

Urinary Disease in Small Ruminants

Dr. Misty Edmondson reviews the most common causes of urinary tract disease in small ruminants.

Speaker Bio:

Dr. Edmondson is a native of Alabama where she grew up on a commercial cattle farm. She received a BS in microbiology from Auburn University in 1997 and DVM from Auburn University in 2001. She worked in a mixed animal practice in rural Alabama for 2 years before returning to Auburn University to complete a residency in food animal theriogenology. Dr. Edmondson completed her residency and MS in biomedical sciences from Auburn University in 2006 and became a Diplomate of the American College of Theriogenologists in 2005. She joined the faculty at Auburn University's College of Veterinary Medicine in 2006 and was promoted to professor in 2018. Dr. Edmondson taught in numerous courses (including theriogenology, reproductive anatomy, urogenital surgery, production medicine, and others) for veterinary students, as well as clinical rotations in food animal medicine and surgery and advanced rotations in advanced beef production and advanced small ruminant medicine and surgery. She was also involved in theriogenology resident training through clinical instruction, graduate lectures, and laboratories. In September 2018, Dr. Edmondson left Auburn University to join the Alabama Department of Agriculture and Industries as an Associate State Veterinarian. Dr. Edmondson has been married to her husband, Jason, a beef nutrition specialist, for fifteen years. They have two children, Wyatt and Laken, and have a farm where they raise Angus and SimAngus cattle, horses, and a few chickens.

Learning Objectives:

1. Attendees will be able to diagnose common urinary tract diseases in small ruminants based on key physical examination findings and diagnostic tests.
2. Upon diagnosis, attendees will be able to offer treatment strategies to their clients for each of the common urinary tract diseases.
3. Attendees will also have a basic knowledge on the best methods to prevent the urinary diseases covered in this discussion.

Urinary Disease in Small Ruminants

Misty A. Edmondson, DVM, MS, DACT

Hi, everyone, thank you so much for joining us today. Our speaker tonight is going to be Dr. Misty Edmondson. She is a theriogenologist and she actually was an instructor when I was at Auburn. She's a fabulous person, she knows all things small ruminants and she's going to be speaking today on urinary disease in small ruminants. Currently, she's actually working as the associate vet for the state of Alabama. I will turn it over to her. If you guys do have questions, go ahead and type those in the Q&A section we'll get to them at the end if we have time, thanks.

Thank you Katie it's great to be presenting today on

urinary disease in small ruminants, definitely something that if you're seeing any small ruminants you're going to be confronted with some of the issues that we're going to talk about today.

1. Physical examination
2. Diagnostic tools
3. Obstructive urolithiasis
4. Ulcerative posthitis
5. Other diseases
 - Hypospadias
 - Renal Amyloidosis
 - Neoplasia

The first thing we're going to go over this is just kind of a brief overview of the talk for today is physical examination, important factors and how small ruminants may be a little bit different than our other ruminant species and definitely from small animals.

Some of the diagnostic tools that we have available for use in small ruminants, and then we're going to go over some more specific diseases spending most of our time on obstructive urolithiasis that's probably the number one for sure most common urinary diseases that we see in small ruminants and very common to see all sorts of posthitis and then a few other diseases

that kind of fall at every category but hypospadias in young animals, renal amyloidosis and then go over a couple of instances of neoplasia that we see in small ruminants.



Physical Examination

So to begin with, we're going to start with our physical examination and we talk about their physical exam.

- Urinary tract disease very common in small ruminants
- Abdominal palpation
- Urethra
 - Male
 - Observe indirectly as exits pelvis and stays along body wall to external urethral orifice
 - Pulsations & generalized or focal swellings along length (obstruction, urethral rupture, hematoma, abscess)
- Vulvar or Sheath Hairs
 - Examine for grit, blood, purulent exudate, urine

The first thing we want to do is to obviously take a look at the animal in general and then focus in on our external genitalia. Part of that exam is going to be involving abdominal palpation as well as palpation of the external genitalia as well. Most of the urinary disease that we see specifically urolithiasis is involving the male. So we want to make sure that we do a very good physical exam of the entire reproductive tract the external genitalia the urogenital tract.

We want to make sure that we follow the urethra. So remember that the penis is located physically as it exits the pelvis and it stays along the body wall to the

external urethral orifice through the preputial opening. One thing that we can look for in small ruminants, ruminants in general are pulsations when those animals urinate. That's normal, that's that normal pulsatile fashion of urination.

And if we see that urethral pulsation without the production of urine that's a very good indication that this animal is most likely obstructed and has urolithiasis. Again, we can see generalized or folklore swellings along the length of the urethra along the penis in the male. So obstruction we can see urethral rupture where that urethral rupture that may be more of a generalized swelling depending on how much urine has leaked into the tissues and how long that's been going on. We can also see hematomas and abscesses although those are not as common in general.

We also want to make sure we look at the sheath hairs and the vulva of those animals as well. We want to examine for any grit, really crystals that have formed on those hairs with the urine that exits there that grit will appear. Blood purulent exudate and then we want to look at the consistency of the urine as well.

- **Exteriorize penis**

- Examine free portion of penis and prepuce
- +/- Sedation: set up on rump or lateral recumbency

Continuing with our external genitalia examination, we would want to make sure we're able to examine the free portion of the penis and the prepuce. That is much more readily, more easily done in an intact male or an animal that has been castrated after puberty. The influence of testosterone on the reproductive tract in those males allows for the exteriorization of the penis and prepuce much more easily.

Generally, the way that I have done that is to provide sedation particularly if it's on emergency, which these often tends to tend to be when you don't have a lot of people there to help you, sedate them, just a light

sedation. And then setting them up on their rump tends to provide a really, really good access to the penis and prepuce if you can just set them up just like you would a sheep for shearing or any other type of examination. That makes that much more easy to visualize and exteriorize the penis. Or you can place them in lateral recumbancy and then manually extend the penis and prepuce.

- **Males**
 - Urethral pulsation with urination
- **Females**
 - Standing but often squat to urinate

So again talking about naturalistic behavior, just remembering that in ruminants it is a pulsatile stream of urine that is created, so there is urethral pulsation. If we see that without urination, that would be abnormal. And in females, often times they'll squat to urinate, so that's completely normal. It's not an indication of pain or anything like that if you see them squat to urinate.

Diagnostic Tools



So now we're going to go into some of the diagnostic tools.

- Vaginoscopy
- Cystoscopy
- Radiographs
- Ultrasound
- Urine Collection/Urinalysis
- Chemistry/CBC

This is a pretty long list. Some of this may be limited more to academic settings or referral hospital settings. But there's definitely plenty that you can do in your own practice. If you have an ultrasound and those types of things that will get you to the point of being able to make a good diagnosis.

- **Vaginal examination**

- Use a speculum & light source to examine urethral orifice
- Also visualize cervix, vagina for trauma, adhesions, etc

So as far as the vaginal exam vaginoscopy, just a small speculum and a light source can be used to examine the vagina and the ureteral orifice, want to make sure that we don't see any types of growths or unusual appearance there on a vaginal exam. You can also visualize the cervix as well. And often times you'll see that there may be some adhesions or that there's some type of trauma that's occurred either secondary to breeding or secondary to dystocia. It's not uncommon to see adhesions form across the cervix and into the vagina after that, so that can also affect the ability of that animal to urinate as well.

- **Fiber optic endoscope**

- Visualize inside of bladder
 - Epidural, sedation
 - Void bladder of urine
 - Inject 500cc of air to visualize bladder musoca
 - Rarely used for routine cases
- Rarely used due to requirements for diameter and length of the endoscope

Fiber optic endoscope, this is a pretty specialized piece of equipment just because the diameter and the length that we would be required to enter the urethra through a small ruminants is a very, very small diameter. But if you have access to that, it does allow you to visualize the inside of the bladder to look for and we'll talk about some of the neoplasias or stones or other pathology that might be present. If you do that, obviously sedation, astromonas and epidural would be needed.

