

Canine Red Eye: Internal Causes

Amy J. Rankin, DVM, DACVO reviews common intraocular causes for a dog to present to a veterinarian with the complaint of a “red eye,” including glaucoma, anterior uveitis, and hyphema.

Speaker Bio:

Amy J. Rankin, DVM, DACVO received her veterinary degree from the University of Wisconsin in 1993. She then completed a rotating internship in small animal medicine and surgery at Oklahoma State University. Dr. Rankin spent 3 years in small animal private practice in Washington and Idaho, and then she completed an ocular pathology fellowship at the University of Wisconsin’s School of Veterinary Medicine. She completed her residency training and master’s degree at Purdue University and then spent 6 years in a private specialty practice in Milwaukee, Wisconsin before joining the faculty at Kansas State University in August of 2007. Dr. Rankin is currently a Professor of Ophthalmology at Kansas State University.

Learning Objectives:

1. Recognize the clinical signs of glaucoma and anterior uveitis
2. Differentiate between acute versus chronic glaucoma cases, identify common causes of secondary glaucoma, and select relevant treatment plans
3. Recall elements of a diagnostic work up and treatment plan for anterior uveitis cases

Canine Red Eye: Internal Causes



Amy J. Rankin, DVM, MS, DACVO

Hi. My name is Amy Rankin, and I'm a professor of Ophthalmology at Kansas State University. And today I'm going to talk to you about diseases that occur on the inside of the eye that cause a red eye in your canine patients.

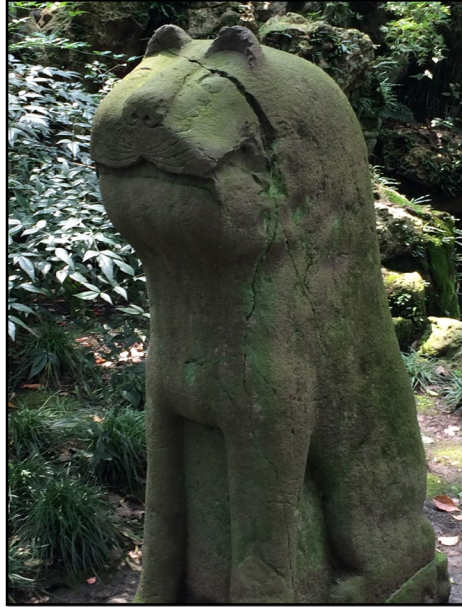
Overview

- **Glaucoma**
 - Episcleral injection
- **Anterior uveitis**
 - Episcleral injection
- **Hyphema**
 - Blood in the anterior chamber



We're really going to concentrate on three different diseases today. We're going to talk about glaucoma and anterior uveitis. These are two diseases that have episcleral injection as the form of redness. The slide on the right is a good example of episcleral injection. We've got these large caliber, really big, straight blood vessels that occur like spokes on a wheel radiating from the limbus towards the back of the eye. And then at the end of the lecture, we're going to talk about hyphema, which is actually blood accumulating in the front portion of the eye, or in the anterior chamber.

Glaucoma



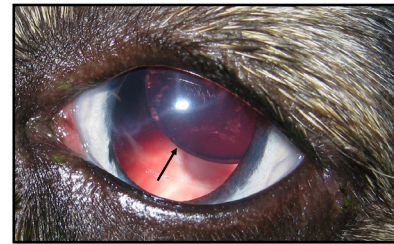
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So we're going to start the talk today by talking about glaucoma. Glaucoma is not just a single disease. It's actually a group of diseases. And the thing that all of these diseases have in common is that the intraocular pressure is too high for the optic nerve in the retina to function normally. I think it's really important when we have a discussion with an owner, after we've diagnosed glaucoma, that we really set their expectations where they should be. So for example, when we talk about primary glaucoma, which is an inherited bilateral form of glaucoma, almost in every case, we're going to lose the battle. Not only in one eye, but probably in both eyes. Hopefully, we're going to be able to prolong the time that that patient is going to be visual and comfortable. But we know eventually that our medical and surgical therapies are going to fail. But I think if you have that discussion with an owner right from the very beginning after your diagnosis this condition, their expectations are going to be right where they should be. I've had so many owners tell me, when the second I develop glaucoma, they'll say, well, Dr. Rankin, you told me this was going to happen, so I'm already prepared. I'm already prepared and I kind of know what chronic or palliative procedure I want to have performed to my patients. So again, I think it's important to, sort of, rip the Band-Aid off on that very first visit and have that conversation with an owner.

Glaucoma

- **Ophthalmic findings**
 - Red eye (episcleral injection)
 - Corneal edema (blue)
 - Mydriasis
 - Lens subluxation/luxation
 - Painful!!!! (blepharospasm)
 - Buphthalmia
 - Retinal and optic nerve changes
 - Decreased vision



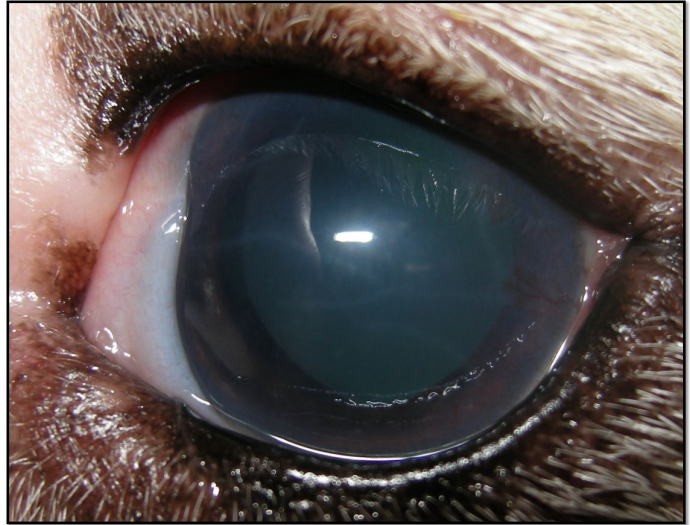
So how do we diagnose glaucoma? Of course, we all know that's by measuring the intraocular pressure. So having a pressure above the normal range. So again, normal range for most dogs is between 15 and 25 millimeters of mercury. There can be times, though, where you can still suspect that a dog has glaucoma and we can have a normal, or maybe even a lower than normal, intraocular pressure reading. Because oftentimes, at least initially, that pressure will be kind of waxing and waning and spiking at different times. So it might be that you're catching it when it's more in the normal range or a little bit lower. Other ophthalmic findings that we can see with glaucoma besides having an elevated intraocular pressure, of course, is episcleral injection, or a red eye. We can see a haziness to the cornea-- so we can see corneal edema. That happens because the pressure is so high, it prevents the endothelium from functioning properly. The endothelium-- its only job is to pump fluid out of the cornea. So when the pressure is high enough to cause it not to function, we're going to get a buildup of fluid, and we're going to get that kind of hazy coloration to the cornea.

We can see a dilated pupil. That happens because the constrictor muscle in the iris is paralyzed when the pressure is too high. If the eye is buphthalmic, or larger than normal, it's been stretched. Now we can see a lens subluxation or a luxation-- and we've got two examples of that on the photographs on this slide. I've got the arrow pointing towards the edge, or the equator, of the lens. In both eyes, the lens is subluxated, so it's only partially out of place. We can also see retina and optic nerve changes with chronic glaucoma. And, of course, decreased vision when we're talking about the second eye. Most of the time, when owners come in with a case of glaucoma, especially primary glaucoma, the first eye is already, probably, going to be lost to glaucoma. Because they're not going to notice all of these subtle signs. And that's why it's important on that very first visit when we're dealing with one eye that has glaucoma or when talking about

the risk of it happening on the other side, that we kind of go over some of these clinical signs so that they know what to watch for. So they know when to call you earlier to bring them in.

Glaucoma

- Acute vs. chronic
- Primary vs. secondary



When we're faced with a case of glaucoma, they are, kind of, two sort of important decisions that we need to make-- is this acute or chronic glaucoma? And is it primary or secondary glaucoma? Our treatment is going to be very different for kind of all four of these scenarios.

Acute Glaucoma

- Potential for vision



Canine Red Eye: Internal Causes

So, for me, acute glaucoma really has nothing to do with the timing of it. If you read in certain review articles or in textbooks, they talk about it being less than 24 hours or less than 72 hours in duration. For me, it's really all focused on, does this dog have the potential for vision? So if I think that they're able to - either we can preserve the vision that they have if they're already visual or if I think this happened recently enough, meaning I don't see any chronic changes inside the eye, not necessarily relying on the owner's history. But if I think that we can regain vision, I'm going to treat them really aggressively. The patient in the photograph on this slide actually has a fairly good prognosis for regaining vision. Not keeping it forever, don't get me wrong about that. But regaining vision for a period of time. Because when I look at the left eye, he's got a dilated pupil, a little bit of corneal edema. We've got some pain. Our third eyelid is elevated a little bit. But what I don't see are changes that fit with chronic glaucoma, and we'll go over those on the next slide.

Chronic Glaucoma

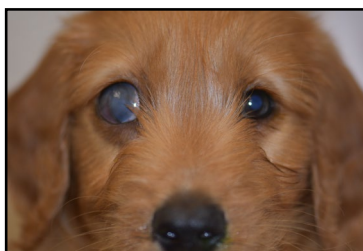
- **The 3 B's**

- Big (buphthalmic)
- Blue
- Blind

- **Duration**

- **Treatment options**

- Palliative procedures



So signs of chronic glaucoma-- I like to think of it as the three B's. So it's big, blue, and blind. So when that pressure has been elevated for a long enough period of time, it's going to cause the eye to stretch. It's going to be bigger than normal. And both of the dogs on this slide have their right eye that's buphthalmic. Usually, when the eye is buphthalmic, that pressure has been high enough to stretch the sclera, which is really pretty tough tissue. Usually, the animal's going to be permanently blind because they've got damage to their retina and optic nerve. There's exceptions to every rule, though. So if you've got a buphthalmic eye in front of you, and you're getting a positive menace response and you're confident that you're doing it correctly, you're not touching their face or putting too much wind towards their eye that they're feeling it with that, then I would still treat them aggressively. Shar Pei's-- Shar Pei's are kind of abnormal. Let's face it, right? Everything is kind of stretchy about them. Their skin, but the same thing is true of their sclera. Sometimes they can become buphthalmic and still retain vision. Puppies and kittens are the same way. Their whole body is, kind of, a little bit more malleable. So they may be buphthalmic and still have some vision.

