Motion Sickness Pathophysiology and Clinical Presentation, in partnership with the American Academy of Audiology

Presenter: Jamie Bogle, AuD, PhD

Thank you for joining today's webinar entitled Motion Sickness Pathophysiology and Clinical Presentation. This course is presented in partnership with the American Academy of Audiology. It is my pleasure to introduce our presenter, Jamie M. Bogle, Aud, PhD Dr. Jamie M.

Bogle is the Division Chair of Audiology at Mayo Clinic, Arizona and an Assistant professor of Audiology at the Mayo Clinic College of Medicine and Science. She earned her doctorate degree of Audiology and PhD from the University of Colorado at Boulder and was awarded the James and Martha Crawford Endowed Clinical Research Fellowship in Otolaryngology at Mayo Clinic, Florida. Clinically, Dr. Bogle evaluates children and adults with dizziness and imbalance due to peripheral and central conditions. You can read more about Dr.

Bogle on the course page on our website. Welcome Dr. Bogle. Well, we're so happy to have you back today. Well, thank you so much for the invitation and I'm happy to speak with you today on a topic that I work with pretty much every day with most of my patients.

Before we get started on content, I do have a brief message from Tish Gaffney, our current AAA President and she's going to give us a little bit of an intro and get us kicked off.

I'm Tish Gaffney, President of the American Academy of Audiology. The Academy is the home for audiologists. We are dedicated to achieving recognition of audiologists as the experts in hearing and balance care and the field of audiology as an independent clinical specialty that is integral to the healthcare team. We offer our members the latest resources for practice management and clinical evidence based practice so that they can grow professionally and improve patient care. We advocate for our members in Washington and throughout the country to ensure that the voices of audiologists are heard and we help connect audiologists to one another through valuable networking opportunities.

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All right everyone, I hope to see you at AAA New Orleans. Should be a really fun time. A couple housekeeping pieces here. These are my disclosures. I have nothing specific to disclose for this talk and these are the limitations and risks that we know are associated with any sort of medical or healthcare related talk.

So those are available for your review and these are learning outcomes for today. I know many of you may be familiar with motion sickness, but I think some of the pathophysiology especially is really quite interesting and something that you may just not have had much exposure to. And so I wanted to provide that for you and apply this for your clinical patients. You know, motion sickness is one of the most common, common types of self reported symptom that we'll see in our dizzy clinic. And this has been around for quite some time.

As soon as people started moving in ways that were not walking, using different sort of vehicles and transports, people started getting motion sick. One of the earlier descriptions of motion sickness was written by Hippocrates who got seasick and was describing that being on a boat was providing some sense of disorder on the body. So this overall is a type of illness that is caused by motion and that motion can really be most anything. The types of sickness or illness that people describe though do vary and we mostly think of things like nausea or maybe dizziness. But there can be a wide range of things feeling sweaty or getting a headache.

Many people describe feeling very tired with motion sickness as well. So there are plenty of symptoms that you can get associated with motion sensitivity. And of course, if you've ever experienced it, know that these are not the most pleasant symptoms.

So in looking at common triggers, there as I said, can be a wide range of things that can trigger motion sickness. We look down the list, there's probably a few things here that you could identify as something that's concerning for you. The most common type of motion sickness is seasickness. And we know that at least a quarter of those passengers who are on larger ships or on cruise ships will develop motion sickness within the first couple of days. This does become a bit more prevalent if you're on a smaller ship, particularly if you're in adverse weather.

So think of those choppy seas and the smaller boats. Nearly, you know, over half, up to three quarters of patients will experience some sort of seasickness. I myself didn't know I got seasick until I went out on a whale watching boat and discovered that that was in fact the case. So, you know, growing up in a landlocked state, you don't really have that sort of exposure. So I do think it highlights the challenges that we have when identifying, you know, some of these triggers and that if you've never been exposed to it, you don't know if it's necessarily a trigger for you for experiencing motion sickness.

Now I think the one that we will commonly describe or because we're experiencing this pretty much daily is travel through the car. And this has been reported in a really a large number of patients up to 46% will describe some sense of motion sickness when in the car. This can be increased in situations that commonly doing so sitting in the backseat of a car or if you're trying to read or navigate on your phone. This does increase if you're in more of a high velocity situation. So things like rally cars.

Depending on how you drive, you may be a more high velocity person. But those situations where you're inducing more velocity, more stop and go with poor visual cues are likely the culprits for this increase in car sickness.