You would need to avoid the bladder for good visualization, and that's aided with injecting 500 CCs of air into that bladder. But again in most cases, that's

not necessary. It's not something that we have to use for these animals to make a diagnosis.

- **Survey Radiographs**

- Evaluate peritoneum, body wall, kidneys, ureters, urinary bladder, urethra if enough contrast with surrounding tissues
- Abdominal effusions (urine, blood) may decrease visualization

- **Contrast Radiographs**

- Excretory urogram to view kidneys (anatomical and qualitative function)
- Administer contrast media IV & serial radiographs (up to 40 minutes)
- Patient should be well hydrated which is often not the case in many cases
- Contrast cystography & urethrography may be used normograde or retrograde with air or contrast media

As far as radiographs are concerned, you can just do survey radiographs that allows us to visualize the peritoneum, the body wall the kidneys, the ureters, urinary bladder and urethra if there's enough contrast with surrounding tissues.

And in the cases that, most of the cases that I saw were pet animals or show animals that were fed high grade diets and generally over conditioned, so there was a lot of distinction made and a lot of contrast with the surrounding tissues. So radiographs can be helpful with those animals it also allows us to visualize abdominal infusions, but again, we have to remember that on radiographs that's not as easily seen. And so if we have that haziness on our radiographs, it may be an indication that there's an abdominal effusion present.

On contrast radiographs, again, this is not something that's probably done very commonly in routine general practice, but they are available. You can do excretory urograms to view the kidneys if you're concerned about the anatomy or function of the kidneys, and that's again just administering IV contrast and then taking Cyril radiographs for 40, 45 minutes or so. It's important to make sure that the patient is very well hydrated and as you probably remember in most of these cases when we're dealing with urinary tract disease, often times these animals are very dehydrated, so it may not be quite as useful.

So we can do that either in normal grade or retrograde with air contrast media. Again, we have to be careful, especially when we're thinking about urolithiasis if we're using that contrast in a retrograde fashion into the urethra. That urethra is very friable in those cases.

- **Transabdominal ultrasound used to visualize bladder & kidneys**

- 3.5-5 mHz curvilinear or linear probe
- Left kidney: dorsal, paralumbar space
- Right kidney: 11-12th intercostal spaces
- Locate both kidneys on right side
- Kidney smooth & lack lobulations

- **Very difficult to visualize urethra & ureters in SRs**

- **Visualize abnormalities such as...**

- Hydronephrosis – dilated ureter with anechoic fluid
- Pyelonephritis – enlarged kidney with dilated renal sinus, echogenic debris present

And again, radiographs probably do have their place but since ultrasound has been available and is becoming extremely common in most all practices are very common in a lot of practices, most people just pick up the ultrasound instead of trying to use radiography.

So transabdominal ultrasound can be used to visualize the bladder and the kidneys. My preference is for a 3 and 1/2 but you can use up to a 5 megahertz, I've again preferred the 3 and 1/2 megahertz curvilinear probe over the 5 megahertz linear probe, but both of them can work very well. Just a review of the anatomy that

the left kidney is located in the dorsal parallel bar space, and then that the right kidney is located a little further back the 11th to 12th intercostal spaces.

Both of those are visualized on the right side. If you'll remember that the ruminant kidneys, that left kidney is on midline and the right kidney just sits off to the right and is caudal to that left kidney. So you can visualize both of those kidneys from the right just with a simple ultrasound exam.

Remembering that both the kidneys are smooth and like the labulations that we see in cattle, so that's completely normal in small ruminants. As far as visualizing the urethra in the ureters, that's in a normal animal that should not be very easy to do. If we have hydronephrosis, then we may be able to see that but it's not terribly easy. So again by ultrasound and the kidneys, we can look for hydronephrosis and see that dilated ureter with an anechoic fluid, that Black fluid present.

And then it can also clue us in that we may have some more serious infection or pyelonephritis. If we see an enlarged kidney with a renal sinus that's very large, and there may even be some echogenic debris, some exudate present in there, that could be an indication as well. And one thing I always do I always ultrasound the kidneys before, for urolithiasis especially. Want to

make sure we do a good thorough ultrasound of the kidneys because I have seen nephroliths present in the kidneys.

And obviously that has a much more guarded prognosis than more straightforward urolithiasis cases. So if you get the ultrasound out on the abdomen and you're looking at the bladder, save a little bit higher up on that right side. Take a look at the kidneys, make sure that there's no evidence of nephroliths or severe pyelonephritis again things that could change your clinical outcome.

- **Bladder**

- Right inguinal area transabdominally, may be viewed transrectally.
- Evaluate wall thickness (varies with fullness), contents, distension
- Bladder may contain hyperechoic debris with hematuria, pyuria, or stones/crystals
- Excessive free fluid in abdomen
 - Anechoic fluid – transudate or modified transudate → urine leakage
 - Echoic fluid – cells or protein indicate inflammation or exudate

As far as the bladder is concerned, most of the time I'll start in the right inguinal area with a transabdominal, again at 3 and 1/2 to 5 megahertz probe and just kind of move it around and go towards midline from the inguinal area to the midline. That right inguinal area is a good place to look because there's less hair in the inguinal area, you don't have to shave. You just get a real quick and dirty view there, but you can also go trans-rectally in small ruminants to visualize the bladder as well.

I don't typically do that, unless I'm doing a reproductive exam. But if I'm suspecting urolithiasis or primary

bladder disease, I'm going to go trans-abdominal. And so what we look for there is the wall thickness and obviously it varies with the fullness of the bladder, but if I have a grossly distended bladder and very thickened walls, that clues me in that there's obviously some pathology going on there, versus it would be very thin in a normal bladder that's distended.

And that helps us look for the contents in the bladder, oftentimes if there's crystals or there's infection present, we'll see that sort of snow globe effect. We'll get some varying recognized in that swirling effect of the contents in the bladder and again looking at station as well.

Well, we do see that snow globe effect or those hyperbolic contents that clues us in that we may have hematuria or pyruvia or stones or crystals that are present in the bladder. We can also when we're ultrasound the abdomen, look for excessive free fluid in the abdomen. There's normally a little bit of free fluid there, but if you ultrasound very many animals you'll notice quickly, especially in cases with urolithiasis, that they can have a leaky bladder, or actually rupture of the bladder. And you'll see quite a bit of fluid even from those leaky bladders where they've just been distended for a prolonged period of time.

So again, if we see that again it can affect our prognosis for that animal and what we may need to do as far as surgical intervention.

● Cystocentesis

- Better sample vs free-catch for urinalysis and bacterial culture
- Short-term relief in cases of obstructive urolithiasis
- Good restraint, aseptic prep, 18-gauge, 2-3.5-inch needle with syringe attached
- Quick insertion & withdrawal needle → ensures small perforation of bladder, seals quickly

As far as urine collection and urinalysis, we're going to go into the various ways to collect urine and what those mean. Cystocentesis obviously is a better sample, particularly if we want to do bacterial culture than the free catch. So oftentimes, we'll try to do in cystocentesis particularly if we have ultrasound to guide us, to aid us rather in an ultrasound guided cystocentesis.

There's some debate as to whether or not we should perform a cystocentesis in animals that are suffering from obstructive urolithiasis, it can be a temporary relief to that animal and if you suspect that this animal

is approaching the stage that this bladder looks extremely large, I'm fearful of it rupturing and surgery or whatever treatment option may be delayed, then that might be a good approach to go ahead and relieve some of that. There's some folks that and I guess I'm of the mindset that this is a true emergency and we'll just go ahead and go straight to surgery.

But if that felt like that was going to be delayed at all, that cystocentesis would be an option. To perform the cystocentesis, make sure you have good sedation or restraint, so you have enough people around just adequate restraint and aseptic prep, and usually using an 18 gauge needle with at least two to 3 and 1/2 inch needle with the syringe attached. You want to quickly insert that and withdraw the needle.

