Hi, everyone. Thanks so much for joining us today. My name is Katie Krothapalli. I'm the director of veterinary education for Vetcetera. Our speaker today is Dr. Misty Edmondson. She's back with us. She's kind enough to give us her lunch break and talk about common emergencies in small ruminants.

But hope you'd had a chance to check out her urinary disease in small ruminants webinar that we had a couple of weeks ago. This will be a little bit more in-depth information about other things that you're going to find in small ruminant practice. She's a board certified theriogenologist. And she was all things small

ruminant guru for when I was in vet school at Auburn. Now she's an Associate State Vet for the state of Alabama. And I will turn it over to her, thanks.

Thank you so much, Katie. It's great to be with you guys today. So without any further ado, we'll go ahead and get started. It seems to me a lot of times our first introduction to some of our small ruminant patients is on emergencies. And so we're going to get into just a few of the most common emergencies that, at least, I saw in practice.

These will include obstructive urolithiasis, the block goat, anemia due to internal parasitism.

And this seems to be a continuing and growing problem due to resistance issues with our internal parasites, pregnancy toxemia and then finally, dystocia.

So again, our first topic is obstructive urolithiasis.

Obstructive Urolithiasis: Clinical Presentation

vetcetera

- Often present as a "sick goat" – disease of males
- Restlessness, vocalizations, tail switching, colic
- Dribbling urine or anuria
- **Urethral pulsations without urination (male)**
- +/- urethral swelling at site of obstruction
- Crystals/blood on preputial hairs
- Preputial/rectal prolapse
- Signs of pain – stretching out bruxism, ↑ HR & RR; pain may subside if rupture

Figure 1: Dr. Dwight Wolfe, Auburn University

So as far as the clinical presentation, oftentimes are some of the pet owners, they're not really aware of obstructive urolithiasis being a problem in goats. So oftentimes, it's just a sick goat. Something's not right with the goat.

It is a disease of male goats, particularly castrated male goats. Sometimes the owners, the only thing you'll notice is that they're just restless. They're vocalizing more than usual. They don't want to lay down, or they lay down and they get right back up. There's tail switching or even signs of colic. They may notice dribbling urine or maybe that they haven't produced

any urine at all and urea. But a lot of times, they're not observing them that closely, especially if they're out on pasture.

If on a physical exam and clinical presentation, we notice urethral pulsations without urination in the male goat, that is almost pathognomonic for obstructive urolithiasis. If you'll remember that normal urination in ruminants is in a pulsatile fashion. When they urinate, it's a pulsatile strain. And so these black goats, if they're still trying to urinate, they still have that pulsatile urge. And so what we do with these guys is we want to look just underneath the anus in the perineal area. And if you see pulsing in that, that's our urethral pulsation.

Again, if you see that, that is a great indication that this animal is suffering from obstructive urolithiasis in the male. On physical exam, you may also notice some urethral swelling at the side of obstruction. If they have ruptured urethra, there can be significant swelling there. Other factors that could clue you in are blood or crystals, that sand-like grit on their preputial hairs. And if they're straining long enough without any intervention, we can see a preputial prolapse or rectal prolapse. I've also had owners who report that their goat is constipated because they see them straining and they think it's constipated.

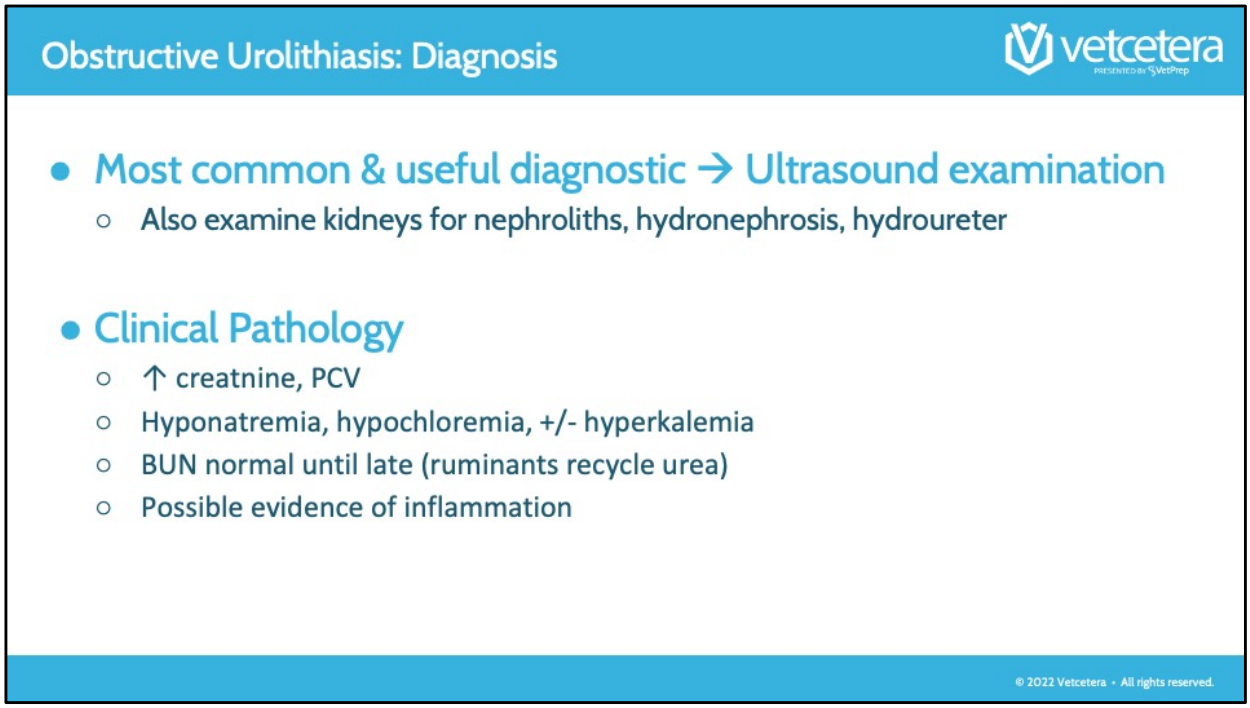
I've never seen a constipated goat, knock on wood. But

that doesn't mean that that's probably not possible. But far and away, a straining goat, you need to rule out obstructive urolithiasis if you see one that comes in for quote, "constipation." Signs of pain in goat, oftentimes, they don't show us very many signs. The picture that you can see here on this slide is a good representation of a painful goat with obstructive urolithiasis. They are stretched out. They don't really want to move around very much because they are painful.

They have a large bladder, and so we can see some bruxism with them, of course, increased heart rate and respiratory rate. And then again, we can see-- if they do rupture the bladder, we can see that the pain subsides. And I think I had told this story previously, but I had a goat owner call me because the goat was constipated. And so I related to her my concerns about him potentially being obstructed. And she was going to bring him in, and we had set a time for her to bring him on in to the clinic. And she never showed up. She never showed up.

So finally, we called her back after a couple of hours to find out, and she said, oh, I'm sorry. He's 100% fine now. He's better. Well, she went ahead and brought him in because we were concerned about a rupture. And sure enough, he had ruptured his bladder. So those are all things that can happen as far as the clinical

presentation on these goats.



As far as diagnosis, the most common and useful diagnostic tool, far and away, is just an ultrasound exam. But we can ultrasound for free fluid in the abdomen. We're going to ultrasound the bladder itself.

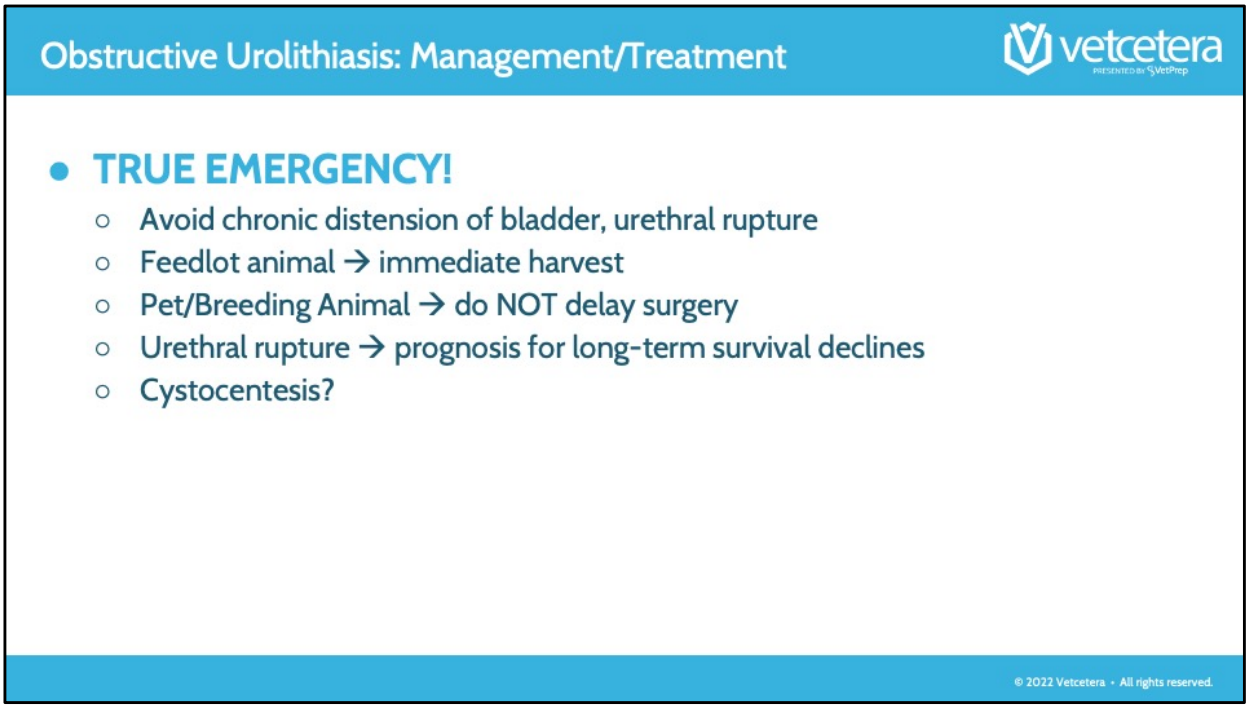
And oftentimes, we'll see a grossly distended bladder, lots of free fluid. It's important to remember, though, when we're looking at these guys, that we also need to examine the kidneys. And maybe not so much as an indication of obstructive urolithiasis because that's going to be in the lower reproductive tract or your genital tract, but it does give us more of a prognosis. So if we see nephroliths within the kidneys, obviously,

that's going to carry a poor prognosis.

If we see hydronephrosis and hydrourolithmia, it's something we need to communicate to the owners that this could be a little bit more in-depth medical management even after surgery. As far as clinical pathology, increased creatinine and increased CVE, the BUN really does it increase very much until late in the course and that's because ruminants are recyclers of urea. We can see hyponatremia, hypochloremia.

And we can see hyper-- or hyperkalemia may or may not be present. Sometimes it just depends on the duration of that. If it's a very recent blockage, a lot of times, we don't see hyperkalemia. If it's been transiently blocked or just a partial obstruction over a prolonged period of time, then we can see that. But it's just something to be mindful of as we potentially take this animal to surgery to be mindful of that potassium.

And then, of course, possible evidence of inflammation, these guys usually have a pretty good urinary tract infection that would be evident. And if they've ruptured, of course, we can see peritonitis and things like that.

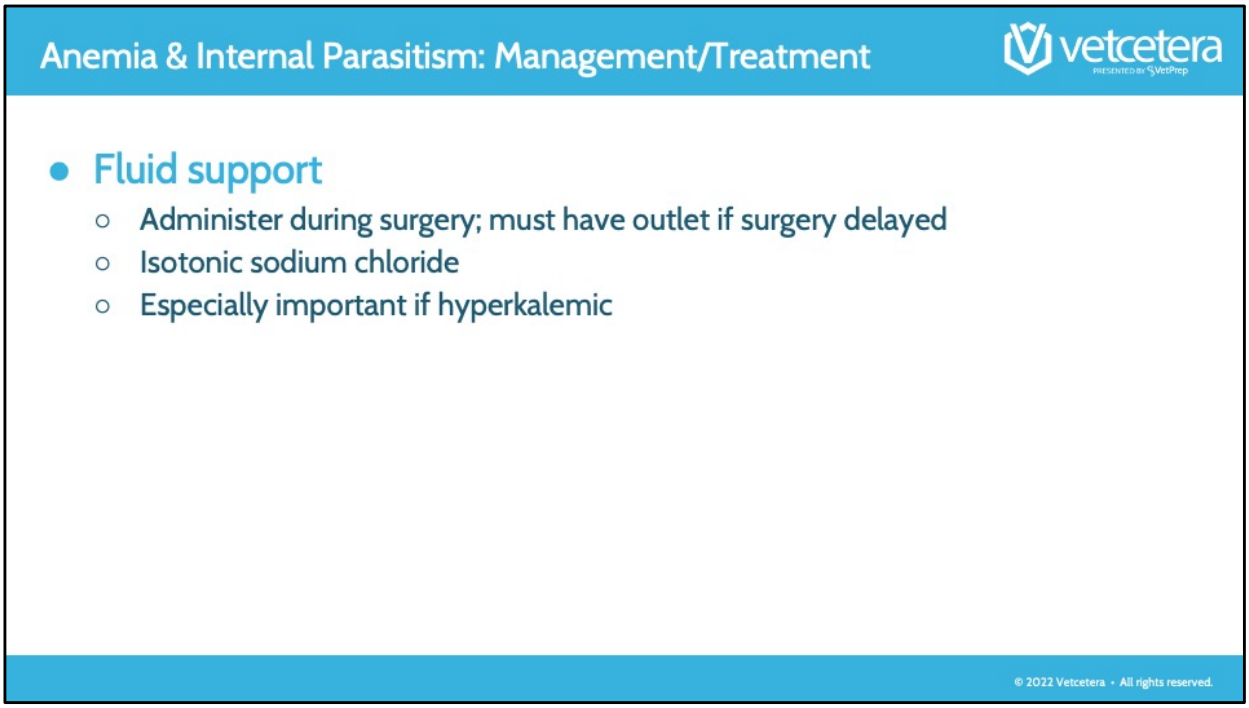


As far as management and treatment with urolithiasis, this is a true emergency. This is not something that can wait till the next day. Owners are going to wait till they get off work.

If they notice this, this is something that needs to be seen as soon as possible. And that's to help avoid chronic dissension of the bladder and potential rupture of the bladder or the urethra. For feedlot animals, one potential would be for immediate harvest of that animal if that's what is-- if it's destined to be harvested, we could just go ahead and do that. Maybe it's a little smaller than ideal, but that could be performed.

If it's a pet or breeding animal, we don't want to delay surgery because again if we have a urethral rupture or a bladder rupture, that does increase the risk for complications by delaying the surgery. And if we can go ahead and do that as soon as possible, it does improve our outcome. And I have cystocentesis listed here with a question mark. If you're fearful that this animal's bladder is going to rupture, it's imminent, and it's going to happen any second now, should we pull fluid off from that bladder?

And so the question mark there is if you think it's going to be-- surgery will be delayed, then maybe cystocentesis might be an option. The risk that we have there is having a leaky bladder and leaking urine into the abdomen. So as long as you know that you're going to get this animal into surgery pronto, it's like, again, emergency surgery, we're going to get him in as soon as possible, then cystocentesis may not be necessary. However, if there is going to be a delay in surgery, then we may need to perform cystocentesis just to prevent rupture of the bladder.

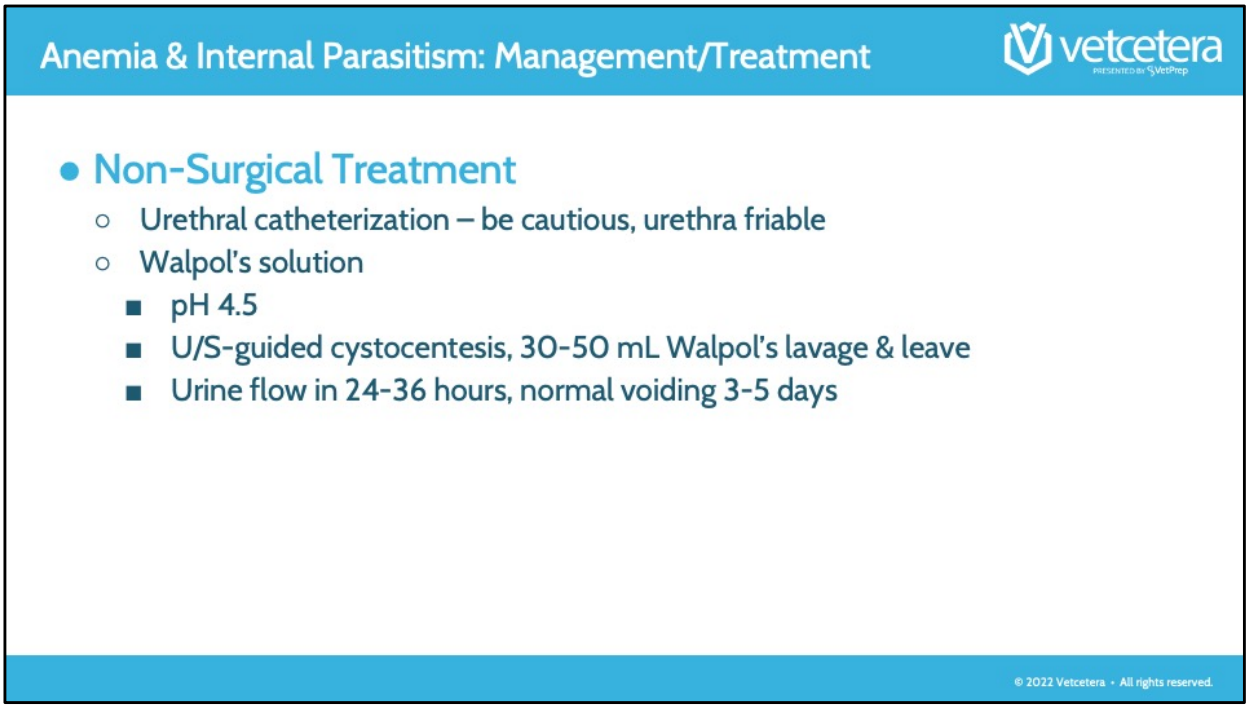


As far as fluid support, we do want to administer fluids during surgery. Again, we must have an outlet if surgery is delayed. So we would not want to start fluids on this animal until we know for sure. Usually, we wait until we start surgery. And then we can go ahead and feel comfortable that we're going to have that outlet here quickly. Fluid support for most of these guys is going to be isotonic sodium chloride, and that's going to be especially important.

Fluid therapy is especially important if they're hyperkalemic. Most of the time, I haven't had a lot of-- and knock on wood, I guess, had a lot of problems with

hyperkalemia in goats. It is described that, I think, I've had one anesthetic death that we did think that it was probably related to the hyperkalemia.