Really, when we're talking about chronic glaucoma-- so if we've got a blind, buphthalmic eye, our treatment options are going to be really radically different than acute glaucoma. Doesn't mean that we can't start medical therapy on them, at least to buy us some time before we talk about the different palliative procedures that we can perform on that patient. Because our goal is really shifted. For acute glaucoma, my goal is to regain and maintain vision for as long as possible. My goal with chronic glaucoma, is actually shifted away from vision, because that's already-- that ship has sailed. My goal, then, is to make that patient comfortable. In the bottom right hand photograph, I've got a little arrow drawn towards these little striations, or white lines in the cornea, and that can happen with chronic glaucoma. It's called Haab's

striae. It's actually little breaks in Descemet's membrane. You can think of it kind of like a stretch mark on your skin. So when the eye is becoming stretched, Descemet's membrane kind of splits, and we get these little lines of scar tissue present.

Primary Glaucoma

- Abnormal iridocorneal angle

- Breeds

- Cocker Spaniel
- Basset Hound
- Beagle
- Boston Terrier
- Many more....

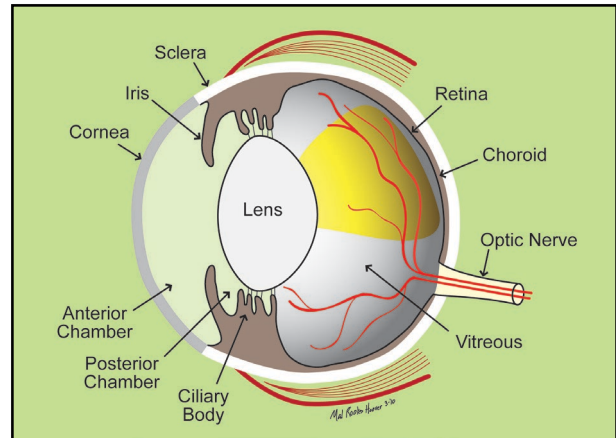


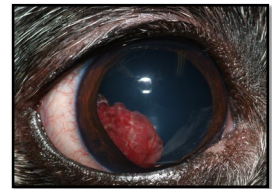
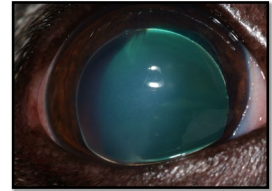
Figure 1

So acute versus chronic. Again, acute is really focused on, do we think that animal has the possibility of retaining or regaining vision? Chronic glaucoma is a permanently blind eye. Now we need to talk about primary versus secondary glaucoma. Primary glaucoma is a breed-related, inherited abnormality of the iridocorneal angle. If you remember back to vet school, aqueous humor is made by the ciliary body, flows through the pupil and into the anterior chamber, and it leaves either one of two routes-- either through the iridocorneal angle, that little formation between the cornea and the base of the iris, or it gets absorbed into the uveal tissue, into the iris and the ciliary body. And it flows out through the nonconventional pathway, into the systemic vasculature. The important thing to keep in mind when we're diagnosing primary glaucoma is that, it's going to be breed-related, and the list is really long. I think we're up to over 51 different breeds that have primary glaucoma as one of their problems. And depending where you practice in the country, you're going to see certain breeds more often than other breeds. Cocker spaniels are kind of the poster child for primary glaucoma in the United States, but we see it in a lot of other breeds. We see them bassets, we see it in labradors, siberian huskies, all different types of terriers. The important thing to keep in mind is that this is a bilateral disease. So when this patient comes to you, oftentimes, that very first visit one eye is going to have chronic glaucoma. Because they didn't know that their dog was going to develop glaucoma. They weren't looking for those signs that you're hopefully going to help them look for when the second eye has a problem. So our goal is going to be to treat the chronic glaucoma eye by doing something palliative to make them comfortable. And then, starting the second eye on a prophylactic medication to, hopefully, prolong the period before they develop glaucoma on that side. And again, really important on that first visit when you diagnose primary glaucoma that you have that discussion with them, that the second eye is going to

be affected at some point in the future. So primary glaucoma, again, breed-related, physical abnormality inside the eye.

Secondary Glaucoma

- Anterior lens luxation
 - Terriers
 - Border Collie, Poodle, Spaniels
- Anterior uveitis
- Neoplasia
- Hyphema



Secondary glaucoma, just as the name suggests, means it's secondary to something else. So we can have an anterior lens luxation. Got an example of that on the top, right hand side of the photographs. We got a lens sitting in front of the pupil. So fluid can't flow from the back of the eye through the pupil, because it's physically blocked by the lens and the pressure is going to be elevated. We see that primarily terriers. But, of course, if you remember from the slide before, terriers also get glaucoma so they can have both abnormalities. We can see it with severe anterior uveitis. So this usually isn't going to occur in one of those cases where, let's say, you've got a pressure of 50. You're taking a look at the eye. And you're calling in maybe somebody else in your practice to come and take a look. You're like, oh, I think this animal might have a little bit of uveitis. I think there's a little bit of flair. I'm having a hard time telling. It's usually going to be fulminant anterior uveitis that's going to cause secondary glaucoma. So if you're, kind of, second guessing yourself and you're not really sure, I'm going to put uveitis probably pretty far down on that list. We can have intraocular tumors that physically block the ear to iridocorneal angle. Or, like the picture in the center of the right-hand column, we've got a tumor behind the iris that can push the iris forward and physically close off that iridocorneal angle. That, too, will cause the pressure to go up. And hyphema-- so if we've got blood in the anterior chamber, you can imagine how that picture, kind of, right in the center-- there's not going to be a lot of the iridocorneal angle that's going to be open for aqueous humor to leave.

Diagnostic Test

● Tonometry

- Normal 15-25mmHg
- Ideally both eyes should have roughly the same IOP
- >5mmHg (or >20%) difference should make you wonder why....



So, of course, we diagnose glaucoma by measuring the intraocular pressure. Normal intraocular pressure in dogs is usually between 15 and 25 millimeters of mercury. Ideally, both eyes should have about the same intraocular pressure. If there's greater than 20% difference between the two eyes, which, for me, sometimes it's hard to do math that quickly in my head in the exam room in front of an owner. So I like to just have the number five in my mind. So if there's greater than 5 millimeters of mercury difference between the two eyes, that's usually going to make me wonder what's going on with that patient. Is one eye too high, and that's an eye that has glaucoma? Or is one eye too low, and maybe we're thinking anterior uveitis? There's lots of different tonometers that are available for us to use in our veterinary patients. I think the rebound tonometers are a little bit easier for most of us to learn on. So it's got a shorter learning curve than the applanation tonometer, like the Tono-Pen on the far left. The example on the right is one of the newer rebound tonometers. And what's really nice about that one is, it's a little bit idiot-proof, if you will. So there's a green light and a red light that tells you when you should be taking your measurements-- when you're at the right distance from the patient.

- Assess vision/potential for vision

- Menace response
- Maze test
- Tracking
- PLRs direct and consensual
- Dazzle reflex
- Fundic examination



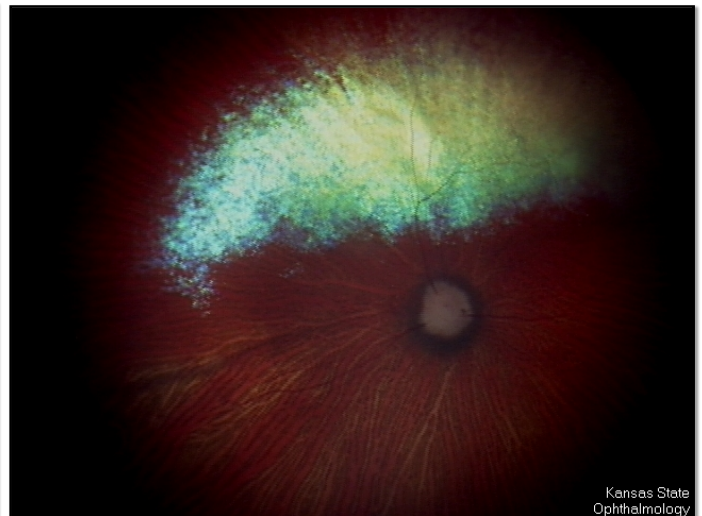
When we're looking at a patient after we've diagnosed glaucoma, it is really important for us to accurately assess vision, or maybe accurately assess the potential for vision, might be a better way of phrasing that. And there's a lot of different things that we can look at that can help us determine whether or not that animal is visual or is going to potentially be visual in the future if we can bring the pressure down. So doing the menace response. Of course, this is a learned response. Most of our glaucoma patients are not going to be really young puppies, so that's going to be OK. They should be old enough to know that they should blink when something is coming towards their face. It's really important to cover up the other eye well, otherwise, they're going to be able to see with that eye and you might get a false positive. It's also really important not to touch any of the hair on their face or blow too much air towards them. Because they can sense that or feel that, and then they're going to blink in response to that. So really you're stimulating a palpebral reflex rather than a menace response. You can set up a maze test if this is their second eye-- really difficult to do on their first eye. Every once in a while, I'll have some students or some residents that want to patch a dog's eye, their good eye, and see how they navigate a maze with the affected eye. Really difficult to do because what is a dog going to do when it gets off the table after its eye has been patched? It spends the entire time digging at the patch trying to get it off. So really only appropriate to do when it's the second eye that's involved. You can evaluate whether or not they can track an object in the room. So what I'll do is, I'll cover up the good eye and I'll drop a piece of cotton or piece of gauze-- something that doesn't have any smell to it or make a sound when it hits the ground-- and see if they can follow it with their head or with their eye. It is really important to assess their PLRs when you've diagnosed glaucoma. So we look at both the direct and the consensual PLRs in both eyes. And probably the most important PLR if I had to pick, would be the

consensual PLR from the eye that has glaucoma looking for a response in the other eye. So shining a light into that eye that has a pressure of 50-- does the message get through to the other side? If it gets through the other side, that doesn't mean that we are going to be able to regain vision, but it tells me that at least there's a chance. If we have a lack of a consensual PLR to the other side and lack of a dazzle reflex-- dazzle reflex is just shining a really bright light in the eye and they should blink-- that is a really negative prognostic indicator for our ability to regain vision. And of course doing a thorough fundic exam is really important. Because if we see chronic changes in the optic nerve or the retina, that patient is not going to be able to regain vision.