When you do that quickly, again that's going to ensure that there's a very small perforation in the bladder that is able to seal very quickly. We try to do that at an angle going in again just so those layers of the bladder can close over each other and hopefully seal that. And you would want to take a significant amount of urine if it's grossly distended, significantly distended to help that bladder kind of just decrease the volume and allow it to seal over.

- **Abdominocentesis**

- Analysis of free peritoneal fluid (uoperitoneum)
- Aseptic preparation of abdomen, local anesthesia (lidocaine)
- 18-20 gauge, 1-1.5 in needle; teat cannula if significant debris present
- +/- ultrasound-guided
 - Collection of fluid from visualized pockets
- Blind sample
 - Cranial: just caudal to xiphoid & 1-2 in to lateral of midline
 - Caudal: just cranial to udder or scrotum & 1-2 lateral of midline
- Testing: Creatinine concentration > 2X serum creatinine = Uroperitoneum

As far as abdominocentesis, we will do this when we see evidence of free fluid in the abdominal cavity and suspect a uroperitoneum potentially. So again, aseptic preparation of the abdomen, and then we're going to use a local probably just one to two CCs of a bleb of lidocaine in the skin and maybe into that muscle layer just with a small gauge needle. If we're going to perform the abdominocentesis though, we're going to need at least an 18 to 20 gauge one to 1 and 1/2 inch needle.

If you suspect if there is a lot of debris present, you may actually need if you have a T cannula, those work

very well just because they have a larger opening present and allow some of that debris to be collected and it won't prevent you from obtaining your sample. I prefer to do these under ultrasound but you don't have to. It's easy enough to do a blind sample collection.

If you're going to do that, there's two spots that are recommended for collection. Cranially, you would use just caudal to the xiphoid process, and about one to two inches lateral to midline and then a caudal location that's just cranial to the udder or the scrotum, again 1 to 2 inches lateral of midline. So just off midline there to collect those samples.

I do prefer the ultrasound because oftentimes, there'll be pockets of fluid or urine that's present. And so that ultrasound obviously just helps you be a little bit more accurate. And again, if you're suspecting your peritoneal, one thing that you can do with that fluid is a creatinine concentration of the fluid. And if it is greater than 2 times your serum creatinine, then it's truly diagnostic of your peritoneum.

Kind of another I guess quick and dirty trick is if you put some of that urine in and a spoon, a metal spoon and you beat it up, it will have the odor it'll smell like urine, it'll have that uremia type smell that we're used to detecting. So that's another easy way if you can't wait on some of your plant pathology to detect the

creatinine level in that fluid.

- **Urinalysis**

- Gross examination for color & clarity
- Urine dipstick for biochemical testing
- Refractometer for specific gravity
- Centrifugation at 450G for 3-5 minutes to examine the sediment & supernatant

- Urine Specific Gravity (USG)
 - Use refractometer vs dipstick (dipstick upper limit 1.025-1.030)
 - Determine origin of azotemia
 - Ability to concentrate urine lost prior to azotemia, USG > 1.025 adequately concentrated in ruminants
 - Some clinically normal animals may have USG 1.005

As far as urinalysis, it's just like with any other animal for the most part, as far as we're going to visually examine it for color and clarity. We can use a urine dipstick for biochemical testing for most of the things we want to look at. For specific gravity, the dip sticks aren't very accurate and we'll talk about that a little bit more. So I like to use a refractometer for those, and then we obviously want to look at a sedimentation.

So we can centrifuge our sample three to five minutes to examine that sediment in the supernatant just to get a better idea of what may be going on. So as far as the urine specific gravity is concerned, again I prefer the

refractometer because it does have an upper limit of maybe 10:25 to 10:30. The urine specific gravity obviously is necessary because it will allow us to determine the origin, if we have azotemia present.

And remember with small ruminants, ruminants in general they're a little bit different in their ability to concentrate urine it may be as low as 10:25 is considered a concentrated urine in ruminants. So that's just a little bit different. And then we see sometimes we see specific gravity as low as 10:05 and that can be normal in a clinically normal animal. So they're a little bit different, we need to make sure we have an accurate urine specific gravity. But we also have to remember how to interpret that, a little bit different.

● Biochemical Tests for Urinalysis

- pH
- Protein
- Glucose
- Ketones
- Occult blood
- Bilirubin
- Urobilign
- Nitrites
- Urine Specific Gravity

So some of the biochemical tests, and we'll go through each one of these. We're going to start with the pH.

- Biochemical Tests for Urinalysis (dipstick)

- pH
 - Normally alkaline (7.5-8.5)
- Protein
 - Alkaline urine of SRs can falsely elevate protein to 1+ to 2+
 - Other tests (sulfosalicylic acid turbidity test or colorimetric assays) would need to be performed to definitively determine if true elevation in protein
 - Post-renal proteinuria: cystitis, urethritis
 - Pre-renal proteinuria: intravascular hemolysis and myoglobin
 - Proteinuria present in neonatal lambs & kids until 2 days of age (renal permeability to colostral proteins)

So normally small ruminants have an alkaline pH 7 and 1/2 to 8 and 1/2 is not unusual, and that's because the herbivores they're on a protein diet, all of those things can increase the pH and so that 7 and 1/2 to 8 and 1/2 is not unusual to see.

If we're looking for the presence of protein in the urine. So when an alkaline urine of small ruminants, they can have a falsely elevated protein at 1 plus 2 plus. Now if you see a 3 plus or greater, then that's real, we need to be worried about that. But if you see everything else on your urinalysis is normal and you have a 1 plus or 2 plus protein and it's on that dipstick, it's probably not a

whole lot to worry about.

There are some other tests that we could perform for protein detection, but again that's probably more of a referral type situation where we would utilize those types of tests. But post renal protein urea we would see in cases of cystitis or urethritis, and then prerenal proteinuria if we have intravascular hemolysis in myoglobin. One important thing to remember if we're examining neonates is that proteinuria is present in lambs and those young kids until two days of age. And that's just a renal permeability to plasma proteins, so that's normal in those young animals.

- **Biochemical Tests for Urinalysis (dipstick)**

- Urine Glucose
 - Normally negative
 - Renal threshold 100-140 mg/dL (may be as low as 81 mg/dL)
 - Causes: *Clostridium perfringens* type D enterotoxemia, corticosteroid, xylazine, or dextrose administration; less common causes include stress and renal tubular disease

As far as urine glucose, usually that should be negative. The renal threshold for these guys is 100 to 140. It may be as low as 80 milligrams per deciliter, but usually that 100 to 140 milligrams a deciliter is a pretty good number. Some of the calls is what could cause us to see glucose in the urine. So *Clostridium perfringens* type D enterotoxemia, corticosteroid use, xylazine or dex administration and then less common causes would be stress in renal tubular disease. But some things to think about if we see glucose in the urine.

- **Biochemical Tests for Urinalysis (dipstick)**

- Urine Ketones

- Detect excessive fat metabolism (pregnancy toxemia, starvation)
- BEST test for diagnosing pregnancy toxemia in SRs
- Urine ketone strips do not detect β hydroxybutyrate (primary ketone produced)
 - only detects acetoacetate & acetone
 - false negatives may occur due to volatility of ketone bodies

As far as urine ketones, it is used most commonly for pregnancy toxemia in the small ruminants, but it helps us detect excessive fat metabolism against starvation cases or pregnancy toxemia is when we would use that most likely. Remember that the urine ketone strips do not detect beta hydroxybutyrate, which is the primary ketone that is produced in ruminants, it only detects the Co acetate in the acetone. So you may have some false negatives just due to that fact.

If we're wanting to measure beta hydroxybutyrate, there is a blood ketone that is available and it can be used to help measure beta hydroxybutyrate. We use

that more commonly when we suspect pregnancy toxemia. Excuse me. So urine ketones are very good for diagnosing pregnancy toxemia, but we can also use a blood test to detect for better hydroxybutyrate for that.