But it's just something to be mindful of but nothing to scare you or stop you from performing that surgery just, again, something that owners need to be aware of that the electrolytes are out of whack with these animals. And there are some inherent risk with doing the surgery and putting an animal under anesthesia.



As far as the treatment itself, non-surgical management, there have been some papers, particularly out of Oklahoma State, that looked at urethral catheterization.

I think K selection if you can catch these early on where it hasn't been partially obstructed or obstructed for a prolonged period of time, those offer a better prognosis. If it has been instructed for a prolonged period of time, I found out that urethra is quite friable, and so there's all there's a pretty good risk of actually rupturing that urethra with the catheter. So you're going to try that to be careful.

Using Walpole's solution, I think Walpole's like a glacial acetic acid solution at pH 4.5. And the theory is that solution placed into the bladder can help dissolve the stones, particularly if they're struvite stones. This is performed by ultrasound-guided cystocentesis where you would remove urine from the bladder, place 30 to 50 mils of Walpole solution into the bladder and lavage that, and then maybe place another 30 to 50 mils after that's been washed out of the bladder, the first 30 to 50 mils has been lavaged out, place a second 30 to 50 mils of Walpole solution into the bladder and leave it there.

The cases that I've seen this work best on or animals that have been partially obstructed, I would say that, at best, probably a 30% to 40% success rate of establishing urine flow within 24 to 36 hours and potentially normal voiding of urine within three to five days. This is probably not my first go-to in pet cases. If there were some financial constraints and the animal's partially obstructed, and we can monitor that very closely in hospital, then I think Walpole's is a good option.

But if it's a pet animal, and if it's end uric, obviously, that's probably not a good use of this Walpole solution.

Anemia & Internal Parasitism: Management/Treatment

vetcetera

- Surgical Treatment
  - Amputation of urethral process – high recurrence rate
  - Salvage (Feedlot Animal) – temporary due to stricture formation (weeks to months)
    - Urethrostomy at site of obstruction
    - Penile amputation
  - Breeding or Pet Animal
    - Tube cystotomy
    - Bladder marsupialization

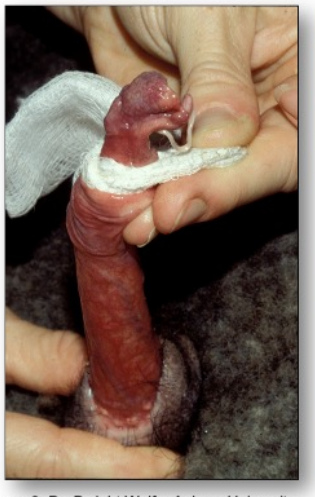


Figure 2: Dr. Dwight Wolfe, Auburn University

As far as surgical treatment, the first thing I always try is amputation of the urethral process. I do this with the animal under just light sedation. Unlike diazepam for sedation, when I was by myself on emergency, that allowed me to-- I could set the animal up on their haunches just like you would normally sitting up a sheep.

And that helped exteriorize the penis very easily in those animals that you would be able to exteriorize so that you can visualize that vermiform appendage. In animals that have been castrated prior to puberty, it's virtually impossible to extend the penis. And you can

do a lot of damage to the prepuce and potentially penis by trying to extend an animal where that perputial attachment is still in place.

So not every animal we will be able to amputate the urethral process just because of lack of exposure. But in animals that have delayed castration until after puberty or intact males, we can try to amputate that your urethral process. The method that I like to use, you don't want to use a pair of scissors that could cause a crushing injury to that urethral process and effectively narrow the opening of the urethral process or the diameter.

Cut that at a large angle, a very large angle, not a very acute angle. We want it to be probably at least 130 to 140 degree angle with-- I always use a scalpel blade and maybe a microscope slide to make a good clean cut on that urethral process, and you prayed for urine to leak out after that. The problem with this is that there is a higher recurrence rate. So we just have to be mindful of that that it may not be curative. It may be a temporary fix.

As far as feline animals, our salvage procedures, this is going to be temporary procedures because these two procedures I'm going to mention will eventually form a structure in weeks to months. So urethrostomy, actually performing the incision over the side of the

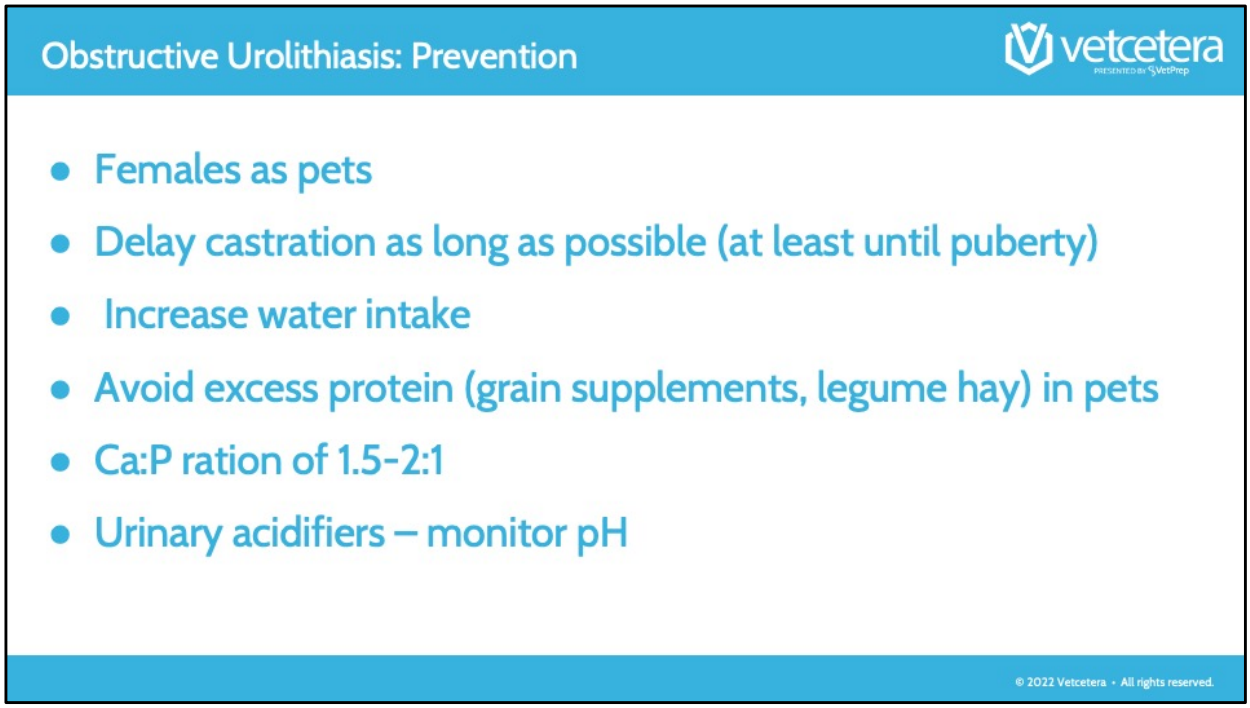
obstruction to remove if it's a single, solitary stone, that's very easy to do. If it's grit-like struvite crystals, then it may be a little bit harder to accomplish that. But that's one option or a penile amputation.

And again, those will stricture down, but that'll give you time to clear the uremia that may be present in these animals and then get them to harvest. Most of the time, the animals that we're seeing in practice are going to be breeding animals or pet animals, the ones that they're willing to treat or have the financial means to treat these animals. And so the surgical options for those are going to be a tube cystotomy, where we actually place a tube into the bladder that exits through the external body wall.

And that allows the urethra time to rest up, stop those urethral spasms as we change the diet and do other things to hopefully get rid of those stones, and then over time, we can close that tube. And they can urinate on their own, and then we can pull the tube. And then bladder marsupialization, this is something that we really have to communicate to owners is that this is a procedure that does require lifelong management of the animal. This is where the bladder is actually marsupialized to the skin of the abdomen of the animal. It's an easy procedure to perform, and it's very quick. Animals feel better almost immediately.

But we do have to treat the area around the marsupialization site with some type of emollient cream, just lanolin or diaper rash ointment to prevent urine scald. That urine scald can be quite severe, and so daily application of Vaseline or lanolin or something along that line to help prevent that urine scald. The tube cystotomy is a more expensive procedure because it requires longer hospitalization. But the bladder marsupialization may be a good secondary option if the owners are willing to take care of it.

If the tube cystotomy fails, then really the only other option would be the bladder marsupialization.



As far as prevention of obstructive urolithiasis, recommending females as pets. This is not a disease of females. It's a disease of males. So if we can have owners that have females as pets, then we can help decrease the frequency of this. Delaying castration as long as possible or at least until puberty-- and a lot of that puberty is very dependent upon the breed.

A lot of the pygmy breeds reach puberty a little bit sooner. So I usually tell people when the little bucklings-- when you can't stand the smell of them anymore is a good time to castrate them. That allows under the influence of the hydrotestosterone that

preputial attachment breaks down. It also increases the urethral diameter, so it can-- just another way we can help prevent obstructive urolithiasis in these male goats. Anything we can do to increase water intake. Oftentimes, when I would see show animals that had been on the show circuit, so to speak, and had been traveling a lot, they would block almost a week to two weeks later.

And so we would try to work with the owners to find ways to increase water intake during those stressful times. Maybe where they're not used to the flavor of the water, this chlorinated water versus the water that they had at home from a well or from a creek. So flavoring waters, if you have show animals, a lot of people will flavor them with Gatorades and Kool-Aid, things like that, to make it more palatable and make them wanted to drink more.

Taking water from home, I've had some clients that would load up containers of well, water from home because their animals didn't like the taste of chlorinated water. So anything we can do to increase water intake, that also includes having salt blocks or loose salts available to help increase water intake; avoiding excess grain, particularly in pet animals. Pet owners of dogs are a lot like pet owners of goats. We tend to see weight issues and over conditioning in

goats just like we do in dogs.

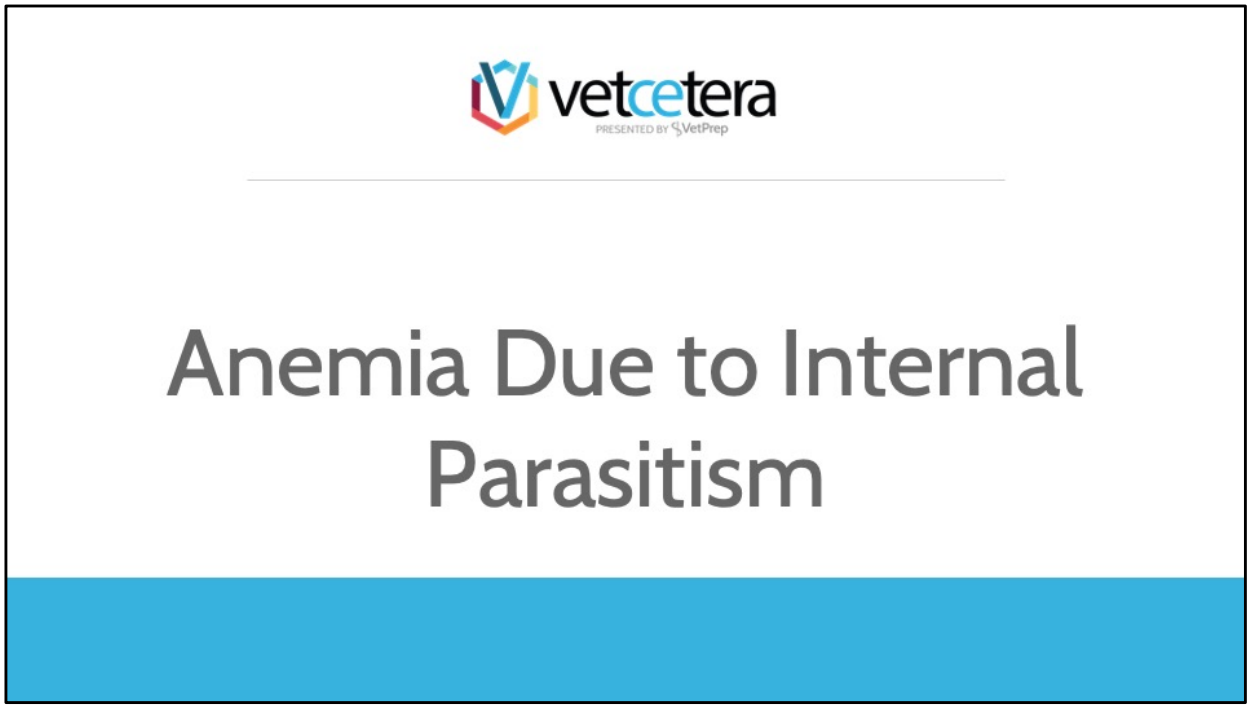
So brain supplements should be very limited in pet goats. Unless they have a job, they don't probably need a lot of extra grain. We can also body condition score sheep and goats to help determine who needs grain and who doesn't. Now if they are pregnant or they are lactating, they will probably need some supplementation along the way. And another part of this is the nutritionist, as far as the calcium to phosphorus ratio, maintain that at 1.5 to 2 to 1 ratio of calcium to phosphorus.

I've had several, particularly show good owners, and I don't mean to pick on show good owners, but that's just the example that comes to mind that they'll buy really, really good, almost expensive, feed that's balanced and it's complete. And then I had, sunflower seeds or beet pulp or what have you. Sunflower seeds, in particular, come to mind. And they just really have a really, really abnormal calcium to phosphorus ratio from messing with that complete ration. So making sure they're not adding extras to the ration, if they do, that's, at least, factored in.

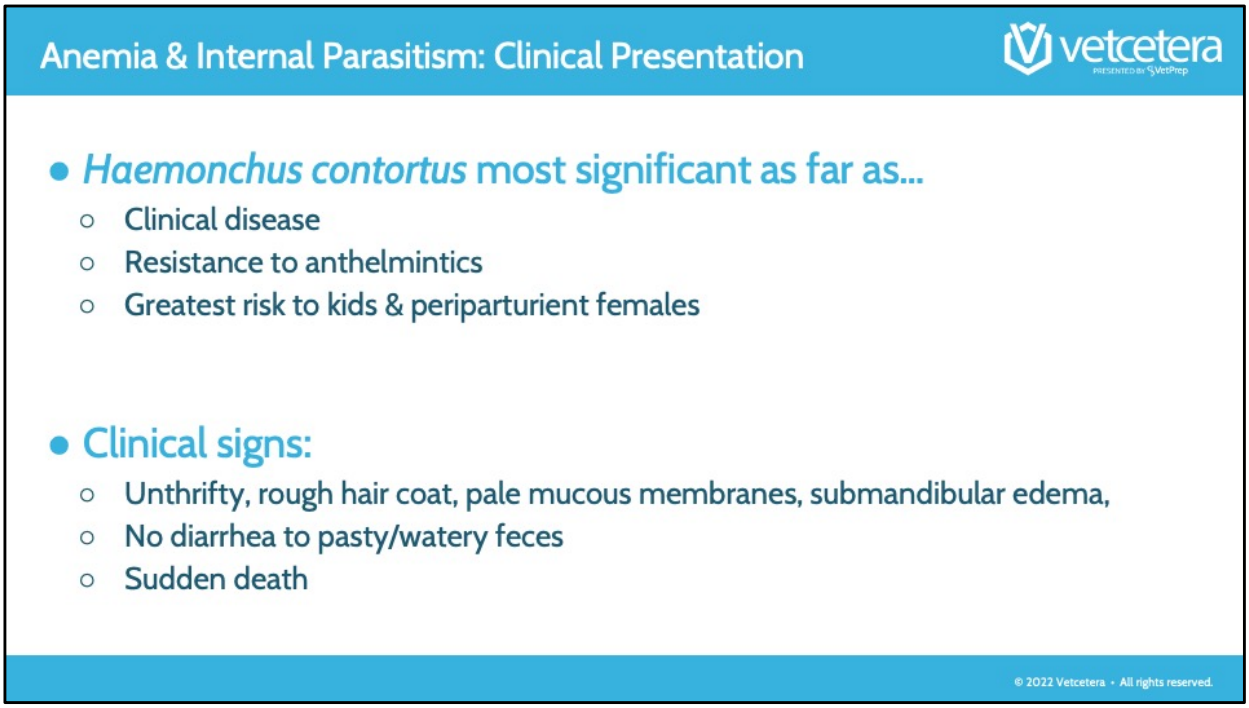
And you can have a nutritionist. All these companies have nutritionists that can do that for you, guys, and for the producers. It's worth making sure it is a balanced ration. And then urinary acidifiers to help monitor the

pH. We'd like to keep the pH around 6 and 1/2 or so, but that's sometimes unrealistic with ruminants. We know that ruminants tend to have a much higher pH closer to 8 to 8 and 1/2. So if we know we have an animal that's predisposed or has had previous problems with obstructive urolithiasis, we can place them on urinary acidifiers, like ammonium chloride.

And typically because there is a suggestion that these animals can develop some resistance to ammonium chloride, potentially putting them on that for two weeks, all for two weeks and back on for two weeks, may help prevent some of that tolerance-- probably not resistance, but more of a tolerance to the urinary acidifiers, like ammonium chloride. So that's all I have for obstructive urolithiasis. That's the quick and dirty.



I'm going to move on now to anemia due to internal parasitism.



