



## **Vestibular Disease**

**DRIP** 4

April 18, 2022

Instructor: Logan Donaldson DVM, Diplomate ACVIM (Neurology)

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Now let's say, OK, we haven't seen that. We've gotten through everything, and we find out that the patient eats an entirely raw fish diet. And they get a massive amount of thiaminase, and we can say pretty clearly that these patients have a thiamine deficiency, or we found it on MRI because we can absolutely see some changes associated with thiamine deficiency on further testing. A lot of these patients also come in either bradycardia, tachycardia or have an arrhythmia. And that's usually due to the thiamine necessity for cardiac supplementation. And so how do we treat this? Usually, if we discovered early and we just supplement the medication, the patient with thiamine and we get them on an appropriate diet, usually have a pretty good prognosis.

If we have severe neurologic deficits when they come in, sometimes that damage is irreversible. And so that's why it's important for a lot of these patients to get them on a good diet to start with. And if somebody is going to do an at home diet or a fully raw diet, we don't really have these conversations with them, that they're using a nutritionist and we're making sure that we're having thiamine supplement it into the diet, if need be, or we have the appropriate food sources in order to have that thiamine in there.



Similar to thiamine, hypothyroidism, usually a very good prognosis as long as we identify it early. And this is where we always want to get full thyroid panel. You should start to get back results within a day or two of submitting it, in my experience, once they receive the sample. Usually, I get final results back within about four or five days.

If we get those initial results back and they're concerning thyroidism, you can usually start to supplement it fairly safely. If you're worried about it, you can always start them with it that same day and wait for the results to come back. I usually wait for the full thyroid panel, especially if the patient doesn't have any other clinical signs of hypothyroidism.



So once we get past that, what is the other big one? We always worry about otitis media and interna. So when we're dealing with otitis media and interna and I'm staying on this side of everything, these are typically not primary neurologic diseases or due to other underlying issues. Most of these patients need to be treated with prolonged antibiotics.

Sometimes this does require myringotomy or a ventral bulla osteotomy in order to go in and remove that material there. If you're talking about doing a ventral bulla osteotomy on these patients, a lot of those patients will come out of surgery with facial nerve deficits and vestibular deficits. That usually are self-limiting, but some of those deficits are retained as well. So before performing that procedure or recommending that procedure, it's another important conversation to have with the family.

Myringotomies are usually fairly straightforward. And I don't personally perform them. I always refer them to a dermatologist because then they are also able to handle some of the other underlying issues as to why they needed this in the first place, which is usually allergies.



So now we talk about some of the primary neurologic diseases. Let's talk about infectious or inflammatory disease. One of the things that I will commonly refer to this as a cerebral ataxia or inflammation of the cerebellum. It can be tough to find this on our MRI and spinal tap in some cases. If I'm going to get a false negative on a spinal tap, I usually get it due to cerebellar inflammation, but I absolutely get positive results as well in these cases.

Most of these patients' prognosis is based upon their deficits and their response to treatment. As a standard conversation for encephalitis patients, about a third of patients with inflammation of the central nervous system do very well on steroids. But third we have to reach for stronger drugs and a third don't respond well to the medications. When you start to break it down into the actual differences between the individual inflammatory diseases, you start to get very different prognoses, which is why it's sometimes important to get this answer before treating these patients.



We talk about vascular disease. Most of the time, it's just supportive care and time. Most patients have a good excellent prognosis, but some of these patients take weeks to months of intensive care and some families are capable of doing that. And so this is a conversation to have with them before they jump into the full care for the patient. If they have a vascular disease, we then recommend full systemic workup to do a hypercoagulable workup to find out is there an underlying reason because the best way to prevent this from happening again is to treat the underlying disease process.



Let me talk about metabolic disturbances. These are usually pretty rare, in fact, I've personally never seen a primary case in my career. But most of these patients will retain deficits. And this typically affects both vestibulocochlear and facial nerve. And so if we see what we think might be idiopathic 7 and 8, we still want to do CBC in chemistry and thyroid to make sure we're not missing something there. The treatment here is just treating that underlying disease.



Neoplasia is a big one. These patients unfortunately usually have a guarded to poor prognosis and the lifespan is pretty poor for infratentorial tumors. It is significantly less than infratentorial tumors. I'm sorry, supratentorial tumors. Those are tumors more towards the cortex versus those under the tentorium cerebelli and affecting the cerebellum.