- **Biochemical Tests for Urinalysis (dipstick)**

- Bilirubinuria
 - May be result of hemolytic disease, hepatic insufficiency, biliary obstruction
- Urobilinogen, nitrates, and Urine SG (via dipstick) → not considered diagnostic
- Urine Sediment Examination
 - Presence of cells, bacteria, casts, crystals, other debris

As far as bilirubinuria, we see that as a result of hemolytic disease or hepatic insufficiency or biliary obstruction, which again those are not very common in small ruminants and we don't see that very often.

The others that are, excuse the, other biochemical tests that are available on the urine dipstick-- those being urobilinogen, nitrates, and specific gravity, as I mentioned previously, we're not considering those to be diagnostic. And so if we have concerns, we would probably want to use other means of testing that rather than the dipstick. And then of course, as we mentioned previously, the urine sedimentation exam is very, very important that helps us determine if we have crystals present, if there's bacteria, what cells are

present if we're worried about some type of neoplasia. Oftentimes we'll get some sloughing of those neoplastic cells, and we can see those individual cells in our sedimentation.

- **Biochemical Tests for Urinalysis (dipstick)**

- Occult Blood
 - Indicates presence of hemoglobin, myoglobin, or whole blood
 - Color alone cannot differentiate
 - Centrifuge & examine sediment
 - supernatant loses pigmentation: hematuria (hemorrhage, inflammatory)
 - supernatant remains red/brown & no sediment: hemoglobinuria, myoglobinuria
 - Hypophosphatemic hemoglobinuria (*Brassica* sp.), postparturient hemoglobinuria, cold water isoerythrolysis

If we are looking at occult blood that would not indicate the presence of hemoglobin, myoglobin or whole blood which oftentimes with urolithiasis we're going to see that B positive because of the irritation to the bladder into the urethra. With color alone, we know we cannot differentiate whether it's hemoglobinuria or myoglobinuria so we can do a centrifuge and examine the sediment that's present. If that supernatant after centrifugation loses its pigmentation, that's an indication of hematuria and so we want to investigate that further.

We have the supernatant remains red or brown and

there's no sediment present, then that's more of an indication of hemoglobinuria or myoglobinuria. Again, which would lead us into different directions as far as our diagnostics are concerned and our treatment options. So hypophosphatemic hemoglobinuria, I have seen some cases of that in small ruminants.

Postparturient hemoglobinuria specifically and then you can also see it with cold water ice over urethrolisis, we just get lysis of those red blood cells and the same is obviously the case with the postparturient hemoglobinuria.

But just some helpful hints to how you can handle those urine samples that may appear to be offloaded or if you think you have blood in the urine, spin it down just make sure you don't have a hemoglobinuria or myoglobinuria.

- **Hematuria**
 - Urolithiasis, cystitis, pyelonephritis, enzootic hematuria, etc
- **Hemoglobinuria**
 - Leptospirosis, bacillary hemoglobinuria, copper toxicity, post-parturient hemoglobinuria
- **Myoglobinuria**
 - Toxic plants (*Cassia* plants)

As far as discolored urine as we just mentioned with hematuria, urethrolisis, cystitis, pyelonephritis and then the enzootic hematuria would be diseases that we would see hematuria with.

With hemoglobinuria, leptospirosis is probably the biggest one that jumps up on the top of our differential list followed by bacillary hemoglobinuria, some infections causes copper toxicity seen that quite a bit unfortunately with just some mixing your own minerals where we had animals that came in. And the first thing that owners noticed was like yeah, maybe the is a little bit darker than usual, but their urine looks really, really

weird, this dark brown color. And then again, I've seen a few cases of postpartum hemoglobinuria.

And then myoglobinuria, we usually associate that with toxic plants. And Decaisneas are the species that we think about more commonly. They're not located everywhere, but if there's a temperate or subtropical environment, those types of plants can grow there are some ornamentals as well. So you just never know. And unfortunately, small room is just about anything so sometimes toxic plants need to be examined as well.

- Renal Function

- BUN – 10-30 mg/dL
- Creatinine - < 2 mg/dL

- Creatinine may increase more quickly than BUN
 - Ruminants recycle urea through rumen

As far as our CBC chemistry, we're thinking about renal function our BUN and creatinine are our standard tests that we're going to look at. The BUN should be 10 to 30 milligrams per deciliter and the creatinine should be less than 2 milligrams per deciliter. And I really focus more on the creatinine as a better indicator, because it increases more quickly than the BUN does when there's renal disease in ruminants. And that's because ruminants recycle urea through the rumen, and so we'll see that BUN just doesn't get as high or get high as quickly in ruminants.

Obstructive Urolithiasis



All right so now we're going to move on to obstructive urolithiasis. This is definitely one of the major diseases other than parasites in small ruminants that we see and saw the most of--

- **Debilitating/life-threatening**
 - Recognize & treat early

And one thing we want to remember is that this is truly debilitating and a life threatening disease. If an animal that has a urinary obstruction, if it's not recognized early and treated early, obviously can have disastrous outcome. And so I would always try to remind our small ruminant owners, particularly those that have castrated males what to look for. What are the signs, and what we can do to prevent it?

So some good education for clients just so they know what to look for, if they see some of those subtle early signs they need to give us a call and let us talk them through it and of course recognizing it early so that we can begin treatment early on.

● Pathophysiology

- Disease of males, especially castrated males
 - Decreased diameter of urethra
 - Preputial attachment still intact
- One large stone to many small, sand-like stones
 - Depends on composition of calculi
- Most calculi form readily in alkaline urine
 - Herbivores
 - High protein diets
 - Urinary tract infection
- **Single most important factor = decreased water intake → dehydration**

So as I mentioned, this is primarily a disease of males, especially castrated males and that's because of the decreased diameter of the urethra again without the influence of testosterone, we see a decrease in the diameter of urethra.

And there's been some pretty good studies that have looked at that and delaying castration, instead of early castration I think one study was at two weeks they delayed it to three months and that increased the urethral diameter at the sigmoid flexure, that particular location about 2 and 1/2 times, so it was 2 and 1/2 times as large.

And then that same study, they looked at it delaying it from two weeks and then to five months, it actually increased the diameter by 3.5 times. So I know in other species early castration doesn't seem to have an effect on urethral diameter, but the studies would suggest that it definitely has an impact in small ruminants. Another reason to delay castration is that if they're castrated early, that preputial attachment will still be intact.

Again, the influence of testosterone in the animals that are intact allows that prepuce to break down, they can actually extend the penis and so we went back to our physical examination about examining the free portion of the penis and the prepuce, well that's very, very difficult if not impossible to do in males that have been castrated early. So we want to some of our recommendations and we'll get to that a little bit later on, would be to delay castration in those animals.

And so we can see anything from one large stone to many small sand-like stones and here in the Southeast where we're most likely to see struvite stones that's typically what we see is a small sand-like stones. But it really depends on the composition of the calculi and you can submit those for analysis, I believe it's the University of Minnesota that's still doing that. But again, most of our calculi form readily in an alkaline

urine, which we definitely have it with a pH of 7 and 1/2 to 8 and 1/2 in our small ruminants, because they're herbivores. A lot of these animals, particularly our pets and our show animals are high protein diets, and then if they have urinary tract infections that also predisposes them as well.

But the single most important factor is decreased water intake and that leads to dehydration, and that is what predisposes these animals to urolithiasis because all of our small ruminants are herbivores we have a lot of them that are on high protein diets. So what's the difference? What predisposes some animals won't get obstructive urolithiasis and others will well it's those episodes of dehydration. And those episodes of dehydration may be that the animals gone to a show and had a long trailer ride and it was a hot day or they didn't like the taste of the water oftentimes I'll hear owners refer to that.