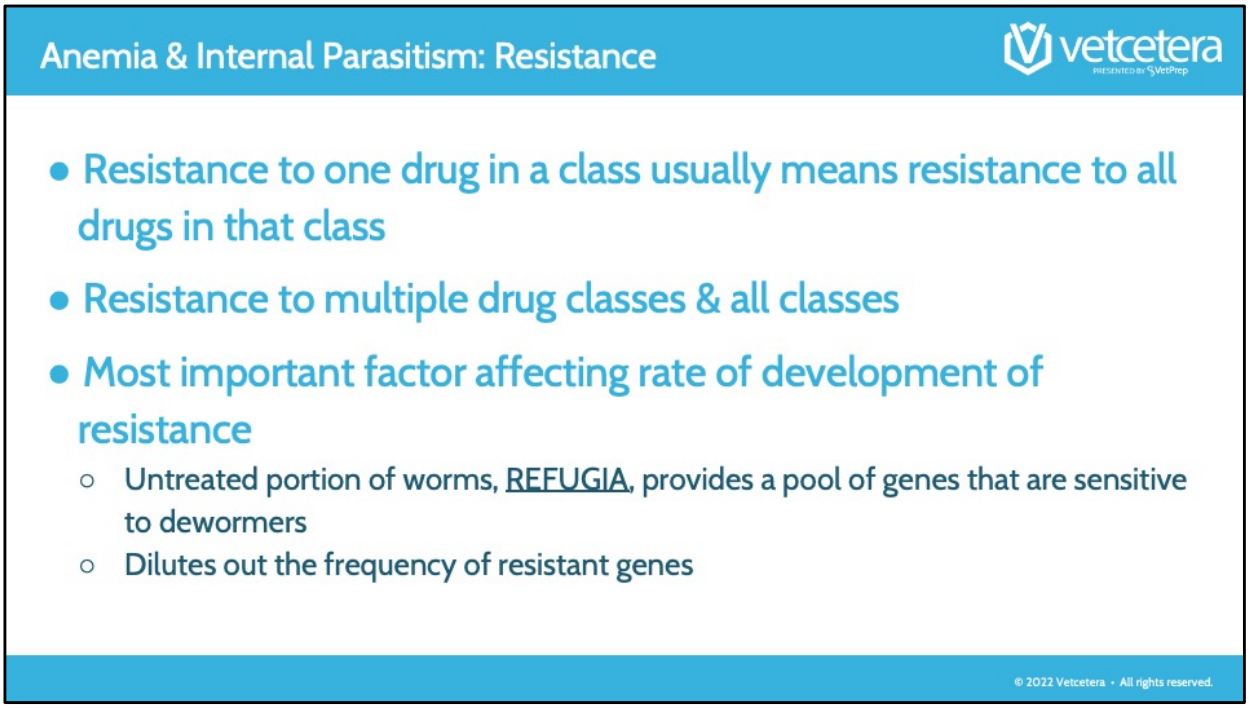
So as far as clinical presentation, haemonchus contortus is the parasite that we are most concerned with. It's most significant because the clinical disease that we see, that is anemia. And then as far as resistance to RX Dominick's or dewormers. Then the greatest risk to kids and impaired parchment for females. And that's probably not a surprise. Those periparturient females under the influence of progesterone, they have a little bit of a weakened immune system because of that and in kids as well. And so they're much more likely to have problems with parasites.

Although we do see plenty of bucks and weathers that have issues as well that we need to pay particular attention to our recently weaned kids and periparturient females. As far as clinical signs of parasitism with haemonchus contortus or the barber pole worm, these animals will look in thrifty. They have a rough haircoat, pale mucous membranes, of course, due to that anemia, and submandibular edema are probably the most common ones that we see. That bottle jaw appearance because of hyperprolinemia.

Oftentimes, when we think about these parasites, we think, well, our owners will think that well, she doesn't have diarrhea. So it's just not going to be parasite. Parasites or not the issue. But we know that absolutely they can have normal looking food scenes or consistency of their feces and still have major parasite issues. So that's not always a dead giveaway to parasitism. And then sudden death is also, unfortunately, one of the clinical signs that we can see. A lot of times these animals will be just rocking right out along, doing great there, have a parasite burden, and having anemia, and then it just gets to-- it reaches a threshold.

Even in chronic cases of parasitism, we can see animals with PCBs of 12 or maybe even as low as 10. They'll be fine until there's some stressor. Maybe a dog runs into

the pasture and they just don't have the reserves to deal with that. And so that's when we can see sudden death just after a stressor that they've been dealing with a chronic anemia and then a sudden stressor.



As far as resistance, we do know that resistance to one drug in a class usually means resistance to all drugs in that specific class.

We have seen resistance to multiple drug classes and to all classes. There has been complete resistance on some forms, particularly in the southeast, which is the ones I'm more familiar with. The most important factor affecting the rate of resistance or the development of resistance is the number of untreated portion of worms. A refugia, you've probably heard that term before, it's basically the naive population of parasites within that herd. So that's a pool of genes that are sensitive to dewormers or antihelmintics. And that's a

good thing. We want to have that refugia because it allows us to use that to dilute out the frequency of resistant genes.

Anemia & Internal Parasitism: Diagnosis

- **Fecal Flotation**
  - Qualitative (1+ to 4+)
  - Okay but not information on resistance
- **Modified McMaster's Fecal Egg Count Reduction Test**
  - Quantitative (EPG)
  - 2 tests performed 10-14 days apart
  - No resistance = 95-99% reduction
- **DrenchRite® Larval Development Assay**
  1. What parasites are in the herd/flock?
  2. Which drugs are effective on the farm?

Figure 3: https://www.wormx.info/drenchriteassay

So ways that we can diagnose internal parasitism is simply with a fecal flotation, a qualitative assessment 1 plus to 4 plus. It's OK to use, but it doesn't really give us a lot of information on resistance. To do that, we need to look to a more quantitative test. And probably the most common one is the modified McMaster's or modified Wisconsin but an egg count reduction test. And that gives us the number of eggs per gram of feces that we have. So we would use the modified McMaster's to perform. So we do one today, and we would do one 10 to 14 days after we dewormed them.

And if we did not have any resistance whatsoever, we

would expect a reduction in the number of eggs per gram of feces of at least 95% up to 99% reduction. If we have a reduction, we calculate our reduction between those two, between the first test and the second test after deworming. If we have anything below 95%, we know we have, at least, a partial resistance to that dewormer. Another one-- and I've really started trying to, I guess, maybe influence people in this direction is with the DrenchRite larval development assay.

It lets us know which parasites are actually in the herd or flock and then which drugs are effective on the farm. So it's performed, like I said, to detect the resistance that's in that farm. We gather sample from multiple animals on the farm. And it actually utilizes the eggs that are isolated from that feces. And so they isolate them, the eggs, and they place them in this 96 well plate that you can see here on the slide. They actually put the drug within those wells in different concentrations.

They incubate the plates for seven days to allow for those eggs to hatch and then subsequently develop into the third stage larva, and then-- because that's the third stage or larva. If you remember, it's the stage in which the animal ingests them on pasture of that blade of grass, and so they serve as the major source of

infection. At the end of that seven-day incubation period, the plate is then examined underneath a microscope, and the parasites are identified by species based on the development of the larva, and then they also look at the stage of development of the larva.

And so using all that information, they can determine the susceptibility of that particular parasite to that drug that was in that well. And if there's any suspected resistance or-- and they look at all the different classes, all three classes of dewormers that are currently available. And so you get a lot of information from that DrenchRite larval development assay. This was originally performed at Dr. Kaplan's lab at the University of Georgia. And if you're going to write anything down about parasites, this website, it's www.wormx.info, has a lot of information. It's the American Consortium for Small Ruminant Parasite Control.

A lot of information there, but since Dr. Kaplan has retired from Georgia, this larval development assay is now offered at LSU, Louisiana State University. And there's some information. You can look that up and get the email for the person that now offers that larval development assay at LSU. So that's a good-- it's a more expensive test. It's a few dollars to perform that. But if you're running the McMaster's, a modified

McMaster's of Wisconsin, it's actually performed on individual animals. So the larval development assay may be-- it definitely gives you much more information, and it may be cheaper in the long run versus doing multiple McMaster's fecal egg counts on multiple animals within a herd.

Anemia & Internal Parasitism: Management/Treatment

vetcetera

- FAMACHA©
  - Tool to ID anemic animals
  - Classifies animal based on level of anemia (1-5); laminated card
  - Animals needing multiple treatments = Cull

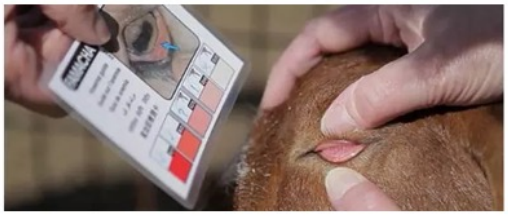


Figure 4: www.wormx.info/famacha

Clinical Category	Eye Lid Color	Packed Cell Volume	Treat?
1	Red	≥ 28	No
2	Red-Pink	23-27	No
3	Pink	18-22	?
4	Pink-White	13-17	Yes
5	White	≤ 12	Yes

As far as management and treatment of internal parasitism, FAMACHA, I'm sure everybody's heard of FAMACHA by now. It stands for Faffa Malan's Chart. Faffa Malan was the one that developed this chart. It is a tool. And you can see in that picture there the chart that we can use. But it's a tool to identify anemic animals. And it classifies these animals based on the level of anemia. So category 1 through 5 is a laminated card that you get from-- we used to get it from the University of Georgia.

And then what that helps us to do, we have to keep up with the animals. So good husbandry is a must from

FAMACHA. We have to be able to identify animals and identify them over time. So I had a herd that-- a herd of, I think there were 13 or 14 goats in a herd, and they all had names, and they all started with the letter L. They had no type of scrappy tag, no ear tag, anything else. So we had to go about and institute some tagging in order to keep track of those animals for their FAMACHA scoring.

So you're going to look at these animals usually every couple of weeks or so and do this FAMACHA scoring. The clinical category, you can see here a category 1. Their eyelid color is red. It would correspond to that bright red on the left of the card. And that correlates to a packed cell volume at greater than or equal to 28. And so they would need treatment. And you can see, as we move down this clinical chart here, we go to a 3. Their eyelid color, they're a little bit more anemic, packed cell volume of 18 to 22.

We have a question where, should we treat them? And as we're going over this FAMACHA over time, we would look back to this same animal and see what their FAMACHA score was two weeks ago or three weeks ago. And if they were a 1, two or three weeks ago when we last looked at them and FAMACHA scored them, and now they're a 3. That question mark may turn into definitely, we need to do that this animal was

progressing. Over the last three weeks, their anemia has gotten more severe. If we wait another two or three weeks, we may be in trouble.

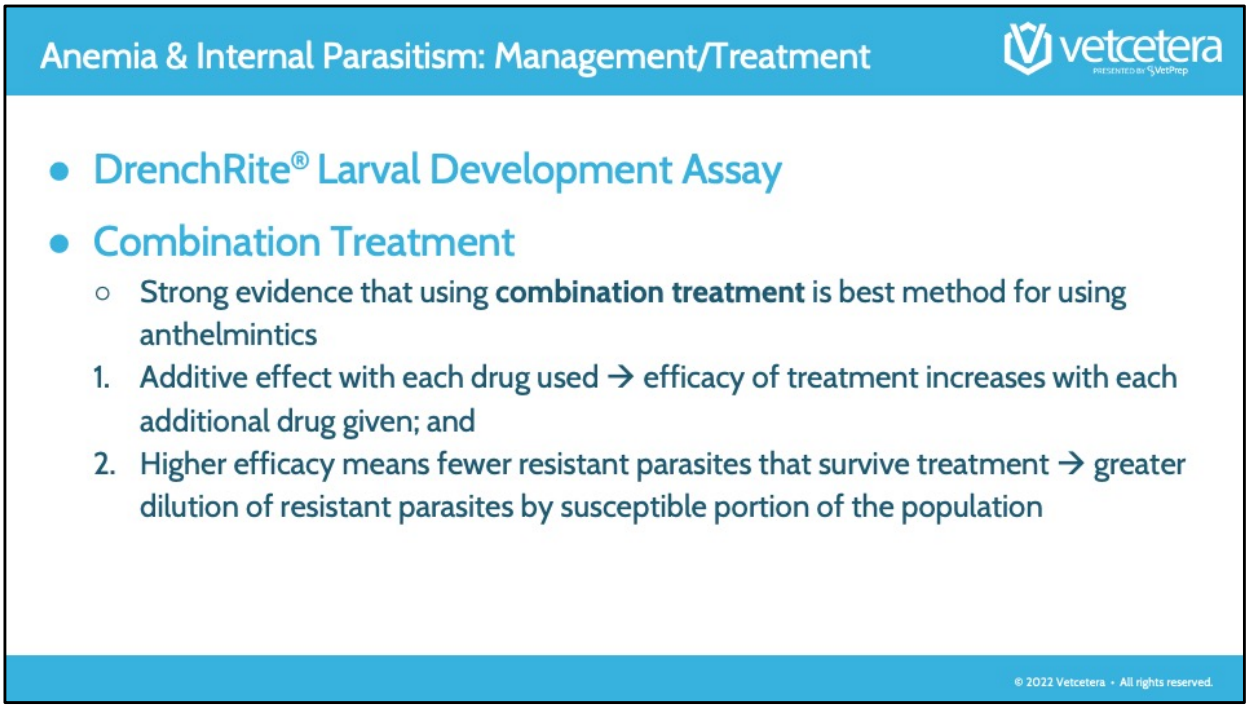
So we probably want to go ahead and deworm that animal. Now if that animal was, let's say, a 4 three weeks ago, and we did dewormed because that's what we would call-- we'd call it to do with the category 4 animal. We dewormed it, and now it's improving. It's a 3. So we wouldn't necessarily want to deworm that animal at this point because it has improved. And then, of course, a clinical category 5, eyelid color is white, packed cell volume, less than or equal to 12, then we would need to treat those.

These are the ones that we often see brought into our clinic for emergency treatment. So we're going to treat those animals that are most at risk, and that's again those categories 5, 4's, and potentially 3's. We don't want to necessarily use the FAMACHA chart on young animals because anemia in kids can progress very rapidly. So we need to keep a really close eye on them, looking for dirty ruminants, animals that just aren't moving around very much that look a little lethargic, those types of things.

Again, keeping a really close eye on our parturing does, again, because they have that decreased immunity to our gastrointestinal nematodes and very high

nutritional demands. So all that sets them up for potentially severe infestations with these parasites. Any animals that are suffering from some other disease, say, a pneumonia or some type of injury, something like that, or if they're in poor body condition, if they score as a 3 on the FAMACHA chart, then you should always treat them.

So FAMACHA can be used to decrease the use of antihelmintics or dewormers on a farm. And we know that means we have a greater refugia on the farm and hopefully preserves the use of our dewormers that do work.



So again, we talked about the larval development assay. There's some really good evidence, particularly out of Australia and New Zealand that combination therapy or combination treatment is going to offer us the best success for dewormers. It's an additive effect with each drug that is used, and it improves the efficacy of that treatment with each additional drug.

And again, if we have greater efficacy, we have fewer resistant parasites. We have an increase in the refugia, and so we have better success at treating our animals. As far as treating an anemia, if they came in and it's a chronic infection and their packed cell volume is 12 to 15, I wouldn't necessarily think that I absolutely have to give them a blood transfusion. If it's 12 or less, the animal's lethargic and down and can't stand, then I

would want to go ahead and transfuse them.

And having some ways at the University, we kept a small goatherd and sheep-herd just for blood transfusions. And we castrated-- we had some weathers around, and those guys did great. They didn't have really any parasite problems. They were able to grow and not have really any stress around because they were weathered. And we used them. They get to be really good size animals, 150 pounds or so. We used some boer class goats. Boer-Kiko crosses, they did great, and they served as our blood donors.

So having a small little herd of goats handy for blood transfusion was really, really nice. And those guys saved a lot of animals' lives.

Anemia & Internal Parasitism: Management/Treatment

vetcetera

Table 1: Impact of using dewormers in combination on the efficacy of treatments.
The increases in efficacy are due to a simple additive effect as per the equation below:
Where D1 = efficacy of dewormer 1, D2 = efficacy of dewormer 2, D3 = efficacy of dewormer 3, C2 = efficacy of D1+D2, and C3 = efficacy of D1+D2+D3
C2% = D1% + (100-D1%)*D2%
C3% = C2% + (100-C2%)*D3%

Drug 1 (%)	Drug 2 (%)	Drug 3 (%)	Combination (%)
80	80		96
80	80	80	99.2
90	90		99
90	90	90	99.9
60	95		98
60	60	95	99.2
99	99		99.99
60	60	60	93.6
50	50	50	87.5
40	40	40	78.4

Table 2: Impact of combinations on percent of resistant worms that survive.
Table shows the % of worms killed by a single dewormer vs a combination treatment with two dewormers both with the same efficacy, ranging from 80% to 99%. The last column shows the magnitude of the difference between % of worms killed and % surviving when one or two dewormers in combination are used. Note that the higher the efficacy of the drugs, the smaller the difference in efficacy when used in combination, but the greater the difference in the % of resistant survivors.

Efficacy of Dewormer		Single Dewormer	2 Dewormers in Combination	Fold Difference
99	% Killed	99	99.99	1.01x
	% Surviving	1	0.01	**100x**
98	% Killed	98	99.96	1.02x
	% Surviving	2	0.04	**50x**
95	% Killed	95	99.75	1.05x
	% Surviving	5	0.25	**20x**
90	% Killed	90	99	1.1x
	% Surviving	10	1	**10x**
80	% Killed	80	96	1.2x
	% Surviving	20	4	**5x**

Figure 5: https://www.wormx.info/combinations; Ray Kaplan, Combination Dewormers: The Time is Now, January 2017.

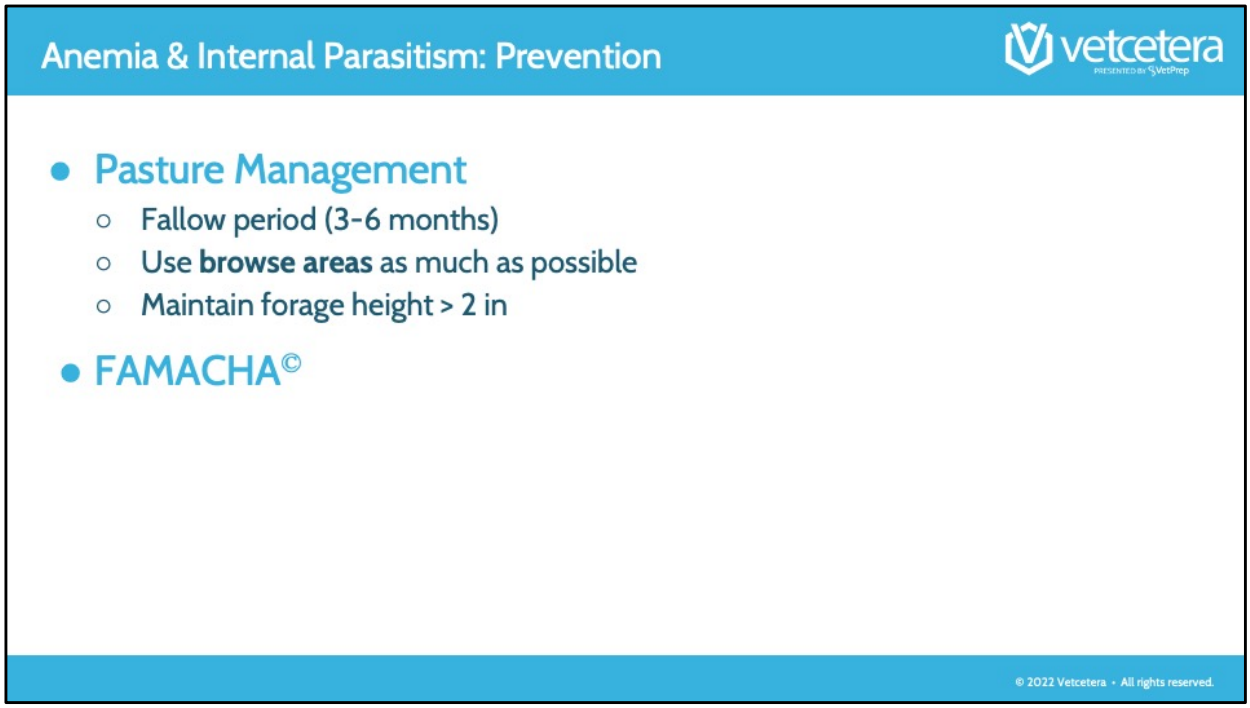
But getting back to our combination treatment, this is a study that was done by Ray Kaplan, where actually they looked at different drugs. And if we had an 80% efficacy of drug 1 and an 80% efficacy of drug 2, by combining those, we can increase that to 96% from 80% efficacy. Now if we had three drugs, we added a third drug. And that 80%, you can see would increase that to 99.2%.