Fundic Examination



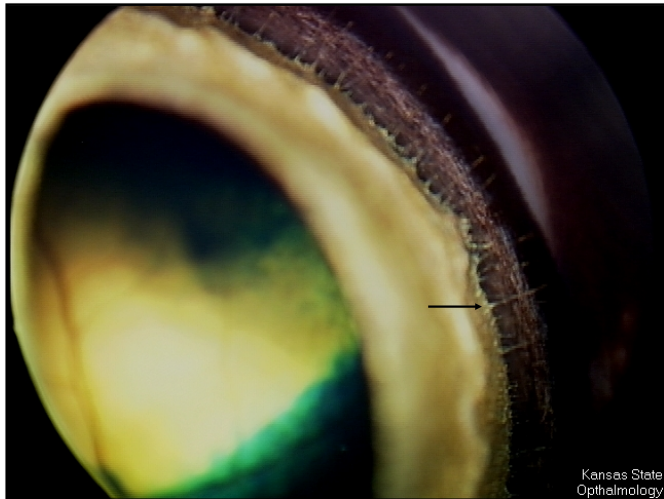
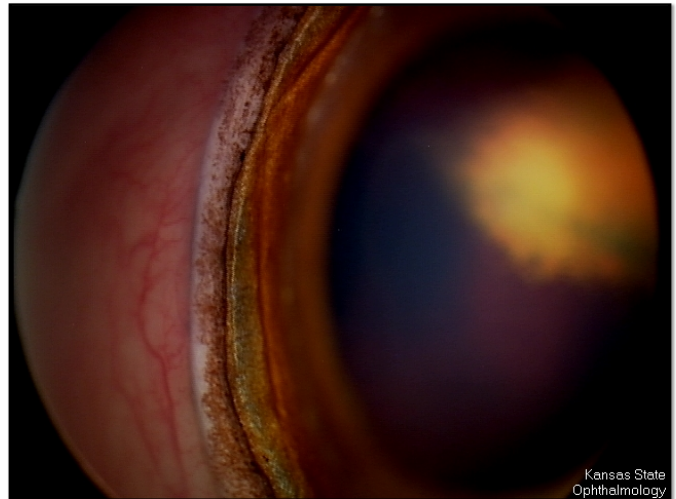
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So this is an example of a normal fundus on the left hand side of the slide. So we've got the tapetum on the top, non-tapetal portion on the bottom. We've got, kind of, a triangular-shape optic nerve. There's a lot of myelin on it. That's what's giving that, sort of, triangular shape and kind of puffier, elevated appearance to the optic nerve. The blood vessels are really robust. They go all the way to the periphery, they're branching at the end. In comparison, when we look at the side on the right, we've got to tapetal hyperreflectivity, because the retina is extremely degenerated. So the retina is very thin, which is allowing us to see the tapetum better than we should be able to see it, because we're looking through much thinner retina than normal. The blood vessels-- you can just barely make them out as they're going over the surface of the optic nerve and into the retina, but they certainly don't branch all the way to the periphery of the retina. And the optic nerve is really lost its normal characteristics. So it doesn't look, kind of, fluffy, there's a lack of myelin that's present. All of those things fit with an eye that is not going to regain vision. Even if we bring the pressure down, the optic nerve and retina have been permanently damaged.

Gonioscopy

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Gonioscopy is something that we can do if you refer a patient to an ophthalmologist. So it's not something that you'll be doing in practice. But I just wanted to kind of show you what we evaluate when we're looking at the iridocorneal angle. So when we do gonioscopy, we place this funny shape lens on the surface of the eye or on the cornea. And what we're doing is just bending the light so we can look at the angle. The picture on the left is a nice, normal angle. So we've got the iris that's the kind of yellowish, golden kind of color. And then we've got these little strands, which are pectinate ligaments. And I've got a black arrow that's pointing towards one of them. And you can see all of those different flow holes in between the pectinate ligaments where aqueous humor can easily flow out of. Verses on the right-hand side, we've kind of the opposite. We don't see any pectinate ligaments. So we see the base of the iris right up against the sclera. And we see that pigmented line, there, but we don't see any flow holes. So that's an example of a closed angle. When we talk about primary glaucoma in dogs-- in humans, there's primary open angle glaucoma, which we do see in dogs, but it's in a select group of dogs-- beagles primarily. But we rarely see that type of glaucoma in our veterinary patients. Most of them have an angle like this. So they have closed angle glaucoma.

Acute Glaucoma Treatment

● Emergency treatment

- Topical prostaglandin analogues
 - Latanoprost 0.005% (Xalatan®)
 - Travaprost 0.004% (Travatan®)
- Topical carbonic anhydrase inhibitors
 - Dorzolamide 2% (Trusopt®)
 - Brinzolamide 1% (Azopt®)
- Beta blocker
 - Timolol 0.5 & 0.25%
 - Betaxolol 0.5%



So when we talk about treatment for acute glaucoma-- again, this, for me, is reserved for patients that I think can regain vision. So they do not have a buphthalmic eye, they possibly have a positive menace response, they've got a consensual PLR from the eye with glaucoma to the other side, they've got a positive dazzle reflex, they don't have any changes inside the eye that tell me this eye is permanently blind. Then I'm going to treat that patient as aggressively as I can to bring their pressure down to see if we can regain vision. So there are a lot of different anti-glaucoma medications that are on the market for humans. All of these are human medications. No company is going to market something just for dogs, because glaucoma is not something that's going to make them a whole lot of money in our veterinary species. Depending on which ophthalmologist you work with, there might be other medications that they use, as well, which can be totally appropriate. These are the three that we tend to use here. So we use something from each of these categories. So I'll choose a topical prostaglandin analog, topical carbonic anhydrase inhibitor, and a topical beta blocker, as well, unless there's a contraindication in my patient to use these.

When we treat glaucoma, we treat it with, sort of, a polypharmacy approach. So we don't choose just one medication and sort see how things go, we're going to treat with something from each of these categories. So Latanoprost and Travaprost are the two prostaglandin analogs that have been studied in dogs. There are others that are available for humans. Latanoprost is now available as a generic. So it's relatively cost effective. It's a 2.5 ml bottle, so a very small bottle. And it used to be over \$120 per bottle. Now that it's available as a generic, we can get it for sometimes as low as \$15 or \$20.

We'll use a topical carbonic anhydrase inhibitor-- dorzolamide is what we use most commonly, because it's available as a generic. So just more cost effective for our clients. Brinzolamide recently became

available as a generic. It's still a little bit too pricey for most of my clientele here in Kansas, but I think eventually that price will come down with time. The advantage of brinzolamide is that the pH is closer to a physiologic pH, so it's a little bit better tolerated by most animals compared to dorzolamide. Usually, when an owner comes in and they're complaining about the stinging, or the fact that their dog blinks a little bit after we give dorzolamide and I tell them the cost difference between the two medications, most owners are going to change their mind. They're going to think that they're not squinting quite as much, because it's a several hundred dollars difference between the two bottles of medication. And then a topical beta blocker. So either timolol or betaxolol are the two that we use most commonly. So contraindications for these medications, for me, a topical prostaglandin analog is always contraindicated if you have an anterior lens luxation. These drugs are really potent miotics, so they're going to make the pupil smaller. And then you're going to trap that lens in the anterior chamber, causing the pressure to go up. So if you can't evaluate where the lens is sitting, if that animal has so much corneal edema that you can't see inside, I would not use a prostaglandin analog. I also avoid beta blockers in animals that have underlying cardiac disease.

Acute Glaucoma Treatment



● IV mannitol

- Used less commonly
- Osmotic agent -dehydrates vitreous
- Dosed at 1-2 grams/kg
- Administered slowly over 20-30 minutes
- Withhold water for 4 hours
- Contraindications
 - Renal disease
 - Cardiovascular disease
 - Dehydration
 - Other debilitating illness



IV mannitol or oral glycerin are hyperosmotic agents that we can give to help decrease the intraocular pressure. We don't use these as commonly anymore since the advent of the prostaglandin analogs, which have been available since the 1990s. So I only use this if the pressure doesn't come down after I give one or two rounds of latanoprost, dorzolamide, and timolol. So IV mannitol is sort of labor intensive, because you have to have the mannitol nice and warm or it's going to crystallize when it's at room temperature. You have to draw it up through a filter, place an IV catheter, and you have to administer it slowly. So if you don't have a syringe pump available, that means one of your nursing staff is tied up for 20 to 30 minutes administering mannitol slowly. It's important if you do that, that you have to withhold water for four hours. Your goal is to dehydrate the whole animal, but specifically the vitreous, which is primarily water. So you're going to remove that. So if you allow them access to water right away, they're just going to consume an entire bowl full of water, they're going to rehydrate themselves, and then the pressure is going to go back up. Usually when we give a hyperosmotic agent, our goal is just to lower the pressure, if that animal is going to respond, for probably 10 to 12 hours. No longer than that. It just buys us a little bit of time to hopefully get our medications on board to help lower the intraocular pressure. Of course, there's several contraindications to giving IV mannitol. Animals that have cardiac or renal disease, we shouldn't use this product in. And also, animals that are severely dehydrated.

