be directed against the ketoacidosis and hypoglycemia. If the fetuses are alive and within 3 days of their due date, a cesarean section may be a viable option. If the fetuses are dead or too premature, inducing early kidding is an option. An antibiotic such as procaine penicillin G at 30,000 IU/kg once a day is appropriate if the fetuses are thought to be dead.

While hypocalcemia and recumbency is often found in cases of pregnancy toxemia, other causes for recumbency should be considered such as hypomagnesemia (also a finding in pregnancy toxemia), polioencephalomalacia (goat polio), pulpy kidney disease, rabies, lead poisoning, chronic copper toxicity, and listeriosis. These can be differentiated based on specific clinical and diagnostic findings. Glucose test strips are available over the counter. However, hypoglycemia is not a consistent finding in pregnancy toxemia cases, with up to 40% of cases having normal glucose levels and up to 20% having hyperglycemia.

Hypoglycemia can be treated by a single injection of 60–100 mL 50% dextrose IV, followed by balanced electrolyte solution with 5% dextrose (see below). Repeated boluses of IV glucose should be avoided because they may result in a refractory insulin response and make the situation worse.

If early discovery of the condition has occurred, treatment can be done by an oral drench of 200–300 mL of propylene glycol as an immediate energy source to prevent body fat from being metabolized. The drawback is the GI tract must be functioning properly to take advantage of the oral drench. If the disease has progressed and is in later stages (neurological signs), the next option is IV fluids (or bolus as stated above). An IV fluid infusion is the best option as it provides instantly available energy without having to be digested. Glucose (5% dextrose or 50–120 mL of 23% calcium borogluconate solution into a liter of 5% dextrose IV) is the treatment of choice. Ongoing treatment will include adjusting the ration to provide for more energy once the condition has stabilized. It may be necessary to continue with oral supplementation of rapidly mobilized energy. Note: there are several "recipes" for recovery and calories enhancers, but many are mainly contain oils (fats) and may not be the best solution when it is immediate calories required (carbohydrates). Karo, syrup and molasses are often used as a rapid calorie source and can be appropriate when glucose and gluconate are not available. Calcium gluconate is OTC at many feed stores.

**Prevention**

Grandmother was right - an ounce of prevention is worth a pound of cure. The goal of prevention should be directed at maintaining a proper body condition score before, and during pregnancy, identify does with twins and triplets (history or ultrasound) and feed them accordingly, maintain overall health during pregnancy (parasite control, hoof trims, etc.) and possibly adding niacin (1 gram per day) in late gestation via feed. Adequate winter feeding to provide sufficient calories for producing warmth as well as maintaining body condition during gestation is a primary factor in preventing the disease. The last 4 to 6 weeks of gestation will require supplementing calorie intake with a source of carbohydrates such as grain. The amount fed as a supplement will vary according to the forage quality, body weight of the doe, number of fetuses and adult body condition score. Protein must also be balanced so that rumen micro flora can utilize the carbohydrates properly. The herd can also be tested for risk of developing pregnancy toxemia by testing serum levels of BHB. Goats off feed should be feed separately from the herd but in eyesight of the herd to avoid stress from isolation. Other contributing factors such as housing, nutrition, etc. should be corrected for the group and overall feeding practices should be assessed (type, amount, frequency, space for feeding, protection from elements).

Feeding less to prevent "large kids" and avoiding dystocia is not an effective strategy for future kid health or to prevent the disease, as genetic factors for large kids or breeding to a large buck, will determine kid size more than actual feeding and the fetus has a priority over the doe for receiving glucose (energy). Caveat: overfeeding can result in large fetsuses, so proper calorie intake (enough, but too much) is important and is a factor to prevent dystocia due to fetal size.

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