So even if we do have resistance to a particular dewormer, let's say, we did the DrenchRite larval development assay. And we got our information back. And we had, let's say, 60% resistant to all three classes

of dewormers. If we use all three of them, we can see a really good efficacy when used in combination. We need to be very cautious about how we're using our dewormers. We don't want to overuse them. But we still have some options available by using these in combination.

And in Australia and New Zealand, they actually have combination products. We don't have those in the United States. But we can use three separate drugs at the same time to have the same effect, that combination effect. So again, you can say that the efficacy of the dewormer here for a single dewormer. And then as we go down and have a decrease in the efficacy, what we can do with combinations. So combinations right now seems to be the key to helping improve the kill on the parasites that we have in improving the perfusion by using FAMACHA so that not every animal gets dewormed.

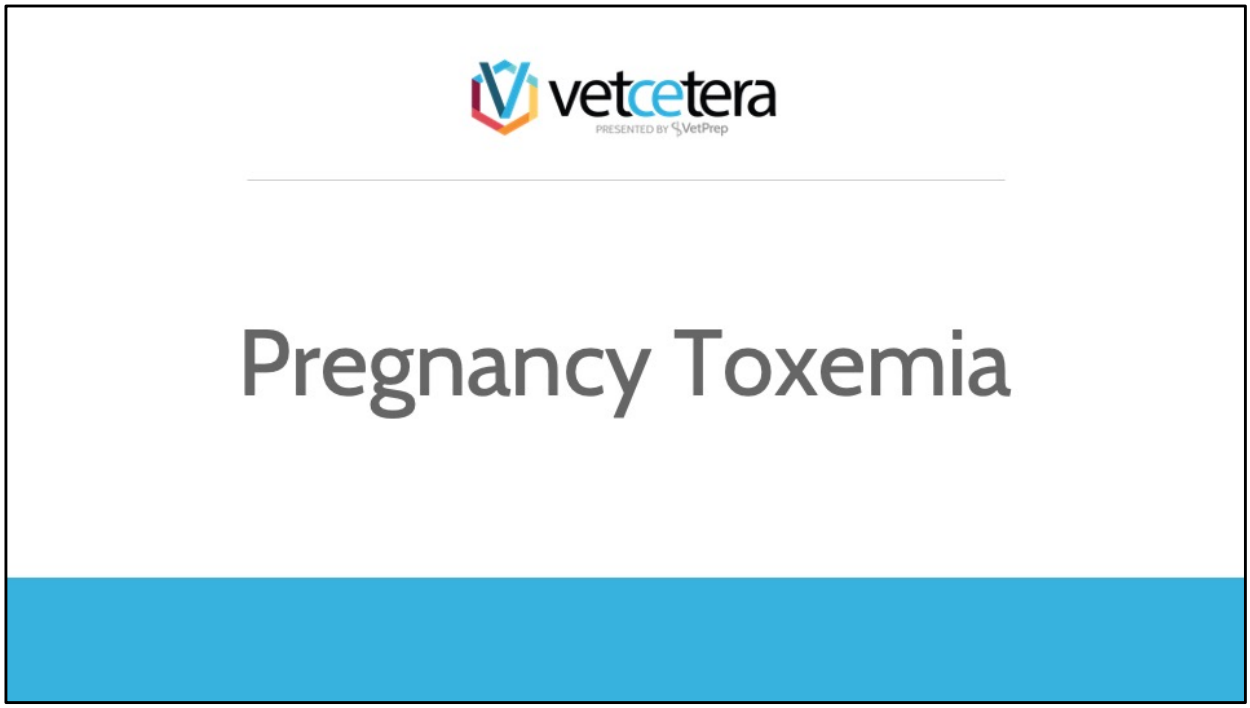
And that's a change. It's very different than when I was coming through vet school. We thought we'd had to deworm every animal multiple times a year, and we'd switch dewormers every time. And we now have learned that was absolutely the wrong thing to have done. So we have to do a good job in managing that better.



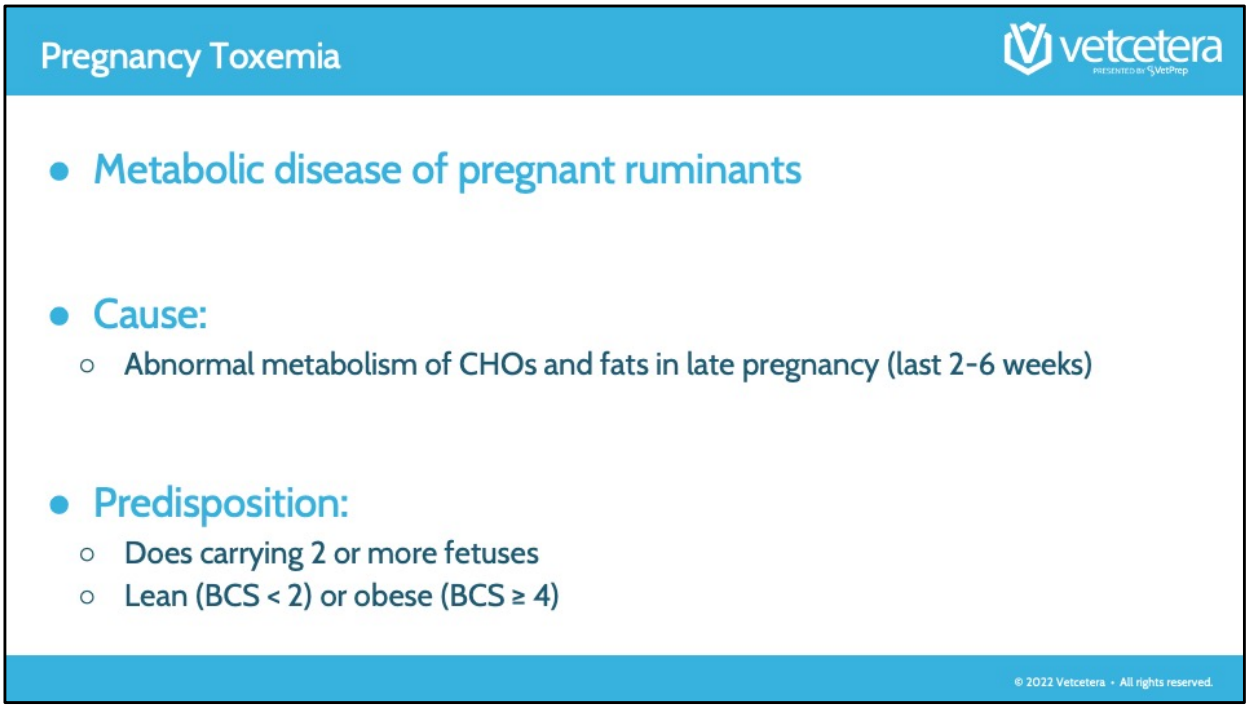
As far as prevention, pasture management, allowing land to lay fallow for three to six months if possible. Oftentimes, that's not possible. Using browse areas as much as possible, maintaining a forge height of greater than 2 inches, and that's because that larva, that third stage larva, can't migrate any higher than that on the forage.

And so if we keep our forge height higher than that, then we decrease the ability of that parasite to cause infection. And then, of course, FAMACHA, we're never going to get rid of the parasites 100%, but hopefully we can. So using FAMACHA, we can select for animals

that are resistant to the parasites. Animals that need continuous or multiple treatments really need to think hard about calling those animals from the herd.



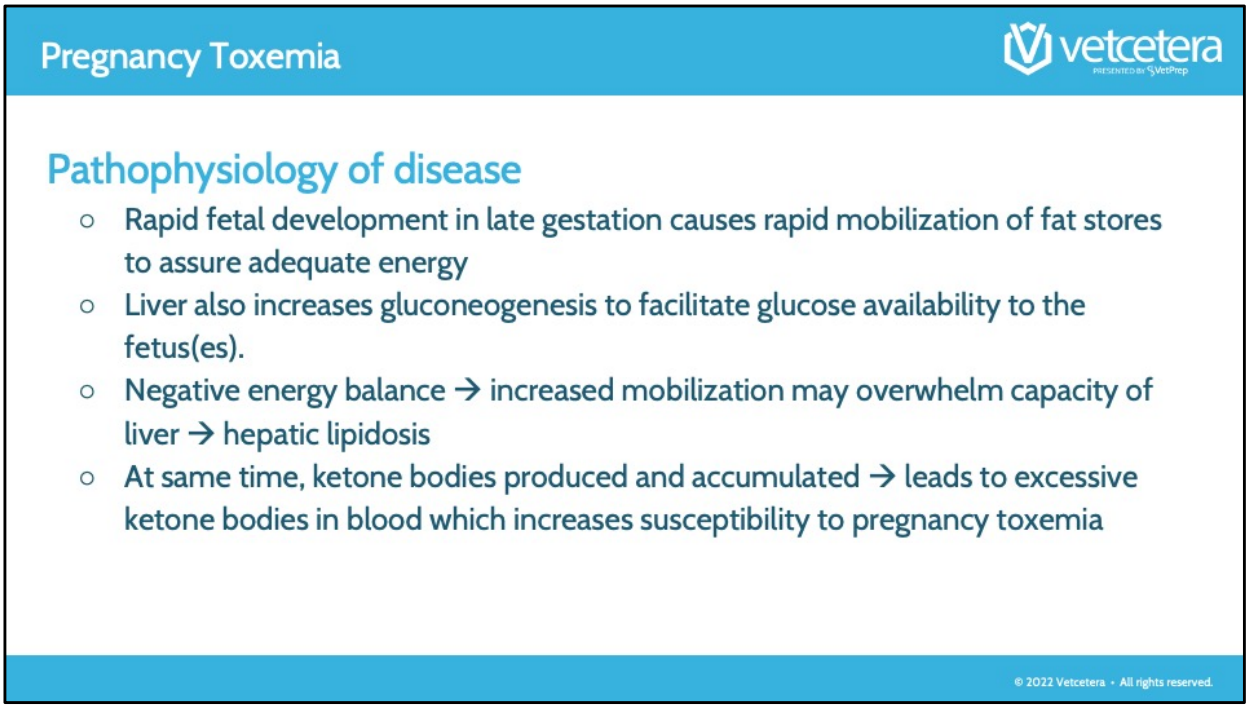
All right, so next topic is pregnancy toxemia.



This is a metabolic disease of pregnant ruminants. We tend to see this a lot in our small ruminant species. And it's just abnormal metabolism of carbohydrates and fats and late pregnancies. It typically lasts two to six weeks in our small ruminants that causes this. We know that those that are carrying two or more fetuses, those energy requirements carry more than the two fetuses. That's a tremendous strain on that animal.

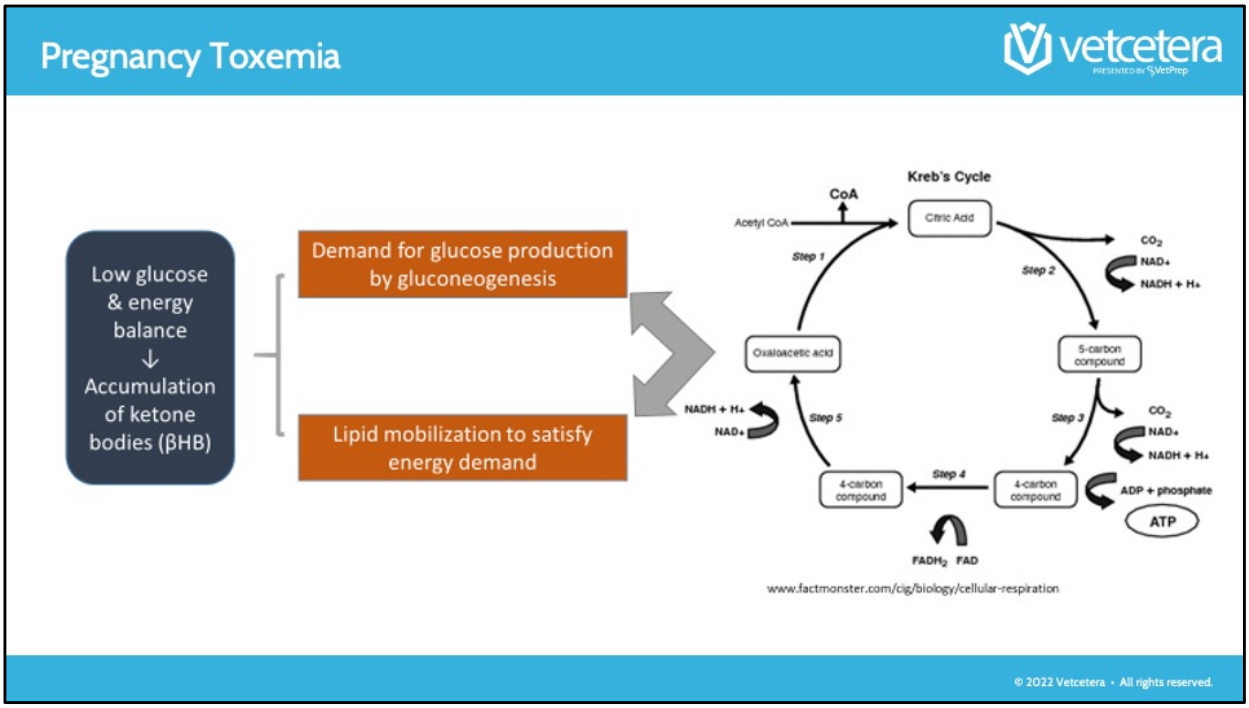
And then animals that are in very lean body condition, body condition less than two or very obese animals, body condition greater than or equal to 4, which tends to be our pet or show animals, those are the animals

that we're most concerned about.



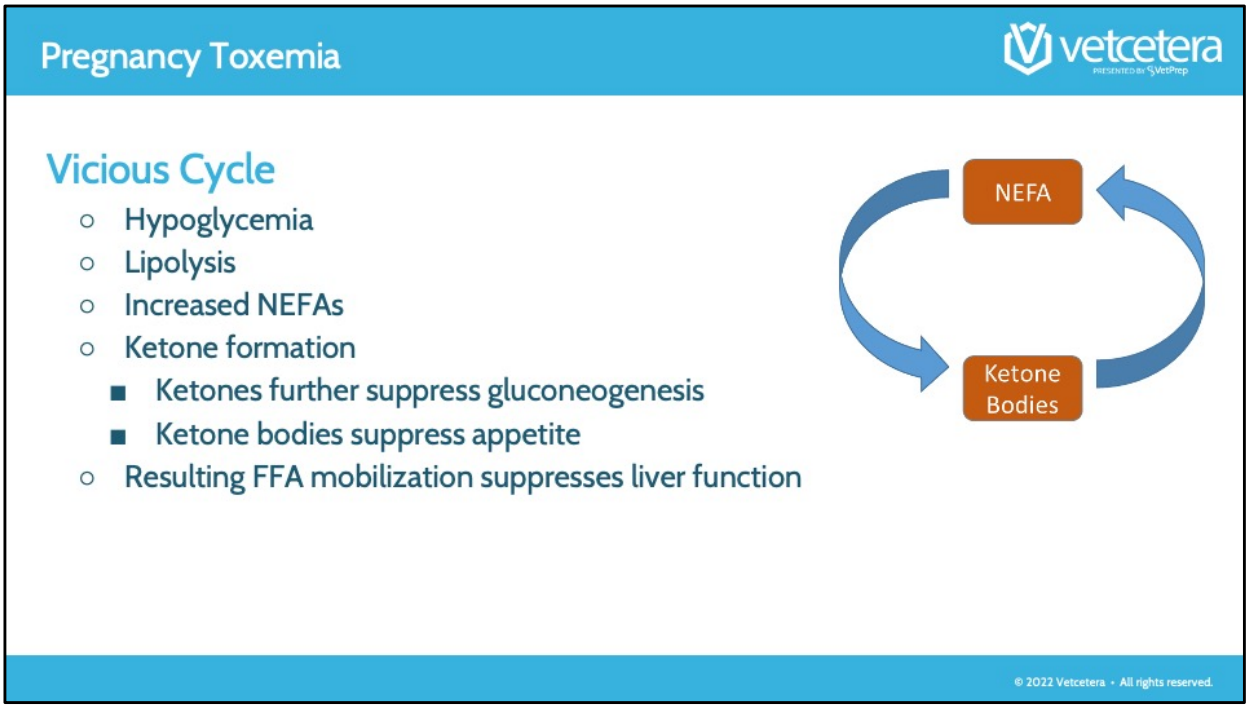
So I won't spend a lot of time on the pathophysiology of disease. We just see rapid fetal development in gestation. It causes the rapid mobilization of our fat stores to make sure there's enough energy there. The liver increases gluconeogenesis to facilitate glucose availability to the fetuses.

That creates a negative energy balance, and so it means increase mobilization that can overwhelm the capacity of the liver and lead to hepatic lipidosis. And then we also have ketone bodies that are being produced at the same time. And those accumulate and that leads to excessive ketone bodies in the blood, which actually increases the susceptibility to pregnancy toxemia.



And this, for everybody to just, I guess, be thankful that we've made it through, and we don't have to memorize the Krebs cycle again.

This is showing that we have low glucose and energy balance and accumulation of these ketone bodies, particularly beta-hydroxybutyrate in the blood.



And it is a vicious cycle. We see that hypoglycemia and then the lipolysis increased non-esterified fatty acids and leads to ketone formation. Those ketones then further suppress gluconeogenesis, which suppress appetite. And it's just again a vicious cycle that just repeats itself. So we have to find ways to break the production of those ketone cycles.

And then those free fatty acids do suppress liver function which adds to the problem.