They just didn't like the taste of the heavily fluorinated water versus what they have normally at the farm. Or it may be due secondary to another disease. If there's a respiratory disease going on and the animals aren't eating and drinking like they normally would, that can lead to dehydration, which can then lead to urolithiasis formation. So it's not uncommon to see these animals have some history of another disease weeks prior or

trailing the animals to a show or to some event that could lead to that.

● Pathophysiology

- Most common sites of lodging
 - Urethral process
 - Sigmoid flexure

- Three potential outcomes
 1. Urethral obstruction – partial or complete
 2. Urethral rupture
 3. Bladder rupture

- Prolonged partial obstruction → hydroureter, hydronephrosis, bladder wall damage, urethral strictures

So the most common sites of the lodging of the urolith or at the urethral process, if you'll remember that's the little tip on the vermiform appendage or the urethral process is that the small, narrow opening at the end of the urethra in ruminants, in small ruminants this urethral process is a very, very commonplace for obstruction. And then the sigmoid flexure, again, there's a narrowing of the urethra there.

So there's three potential outcomes with obstructive urolithiasis. We can see urethral obstruction is either partial or complete, and if it's complete, we can see urethral rupture or bladder rupture. We also need to be

aware that prolonged partial obstruction can lead to hydroureter, hydronephrosis, bladder wall damage and urethral strictures. And so again, part of our physical exam is going to be making sure we identify if those are present or not with their ultrasound or with a good physical examination as well.

● Clinical Signs of Urethral Obstruction

- Restlessness
- 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
- Vocalizations
- Signs of pain – stretching out, bruxism, ↑HR & RR



Dr. Dwight Wolfe, Auburn University

So more importantly, and I think this is one of the things that is important for our producers and owners to understand on the clinical signs of urethral obstruction. Most of them are very subtle. Restlessness tells which in colic, dribbling urine or anuria. A lot of times owners may not notice that, they are in a pasture and they just may not notice the urination habits of their animals. But if they're acting unusual, if you'll tell owners look for urethral pulsations without urination.

And so I tell them to look just underneath the rectum and if it looks like it's pulsing, are they producing urine or are they not producing urine? And so that's a good

way. Now that is almost pathognomonic for urethral obstruction. If you see a male animal, male ruminant with urethral pulsations without urination, then that is obviously a urethral obstruction. You may or may not see urethral swelling at the site of obstruction, particularly if there's a tear or urethral rupture. But crystals and blood on their preputial hairs oftentimes we'll see that.

Preputial or rectal prolapse, I've had producers call and complain that their goats constipated and they are straining. Yes they are straining, and that's what leads to that preputial and rectal prolapse, with their straining to urinate. So if you get the call about a constipated male goat, then think urolithiasis. Vocalizations of these guys are painful. So the picture that's that you see here on the slide is that animal is in that saw horse type stance or stretched out. They may be grinding their teeth, you'd see an increase in heart rate and respiratory rate, they really do not want to move. They're very painful, their bladder is significantly distended, and they're very, very uncomfortable.

- Clinical Signs of Ruptured Urethra

- Ventral & preputial edema
- Aspirated fluids smell of urine when heated
- Signs or uremia
- Chronic cases – skin slough & SQ tissue slough
- Preputial adhesions



<https://www.progressivecattle.com/topics/herd-health/watch-for-unusual-health-issues-in-the-feedlot>

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So clinical signs of a urethra that is ruptured. Ventral and preputial edema that cannot find a great picture that I thought I had one of a small ruminant with this, but this picture depicts a bull but they look very similar. Just a lot of significant swelling along the ventrum of the animal. If you aspirate those fluids, it's going to smell of urine when it's heated.

The animals will oftentimes be sick if they're uremic. And then in chronic cases if it's been going on long enough, you'll actually see the skin and the subcutaneous tissue begin to slough off. Obviously it's not designed to handle urine, and so that tissue does not fair well. And then you can also see preputial adhesions as well.

- Clinical Signs of Ruptured Bladder

- Pain may subside
- Gradual abdominal distension
- Depression, anorexia
- Signs of uremia
- Large amount of fluid in abdomen

Clinical signs of a ruptured bladder. The pain may subside, and I've had producers call, the one in particular that I remember was a really, really good client. She had a goat had called because she thought he was constipated, he was straining. And I told her my worries about him being obstructed and she was supposed to come at 10 o'clock and then lunchtime rolls around and right after lunch I gave her a call back and she said Oh well he's fine now, he's completely normal.

And so we did convince her to bring him on in and sure enough, he had ruptured his bladder. And we went in

and did surgery. So even though the pain may subside, that may be actually a worsening of disease. Gradual abdominal distension is another sign that you might see just as the urine fills the abdomen. Depression and anorexia and then weakness because of the arrhythmia, and then of course large amount of fluid in the abdomen on your ultrasound. Already, yes.

- **Diagnostics**

- Clinical Pathology
 - ↑ creatinine, PCV
 - Hyponatremia, hypochloremia, +/- hyperkalemia
 - BUN normal until late (ruminants recycle urea)
 - Possible evidence of inflammatory process
- Most common & useful diagnostic → Ultrasound examination
 - Also examine kidneys for nephroliths, hydronephrosis, hydroureter
- Others: radiographs, contrast urethrography

So as far as diagnostics on clinical pathology, again we talked about the increase in creatinine and we may not see a tremendous increase in our view until late in the process. They're going to be dehydrated, increase peak packed cell volume, hyponatremia, hyperthermia. We might or might not see hyperkalemia. We need to be mindful of that if we do take them to surgery, we can see fatal arrhythmias with that as well. And then possible evidence of an inflammatory process on our CBC.

Again, I mentioned earlier that I found the ultrasound to be the most useful, I think again that's become an

extremely common in most practices to have an ultrasound, and again we're going to examine the kidneys to make sure we don't see any nephroliths or hydronephrosis or hydroureter. We can also use radiographs or contrast urethrography, but I would say 99.9% of the cases that I've managed have just been with an ultrasound. The diagnostics were sufficient with just an ultrasound.

- **Treatment**

- **EMERGENCY!**

- Avoid chronic distension of bladder, urethral rupture
- Feedlot animal → immediate harvest
- Pet/Breeding Animal → do NOT delay surgery
- Urethral rupture → prognosis for long-term survival declines
- Cystocentesis?

As far as treatment, again this is an emergency, we need to keep that in mind. We want to avoid chronic extension of the bladder or urethral rupture or bladder rupture. So we need to be mindful of getting our surgery if that's the plan done as quickly as possible. For feedlot animals, we might just go straight to immediate harvest of that animal if they're not uremic. For pet and breeding animals, which was most of the clients that I had in practice and have in practice is to do not delay surgery.

You need to make a decision as quickly as possible, diagnosis as quickly as possible, go over the treatment

options and then get started with surgery if possible. If the rupture has occurred in the urethra, the prognosis for long term survival declines and others just need to be aware of that. We can do everything possible, some will surprise us and do well, but oftentimes we'll get stricture formation around the rupture site and that stricture formation within weeks.

So owners need to be aware that we can perform surgery to remove the obstruction or alleviate the obstruction, but if the urethra is ruptured, we may be several weeks down the road and facing similar decisions. And then I mentioned whether or not to do a cystocentesis if we can't go straight to surgery to relieve that prolonged distention of the bladder, then cystocentesis may be necessary.

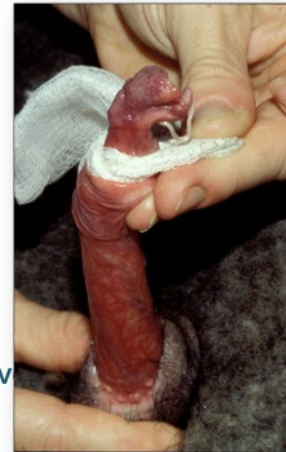
- Treatment

- Fluid support
 - Administer during surgery
 - Must have outlet for urine if surgery delayed
 - Isotonic sodium chloride
 - Especially important if hyperkalemic

So the treatment, we want to make sure that we provide fluid support though I prefer isotonic sodium fluoride during surgery, but again, you have to have an outflow of urine for the urine. So some outlet if surgery is delayed you shouldn't, these animals on fluids until there's an outlet that's going to be provided pretty quickly. And that's especially important if they're hyperkalemic and going into surgery and that hyperkalemia can lead to fatal arrhythmias.