After Initial ER Visit

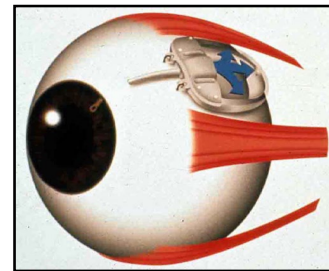


- Ophthalmologist referral
- Maintenance therapy
 - Latanoprost 0.005% q12 to 24 hr
 - Dorzolamide 2% q6 to 8 hr
 - Timolol 0.5% q12 hr
- ****Treat the “normal eye”****
 - Timolol 0.5% q12 hr
- Frequent IOP checks for monitoring of BOTH eyes
 - 1 day, 3-5 days, then weekly, monthly, q2 to 3 months

So kind of after you've diagnosed glaucoma, you've done your first round of medications, I think that's always an appropriate time to offer to an owner the possibility of referring to an ophthalmologist. Doesn't mean that they can't stay with you if that's what they choose to do, but it's a good idea to mention it. Because glaucoma is never ever a good diagnosis. After we've kind of gotten over the crisis, our maintenance therapy is going to be those same three medications. Oftentimes, we're going to use a prostaglandin analog, carbonic anhydrase inhibitor, and a beta blocker, and our frequency is really going to vary depending on how high that pressure was. So initially, I'm probably going to treat really aggressively. And if that pressure stabilizes, I'm going to kind of slowly decrease their meds, but never take them off of it with primary glaucoma. So latanoprost or a prostaglandin analog, anywhere from once to twice a day. Most of the time when they come in with an acute spike, I'm probably going to send them home with twice-a-day therapy because I don't want to err on the side of not being aggressive enough. Same thing with those dorzolamide, I'm going to treat probably four times a day initially, and then try to wean them down to three times a day. Maybe twice a day if we're lucky. And beta blockers, I usually only use at twice a day. It's really important to have that conversation with the owner when you suspect primary glaucoma. So in practice, you're not going to be able to gonioscopy to be able to diagnose primary glaucoma. But if you have a breed that primary glaucoma occurs in and there's a lack of all of those things that we talked about that cause secondary glaucoma, you should treat that case like a primary glaucoma case. Most of the time, causes of secondary glaucoma are going to be really obvious. It's going to be a gigantic intraocular tumor. It's going to be an anterior lens luxation, horrible hyphema, or really fulminant anterior uveitis. So in the absence of any of those things that can cause secondary glaucoma in a purebred dog, I think you can be fairly confident in diagnosing them with

primary glaucoma. It is really important on that very first visit to talk to them about, we need to treat the second eye, or the good eye, if you will. The "normal" eye. Normal in quotation marks, because it's not normal because we know that eventually that eye will also develop glaucoma. We know from the literature and from experience, that animals that are not placed on a prophylactic medication will develop glaucoma in that second eye within about eight months of diagnosis in the first eye. But with prophylactic therapy, with a topical beta blocker, we can delay the onset of glaucoma to a median time of 31 months. So the median time means 50% of the dogs will develop glaucoma before then, 50% of the dogs will develop glaucoma after that 31 month time frame. But 31 months is a huge portion of a dog's lifespan. Almost three years. It's important that we monitor the IOP, kind of, frequently initially. So after we diagnose glaucoma, if we don't have them hospitalized, I'm going to have that patient come back the next day, because I want to make sure that their pressure is coming down. Then I'm going to see them back a few days after that. Then weekly for a little while. Then maybe monthly. And eventually, we'll get to the point where it's every two to three months. And even if we've lost that first eye due to glaucoma, we're going to want to monitor pressure in the good eye at least every two to three months.

- Gonioimplants
- Diode laser surgery



There are also referral surgeries that can be done for visual eyes. So depending on which ophthalmologist you work with, some of us will perform laser surgery. So we're destroying part of the ciliary body, the part of the eye that makes the fluid, to lower the intraocular pressure. And we can also place gonial implants, which are actually made for humans. We tend to use the pediatric ones, they fit our patients probably the best. And we place the shunt on the outside part of the eye and we suture it to the sclera. And then we tunnel that little tube into the anterior chamber so that fluid can flow from the anterior chamber into the subconjunctival space.

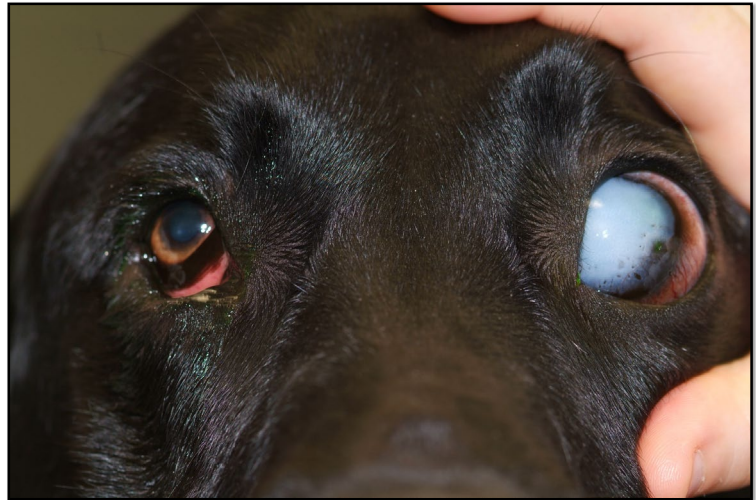
Chronic Glaucoma

- **The 3 B's**

- Big (buphthalmic)
- Blue
- Blind...Blind...Blind

- **Palliative procedures**

- Enucleation
- Evisceration
- Chemical ablation



Chronic glaucoma, our treatment is going to be very different. So for me, this is a permanently blind eye. So it's buphthalmic, lots of corneal edema, and definitely blind. So again, a negative consensual PLR from the bad side to the good eye, negative dazzle reflex, negative menace, and if I can visualize the fundus and I can see chronic changes in the optic nerve and the retina, then I can be confident that this animal is blind. And then our gears are really shifted to a palliative procedure. So either an enucleation, evisceration, or a chemical ablation are the three things that I think are really important for us to discuss with the owner. Oftentimes, when we diagnose glaucoma in our patients, sometimes owners will come in and say, you know, I don't think my dog is that uncomfortable. The eye is wide open, they're still eating well, they're still playing, he's still going for five-mile runs with me. And that's when I think that we, as veterinarians, really have to be advocates for the dog. So we have to have that conversation with the owner that, even though their dog looks like they're not in pain, we know from experience-- and you can say we as in all of us, collectively. Veterinary ophthalmologists and other practitioners that you know. You can use the collective "we." We know that from experience, after we do a palliative procedure-- whether it's an enucleation, evisceration, or a chemical ablation-- that that dog is going to feel better. I have had so many owners come back to me and say, you were right, which, trust me, that doesn't happen every day where owners tell you you were right. But they'll tell me that I was right. That their dog does feel better. They're not sleeping as much. They're more playful. They're more excited when they come home. Because we've taken away that source of chronic pain. Humans with glaucoma describe it as being a headache-like feeling. So it's like a migraine headaches. And you can't tell just by looking at somebody if they've got a headache or a migraine, but that person really doesn't feel well. And the same thing is true of our patients. It's also really important to have a

conversation with owners how these three different palliative procedures differ from each other. You know, an enucleation certainly changes the cosmetics of the dog, but they're going to be comfortable after. And that's really the goal with that procedure. An evisceration is probably a referral procedure. I always tell owners that it truly, really is just for cosmetics, and it does have a longer healing period after we do that procedure. So with an evisceration, we're removing all the intraocular contents and replacing a black silicone ball inside the globe. And a chemical ablation is injecting either gentamicin or cidofovir. Gentamicin, of course, is an antibiotic. Cidofovir is an antiviral medication inside the eye along with a small amount of a steroid to help kill the part of the eye that makes the fluid. The important part that we need to discuss with an owner before we do a chemical ablation is the cosmetic outcome is incredibly variable. Sometimes from across the room, I can't tell which eye we've injected because the eye looks so normal in size and in shape. And then in some patients, the eye can become really kind of small and shrunken or phthisical, and it can look very different from a normal eye. So we have to explain to owners that there's a lot of different ways they can look after a chemical ablation. And most of my owners that choose a chemical ablation are doing it for cost reasons. So it's less expensive, a much shorter anesthetic procedure, and they are accepting of how their pet will look after that procedure.

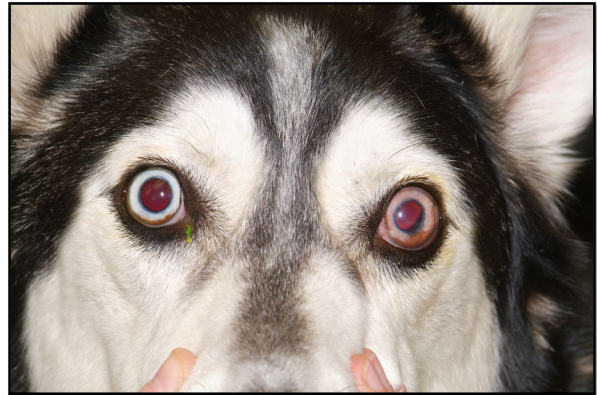
Anterior Uveitis



So now we're going to shift gears and talk about anterior uveitis, or inflammation in the front portion of the eye.

Clinical Signs of Anterior Uveitis

- Aqueous flare
- Episcleral injection/conjunctival hyperemia
- Cells in anterior chamber
 - Hypopyon/hyphema
- Fibrin in anterior chamber
- Keratic precipitates
- Miosis
- Hypotony **



So there are a lot of different clinical signs of inflammation of the iris and ciliary body. Aqueous flares pathognomonic with anterior uveitis. If you see protein content in the aqueous humor, that animal has breakdown of the blood aqueous barrier, and by definition, has anterior uveitis. Probably the best way to evaluate for aqueous flare is in a dark room using a slit beam of light. And most of us have those direct ophthalmoscopes in our exam room, you can set it to the slip beam of light and shine the light into the eye, and look at it from the side. If you can see that beam of light traversing or crossing through the anterior chamber, and you can see a haziness or an opaqueness there, that animal has aqueous flare. We can see lots of other clinical signs, as well. And we're going to go over some of those in the next couple of slides. The Husky on the photograph, here, has anterior uveitis in the left eye. You can tell he's got a pretty dramatic color change to his iris compared to his right eye.