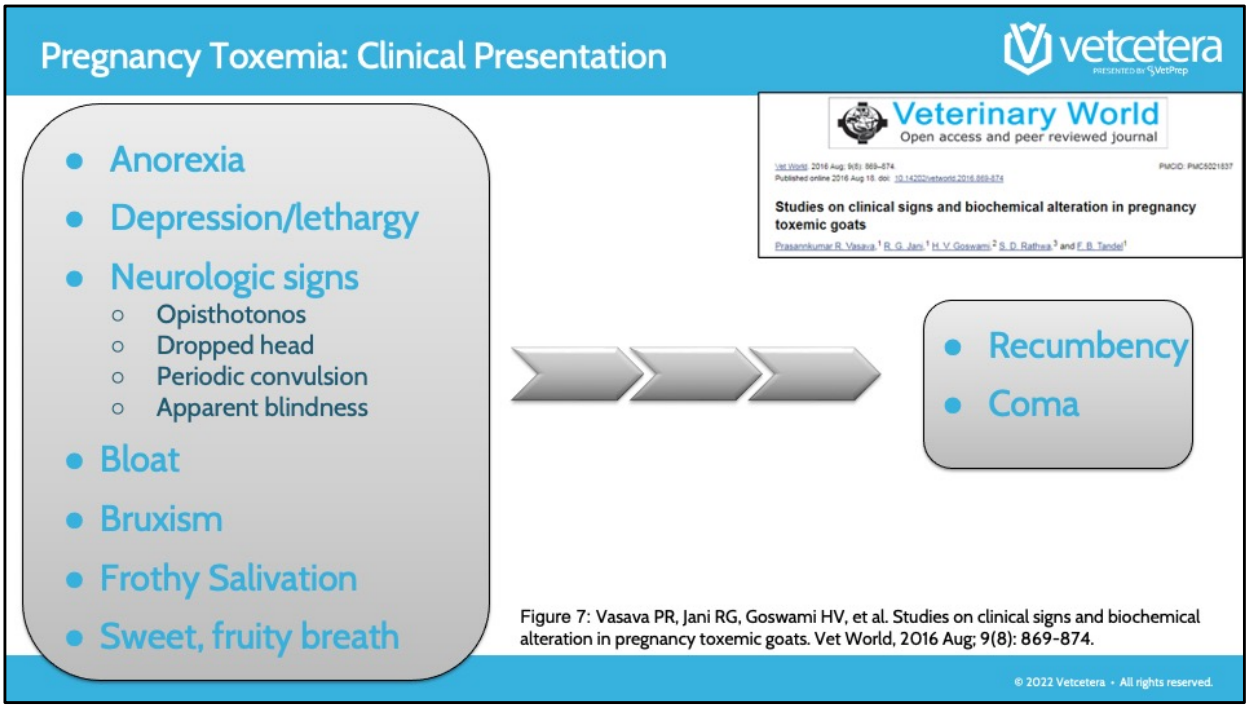
Pregnancy Toxemia: Clinical Presentation

vetcetera

- May be vague initially
  - Weakness, lethargy
  - May separate from herd
  - Mild ataxia

Figure 6: Dr. Thomas Passler, Auburn University

As far as clinical presentation, besides may be pretty vague initially, oftentimes, owners don't notice this because there's just a little bit of weakness. And so maybe they'll write it off to well, it was really hot, or it was really cold. But these animals will be a little bit lethargic. They may separate themselves away from the herd, have some mild ataxia, mild neurologic signs that something that owners need to be taught to look for as well.



Eventually, those things in neurologic signs are on opposite thoughts, drop dead. They can leap. You can see convulsions and blindness as well. But first things to look forward are depression and lethargy--

Excuse me, in the pregnant goat. And then eventually, it can lead to recumbency and coma.

Pregnancy Toxemia: Clinical Presentation

Clinical Signs:	Pathophysiology of CS:
Anorexia →	Ketone bodies
Depression/lethargy →	Low glucose
Neurologic signs →	Cerebral hypoglycemia
Bloat →	Hypocalcemia
Bruxism →	Cerebral hypoglycemia
Frothy salivation →	Frothy salivation
Sweet, fruity odor to breath →	Ketone bodies
Recumbency/Coma →	Low energy, hypocalcemia

Figure 8: Vasava PR, Jani RG, Goswami HV, et al. Studies on clinical signs and biochemical alteration in pregnancy toxemic goats. Vet World. 2016 Aug; 9(8): 869-874.

Most of those clinical signs that we see are related to hypoglycemia and the ketone body formations.

Pregnancy Toxemia: Clinical Presentation

Figure 9: Dr. Thomas Passler, Auburn University

And then again, like we said, the opposite thought is it can progress pretty quickly. So trying to teach owners to recognize this fairly quickly in the pregnant animal, can be very helpful to help prevent any progression.

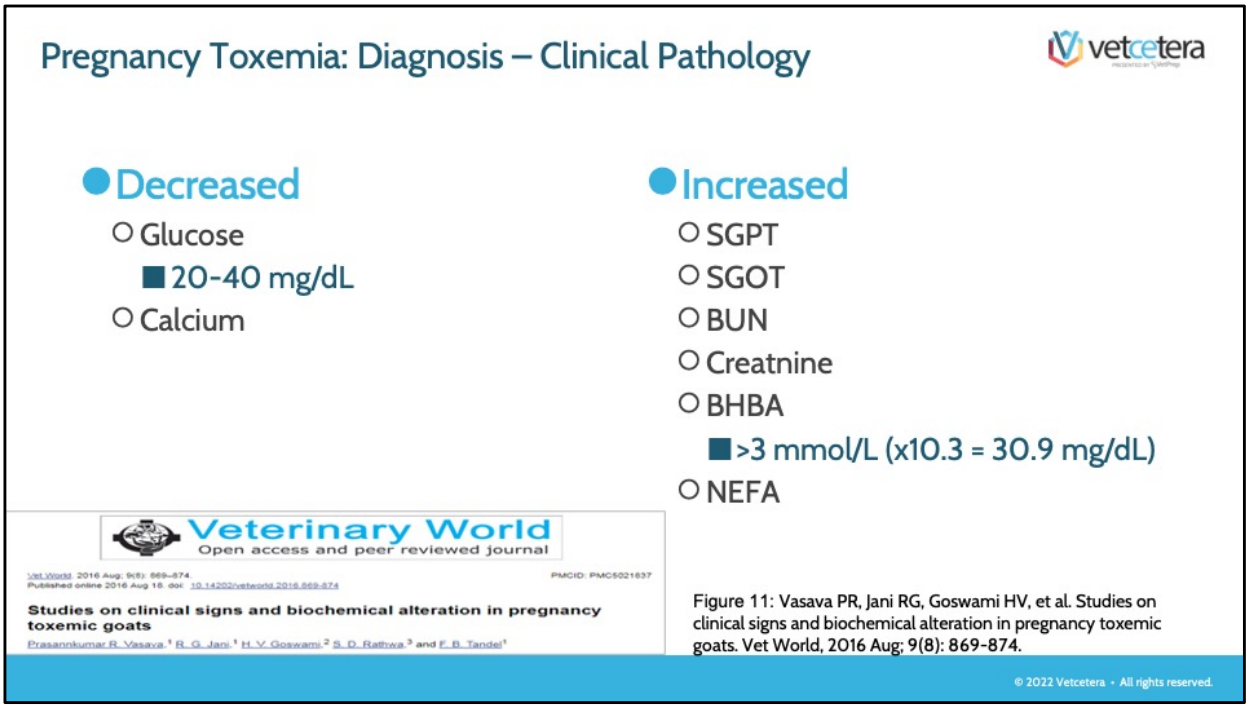
Pregnancy Toxemia: Diagnosis

- Dehydration
- Stress leukogram
- Metabolic acidosis
- Increased ketone bodies in urine
- Blood ketones

Figure 10: www.abbottdiabetescare.com

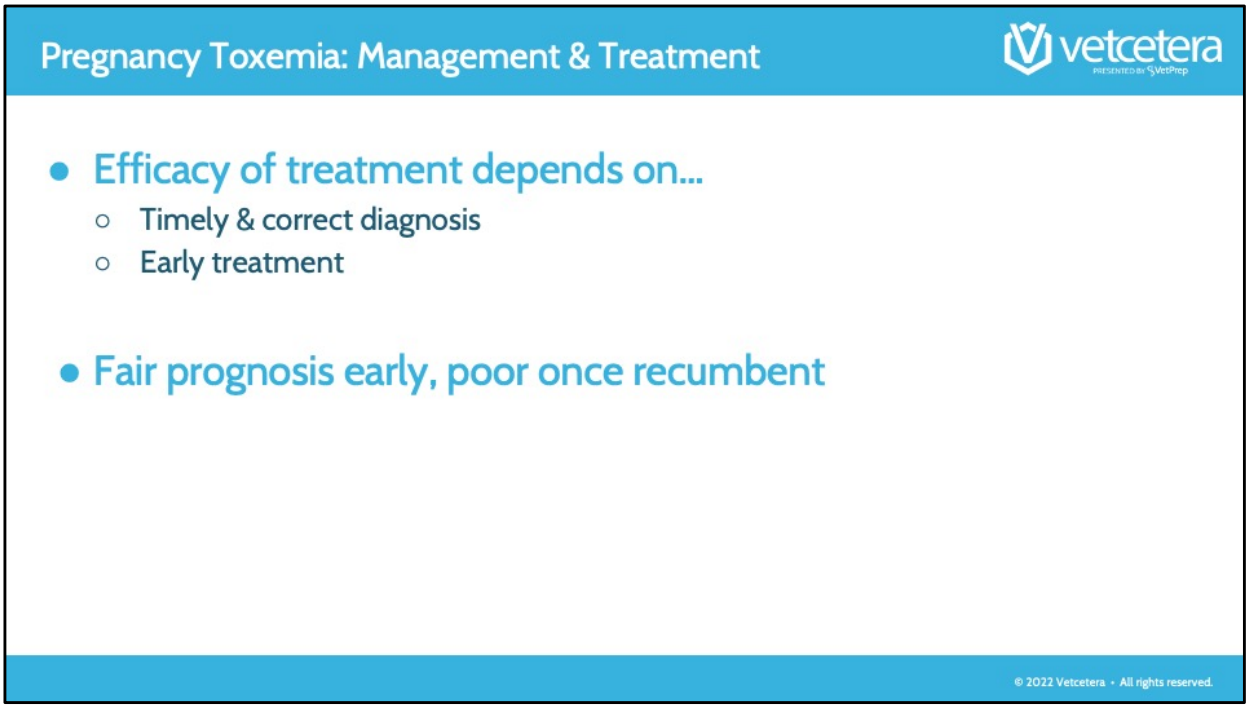
So these animals will be dehydrated. They may have a stress leukogram, metabolic acidosis, and an increased ketone bodies in the urine.

And it always seems like every time I've tried to obtain urine from a doe, they never cooperate. It's always my kind of bad luck. And so we started using this-- no, I don't get any compensation from Abbott or from Precision Xtra. But this is a very handy tool. The Precision Xtra allows us to measure blood glucose and blood ketones and extra measures beta-hydroxybutyrate.



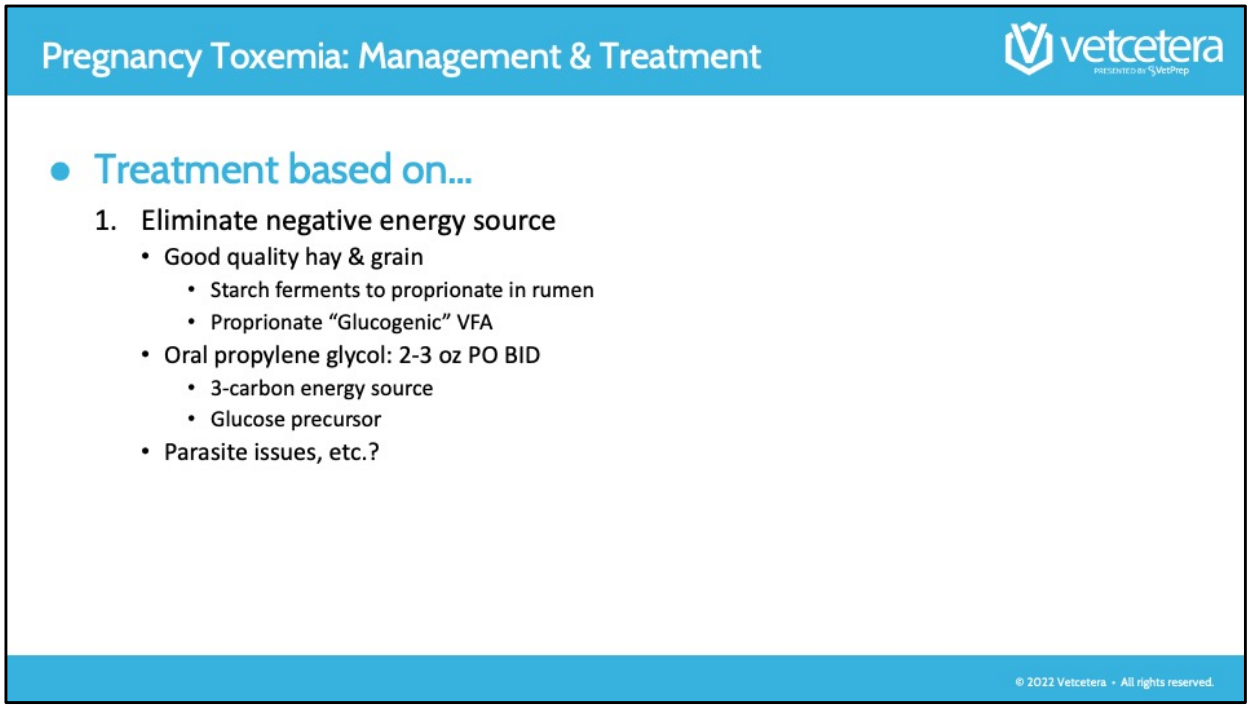
So very quickly, with just a drop of blood, we can get our ketone levels and know for sure definitively that this animal does have ketones in the blood, and then we can institute our treatment.

So we may see again decreased glucose and calcium, increased liver enzymes, BUN, and creatinine, again, that beta-hydroxybutyrate, that ketone body greater than 3 millimoles per liter is a great indication that we have pernicious toxemia that we need to begin treatment.



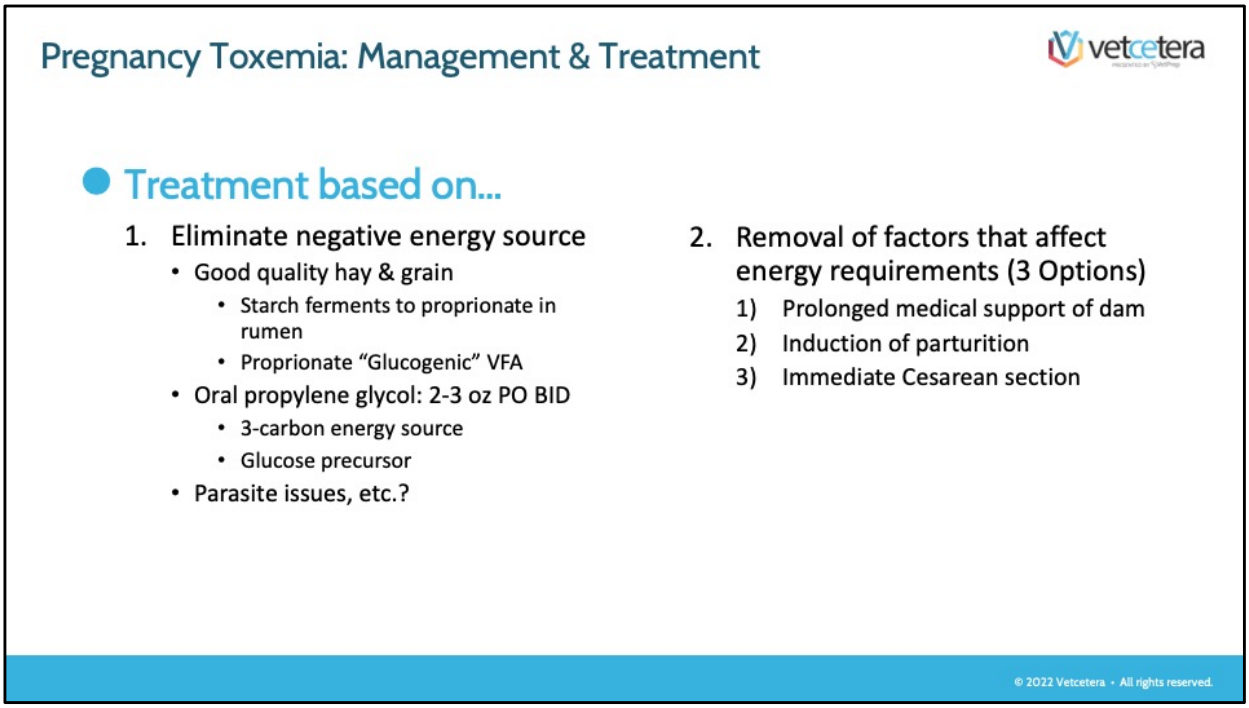
So our efficacy of the treatment depends on identifying and diagnosing this early on and beginning treatment early on. If we can identify it early on, and we typically have a pretty good outcome.

Once they're recumbent, the outcome is very, very poor. We have very poor prognosis.



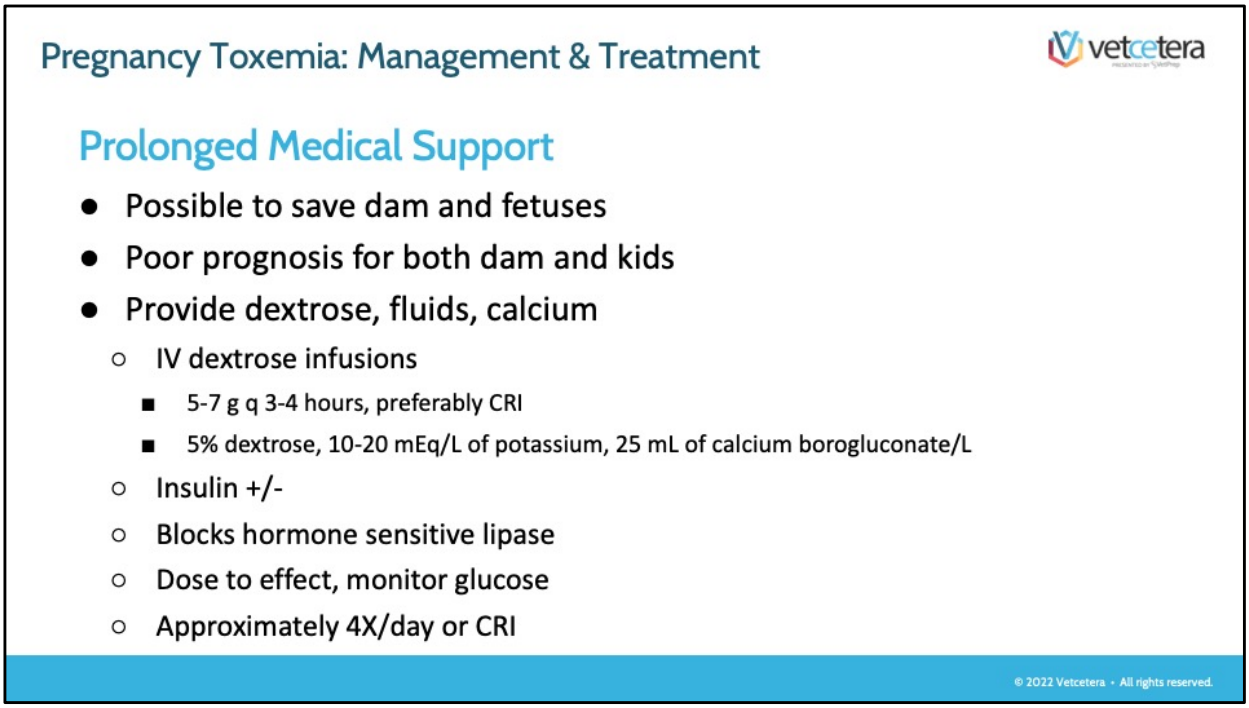
So treatment is based on eliminating negative energy source, making sure, late in gestation, those animals have good quality hay and grain. We can also supplement with oral propylene glycol, 2 to 3 ounces orally, twice a day. And that just serves as a glucose precursor. And oftentimes, we'll get owners to measure ketones in the urine if they start seeing ketones in the urine at home. They'd have a bottle of oral propylene glycol ready to use and give at home.