Air sickness is actually not incredibly common. It is possible. We see that there are travelers in commercial, commercial cabins. So think bigger planes that do get get motion sick. But it's not, not as

common as the, the others that we've described before.

Now with smaller planes and perhaps less pressurized or less temperature controlled cabins.

A larger number of student aviators who describe a sense of air sickness. Over time though, this does decrease. So as people habituate and are exposed to this environment. But air sickness is not something that we're typically worried about. Now the one I think that we're going to see actually quite often and will grow in terms of prevalence is related to cyber sickness.

Now we've known about cyber sickness or simulator sickness for a long time associated particularly with aviation and flight simulation. Now that we're talking about virtual reality, full immersion gaming. These technologies are in everyone's hands and they can be very provoking. So if you haven't tried virtual reality, give it a go. It can be very immersive and can really quickly trigger cyber sickness.

Another area that kind of falls within the same category would be things like automated cars. Where we're in these situations where there's actually no necessary visual reference. The seating may not be like a typical car. There may be augmented reality associated with this. So I do fully expect that we're going to see increases in self reported motion sickness associated with these altered visual and motion environments.

So this is an area that you know, when we talk about, about sensitivity to motion. In terms of patient care, I go through the list, right? How do you do in cars? How do you feel in boats? Have you done any sort of virtual reality?

Most people who get motion sick avoid virtual reality pretty naturally. But it is something that, particularly in our younger patients is part of the culture, their landscape and they want to participate with their friends. So it is something that you may see pop up.

Now one of the things that I do want to emphasize is motion sickness is incredibly common. Absolutely everyone can be motion sick. It just takes the right stimulus at the right time. There are a bundle of us that are a bit more highly susceptible to motion sensitivity. But nearly everyone is at least going to experience this one time.

And that may be a stimulus that they've never been exposed to, or a combination of, of symptoms or conditions that set the tone. But absolutely everyone can reach that threshold. It's just a matter of how well or how quickly do we get there in terms of triggering those symptoms.

So in terms of motion sickness, there's likely a few different things that put us more at risk for the types of symptoms that we associate with, with movement. And so there's likely a relationship between overall susceptibility and underlying genetic predisposition. We do know that women tend to be a bit more prone to motion sensitivity. And this does happen a bit more often during times of hormonal fluctuations, Menstruation and pregnancy being two big ones. There have been some studies looking at associations with race and ethnicity and motion sensitivity.

And that likely then ties back into some of the genetic underlying gene environmental interactions that can be prevalent. And so there are some different components that we know are associated with differences in motion sickness sensitivity. One of the bigger ones, again, related to genetics, is going to be family history. So for individuals who have parents with motion sickness, they're two times likely to experience that themselves. Identical twins are generally going to be concordant, or they either both have motion sickness or they both don't.

And so that again, highlights the genetic component. There's a really high heritability estimate. So upwards of 70% of individuals, individuals are thought to have motion sickness associated with the genetic predisposition. And many, many polymorphisms have been associated with motion sickness.

So we know that there are some genes that are specific for, or at least associated with, so particularly along chromosome 4 and numerous polymorphisms that can contribute to that as well.

So a lot of underlying parts to motion sensitivity and likely many different underlying reasons which can make it a little more challenging when trying to identify maybe risk or presentation. But I think the reality of it is that there's likely a few different things that have to go wrong all at the same time. And being in the right environment before we're triggering that sense of motion sickness.

Now, in terms of this, I think is really quite interesting. There, there isn't seem. There seems to be an age effect associated with motion sickness and that it's incredibly rare for infants and very young children to experience motion sickness. And the thought is that with these kids, they're likely, hopefully sitting in the back of the car, in a car seat where they're recumbent. So they don't really have much visual information.

They're mostly looking at the roof of the car. And so they're not getting any sort of outside visual cue that could trigger some of these symptoms. It's also possible they just don't complain about it because they don't have the language to do that. So there are some limitations to this. We know that kids between 6 and 12 seem to be the most susceptible to motion sensitivity.

I think this is really an interesting time. There's a lot of growth happening at this point. When we think about kids, we're typically putting them in the backseat through this age, they're mostly out of car seats or out of boosters at least, but they're in the back. And we know that sitting in the back of the car is where most people tend to feel the most symptoms. But there is generally about a peak around 9 to 10 years of age.