Aqueous Flare



This is an example of really severe aqueous flare. This is a bichon that had diabetic cataracts. He's got a small miotic pupil you can just barely make out through all the aqueous flare that's present.

Red Eye

- Conjunctival hyperemia
- Episcleral injection
- Corneal vascularization
- Hyphema



You can see different types of redness. Oftentimes, they'll have conjunctival hyperemia in association with episcleral injection. You can also see corneal vascularization. You can see vessels 360 degrees around in really chronic uveitis cases. And sometimes you can have a breakdown of the blood aqueous barrier severe enough that you can develop hyphema. You can also develop hyphema from these little vascular membranes that form on the surface of the iris, and sometimes those can break open.

Hypopyon



We can see hypopyon, or purulent material in the anterior chamber. So this is just an accumulation of white blood cells, primarily neutrophils, that settle out in the bottom of the anterior chamber.

Fibrin in Anterior Chamber



We can see fibrin. I've got a little arrow that's drawn to it on this photograph. So it can look either like a white, sort of, fluffy material, cloud-like material in the anterior chamber. But sometimes it can be more kind of brown or sort of tan in color and sort of lacy in texture.

Keratic Precipitates



Canine Red Eye: Internal Causes

We can also see keratic precipitates. These are white blood cells that are adhered to the endothelium. They always adhere to the bottom portion of the cornea, so that's the best place to look for them. This is a dog that we suspected had a rupture of its lens capsule, which caused severe amount of inflammation. You can see there's a lot of keratic precipitates present. And I've got a little arrow that's drawn to that area, as well.

Miosis

- **Constricted pupil**
 - Inflammatory mediators
 - Prostaglandins
- **Iris color change**
- **Posterior synechia**



And we can see a small miotic pupil. And that's due to inflammatory mediators, primarily prostaglandins, that caused the pupil to become smaller. We can see a change in iris color. So if it's a blue-eyed animal, it's going to become kind of this yellowish or red color just like the photograph of the dog on this slide. And with chronic uveitis, a brown-eyed dog is going to become even darker brown. So it'll take on a different, sort of, appearance. We can also develop posterior synechia, which is an adherence to the iris, to the lens.

- Glaucoma....



This is an example of kind of extreme posterior synechia so iris bombe can occur when 360 degrees of the pupil is actually physically stuck down or adhered to the anterior lens capsule. And then what happens because aqueous humor can't leave through the pupil because there isn't an opening anymore. That iris is going to billow forward. That usually indicates that the animal either has secondary glaucoma already, or it's pending glaucoma. So that's a patient you're going to want to watch closely.

Anterior Uveitis Causes

- **Endogenous causes:**

- Infectious, neoplastic, toxic, metabolic, autoimmune

- **Exogenous causes:**

- Trauma, perforating corneal wounds, corneal surgery, corneal ulceration (reflex uveitis)



There are a lot of different causes of uveitis. So there are both endogenous and exogenous causes of anterior uveitis in our canine patients. Endogenous causes include infectious agents, neoplastic conditions, metabolic diseases, as well as immune-mediated or autoimmune conditions that we can see in our patients. And then we can also have external factors, like trauma, whether it's blunt force trauma, penetrating trauma, corneal ulcers, corneal surgery.

Anterior Uveitis Causes

- **Most common**
 - IDIOPATHIC!!!!

By far and away, the most common thing that you're going to diagnose as a cause of anterior uveitis in your canine patients is going to be idiopathic. That just means that we can't sort out or figure out what it is. It doesn't mean that there isn't necessarily something underlying that's causing it. It also doesn't mean just because it's the most common cause of uveitis that we shouldn't still look for causes of inflammation inside the eye.

Causes of Uveitis

● Infectious

- Bacterial
 - *Rickettsia rickettsii*
 - *Borrelia burgdorferi*
 - *Ehrlichia canis*
 - *Brucella canis*
 - Leptospirosis
 - *Bartonella* spp.
- Algal
 - *Prototheca* spp.
- Fungal
 - *Blastomyces dermatitidis*
 - *Cryptococcus neoformans*
 - *Histoplasma capsulatum*
 - *Coccidioides immitis*
- Viral
 - Canine adenovirus-Blue Eye
 - Canine distemper virus
 - Rabies

So when we talk about different things that can cause inflammation in the iris and ciliary in and dogs, again, the list is really, really long. So the infectious diseases that we're concerned about or the infectious diseases that you're going to test for are really going to be dependent on what type of diseases do you see in the area of the country that you practice in? And does that dog have any travel history to other places? So for example, when we look at the fungal list, the most common thing that we see here in Kansas would be Histoplasma. But in other parts of the country that I've practiced in-- I have been in Indiana and I've been in Wisconsin-- Blastomycosis is by far and away more common than the other types of fungal diseases. So it's really going to be dictated by where you practice in the US.

Causes of Uveitis

● Infectious

○ Parasitic

- Ocular larval migrans
 - *Toxocara canis*
 - *Baylisascaris procyonis*
- *Dirofilaria immitis*
- Onchocerciasis
- Ophthalmomyiasis interna
 - *Cuterebra*

○ Protozoal

- *Toxoplasma gondii*
- *Leishmania* spp.

Same thing when we look at parasitic and protozoal diseases. Again, it's going to be the lifestyle of the dog that we're going to be looking at, as well as where are you in the country.

Causes of Uveitis

- **Non-infectious causes**

- Hyperlipidemia
- Coagulopathy
- Vasculitis
- Diabetes mellitus (LIU)
- Hyperviscosity syndromes
- Histiocytic proliferative syndromes
- Metastatic neoplasia (lymphoma)

There are also some non-infectious causes that we should keep on the list as well. So animals that are diabetic, for example, can develop lens-induced uveitis. So their cataracts are rapidly progressing. They can leak some of the lens proteins into the inside of the eye, and that's going to set up an inflammatory cascade.

Causes of Uveitis

- **Immune mediated**
 - Lens-induced uveitis (LIU)
 - **Cataract**
 - Phacolytic uveitis
 - **Lens capsule rupture**
 - Phacoclastic uveitis
 - Lens luxation/subluxation
 - Uveodermatologic syndrome
 - **Ocular and skin lesions**
- **Neoplasia**
 - Primary ocular neoplasia
- **Miscellaneous**
 - Idiopathic
 - Pigmentary uveitis
 - **Golden Retrievers**
 - Keratitis-associated reflex uveitis
 - Scleritis
 - Radiation therapy
 - Trauma

We also have immune-mediated diseases. So again, cataracts can cause lens-induced uveitis if we have a lens capsule rupture. So that can happen from a penetrating wound, for example, a thorn, or a foreign body, or maybe a cat claw that will cause a rapid release of lens proteins. Also, animals that have rapidly developing cataracts. So for example, a diabetic that has a rapidly developing cataracts. Sometimes that lens capsule can stretch, and it can only stretch so far before it breaks. And that's going to cause a really severe form of uveitis. We've got uveodermatologic syndrome or VKH-Like syndrome that can cause inflammation inside the eye, we can have primary ocular neoplasia, we can also have metastatic tumors -lymphoma would probably be highest on the list that can cause inflammation inside the eye. And then there's a long list of miscellaneous things as well. I put pigmentary uveitis on the list. That's something that we see primarily in Golden retrievers, where they get pigment dispersion inside the eye. That can be a difficult disease to diagnose because for owners, it's really hard for them to be able to tell that that animal has iris cysts and they've got this pigment deposits on the back of the cornea and the anterior surface of the lens capsule because the dog's eye is already brown. So oftentimes they don't present until they're very late in the stage of the disease.

Uveitis Workup



- Thorough medical history
- Complete physical examination!
- CBC/chemistry panel, urinalysis
- Selected infectious dz screening
- Aspirates/impression smears
- Enucleation with histopathology
 - Diagnostic AND therapeutic in blind eye

So when we work up a case of uveitis, there's a lot of different things that we need to think about. I think taking a thorough medical history and doing a complete physical examination is really just an absolute must for any animal with uveitis. And let's face it, they've already paid for that when they've come in. And you're doing their ocular exam. There are so many things that you can learn by taking a thorough history. Finding out what is that animal used for? The indoors or the outdoor? How have they been feeling? Has there been a change recently? Are they gaining weight or are they losing weight? Travel history. Have they gone anywhere? Have they been on any medications? Maybe they've had vaccines recently? Something that they could be reacting to. So sorting out all of that is really important. And then doing a complete physical exam is important because maybe we're going to find something that we can chase down. Maybe they've got enlarged lymph nodes. Maybe they have a mass somewhere that we could aspirate and send off for cytology to be able to diagnose what's going on. Do we hear harsh lung sounds? Maybe we should really encourage taking thoracic radiographs. Maybe we feel something abnormal in the abdomen, and doing an abdominal ultrasound might be a good way to go. If an owner can afford it, I think doing a CBC, a chemistry panel, and a UA is also just a good part of our minimum database. Not every client can afford to do that, of course. And again, I think we really need to rely on our good medical history that we're going to take, as well as our physical exam to help guide our owner into doing what steps might be next. For me, cases that I really want to work up-- so for example, I want to do thoracic films, abdominal ultrasound, maybe infectious disease screening-- is really going to be based on a couple of different things. So animals that come in that don't feel well-- that's obvious. If they're systemically not normal-- they've got a fever, they haven't been eating well and the owner has other complaints-- whether it's joint pain or coughing or other symptoms-- those are cases if I can, I'm