And then if there's other diseases, pneumonia, parasite issues, other things like that, we need to treat those problems as well.



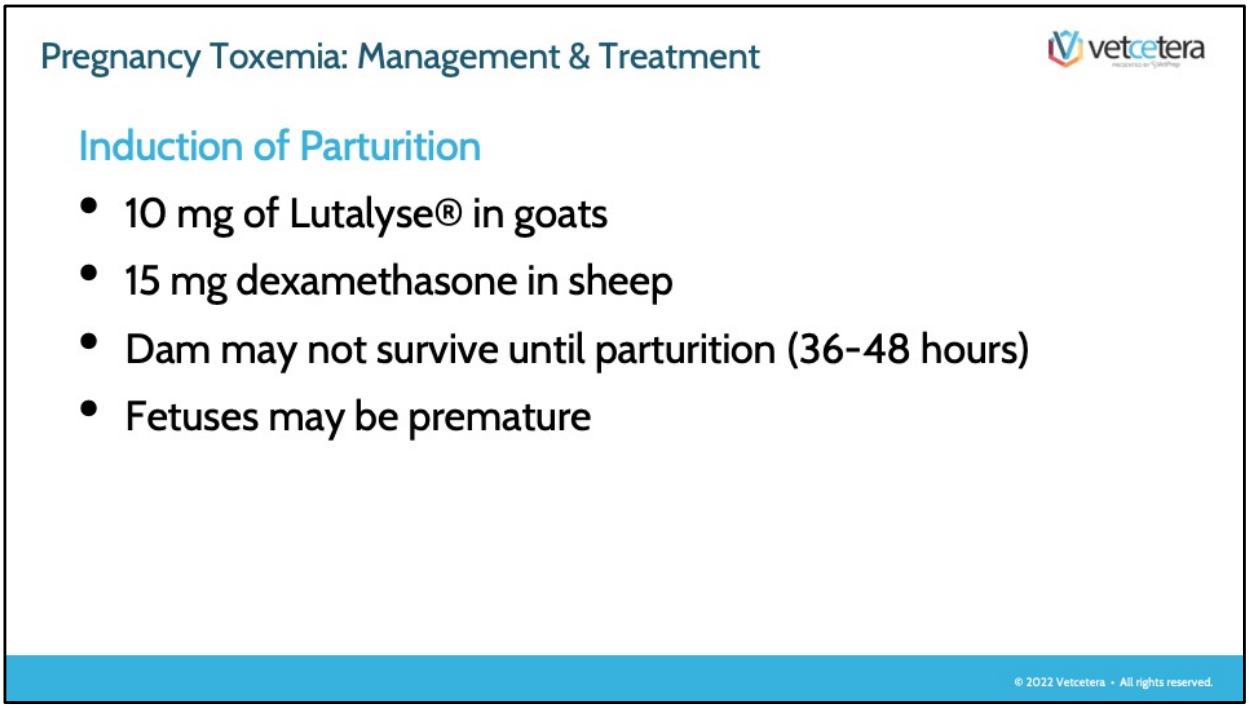
Some other things that we can do, removal of factors that affect the energy requirement. There's three options, prolonged medical support of the dam which can be expensive. Induction of parturition and immediate C-section. If we get rid of the energy requirements, which in this case, are going to be the fetuses, then this animal will recover very quickly.

So oftentimes, we have to have a difficult conversation with owners, producers that all right, what's more important here, the dam or the fetuses because they may not have a good breeding date, or they just may not know how far along she is for sure. And so then we have to help them make some educated decisions about what we need to do.



Medical support is hopefully we can save the dam and the fetuses. Again, it does affect the long term prognosis for both the dam and the kids. It does decrease with prolonged medical support.

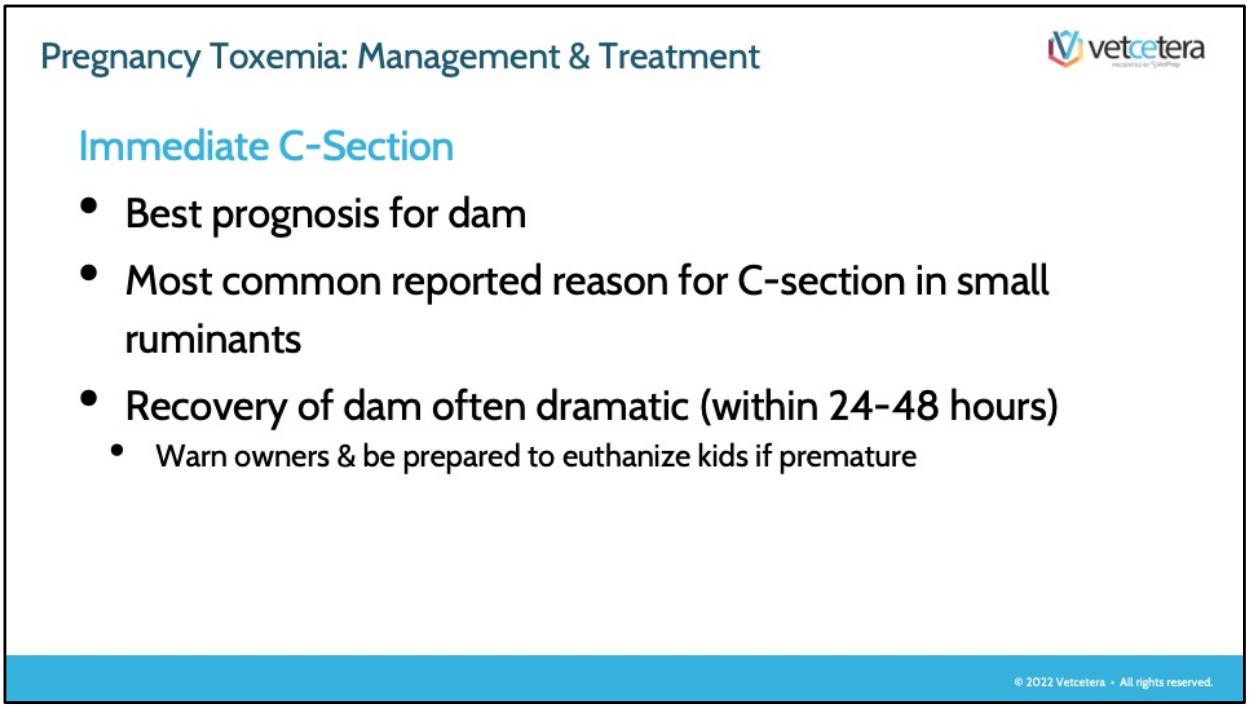
But we can provide. I'll give dextrose, fluids, and calcium. Those, too, affect with insulin. We haven't used that very much recently as something that we had done previously. But I don't know that I had a lot of success in some of these more difficult cases.



Really, induction of parturition or a C-section, we'll talk about those. Induction of parturition, if the animal is very lethargic, if she's down, oftentimes, induction of parturition can't be enough just to push them over the edge.

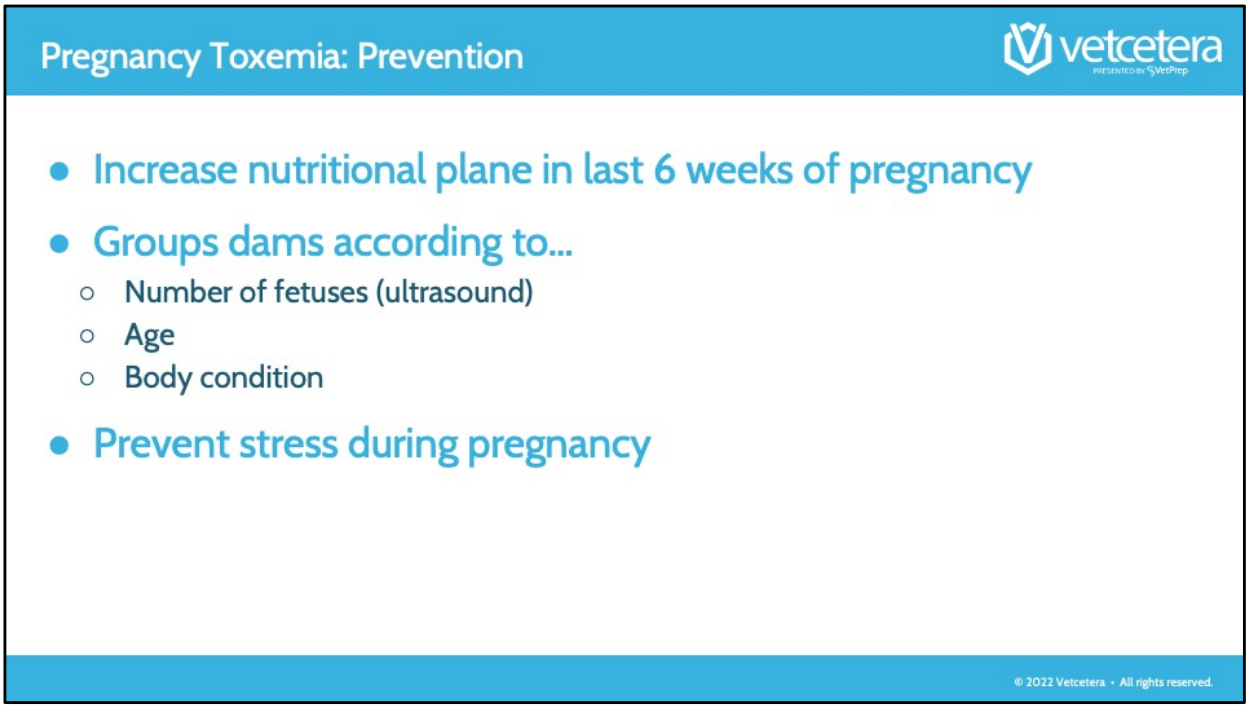
But we can try that especially if money is a concern. They can't afford to take him to surgery. With 10 milligrams of Lutalyse in goats, remember, they're still dependent throughout. 15 milligrams of dexamethasone in the sheep. Again, we would expect them to go into labor within 48 hours. Again, another conversation we need to have with the owners if they don't have a farm breeding date, if that, though isn't within 7 to 10 days of her breeding date, we will probably have premature fetuses that may need to be euthanized.

So again, another conversation we have to have with our owners.



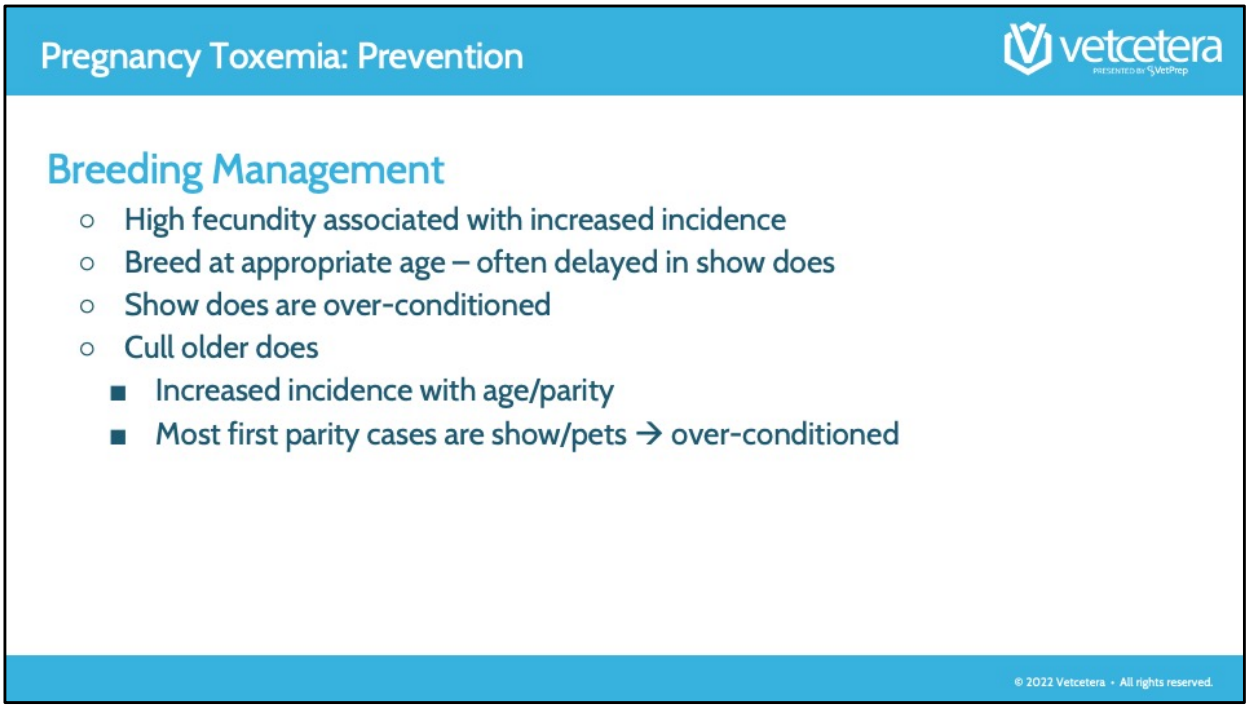
C-section, an immediate C-section offers the best prognosis for the dam. And it's very dramatic. Oftentimes, once you perform a C-section, how quickly these does recover.

Very dramatic and again, we have to have the conversation about if these animals, these fetuses are premature, that we may have to euthanize them.



So as far as prevention, increasing the plan of nutrition in the last six weeks of pregnancy. This would be a time where concentrates, grain, or high-quality hays would be a good idea. Grouping the dams according to the number of fetuses, if we know they're carrying a singleton versus twins or triplets, then we would need to feed them separately.

Based on age and body condition, we know that those older does tend to be more predisposed to this and then preventing stress during pregnancy as well.



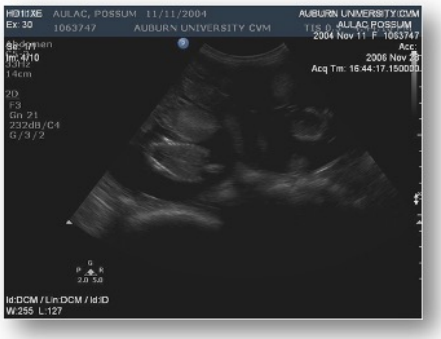
PUFA Kennedy is associated with an increased incident. Again, we're increasing our energy demands because of multiple fetuses. Breeding at an appropriate age oftentimes with show goats, they will delay breeding. And so they're over conditioned by the time they're bred. And over conditioning, in general, predisposes these animals to pregnancy toxemia.

Choline overdose because they do have an increased incident with age and parity.

Pregnancy Toxemia: Prevention

Nutritional Management

- Begin with good quality forage
- BCS and feed accordingly
- Ultrasound
  - Pregnancy status
  - Singletons/twins/triplets
- Establish feeding groups
- Increase feed as needed for weather conditions



As far as nutrition management, I've already spoken about monitoring the body condition and feeding them accordingly, ultrasounding them for pregnancy status, and a number of fetuses that are present. And then establishing breeding groups based on the pregnancy status and the number of fetuses present.

All right, so our last topic is dystocia. Excuse me, I've got to tickle in my throat.

Dystocia: Clinical Presentation

- Eutocia
  - Normally uneventful - only 3-5% require assistance
  - Lateral recumbency may be preferred
  - Older does/ewes may remain standing
  - Amnion protrudes through vulva → forefeet & head
  - Most cranial, longitudinal presentation
    - Some in posterior presentation (3-9%)
    - Normal if both legs extended
    - Dystocia if flexed hind limb

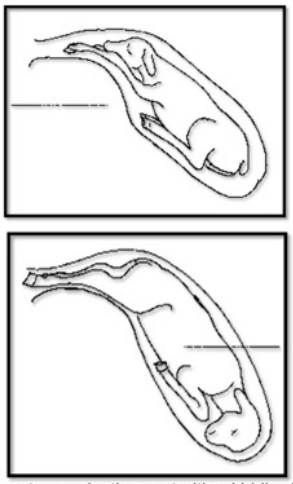
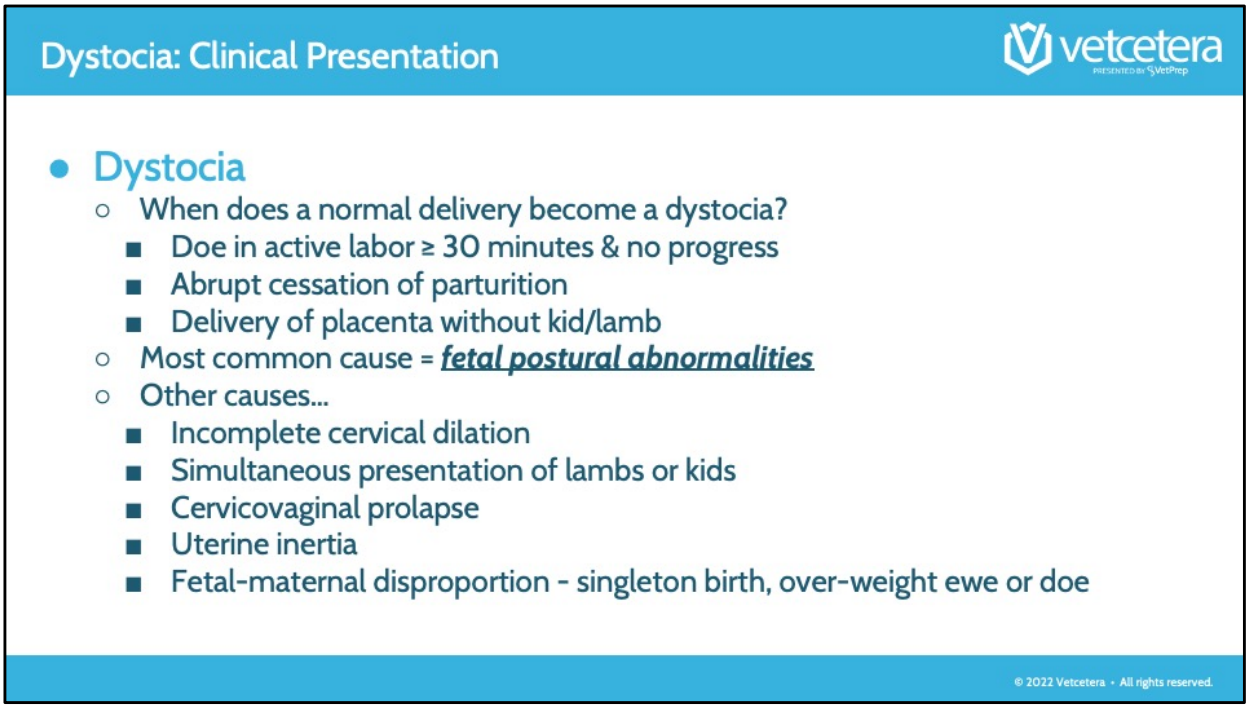


Figure 12: https://goats.extension.org/goat-reproduction-parturition-kidding/

Eustocia is a normal delivery, or it should be uneventful, only about 3% to 5% small ruminant deliveries require some type of assistance. Oftentimes, these does will remain standing. Overdose, particularly, will remain standing, but they may lay down as well. So both of those can be normal The amnion would protrude through the vulva first, followed by the four feet and the head.