As we get older, we do see declines through puberty and adulthood. And these have been associated most often with habituation, Just different exposures to movements and being in the car. Perhaps now

you're driving, you're sitting in the front seat, you have better visual cues, so there is a drop. And so when we think about asking our patients about motion sickness, I, I always ask them about two time periods. One, are you motion sick now?

And two, were you motion sick as a kid? Because many people will tell you that, yeah, I was motion sick as a kid, but I grew out of it. And this is what they're talking about. Where this susceptibility does seem to decline with exposure or just with age.

It's reported to be fairly rare to experience motion sickness over 50 years of age.

You know, likely a relationship between decline in vestibular sensitivity, habituation, exposure, many, many things that could contribute there. It's not very common for motion sickness to trigger at these older ages. It's typically something that's going to occur in our younger, younger ages and then generally decline over time.

So we, when we think about our patient population, There are a couple of diagnoses that we associate with motion sensitivity. First and foremost, migraine in general, Particularly vestibular based migraine symptoms. But migraine just with its heightened sensitivity to really any sort of stimuli Puts someone more at risk for motion sickness. Patients who have vertigo already are likely not going to tolerate motion quite as well, and particularly those with Meniere's disease, which may be a bit more reliant on the migraine component. But Meniere's does.

Meniere's patients tend to describe quite a bit more motion sickness than others. Any sort of additional component that puts you at at a reduced competency, reduced capabilities. So sleep deprivation, dehydration, poor nutrition, all of these components are going to influence your susceptibility to motion sickness. So if you think about it, with motion sickness, we're looking for a threshold before we trigger symptoms. And the less cognitive resources that we have to help bolster that threshold, the more likely

we are to cross it.

So sleep deprivation has been heavily associated with motion sickness. And if you think about your chronic dizzy patients, they tend to be very sleep deprived as well. We know that you can experience motion sickness with significant visual impairment. It's not quite as provoking, but it is something that if you put both blind or individuals in vision denied conditions, you can in fact trigger a very similar of motion sickness. And so that suggests that there's additional underlying components aside from vision that of course are going to contribute to this.

Now, on the flip side, we know that there are groups that are less susceptible to motion sickness, particularly those who have habituated over time. So dancers, acrobats, pilots, people who are suppressing their vestibular system consistently and have had that practice. The other group that is really fascinating are the patients with bilateral vestibular hypofunction. And these patients are generally not going to experience motion sickness because they don't have the vestibular trigger for the subsequent cascade. If you're interested, let me know.

There's some really interesting papers written in the 60s about some of the students at Gallaudet with bilateral hypofunction who are being studied for motion sensitivity as part of the aerospace or NASA program. And just really interesting data coming out. Probably not something you could get through IRB anymore, but really interesting data. So motion sickness, there's a lot of underlying components in terms of genetics, exposure, environment. One of the big ones that we also want to consider is the psychological component.

For those who've been motion sick. It's not pleasant, you don't like it, and you know that getting on that boat again is probably gonna make you s. So this is a really interesting bit of data where they evaluated patients and looked at symptom onset. And for many patients, they can start to experience onset of motion sickness symptoms even before they get on the boat. So they can think about or see a boat and

start to feel as though they're getting motion sick.

Additionally, if you're with someone who's getting motion sick, the people around you are also likely to experience motion sickness. So there's a heavy psychological component associated with this condition. And I think, also fascinating, 45% of patients can benefit from placebo in terms of reducing motion sickness. So that, I think, provides some really good information about the psychological components that are going to play within our motion sickness patients. Now, the motion sickness cascade is actually fairly linear.

The time of onset, though, does vary, but it kind of sneaks up on you. It's not typically something that is like a switch. It's kind of an insidious onset. It kind of can creep in where you just start to feel off. And this is typically going to be following an unfamiliar sort of motion, but there's some sort of motion event that triggers.

Now, early symptoms are kind of vague. You just. You feel uncomfortable, maybe fatigued, not quite as alert. But this does start to then progress into a sense of nausea, stomach awareness, feeling like you could be sick. And there are going to be triggers for this.

So think of, like, those terrible environments where it's hot, maybe there's, you know, smoking, there's, you know, you just ate a really big buffet dinner, and. And now, you know, you're in this motion environment. So there the nausea will start to creep up, but it will be modified based on some of these additional factors. Now, from here, we do start to get some additional symptoms, and they're not ones that I think most of us associated with motion sensitivity, but most people start to yawn, they start to salivate. They may burp.