really going to encourage that we do a good workup in. Animals that have severe uveitis. Again, I'm really going to try to push them if I can into doing a workup. Something that's going to still be within their budget and what they want to do. And of course, animals that have bilateral disease. That too is going to make me more concerned that that patient has an underlying cause that we should look for. It's also important to have that initial communication with the client that even though we do all these things-- we can take radiographs, abdominal ultrasound, we can screen for a lot of the infectious diseases that you see in your area of the country or wherever that dog has traveled to, and we still may come up empty handed. We may have negative results for all of those tests. And then I think it's important to emphasize to the owner, that's a good thing. I don't want your dog to have blastomycosis or histoplasmosis. We don't want your animal to have lymphoma when we find a lymph node that's enlarged and we take an aspirate of it. So turning it into something where yes, we're going to do all these diagnostic tests, and we honestly are hoping that they come back negative because we don't want your patient to have any of these things can set their expectations a little bit differently than if you do just the CBC chemistry panel in UA and you call some owners and you tell them everything is normal. They may be upset with you because they may be thinking that you were going to get an answer just by doing that. And honestly, most of the time when we do a CBC, chem panel, and a UA in uveitis cases, I rarely get the answer. Very, very rarely. It might give me a hint of where to go, for example, if the animal is thrombocytopenic, maybe then I'm going to focus my infectious disease workup on diseases that can cause that. But rarely is that going to give me the answer. For eyes that are permanently blind-- so say, for example, they've got fulminant uveitis, and they've developed secondary glaucoma, enucleation with histopathology can be diagnostic. Hopefully, we can figure out what's going on when the pathologist looks at that globe, but it can also make them feel better.

Anterior Uveitis Treatment



- Treat cause if known
- In most cases the cause is not known... symptomatic
- Goals of treatment
 - Decrease inflammation
 - Relieve pain
 - Prevent complications of uveitis

So how do we treat uveitis? Of course, that there's an underlying cause, we're going to direct our therapy towards that. Most of the time we're not going to know what the cause is. And certainly not initially while we're waiting for some of our tests to come back if we've done a workup. So we're going to treat them symptomatically. And our goal is to make that animal feel better, decrease the inflammation, and hopefully prevent secondary glaucoma and blindness from occurring.

Anterior Uveitis Treatment

Anti-inflammatory Drug Examples	Type	Route	Indications
Prednisolone acetate 1% Neopolydexamethasone 0.1%	Steroid	Topical	<ul style="list-style-type: none"> ACTIVE anterior uveitis Frequency dictated by severity (maximum q4 hours)
Flurbiprofen 0.03% Diclofenac 0.1% Ketorolac 0.5% Bromfenac 0.09%	NSAID	Topical	<ul style="list-style-type: none"> Prophylactic (prevent cataract-induced uveitis) Synergistic use with topical steroid
*Predniso(lo)ne	Steroid	Oral	<ul style="list-style-type: none"> Non-infectious (immune-mediated anterior uveitis)
*Carprofen *Meloxicam	NSAID	Oral	<ul style="list-style-type: none"> Infectious posterior and/or anterior uveitis Other systemic comorbidity precludes use of oral steroids (e.g., DM)

So our treatment is going to be primarily anti-inflammatory therapy. Out of all of the medications on this list, some of these are topical. On the bottom portion of the chart are oral medications. Probably the most important one is going to be a topical steroid. So prednisolone acetate is the gold standard. It's our best anti-inflammatory medication that we have. The frequency of these medications is really going to be dictated by the severity of the inflammation. So if it's really severe, I'm going to treat maybe four, maybe six times a day at the most. If it's mild or mild to moderate, maybe two to three times a day. And then we're going to taper it slowly as they start to improve. We usually don't want to stop them suddenly when they come back in and they look fantastic because the reason why they look so good is because you have them on anti-inflammatory therapy. If we stop that too suddenly, they're just going to have their inflammation recur. Topical nonsteroidal-- sometimes they'll use in combination with a topical steroid because I think they have a synergistic effect. And flurbiprofen is something that, although it's on the top of the list, is actually difficult for us to purchase right now. It hasn't been manufactured here in the United States for the past several months. So diclofenac and ketorolac are the two that we use most commonly. Bromfenac is also an excellent topical nonsteroidal anti-inflammatory medication-- just a bit more expensive than the other two. I generally will treat most of my uveitis cases with an oral nonsteroidal because it's a lot safer to do. In case it is something that's infectious, that's going to be much better for your patients. The only time I feel comfortable personally using an oral steroid in a dog with uveitis is when I am convinced it is non-infectious. So for example I'm treating uveodermatologic syndrome, or golden retrievers with pigmentary uveitis, or lens-induced uveitis. So I specifically know that it's not going to be something infectious.

Anterior Uveitis Treatment

- **Steroid-sparing medications**
 - Azathioprine, cyclosporine, mycophenolate, leflunomide
 - Immune mediated uveitis
- **Antimicrobials**
 - Systemic antibiotics
 - Doxycycline
 - Empirical treatment for common infectious agents
 - Pending titers or if workup declined by owner
 - Note: topical antibiotics are NOT effective for infectious uveitis

There are also steroid-sparing medications, again, only appropriate for immune-mediated uveitis. So you've got to be really convinced that this is not something infectious. So we can treat with azathioprine, cyclosporine, mycophenolate-- are all examples of steroid-sparing medications. If it is immune-mediated uveitis, generally, I'm going to start with prednisone initially because that's going to have a much better anti-inflammatory and immunomodulatory effects initially, so it's going to work a whole lot faster than these other medications and then eventually I'll transition them onto something like this if I need them to be on treatment for a long period of time. Oftentimes we'll start them on oral doxycycline. It covers a lot of the diseases that we're worried about with uveitis-- so tick-borne diseases, it can be used for leptospirosis as well. But it also has anti-inflammatory effects. So a nice medication to use in combination with all of our other medications. It's also important for me to mention that topical antibiotics or topical antifungal drugs are not appropriate for infectious uveitis. So they don't penetrate well enough to be able to combat something we need to treat systemically in those cases.

Anterior Uveitis Treatment



- **Mydriatic/cycloplegic**
 - Atropine 1%
 - Duration of effect is long in a normal eye
 - Eyes with uveitis are refractory to atropine effects
 - 3 main benefits:
 - Eliminates ciliary muscle spasm/decrease pain
 - Dilates pupil/prevents synechia
 - Stabilizes blood aqueous barrier
 - Contraindications
 - Ocular hypertension/glaucoma
 - KCS



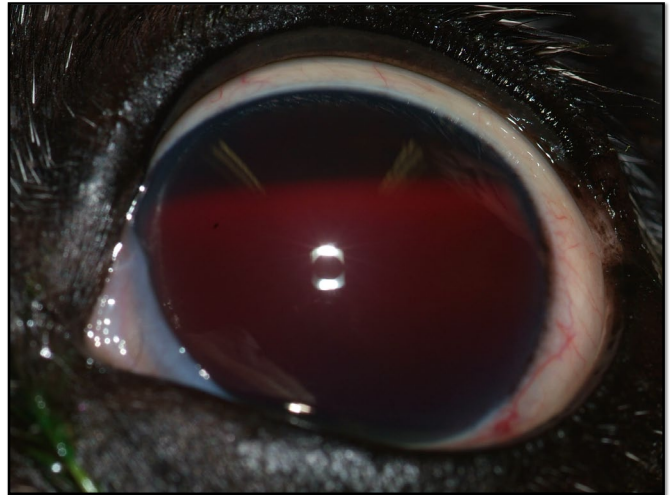
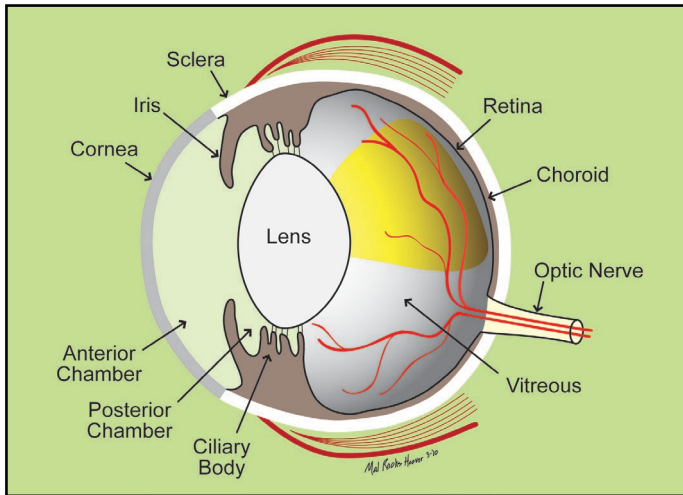
We are also going to treat with topical atropine because that's going to help decrease their pain. It's going to paralyze the ciliary body muscle that spasms when they're uncomfortable. It's going to dilate the pupil. So if they do develop posterior synechia at least their pupil is going to be dilated and actually prevents them from forming synechia that's more likely to happen when the pupil is smaller. And it also has some ability to help stabilize the blood aqueous barrier. Contraindications to using topical atropine - we always avoid it in patients that have a low Schirmer tear test value, or if they have ocular hypertension, or if they already have glaucoma. We're going to avoid using that medication.

Hyphema



So the last disease I wanted to talk about today is hyphema or blood in the anterior chamber.

Hyphema



So this is blood that's trapped between the cornea and the iris and the lens. And it can have a lot of different appearances. This is an example of a dog that has a relatively fresh bleed. So it's a bright red color. And it's already settled out you can see that nice line at the top quarter of the anterior chamber.

Hyphema

- Ocular

- Trauma
- Uveitis/glaucoma
- Retinal detachment
- Intraocular tumor
- Congenital abnormalities

- Systemic

- Hypertension
- Coagulopathy
 - Thrombopathy
 - Thrombocytopenia
 - IMHA
- Metastatic neoplasia

Hyphema over time can actually change color. So if we have a dog that has recurrent bleeds, it may continually be this bright red or fresh blood-like color. But if it's been sitting there for several weeks, sometimes it can take on even a really dark appearance, and it can look almost black. When we look at hyphema, we've got to decide what potentially caused this. And we have both ocular and systemic causes of hyphema. We're going to go over several of these causes on the next couple of slides.