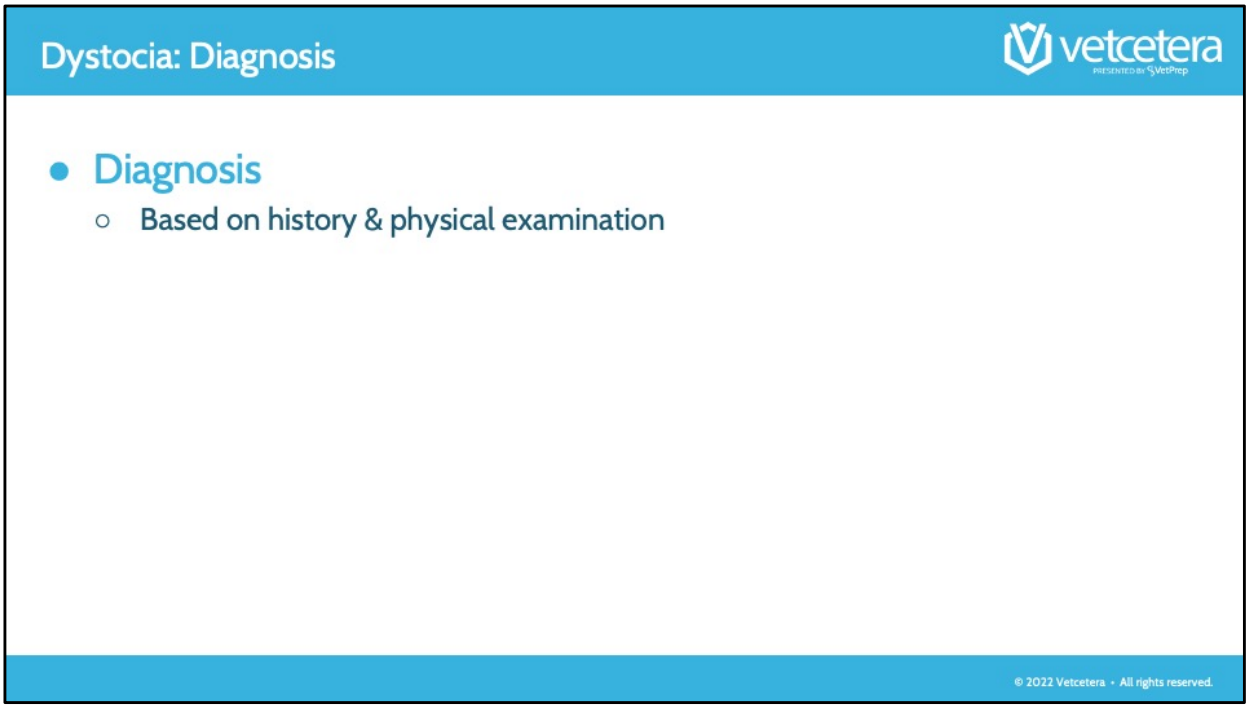
So that's considered a cranial longitudinal presentation. Some through the 9%, depending on what studies you look at, would present in a posterior presentation, which can be normal as long as both of the back legs are flexed. I mean, if they are not flexed, that they're extended as you can see in that bottom picture. If the back limbs are flexed in a true breech presentation, that obviously is a dystocia.



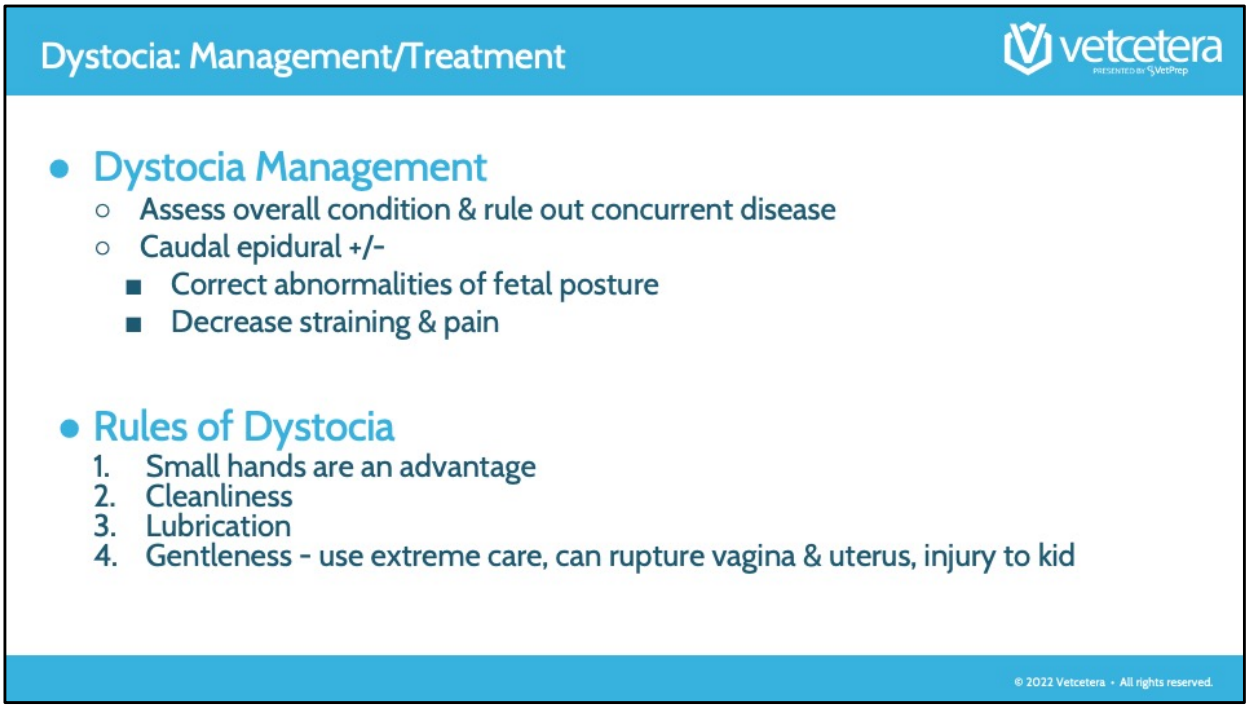
So when does it become a dystocia? And I think this is something that's important for us to talk to our producers, about our owners, about when do they need to call us.

A lot of times, they get a little bit anxious and want to call us as soon as they start showing signs of labor. But when do they need to call us? So does are in active labor, if they're inactive labor for more than 30 minutes and they're not progressing. So let's say, they see two hooves and it's been 45 minutes or an hour but nothing has changed. That's the time that I think it's time to intervene and to get checked out. So abrupt cessation of parturition as well. If they see the delivery of a placenta without a kid or a lamb, those would be the all times to intervene.

The most common calls are fetal postural abnormalities, meaning we've got a leg back, a head back, something of that effect; we can see incomplete cervical dilation as well; with multiples, simultaneous presentation of twins; cervical/vaginal prolapse, where there's been a lot of trauma to the cervix; uterine inertia, and then fetal maternal disproportion. Often, with a singleton birth, we just see a really, really large fetus or we can see this and overweight does in use as well, just have a very large pelvic opening there.

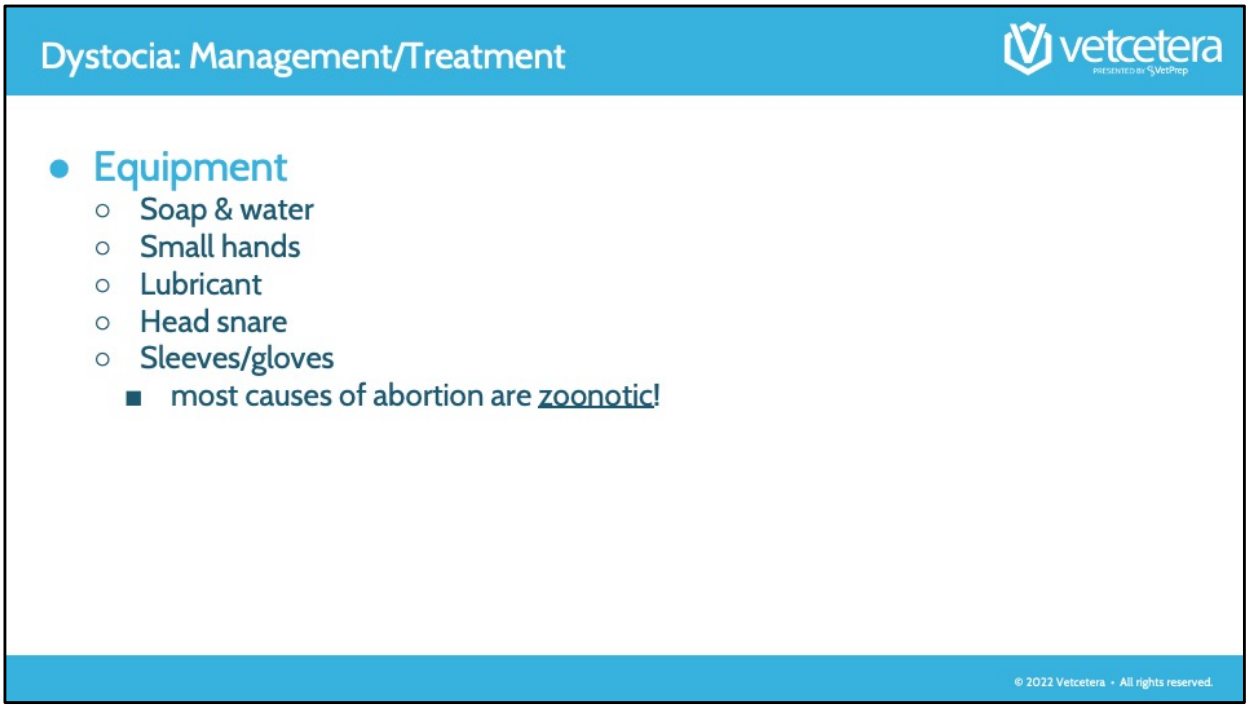


Diagnosis is based on the History and physical exam findings.



As far as management, we're going to assess the overall condition and rule out concurrent disease. Caudal epidural, I love the caudal epidural. It was very easy to do, and oftentimes I have small hands and that definitely is an advantage when dealing with a dissociated small ruminants, cleaning everything up using sleeves, using lots of lube, lots of lube, lots of lube, and being as gentle as you can being very cautious so that you don't cause a rupture in the vagina or the uterus or injury to the kids.

I've used all sorts of tools and things like that to help as well, just making sure we stay clean.



And so the equipment that we need is soap and water, small hands are a good idea if you have small hands around it can be done with larger hands lots of lubrication, a head snare, is very convenient to have. And then just remember, use protection sleeves, gloves, face shields or glasses if you have them. Most causes of abortion and small ruminants tend to be zoonotic. So keep that in mind as well, and advise our clients on that.

A lot of. Times we'll see some type of dystonia secondary to abortions.

Dystocia: Management/Treatment

vetcetera

- "Race to the Finish"
  - More than on fetus attempting to exit the vagina at same time
  - Figure out which legs belong to which fetus & untangle
  - ID 1st kid, repel 2nd kid, deliver 1st kid
- Head Malposition
  - NEVER deliver if the head is retained
    - may cause rupture of the uterus!
  - Push fetus back to gain room to pull head around

Figure 13: https://goats.extension.org/goat-reproduction-parturition-kidding/

So the race to the finish, that's just two fetuses presenting at the same time just have to figure out which legs and head belong to which fetus and then repel that animal back in and deliver the first kid. A head malposition, you never want to deliver when the head is retained because that can cause rupture of the uterus. That pole grabs the uterus at the body, and it will cause a very dramatic rupture. So you want to push that fetus back into game room, and if you've got an epidural on board, you can definitely do that.

And then pull that head around to gain a little bit more room.

Dystocia: Management/Treatment

- Front Leg Malposition
  - One or both front limbs retained
  - Some does may kid normally with one leg retained
  - If both, only head may be protruding from vulva
    - Swollen tongue
  - Elbow lock
    - Elbows are locked in flexion against pelvis
    - Repel fetus & digitally straighten one leg at a time

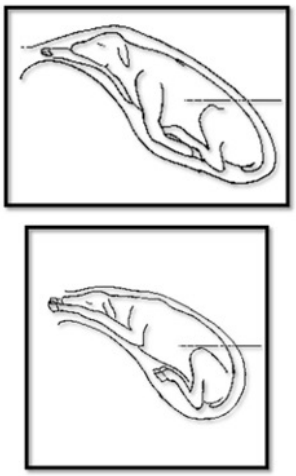


Figure 14: https://goats.extension.org/goat-reproduction-parturition-kidding/

A front leg malposition, some does can kid with one leg retained. I've seen that happen plenty of times. If I am presented with one limb retained, I do try to go and get the second male clarity. Just if it's not a lot of trouble, I try to go ahead and do that. I like to have two handles, and you can use a head snare if you need to. If both front limbs are retained, only the head is protruding from the bowl. And we can have a swollen tongue.

And then we have to go in and grab those legs. If there's elbow lock or the elbows are locked in flexion against the pelvis, we may have to repel the fetus. If the doe has pushed it so hard, its up into the--

Excuse me, --the vaginal cavity, repel that fetus's head back in to go in and grab that leg and force that elbow up. That's a little bit easier.

Dystocia: Management/Treatment

vetcetera

- **Breech Presentation**
  - Caudal longitudinal presentation, dorsosacral position, both rear limbs retained beneath fetus
  - Requires straightening both rear limbs
  - Push fetus cranially & to one side
  - Requires pulling & rotating the hock while the foot is pulled ventrally & medially
  - Careful with fetal hooves

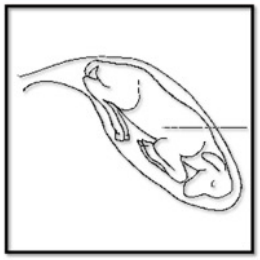


Figure 16: https://goats.extension.org/goat-reproduction-parturition-kidding/

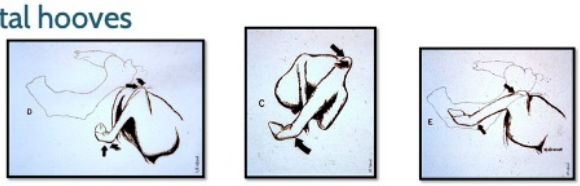
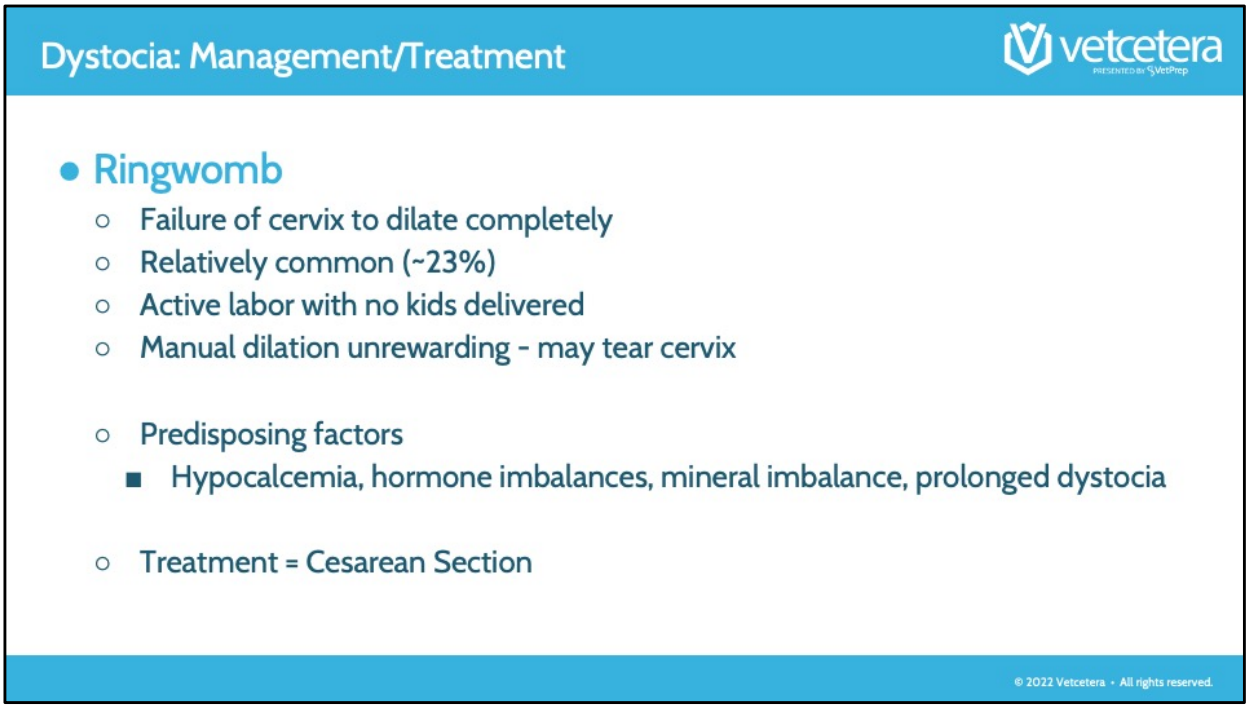


Figure 15: http://www.ansci.wisc.edu/jjp1/ansci_repro/lab/lab13_09/calving/abnormal_positions.html