● Treatment

- **EMERGENCY** – so don't delay surgery
- Amputation of urethral process
 - High recurrence rate
- Urethral catheterization
 - Be cautious → urethra friable
- Walpol's Solution
 - pH 4.5
 - U/S-guided cystocentesis, 30-50 mL Walpol's lavage & leave
 - Urine flow in 24-36 hours, normal voiding 3-5 days



Dr. Dwight Wolfe, Auburn University

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So treatment, as I talked about the urethral process, you can see here this is the glans, penis here and then this urethral process that more of a white tissue that's coming off there. This is one of the primary sites for obstruction. And so what we can do there, is we can actually go in and amputate the urethral process. When doing so, I would advocate for not using a pair of surgical scissors to do that, as it would crush the urethral process and effectively decrease the diameter of the urethral process.

So what I suggest doing is using a blade, sharp surgical blade. I always used a microscope slide to use to help

cut against to make that a shortcut at an angle, a very obtuse angle to make that opening as large as possible. One thing to remember that is even if that doesn't relieve the obstruction and you get urine flow, there is a high recurrence rate. So that may buy you a little bit of time but owners need to be aware that it could recur and it could require more drastic treatment.

The urethral catheterization there are a lot of, there's one institution in particular I think that does quite a bit of urethral catheterization. That's not something that I was told or something that I performed quite extensively at Auburn because that urethra is very fragile and I was always a little bit worried or cautious about rupturing the urethra, making a teared urethra. And then there's been another Walpole's solution that's been around for a long time. Some of you all that have been in practice for a while may have used it in cats, but it's this a glacial acetic acid with a pH of 4.5.

And so the theory behind this is that, that highly acidified solution can go in and "dissolve" or minimize the stones that are present. So what you would do with that is use ultrasound guided cystocentesis, remove urine from the bladder, then infuse 30 to 50 mls of Walpole's lavage the bladder and then eventually after you've lavaged a couple of times, you would leave that Walpole's solution into the bladder, and you would

expect urine flow within 24 to 36 hours and normal voiding within three to five days.

I think that this can be a useful technique particularly when finances are a concern. If you choose your patients wisely I guess, if they're completely obstructed, I don't know that Walpole's is probably the best method. If there is a partial obstruction, or there is dribbling urine, or has been going on just a couple of days and it might be helpful. I have not seen a tremendous amount of success with that Walpole's solution, but I have seen a couple of cases that responded well, so it's another option that's out there.

● Treatment

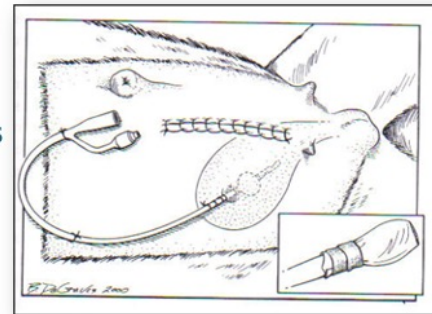
- Salvage (Feedlot Animal)
 - Urethrostomy at site of obstruction
 - Penile amputation
 - Temporary → ***Stricture formation will occur in weeks to months following surgery***

- Breeding or Pet Animal
 - Tube cystotomy
 - Bladder marsupialization

Again, as far as surgical treatment for feedlot animals for salvage procedures, we can perform our urethrostomy at the site of the obstruction, we could also perform a penile amputation. But again, the reason these are salvage procedures is because a stricture will form within weeks to months following the surgery. So it's not a long term success for the breeding or the pet animal. For those animals, the breeding or the pet goat or sheep, we would recommend a tube cystotomy or a bladder marsupialization.

● Treatment

- Tube Cystotomy
 - Foley (16-24 Fr.) into bladder & exit catheter through ventral abdomen
 - Clamp catheter ~day 4
 - Normal urination for 1-2 days before remove
 - Must be in at least 7 days
 - Antibiotics, anti-inflammatories, urinary acidifiers
 - \$\$\$\$



Sheep and Goat Medicine, DG Pugh, 2nd ed

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With the tube cystotomy what we're going to do here, and I think this picture is a good depiction of what that looks like, we're going to place a 16 to 24 French Foley catheter into the bladder and make an incision through into the abdomen and get to the bladder. We're going to make an incision into the bladder and exit that catheter through the ventral abdomen.

So you can see there we're going to put the catheter into the bladder, we'll inflate that cuff with saline and then we'll do a purse string around the catheter to hold that in place. And then pleat the metal around the bladder in the catheter that helps seal that bladder,

and then we're taking the catheter through the body wall and then taking it down with the Chinese finger trap and just suturing that in place.

Again the theory behind this is that it allows for evacuation of urine from the bladder, but it allows that urethra time to heal. So in the meantime, we may be giving antispasmodics such Acepromazine, we're going to be acidifying the urine, it gives that urethra time to rest and heal. We can start clamping that catheter off at about day 4 and just give it as a test. If the animal is able to avoid urine after day four or five, six, then we can remove the catheter on day seven or eight.

We want to leave the catheter in for at least seven days to have good healing around the bladder site and where the catheter was in place, but we need to make sure we have normal urination for at least one to two days with that catheter clamped off before we remove that catheter. With antibiotics, most of the time these animals will have a fairly significant urinary tract infection and so they'll need some antibiotics. They'll need some anti-inflammatories and then urinary acidifiers.

Urinary acidifiers, ammonium chloride is probably the one that's most commonly used, we're aiming at a pH of around 5 and 1/2 to 6 and 1/2 of that. This is a more expensive procedure, probably more because of

hospitalization and in treatment while in hospital, it's an easy procedure to perform. But making sure that you use a belly band or an e-collar or something to keep the animal from chewing on their catheter or trying to pull it out is also something that's very important to remember.

- **Bladder Marsupialization**

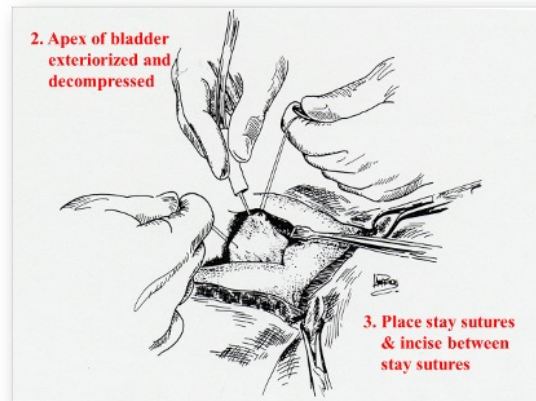
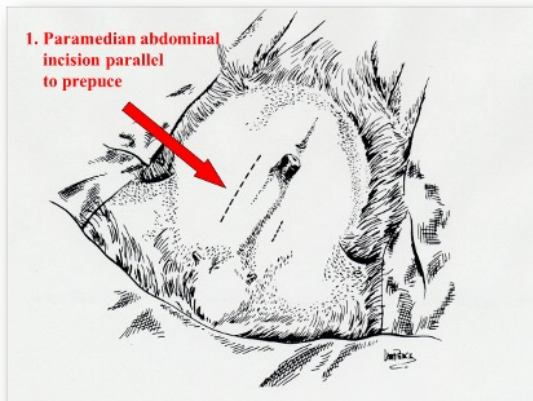
- Marsupialize bladder to external body wall
- Primary procedure or secondary to failed tube cystotomy
- Significant concerns regarding urine scald of skin surrounding the marsupialization site

Bladder marsupialization is another procedure that can be performed where it's basically just as the name describes, you're marsupializing the bladder to the external body wall. It can be a primary procedure or it can be a secondary procedure due to a failed tube cystotomy. One thing that I think before even considering a bladder marsupialization is consultation with the owners regarding your installed and care of the skin surrounding the marsupialization site.