Hyphema

- Ocular

- Trauma
- Uveitis/glaucoma
- Retinal detachment
- Intraocular tumor
- Congenital abnormalities

Blunt force trauma



Penetrating trauma

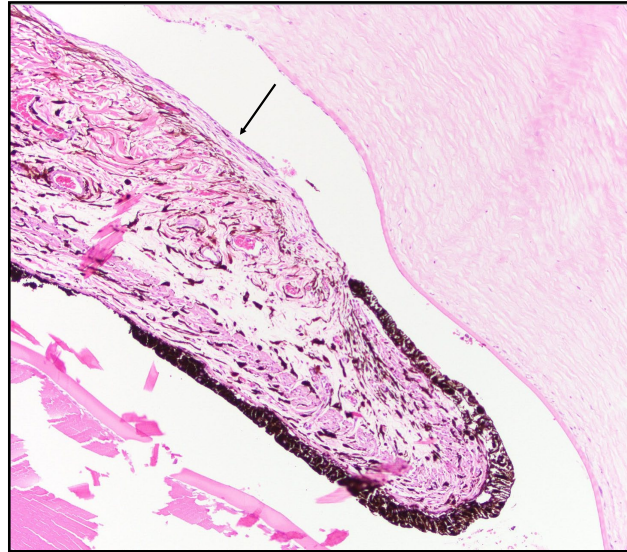


So when we look at ocular causes, trauma, blunt force trauma, or penetrating trauma are both great ways to develop hyphema. Proptosis is also another form of trauma where we can see blood in the inside of the eye.

Hyphema

- Ocular

- Trauma
- Uveitis/glaucoma
- Retinal detachment
- Intraocular tumor
- Congenital abnormalities



Uveitis and glaucoma-- very different diseases. We just spent the better portion of an hour discussing both of those things, but they can both develop hyphema because in both of those conditions, a pre-iridal fibrovascular membrane can form. And this is just a histopathological section of an eye, and the arrow is pointing towards the front side or the anterior surface of the iris. And we've got this vascular membrane that's formed, which oftentimes those blood vessels are a bit fragile, so they can break open and bleed and develop hyphema.

Hyphema

- **Ocular**

- Trauma
- Uveitis/glaucoma
- **Retinal detachment**
- Intraocular tumor
- Congenital abnormalities

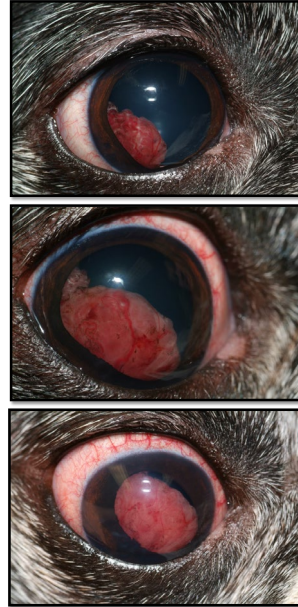


We can have a retinal detachment. So that retina can tear at the periphery and those little vessels then become torn and we can have blood that enters into the vitreous cavity that can then flow through the pupil into the anterior chamber.

Hyphema

- Ocular

- Trauma
- Uveitis/glaucoma
- Retinal detachment
- Intraocular tumor
- Congenital abnormalities

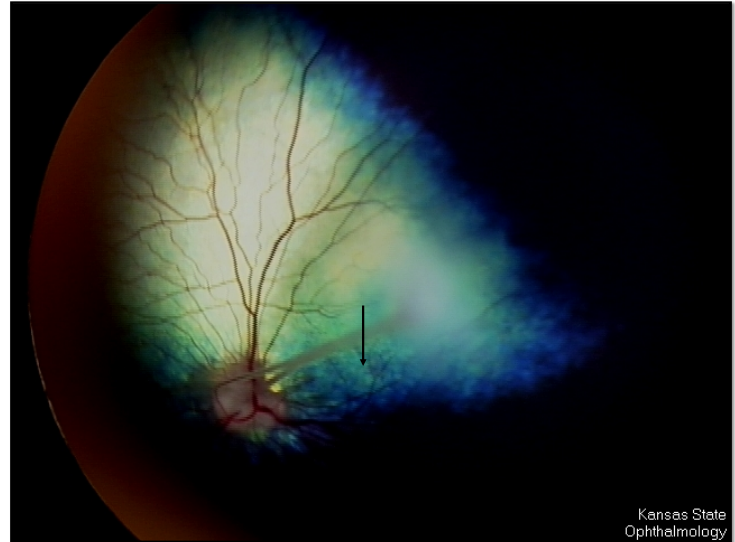
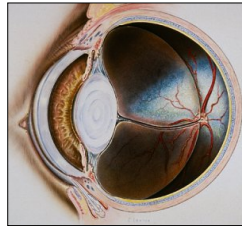


Intraocular tumors are a great way to develop hyphema. So intraocular tumors oftentimes have, again, fragile blood vessels that can break open and bleed. Sometimes we can't visualize the intraocular tumor until the blood is settled or if we can do an ultrasound, sometimes that can help work that out.

Hyphema

● Ocular

- Trauma
- Uveitis/glaucoma
- Retinal detachment
- Intraocular tumor
- Congenital abnormalities

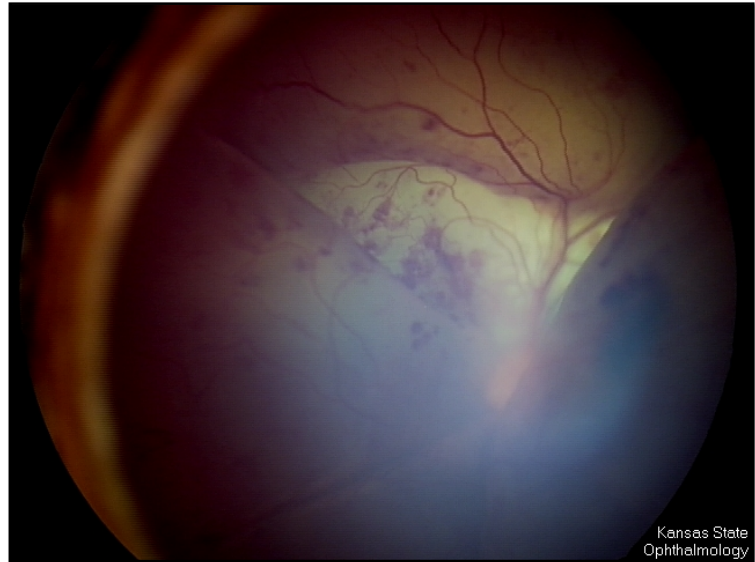


And we can also see congenital abnormalities. This is an example of a photograph of a dog that has a hyaloid artery. In this case, it's only patent right next to the optic nerve. I think I can see a little bit of blood present in that hyaloid artery. But this artery actually goes from the optic nerve all the way to the back of the lens capsule. So sometimes if that breaks open, we can develop some blood that can form in the vitreous and then again can sneak through the pupil and into the anterior chamber.

Hyphema

- **Systemic**

- Hypertension
- Coagulopathy
 - Thrombopathy
 - Thrombocytopenia
 - IMHA
- Metastatic neoplasia

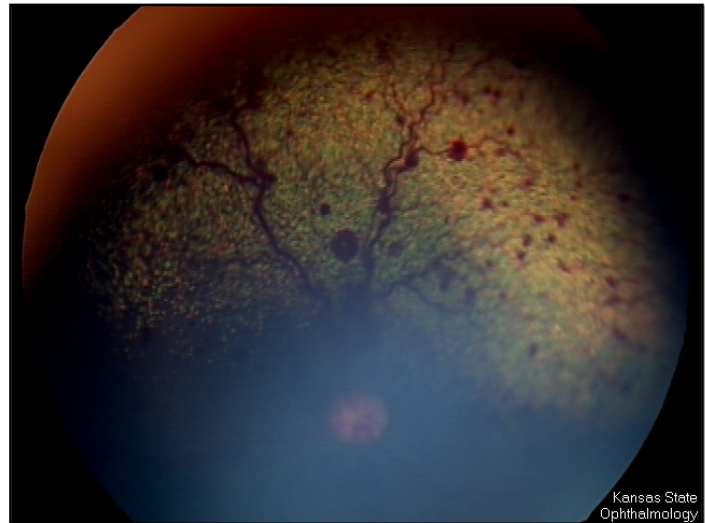


And then when we look at systemic causes-- so systemic hypertension. So having a blood pressure greater than 160 millimeters of mercury can cause retinal hemorrhages. It can also cause a retinal detachment. And sometimes we can have some bleeding inside the eye that can then present itself as hyphema. This is one of the reasons why it's really important to do a thorough ophthalmic exam not only in the eye that has hyphema, but especially in the other side. Because the eye that has hyphema, it may be really difficult for you to see or visualize the back of the eye. And maybe you'll have a better chance of looking at something abnormal in the good or fellow eye, if you will.

Hyphema

- **Systemic**

- Hypertension
- Coagulopathy
 - Thrombopathy
 - **Thrombocytopenia**
 - IMHA
- Metastatic neoplasia



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And of course, we can see thrombocytopenia this is a fundic photograph of an animal that has multifocal circular retinal hemorrhages. And again, the other eye could be filled with hyphema, so you wouldn't be able to see the back of the eye. But if I can do a fundic exam and I see this, it's really going to raise my suspicion for a bleeding disorder.