But there's also additional pieces like their eyes may feel blurry, or they may just feel dizzy or disoriented. They may start to hyperventilate with these sort of symptoms, depending on the individual.

From there, we also do see some changes in blood pressure. We do see a decrease here. You'll also start to see where you start to feel really warm or flush.

You may start to sweat. And this all really kind of leads to the finale where most patients, if we continue down this path, will start to vomit. So once the motion sickness cascade starts, you're kind of stuck. And unfortunately, symptoms will tend to progress until you stop doing whatever it is you're doing and get out of that environment. So the nice thing is, of course, once you're outside of that stimulus, symptoms resolve, and we tend to be back to baseline within a day.

So the cascade tends to vary in terms of the time course per individual and probably depending on the type of stimulus, but also just the particular time course or environment that the patient is in. So post, you know, there are some benefits here, you know, for any parents in the room. You maybe have had to take that really inconsolable child for those car rides to get them to go to sleep. And this is a condition associated with motion sickness called sopite syndrome. And what happens with this syndrome is that we can elicit symptoms associated with motion, so being in the car.

And it tends to be things like drowsiness, sleepiness, fatigue. So we're able to capitalize this on this for our kids by driving them around and getting them to go to sleep. So sopite syndrome in adults with motion sickness covers those components of just kind of vague drowsiness and fatigue. But in our little kids, many of us have used that to get them to sleep. So there's some benefit to motion sickness, I suppose.

You know, with motion sickness, it's something that most of us are going to experience, but you can train it. And we do see this in numerous professions. Especially it would be really hard to be a professional dancer if you got motion sick with spinning. And so dancers tend to have suppressed this by spotting. They use techniques to help reduce their vestibular system and suppress that response.

We also see this in our aviators and pilots over time will also demonstrate this suppression and can give us some really reduced VOR perception at least. And some of that is, of course, associated with their piloting, but also their exposure to simulators and how they've been able to adapt to those environments. So we know that we can suppress this, likely with enough training. All right, so motion sickness is pretty awful. We don't like it, but it's so common.

You know, there. There seems like there should be a reason for why this mechanism exists. And there have been quite a few underlying theories as to why. So the primary reason or the primary theory that we associate with motion sickness relates to what's called sensory conflict. And the sensory conflict theory assumes that there is discontinuity between our visual vestibular somatosensory inputs or between our intralabyrinthine inputs.

We'll talk quite a bit more about sensory conflict. Now, there's a few others.

Another one that you may see referred to in the literature is called neural mismatch. And this one is more based on memory and perception. You know, where we've over time established maybe a movement map or an environmental map for what we expect our perception to be. And this, this mismatch could occur if perhaps that map no longer applies or the environment that you're in doesn't match with the underlying map.

There was some discussion at one point about motion sickness as a defense against poisoning. Realistically, this one doesn't really get talked about too terribly much. But the idea was that if there was a discontinuity between these two sensory systems, this was interpreted as error or hallucination, and could be a sign of something poisonous or something that you shouldn't be ingesting. And then the final one is the nystagmus hypothesis. And this one really relates to a cross stimulation between the vestibular system and the vagus nerve.

And this one, again, isn't as commonly referred to, but there's. These three additional ones are at least presented in the literature and are things that you may run into. Most of us now are really only focusing in on sensory conflict. I think that's the one that for most everyone, makes the most sense, but just to know that there are others out there.

So sensory conflict is going to be your most evaluated. It's the most accepted theory that we have for motion sensitivity. And we typically are thinking about this as a conflict between the visual and the vestibular systems. And you can think about. There are many, many different environments that could provide sensory conflict.

If you think about being in the car, you're getting a lot of visual input, but the vestibular system is not going to be triggered with a constant rate or a constant velocity of motion.

You know, virtual reality is another where you have heavy visual input but no vestibular input. And, you know, you can probably think of many, many others. High action movies can do it, video games, anywhere where you're. You're experiencing a busy visual environment with lack of vestibular input. And so these components are going to all interplay together and provide the, the exposure or the expression of what your environment is like.