This is not a procedure where the animal can then just go back into the pasture and not seen for days or weeks at a time these animals require daily care to

replace some type of lanolin based or zinc oxide based ointment around that marsupialization site, and oftentimes you still get urine smell. So that's one thing that it's a less expensive surgery because typically, the animals can go home within 12 to 24 hours, but that urine scalding I've seen some animals that were euthanized secondary to this procedure, but it was six months out and it was just severe skin infections due to urine smell.

● Bladder Marsupialization



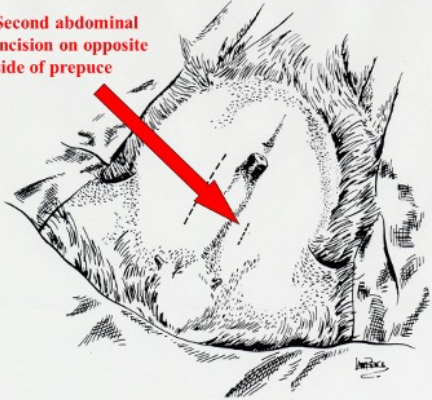
Techniques in Large Animal Surgery, Turner and McIlwraith, 2nd edition

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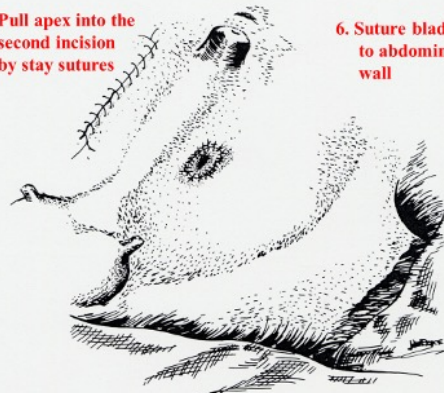
So the bladder marsupialization procedure those where you go in make a paramedian incision that's parallel to the prepuce. You go in, you open the bladder, remove the urine of the bladder, remove the stones that are present, and then suture that up. Place and stay sutures while you're doing that,

● Bladder Marsupialization

4. Second abdominal incision on opposite side of prepuce



5. Pull apex into the second incision by stay sutures



6. Suture bladder to abdominal wall

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you make a second incision that's all on the opposite side of the prepuce from the original incision, and then you use your stay sutures to pull the apex of the bladder into that second incision. You then, again that incision that was made, use that to exteriorize the bladder to the abdominal wall.

So your suturing bladder mucosa to the skin, and bladder serosa to the muscle layer of the skin. So you'll have a peak water mucosa, that is exposed to the outside environment. And a lot of questions I get about the bladder marsupialization are don't you see severe cystitis because this is exposed, or you really don't see

a lot of cystitis and I think that's because the urine that's going through that marsupialization site kind of keeps all the dirt and debris clear of the bladder.

Probably the number one issue other than severe urine scald and significant skin lesions that we would see with the bladder marsupialization is closure or structuring of that marsupialization site. So making sure you have adequate size, I would say at least 3/4 of an inch and a small animal up to an inch more marsupialization site in most of our small ruminants helps prevent that stricture formation.

- Prevention

- Females as pets
- Delay castration as long as possible (at least until puberty)
- Increase water intake
- Avoid excess protein (grain supplements, legume hay) in pets
- Ca:P ration of 1.5-2:1
- Urinary acidifiers – monitor pH

All right, so now how do we prevent it?

Recommending female small ruminants as pets, that we just don't see urolithiasis in our females. So and then delaying castration as long as possible, at least until puberty. And I've joked with owners about how do you know when it's the right time to castrate your male goat? And I usually tell them, when you can't stand the smell of them. So that's how I know they've definitely reached puberty.

Goats and sheep or very precocious and we'll have some mounting behavior and things like that at a very young age, but until again that odor, until they're five

probably 5 to six months of age, is when we would recommend castration. Increasing water intake if you know that you're going on a trip. I have some show owners that will actually transport their own water from the farm to shows, they'll flavor their water with Gatorade or Kool-Aid those types of things. Anything they can do to entice the animal to drink, and then maintaining a calcium phosphorus ratio of 1 and 1/2 to 2 to one.

We know the importance of that calcium to phosphorus ratio, diets that are higher in phosphorus tend to lead to more urolith formations. And one of the things to pet goat owners tend to be really good about following our recommendations and feeding what's in a bag, some of our show goat owners get a little bit more creative with their diets, and may add some things into what was already a balanced diet. And so we've got to be very cautious with that, we can take a complete and balanced diet and then by adding supplements and other things to it really get our calcium phosphorus ratios out of balance. So making sure we pay attention to that is very, very important.

And then urinary acidifiers, some of the sodium chlorides and ammonium fluorides have been used to acidify the urine. We do know that those decrease in efficacy over time. And so some people will

recommend own for two weeks all for two weeks that type of thing, we're just simply monitoring the pH strips for the urine for owners to monitor urine pH can also be used.

Ulcerative Proctitis/Vulvitis



So now we're going to shift gears over again urolithiasis is what I consider to be the most common urinary tract disease in small ruminants to some of the other things that you may see commonly in practice.

- **Other names**
 - Pizzle rot, sheath rot, enzootic posthitis
- **Causative Agent**
 - *Corynebacterium renale*
 - Contagious

All sorts of posthitis and vulvitis, so some other names that those go by are pizzle rot, sheath rot or enzootic posthitis, and that posthitis is just an inflammation of the prepuce.

The causative agent is *Corynebacterium renale*.

● Pathophysiology

- Hydrolyzes urea to form ammonia in urine of animals on high protein diet which excrete high levels of urea → ulcerations of skin of preputial orifice or vulva
- Angoras and Merinos
 - Due to hair at preputial orifice

It is contagious, but we also know that we have to have a high protein diet involved with this infection. The urea is hydrolyzed to form ammonia in the urine of those animals that are on that high protein diet, and so then they excrete high levels of urea and that leads to the alterations of the skin and of their preputial orifice and the vulva.

We see this more commonly in angoras and merinos, and that's just because of the amount of hair fiber they have present at the preputial orifice, so that urea kind of stays closer to the skin than what we might see in other breeds.

● Clinical Signs

- Ammonia irritates skin causing maceration & ulceration
- May spread into preputial cavity
- Swelling ± preputial prolapse
- May lead to stenosis & phimosis
- Ulcerative vulvitis in females
 - Ulcers that potentially distort vulvar commissure

So again that ammonia irritates the skin that causes a maceration or ulceration of that skin in the area. It can spread into the preputial cavity, so it can extend proximal in the prepuce.

We can see swelling, and usually you'll see quite a bit of swelling in the distal prepuce. And even preputial prolapse, and then you have to treat the preputial prolapse secondary to this. And if you have significant lesions, you can see stenosis of the preputial orifice and even phimosis if it's severe. Ulcerative vulvitis can be seen in females, and so with that, I have seen some severe distortions of the vulva commissure and stricture formation just because of the lesions on the skin. And so that in that particular doe it affected her

ability to be bred. So it can be severe if it goes undetected.

- **Treatment**

- Debride and emollient antibacterial ointment
- Dry environment
- Procaine penicillin G (PPG)
- Dietary changes

- **Prognosis**

- Good if early
- Stricture if later

So treatment for this is really just cleaning these up. There are a lot of fiber here around their preputial orifice. We want to clean that up, shave that off, debride the necrotized skin there. And apply an emollient antibacterial ointment. Keep that again as dry as possible. That means changing out shavings frequently, again shaving the hair and fiber off.