Hyphema Thrombocytopenia

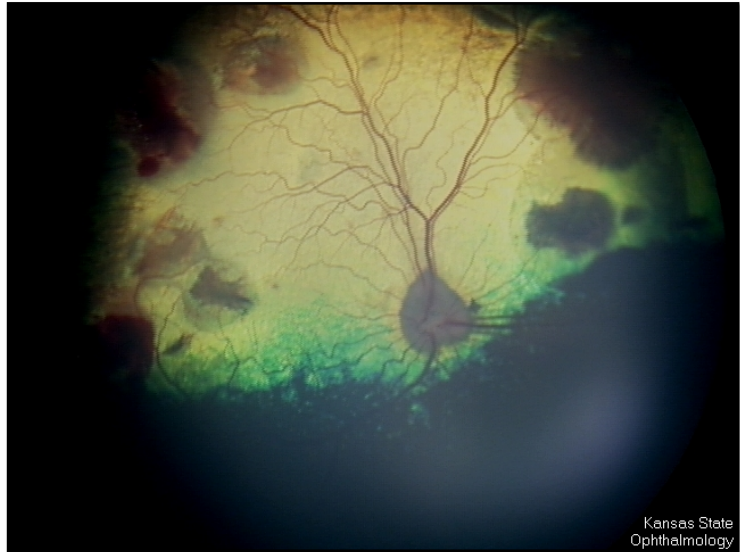
- **Decreased production**
 - Drug toxicity
 - Chemotherapy
 - Sulfa drugs
 - Radiation therapy
 - Chronic infections (*Ehrlichia* spp.)
 - Myeloproliferative disorders
 - Estrogen-secreting tumors
 - Myelofibrosis
 - IM megakaryocytic hypoplasia
 - Chronic renal failure
- **Increased destruction**
 - Immune mediated
 - Infectious agents
 - *Ehrlichia* spp.
 - *Leptospira* spp.
 - *Rickettsia rickettsii*
 - Sepsis
 - Sequestration (spleen)
 - Neoplasia
 - DIC

When we look at thrombocytopenia, this is one of the more common reasons for an animal to present with hyphema to a veterinary ophthalmologist. There are a lot of different things that can cause it. We've got a whole list of things that can cause a decreased production of platelets, leading to hyphema, or we can have an increased destruction of those platelets. Usually, when an animal presents to me, the more common thing that we tend to see is an immune-mediated thrombocytopenia. That doesn't mean that we shouldn't look for other causes. And it also means that we need to be very careful in handling this patient. Generally, if I'm suspicious that an animal has thrombocytopenia, I'm always going to draw blood from a peripheral vein rather than a jugular. We're going to wrap that leg really well, and I'm going to be very careful with my physical exam. This is not a dog that I'm going to have 10 different people palpate his abdomen. We're going to be very gentle in our approach.

Hyphema

- **Systemic**

- Hypertension
- Coagulopathy
 - Thrombopathy
 - Thrombocytopenia
 - IMHA
- Metastatic neoplasia



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And we can also see metastatic neoplasia is causing blood in the inside of the eye. This is an example of a dog that has a hemangiopericytoma that had metastatic lesions to both eyes. So we've got these areas that are bleeding in the back of the eye. And again, that can lead to the development of hyphema.

- Young animal
 - Infectious diseases
 - Congenital abnormalities
- Breed prone to glaucoma?
- Breed prone to retinal detachments
- Breed prone to cataracts?
- Old animal
 - Neoplasia
 - Systemic hypertension

When we're working up a case of hyphema, sometimes our signalment can give us a little bit of help. And I'm never going to let this guide me completely, but it makes sense that older animals tend to have problems like neoplasia or systemic hypertension. So I'm going to be more suspicious of those two things versus a young animal. So if I've got a puppy in front of me, I'm going to be thinking about potentially trauma, maybe an infectious disease, congenital abnormalities. Looking at the breed can be really important as well. Is this a breed that's prone to glaucoma and maybe he's developed that pre-iridal fibrovascular membrane that we saw on that histopath side a few minutes ago that's broken up in blood? Is this a breed that's prone to retinal detachment? So for example, Shih Tzus are definitely overrepresented in that category. Or is it a breed that I know develops heritable or juvenile cataracts? And now we might have, again, lens-induced inflammation. Maybe we've got a pre-iridal fibrovascular membrane that is formed. And again leading to the development of hyphema. So looking at the age of the animal, as well as their breed can sometimes give us a hint as to what's going on.

- What did the owners notice & when?
- General health
- Medication history (esp. new drugs)
- Vaccination history
- Travel history
- Use of the animal
 - Indoor couch potato vs. hunting dog
- Access to rodenticides

It's always important for us to look at lots of other things as well. We want to know from the owner, when did they notice the blood inside the eye? Has this ever happened before? Has it always been the same eye? Has it ever happened in the other eye? Is it something that's waxed and waned? For example, an animal with a retinal detachment oftentimes will have recurrent bleeds. So it may bleed. The blood can eventually drain through the iridocorneal angle and resolve itself, then it can re-bleed and the signs will recur again. It's important to look at their general health, their medication history, vaccination history, travel, all of these things can help us decide how we're going to work up this animal. And of course, their access to rodenticides is also important too when we're talking about bleeding inside the eye.

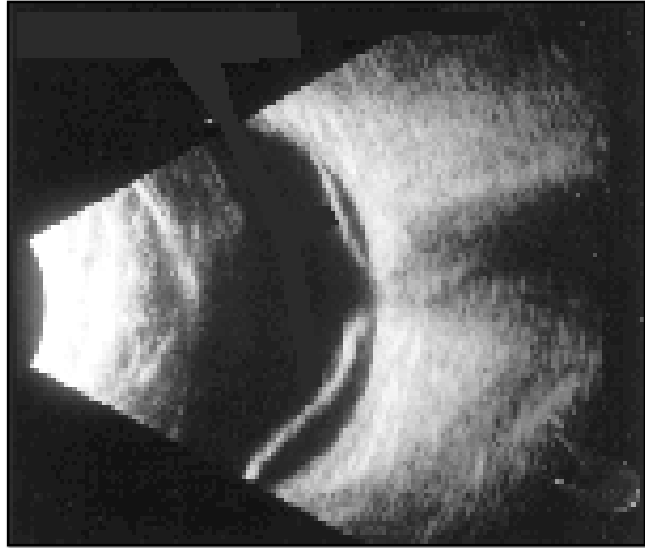
Physical Examination Findings

- **Mucus membranes**
 - Conjunctiva
 - Oral mucosa
 - Preputial/vulvar mucosa
- **Skin**
 - Ears
 - Ventral abdomen
- **Be gentle!**

It's important to do a thorough physical exam. So looking for evidence of bleeding elsewhere, whether that's auscultation of the chest to detect any abnormalities there, looking at mucous membranes on the body, looking for, again, evidence of other hemorrhages, looking in their ears or on their ventral abdomen areas where the hair is sparse and we can see hemorrhages. And of course, it's always important not only for us to be gentle if we're suspecting thrombocytopenia in that patient with hyphema during our physical exam and our handling of that patient, but it's also really important as we start them on therapy, to tell the owner the same thing-- to be really careful with that patient.

Ophthalmic Examination

- Examine both eyes
- Evaluate PLRs
 - Direct and consensual
- IOP
- Fundic exam
- Ocular ultrasound



It's important to examine both eyes. We want to look at the PLRs-- both the direct and consensual, measuring their intraocular pressure. Most of the time with hyphema, the pressure is either going to be normal or maybe a little bit low if the eye has some inflammation, but as we've talked about before when we're talking about secondary glaucoma, hyphema is one of the causes of secondary glaucoma. So we want to be able to address that if the pressure is inappropriately high. It's also important to do a fundic exam if you can in the eye with hyphema if you can see over the top of the hyphema and almost equally as important it is really valuable to take a look at the normal eye and be able to do a good thorough fundic exam because sometimes that can give us a hint as to what's going on. Sometimes we have to do an ocular ultrasound to be able to evaluate the back of the eye when they have hyphema because we can't see through the blood. This is an example of a dog that has a retinal detachment. So the probe is actually on the left hand side of this photograph, and it's going from the front of the eye to the back of the eye. And along the right hand side, we see these two white elevated lines that's actually a detached retina.

Hyphema Diagnostics



- Thorough history and PE
- Complete ophthalmic exam
 - Fundic exam
 - Ocular ultrasound
- CBC/chemistry panel/UA
- Doppler blood pressure
- Tick borne dz. Titers
- Coagulation profile
- BMBT
- Thoracic radiographs
- Abdominal ultrasound

So again, when we're working up a case of hyphema, it's important to do a thorough history, a good physical exam to see if we see any other abnormalities that might help us diagnose what's going on, and do a complete ophthalmic examination not only of the eye that has hyphema, but also the fellow eye. And then it's important for us to do a basic workup. So for me doing a CBC chemistry panel and a UA is important as well as checking their blood pressure. If I detect other abnormalities in my physical exam or maybe I'm suspicious of tick-borne diseases based on my conversation with the owners, then we'll follow up with other possible diagnostic testing. So we can do titers for different tick-borne diseases, maybe a coagulation profile, maybe taking thoracic radiographs or an abdominal ultrasound. Usually, I'm not going to do thoracic radiographs or an abdominal ultrasound until I've got my platelet count back to make sure that that's nice and normal before proceeding.

Hyphema Treatment

- **Underlying cause**
- **Topical anti-inflammatory**
 - Prednisolone acetate 1% suspension q4 to 6 hr
 - Neomycin/polymyxinB/dexamethasone suspension q4 to 6 hr
- **Mydriatic**
 - Normal IOP
 - Atropine 1% q12 to 24 hr
- **Prognosis**

So treating hyphema, if we can detect an underlying cause, of course, we're going to direct our therapy towards resolving that. I'm also going to put them on topical inflammatory therapy. Again, prednisolone acetate is our gold standard for anti-inflammatory medication usually q4 to q6 hours. Neo-poly-dexamethasone would also be an acceptable anti-inflammatory therapy as well. I'm going to use atropine topically if the pressure is in the normal range or a little bit lower than normal. Again, once or twice a day with the same caveats that we've talked about before. So we don't use atropine if they've got decreased tear production or an elevated intraocular pressure. The prognosis for hyphema is really dependent on what the cause of it is. So it's very variable.

Thank You!



And I would like to thank you for your attention today.

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