And if it's not lining up with what we expect, then we start to see abnormalities. So, you know, why does this happen? We have these sensory inputs that are all coming in, and we fully expect there to be discontinuity over time or over experiences. But if the sensory information is different than what we expect, and typically this is based on our past experiences, our past exposures, then we start to look for the conflict. And this is typically going to be between our visual system, which is identification.

We're moving, whether we're actually moving, such as when we're in the car or we have a good visual trigger, like in VR, the visual system is triggering a movement pattern. The vestibular system is

clamping that down, saying, no, there's no movement here. There's been no acceleration, we're stable, and we're not getting much somatosensory information coming in here that's going to contribute. So this intermodality conflict is typically what we're expecting to give us the dizziness here with that difference between our visual and vestibular information. Now, the other one that I think is not as well discussed is related to intralabyrinthine conflict.

And, you know, this is one, I think that's probably really important for our central vestibular patients. Think of like our migraine patients. And in this situation where we're looking at this conflict again between the visual and vestibular system, but also within the vestibular system, a conflict between the canal and the otolith pathways. And this theory is really quite interesting. So the hypothesis to the intralabyrinthine component really is looking at asymmetrical afferent information.

So for whatever reason, the otolith information has been reduced and the semicircular canal activity is read as hyperactive. So it may not be hyperactive. It may be perfectly normal and typical, but because the otolith information is not there to balance it, it's read as a hyperactive response and can then go on to trigger this motion sickness cascade. There is this working hypothesis that our canals and otolith are going to give different roles in terms of motion sickness. And there have been some data at this which has been really interesting to review and to see how this plays out in the clinic.

So this study by Wang and Lewis almost 10 years ago now looked at patients with migraine and compared them to controls. And I won't bore you with the nuances of what the study entailed, but it was looking at the patient's ability to suppress the velocity storage mechanism and accurately identify verticality. And so looking at spatial orientation and also then looking at VOR suppression, essentially, and what they find is that patients, typical patients, are able to do this. We know which way is up and down. We can suppress this response quite easily.

But patients with migraine, vestibular migraine especially, couldn't do this. They had poor understanding of verticality. They couldn't suppress this velocity storage mechanism. And what the authors hypothesized is that the associated motion sickness with patients with migraine related to this abnormal integration between the canals and the otolith. And this, I think for those of you who've worked, worked, or are working in patients with vestibular migraine, this is incredibly important because we know that These patients demonstrate poor spatial orientation.

They have prolonged velocity storage mechanisms. They don't suppress this well. And we know that the otolith pathways are abnormal in this group, particularly when looking at utriculo ocular reflex pathways. So this, I think, is going to play a big role in how we at least understand motion sensitivity in our centrally mediated patients. So this idea comes back to how does the cerebellum in particular mediate the conflict between the otolith and the semicircular canals?

We focus on the cerebellum and the vestibular nuclei, that reflex pathway, because we know that that's where this conflict resolution starts. And particular particularly looking at the nodulus within the cerebellum, because this is where the canal and otolith information is first integrated. And this is what gives us our understanding of gravity, as well as pitch and roll vectors. And roll especially is incredibly important for triggering a sense of motion sensitivity. The role vector is highly associated with motion sickness.

We also know that the nodulus is a key component for not only the integration, but also a piece that helps to inhibit the velocity storage mechanism. And so this is kind of where things are, where the brakes are put on for this perception. So if we're able to suppress the velocity storage integrator, we can. And if we do this, typically by a galvanic vestibular stimulus or sometimes medications can do it. If we can stop the.

We can stop the motion sickness, but only if we do this before the motion occurs. So once you suppress it, you're good to go. But if you start a motion and then try to suppress it, it's not likely going to inhibit that motion sickness cascade. So we know where the problems, but the trick is, how do we reduce that perception and get it so that we're not triggering that cascade? All right, so the vestibular system, of course, has a big role in understanding motion sensitivity.

Of course, the vestibular system is going to encode head and body motion. They're giving us our linear and angular acceleration cues. The vestibular system is also where we're going to understand our spatial and gravitational vectors and perception. And this is also where we start to understand our body's orientation and any sort of understanding of misalignment. So this information needs to be present and it needs to be working well for us to understand where we are in space.

So a lot of this literature on motion sickness comes from aerospace. And particularly with early studies looking at space motion sensitivity and finding that the overall sense of motion sickness came as a response to the body's change in orientation vector. So if the patient or the, the astronaut in this case was perceiving gravity in a slightly different tilt, particularly in the role plane, so just think tipped over to one side, that was likely to trigger then that motion sensitivity. We know that for our astronauts, that space motion sickness tends to be related to the abrupt offloading of the otolith organ. So once you hit that microgravity, the otolith are no longer providing that gravitational vector and are no longer putting the brakes on your semicircular canals.