I've had good luck with procaine penicillin in and these animals as well, and then dietary changes. Decreasing the amount of protein in the diet as well. And the prognosis for these is really good if you catch them early. If your stricture formation and it has gone

undetected, the prognosis may decline over time for those. But again, we do see that particularly in our show lambs, where I saw that more commonly if you have a few practices in an area where there are a lot of feedlot lambs, then this may be something that you see as well, just because of the high protein diet.

Other Diseases



So some other diseases,

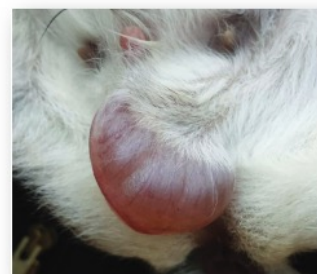
- Common congenital defect in newborn male lambs/kids

- Pathophysiology

- Failure of closure of urethra
- Genetically female intersexes (polled)

- Clinical Signs

- Visible opening on ventral aspect of penis & prepuce
- Concurrent defects
 - Diaphragmatic hernia, brachygnathia, atresia ani



Armstrong, et al. JAVMA, vol 248(10); 2016.

and I don't know if I was a magnet for these hypospadias, but it seemed like I saw quite a few of these in practice, but it is a common congenital defect in newborn male lambs and kids. So it's just a failure of the urethra to close and it can take on a lot of different looks. This picture I have is of a goat kid that we saw at Auburn that had a pretty severe hypospadias, very large, these are the supernumerary teats that you can see here, and this is the back end of the animal. And this was along the urethra, and it just dilated and filled with urine.

And we see that more commonly in intersexes. So

genetically female intersexes they'll have male genitalia and they're polled and they're bred polled bucks tend to-- the pole gene and the intersex gene are the same. And so on poled animals, we tend to see those intersexes and these aberrants of normal development and urethra and so these hypospadias cases. So what we would see is a visual opening on the urethral and the visual aspect of the penis and prepuce begin, it can occur anywhere along the length of the urethra and you can also see concurrent defects with that as well. Diaphragmatic hernia, brachygnathia, atresia in kind of just goes with what we have all been told that when you see one congenital defect, often there are more.

- **Treatment**

- Corrective surgery, if not severe
- Castration

- **Prevention**

- Do not breed polled goats

So as far as correction, what we did with this little guy, what I've done with others, there's corrective surgery. You can go in and open these up and then close the skin over if you can close the urethra, making sure you also castrate those animals as well. Oftentimes if it is severe, animals have to be euthanized. But they still, if it's not severe and you can't correct it, they make good pets. They just don't need to be bred of course, and then prevention don't breed polled goats, that's our number one. We see hypospadias most commonly in females that were bred to polled goats.

Renal Amyloidosis



Moving on, renal amyloidosis.

● Pathophysiology

- Chronic wasting disease caused by tissue deposition of fibrils formed by polymerization of protein subunits
- Protein subunits arranged in beta-pleated sheet biochemical conformation
- Reactive systemic amyloidosis
 - Sequela to chronic inflammatory or neoplastic disease – caseous lymphadenitis

We've seen a few cases of this in practice, it is a chronic wasting disease that's caused by tissue deposition of these amyloid fibrils that's formed by these protein subunits. And so this protein subunits are arranged in a beta pleated fashion as far as the confirmation is concerned, as far as reactive systemic amyloidosis we often think of that as secondary to chronic inflammation and in small ruminants cases like adenitis or CL is where we would see a lot of those cases, is after they've had a history of CL.

● Clinical Signs

- Renal failure, death
- Nephrotic-like syndrome
 - Chronic weight loss, ventral edema, ascites, pleural & pericardial edema, hypoproteinemia, & proteinuria
 - Anorexia

● Diagnosis

- Renal biopsy or necropsy – histologic evaluation of Congo red-stained renal tissue
- Amyloid deposition in glomeruli & medulla

So some of the clinical signs, renal failure. Or just maybe kind of a poor doing animal that with sudden death. Nephrotic like syndrome, chronic weight loss, ventral edema, ascites, pleural and pericardial edema hypoproteinemia or proteinuria. So all of those would maybe be an indication to us that we have some type of renal disease going on, but renal amyloidosis is something that we should put on our differential list. And of course these animals are anorexic and not feeling well.

As far as diagnosis, the only good way to get a diagnosis is to renal biopsy, which can't be performed

easily in small ruminants oral necropsy. And again looking for that amyloid tissue with the copper red stain renal tissue, and you'll see an amyloid deposition in the glomerular and the medulla on histopathology.

● Treatment

- Limited
- DMSO may prevent formation of some affecting proteins & promote solubility of fibrils (legal use)
- If disease associated with chronic infection, appropriate antibiotics
- Prognosis poor

As far as treatment of amyloidosis, it's somewhat limited. I have seen what I would regard as DMSO has been successfully used in some I would call those research cases, it helps prevent the formation of some of those proteins and promotes the solubility of those fibrils but the legal use of that in food animals is something that we try to stay away from of course. So if it's associated with chronic infection, then we'd want to treat that chronic infection if possible, but it does offer a poor prognosis. And if this is what is suspected, a renal biopsy could give you a definitive diagnosis and maybe long term prognosis for the owners to make a

decision on the animal.

Neoplasia



As far as neoplasia concerned,

- **Enzootic Hematuria**

- Pathogenesis
 - Chronic ingestion of bracken fern → hemorrhagic cystitis initially → progresses to neoplasia of epithelial, mesenchymal, and mixed cells
 - Adults grazing the plant or contaminated hay
 - all parts of plant toxic
 - toxic principal unknown
- Clinical signs
 - Hematuria, blood clots present – initially (5-8% of herd affected)
 - Weight loss, exercise intolerance, anemia, dysuria, pollakiuria
 - Papillomas develop after 1-year, invasive carcinomas in 2-6 years

we don't see a lot of neoplasias in the urinary tract of small ruminants. As far as the ones that we do see enzootic hematuria as far as that's the acute form that we see with chronic ingestion of Bracken fern, we can get a hemorrhagic cystitis initially, but then that will eventually progress to neoplasia of the epithelial mesenchymal in mixed cells. So adults that are grazing the plant or hay that is being contaminated with the plant, all parts of the plant are toxic, nobody really understands the toxic principal, but Bracken fern is very common here where I'm at in the Southeastern US, and so it's very common in goats and sheep really

like the taste of Bracken fern. So just be mindful of that, maybe that's something that you could advise owners to go and mow the Bracken fern down with the Bush hog. Just clip it off so that they're not eating too much of it for too long a period of time.

So clinical signs, hematuria with blood clots present. Initially that's the first signs that we would see. We may see as much as 5% to 8% of a herd affected. And then we'd see weight loss, exercise intolerance, anemia, dysuria and leukorrhea. And then the papillomas can develop over a year and then the invasive carcinomas or the long term effect that we see from that, so two to six years. Again, this does have to be chronic ingestion over prolonged periods of time. But if folks aren't mowing their pastures or keeping an eye on that, this is something that we could potentially say.

- **Enzootic Hematuria**

- Diagnosis
 - Clinical signs
 - Ultrasound – excessive bladder wall thickness, asymmetry
- Prevention
 - Reducing or limiting bracken fern in diet

So our diagnosis is based on our clinical signs and obviously with our ultrasound, we can see excessive bladder wall thickness, asymmetry and that would be an indication of that invasive carcinoma and prevention again just limiting the Bracken fern that's in the diet.



Thank you for choosing Vetcetera!

Misty A. Edmondson, DVM, MS, DACT

So all right I think that wraps it up. Hopefully I haven't gone over too long here, and I'm happy to answer any questions.

Thank you so much. It looks like we actually do not have any questions, so that was an excellent review though, thank you so much for taking time out of your lunch to give us a presentation. We really appreciate it. You're going to hop off whenever you're ready.

All right.

We'll just wrap things up with our viewers, thanks.

OK, thank you.