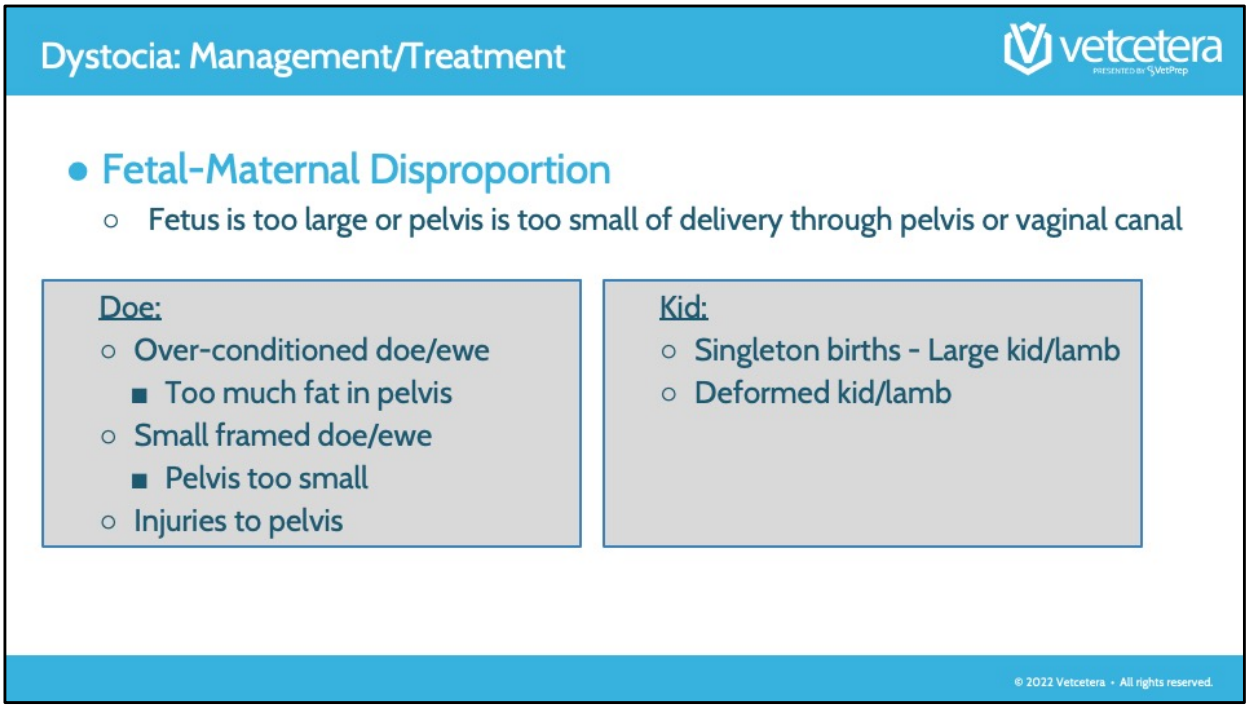
Breech presentation, these are probably the most difficult, a true breech presentation, where you actually have to take the heart and rotate it laterally as you're rotating the foot medially. And that's an easy way to pop that foot up into the pelvic canal. You're going to be repelling that fetus into the uterus, as you're rotating that Hawk laterally as you're rotating the foot immediately.

And just be careful with the fetal gloves that so that they don't cause rupture of the uterus.

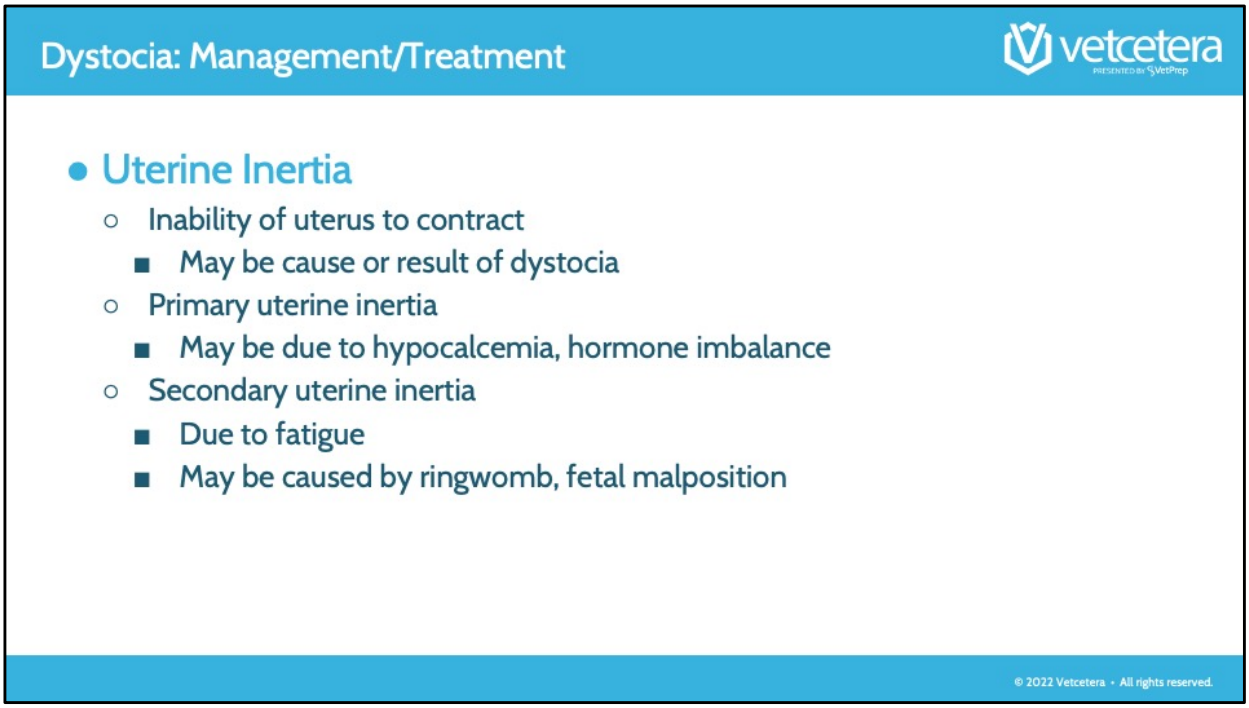


Ringwomb is failure of the cervix to dilate completely, relatively common approximately 23% of dystocias. So if an animal is in active labor with no kids delivered, we need to rule out ringwmb. Manual dilation in most cases is unrewarding. It's probably just going to have to go straight to a C-section if you get in there and you palpate and you feel that cervix is not completely open.

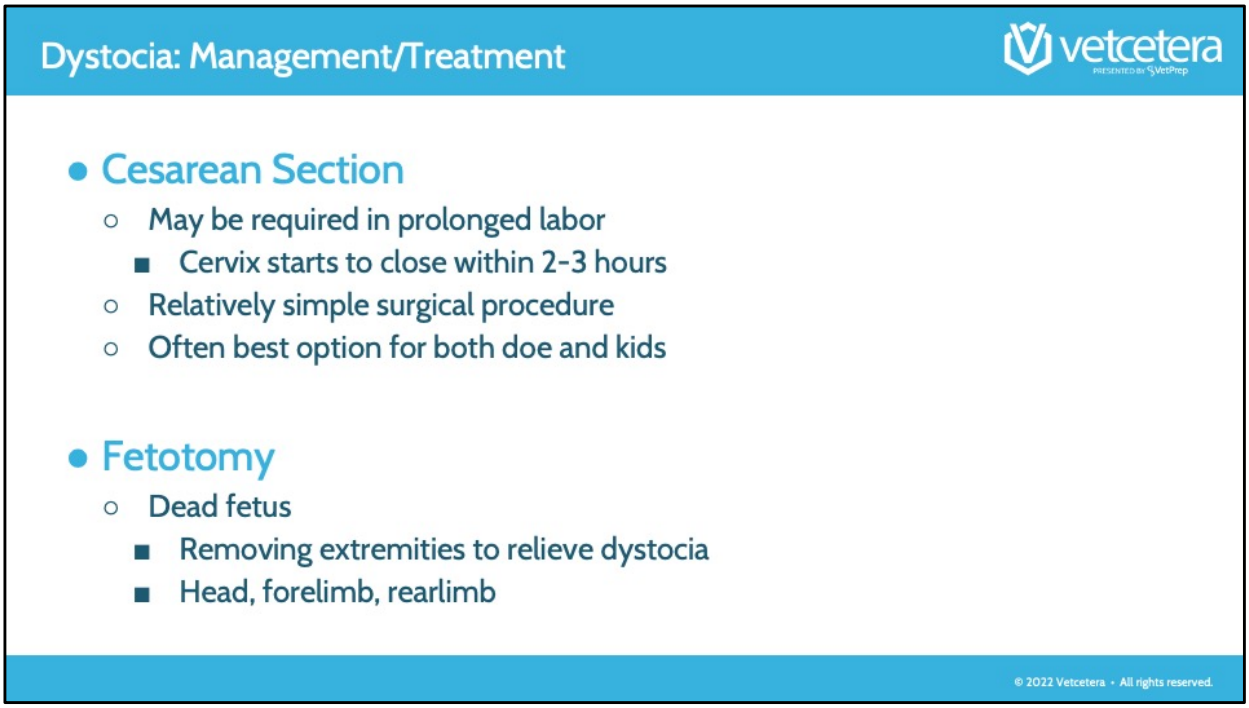
Oftentimes, I think that we see a ringwomb after-- maybe almost haven't recognized the doe that's in labor. And she's maybe been in labor for a little bit longer than we would like. And that cervix is that it was actually dilated. Maybe there was a fetal malposition, and now that cervix begins to close back down. It happens faster in small ruminants than it does in our other ruminant species. So we can see that due to hypercalcemia, hormone imbalances, or prolonged exposure, as I mentioned.



Fetomaternal disproportion, fetus is too large or the pelvis is too small. With the doe, again, over condition does have used which is too much fat in the pelvis to allow the fetus to move through. We may have those with small frames or if they are injured. If there was some type of injury to the pelvis, abnormal anatomy, now that can also cause it, but probably, far and away, I've seen more large kids from lambs born or deformities to the kid itself that's prevented the delivery and the decision that's been cost.

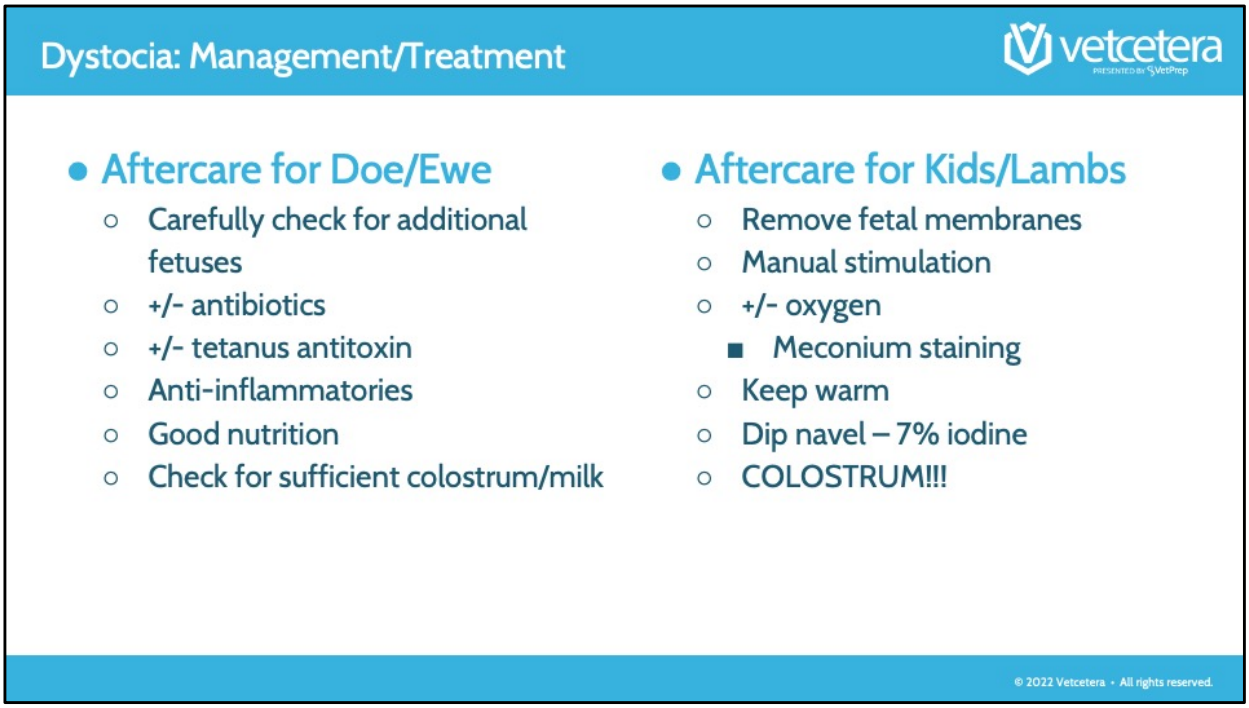


Uterine inertia, and that's just inability of the uterus to contract. Oftentimes, we think about that as a primary factor of hormone imbalances or hypercalcemia. And then secondary uterine inertia due to fatigue and that's again secondary to some type of fetomaternal disproportion or saw a limb being retained or a head being retained. They're fatigue and the uterus cannot contract because of that.



C-section, this is my favorite surgery to do is a C-section, easy to do, very simple. And it's, obviously, oftentimes, the best option for both the doe and the kids.

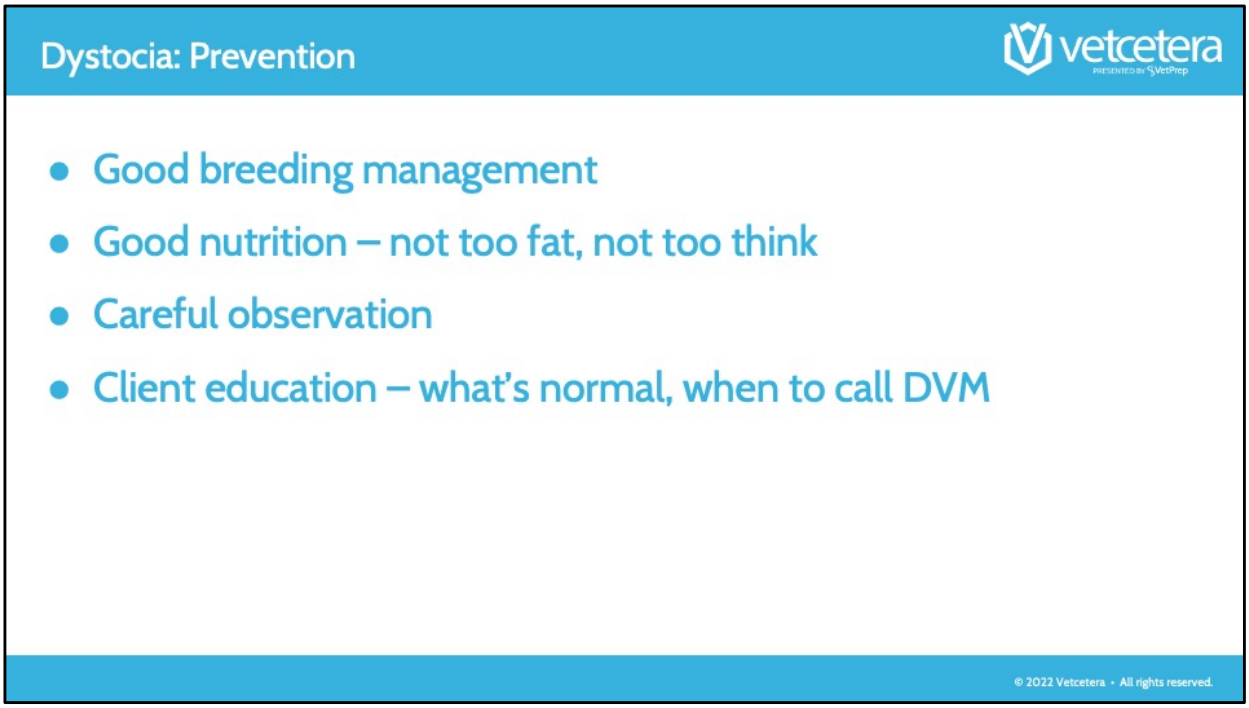
Lithotomies, not my favorite thing to do. But if you have a dead fetus and money is concerned. Then we can do a lithotomy removing extremities to relieve the distortion and then deliver the fetus.



As far as aftercare, we want to make sure we check-- always, always to check for tears and spares, may or may not need antibiotics depending on how traumatic delivery was. Remember, tetanus antitoxin or tetanus toxoid and antitoxin. And these animals are particularly sensitive. Anti-inflammatories, good nutrition as far as the kids are lambs are concerned. Removing fetal membranes from their mouth and nose, obviously manual stimulation,

Meconium staining, they may need some oxygen supplemental oxygen to support if there's some meconium staining. Depending on when they're born, keeping them warm, depending on the environment. Dipping the navel with 7% iodine, that's not absolutely necessary. But if they're born in hospital, we always get the navel just to be on the safe side and then making sure they get colostrum. Ensuring that the dam has colostrum, particularly if we're talking about one that had an induced parturition because of pregnancy toxemia or C-section because of pregnancy toxemia.

If not, then they may need a plasma transfusion or some type of colostrum replacement.



So prevention on dystocia is just good breeding management, good nutrition. We don't want them to fat. We don't want them to thin. We'd like them an ideal body condition around the 3 or a low 4. Careful observation, teaching our producers what to look for when it becomes a dystocia greater than 30 minutes and when to call u, so. things to look for and when to call us.

That's all I have, and I'll take-- I think I've got a minute or two for some questions if anyone has any questions.

We do have one question.

What type of dosage of post-op pain meds for after a c-section do you prefer?

So I think it really depends. And that's a great point. I think that's one of the things that we have really probably need to do a better job of with our remote species is pain management. Oftentimes, I would just use some Banamine or butorphanol. I prefer to do my C-sections in a flank, so a left flank incision versus a ventral midline. And that's because-- especially if I have--

Excuse me, --live, born kids, oh, when they're trying to nurse, they can traumatize that incision a little bit. And that's usually pretty painful to those. So I try to keep my incision away from the udder, off of the ventral midline if possible. There are times that we would need to do that. So my favorite post-op pain meds, orbenamine, and that's a mil per 100 pounds IV or butorphanol 0.05 mg per kg, the standard dose. So usually, they do very well after those C-section. Again, like I

said, I'd keep my incision up off of the midline. If possible.

Perfect, thank you so much. That looks like all the questions we have. So we'll let you hop off. I know we kept you a little bit longer than planned. And we appreciate you joining us again. That was a really great presentation. And I'm sure everyone found it really helpful. Hope that we'll have another one with you soon.

All right, thanks a lot. You have a great day.

Thanks.

Bye, bye.