The systemic health of the patient is very important. And the reason why the prognosis is poor is that many of these are non-operable. They're either in areas that are just too dangerous to try to get to surgically or they're actually of the neural tissue itself and the damage from surgery could be too severe. And so we typically will recommend radiation therapy for these patients, but sometimes decompression surgery is an option.



Last that we worry about is degenerative disease. Some of these patients will stabilize, but the vast majority are progressive. And some of these degenerative diseases can be cerebellar atrophy. They can be degeneration of the peripheral vestibular system. There's a number of things that can occur here, and we don't have great tests for a lot of them. Some of the big things we look for are MRI or changes to the cerebellar structure itself to determine if that cerebellar atrophy or cerebellar hypoplasia in the case of some cats from birth is present.

Most of the time cerebellar hyperplasia stabilizes. The vast majority of the time they're not progressive. Most cerebellar atrophies, which is that early onset death of the cerebellum that typically is progressive. And in those cases, we usually lose the patient over the course of six months to a year due to progressive vestibular disease in cerebellar signs. A lot of these patients if it's peripheral degenerative disease will be deaf as well.



Here's one of the interesting ones for me is metronidazole toxicity. We think about metronidazole toxicity as any patient that's on metronidazole has potential of having this, which in theory is true. But the vast majority of these patients require extremely high doses. And you'll see they're these doses are usually 60 milligrams per kilogram per day for a week to two weeks at a time in order to actually get metronidazole toxicity.

And yes, it can occur at lower doses, but this is pretty common. And so if a patient had wildly overdosed on metronidazole, you might see this. Now what is the treatment? Well, easy ones are supportive care and removal of the drug. But here's the interesting part. Diazepam half a milligram per kilogram IV or per os every 8 hours will bring the recovery time in these patients to less than 40 hours on average from 11 days. And so if we start this treatment, you might be able to get these patients out of your hospital within one or two days because most of the time it's within about 30 to 40 hours versus having them in a hospital for two weeks at a time or having patients at home having real difficulty getting around for this time frame, and so I always recommend it. This works by actively competing with metronidazole inside of that cerebellum and central nervous system.

And so now that we've talked about a lot of these diseases that we can see.



Let's talk about some of the abnormal vestibular presentations. So with this tubular disease, it can occur without head tilts, nystagmus, or the inability to stand, something to always keep in the back of our mind.



And so with that, we're going to talk about a few cases here. These are some subtle deficits. You might come to see a patient with bilateral disease, some patients with physical abnormalities, and in the paradoxical vestibular disease, which might actually be my favorite.



And so the first patient here is Tuck. Tuck is a two-yearold male newborn mixed breed canine. He presented with an acute onset of inappetence and listing to the left.



So the emergency physician came to me and she said, well, Tuck has subtle ataxia and he's listening to the left. He also has anisocoria, but I'm not really seeing anything else on my exam. Is there anything that you're concerned with?

## WHAT DO YOU SEE??



Well, the interesting part here was when we actually looked at this patient. What you'll see is that Tuck is actually a fairly large patient. He was about 80 pounds and so rolling him over and challenging his gravity was difficult. But once we did and as we were getting him rolled over, one of the technicians before we had a chance to look actually pointed out the fact that Tuck developed dysconjugate nystagmus. And so what you'll see here on that left eye is you'll see the dysconjugate horizontal nystagmus in that left eye alone.

It's subtle, but you can see it ticking there. It's better at the beginning of the video. Well, apologies. There you go. You can see it right there on that left eye. The right eye is intact. And so the dysconjugate nystagmus automatically was associated with positional aspect of the patient and gave us concern for central disease.



And so Tuck went to have an MRI and CSF and he absolutely had inflammation of the cerebellum.

Luckily, his infectious testing was negative. He's been treated with a tapering dose of immunosuppressive Prednisone. And he has had a complete resolution of clinical signs. And he's been clinical sign free for a few months at this point.

And so when we see some of these subtle signs, it's always important to say gosh, we do want to try to challenge these patients if we're worried about the severe disease and outside of the anisocoria and some of that listing to the left. There were no signs of central disease until we actually flipped them over. And so that's why it's important to do this. And if you have an 80pound dog, it can be tough. And so you might need an extra pair of hands.