So we've learned a lot from that group in terms of kind of the worst case scenario, we abruptly lose the otolith, what happens. And we know that that impacts your ability to spatially orient. You lose your gravity vector, and it's likely to trigger an onset of motion sensitivity. So all of this does play a role in our patients. It's just not quite as easy see it because we don't have that abrupt on and offset of gravity vector in these patients.

So the understanding of the otolith organs in particular is really a heavy piece to understanding motion sickness. And in our orientation, our understanding of verticality, all of those components are playing a big role in understanding where we are in our environment. Anytime we lose that understanding, we lose that vestibular input and that vestibular reliability. The likelihood of a discontinuity between the visual system is much higher and is something that we likely expect to lead to emotion sickness. Okay, so the vestibular system is quite key for this.

You have to have a peripheral vestibular system to produce motion sickness. It's kind of the must have. As I said before, there are some really interesting studies on vestibular hypofunction and finding that you can't make them motion sick. It's really tough to do. And so there has to be a working vestibular system to produce motion sickness.

That is key, key. We do expect that the velocity storage integrator is particularly important. So vestibular nuclei, nodulus, I think vestibulocerebellum. And this is also going to heavily relate to, you know, one, the breaking mechanism coming from the otolith back to the canals. But also when we think about where our autonomic and vestibular systems integrate, and that's, you know, brainstem, cerebellum, numerous other central pathways.

But once we're triggering these vestibular pathways, we're likely also triggering the autonomic response. And that makes a lot of sense. We're triggering nausea, blood pressure, Changes, respiratory changes, sweating, GI changes, all of those are integrated within the autonomic system. So the, the triggers are, are likely going to be because of this abnormal integration between either the, the inter labyrinth or intralabyrinthine integration or the visual vestibular integration. But regardless, that's what's triggering the autonomic symptoms.

Most of what we associate with motion sickness again is related to velocity storage. And this is a perception of motion that happens after the motion is stopped. And this has been described for

hundreds of years at this point. And it's something that we take advantage of clinically and we'll talk a little bit about that. But the velocity storage integrator is what's responsible for adjusting our angular acceleration that we are, that we're sending in from our semicircular canals and converting that into the velocity signal in the cerebellum.

So this is also really highly important for keeping our eye rotation in line with spatial orientation, with gravity vertical. So this is a really key mechanism for our motion sensitive patients in that this is likely the culprit for many of the abnormal presentations that they see. Now, when we look about velocity storage, there's a few ways you can do it. Some people do a visual velocity storage or visual time decay looking at optokinetic after nystagmus, I'm not successful with that at all. If I look at it, I tend to look at it in terms of step velocity testing when we do some rotary chair results.

So for those of you not as familiar with rotary chair step velocity, what we're doing with this is inducing an abrupt onset of rotation. Typically we're going to, I don't know, around 60 or so degrees per second, although you can go upwards of 240 depending on what you're trying to do. And what you'll find is an abrupt onset of nystagmus, which you can see in the top panel, in this case, right, beating nystagmus that abruptly starts at about three or four seconds and then over time gradually fades. Now what's interesting about this, this test is that once you hit your peak velocity, you maintain that velocity and you'll, you'll keep that rotation happening until you do an abrupt stop, typically at the end of about a minute or so. And what happens with our velocity storage mechanism is that we're able to see a decline after that peak response.

Now, with cupular mechanics, the cupula is not really good at triggering nystagmus over a very long period of time. And if you think about the physics that you learned about the cupula, you're only going to get 6 to 10 seconds of actual stimulation coming from the semicircular canal. And so the canals are their only accelerometers. They're only there. They're only designed to pick up the abrupt onsets and

offsets of velocity change.

So these are not reflexes that sustain perception and motion. They're only giving you the onset and offset. So when we think about that and when we look at the response, that initial peak response is related to the cupula. We expect that. But there's a gap between where we expect the cupula to respond in the yellow line and the blue dots at the bottom, which is the actual nystagmus.

So this red line is actually the neural integration. This is the velocity storage. And you can see here that this is what's going to prolong that nystagmus, and it's going to prolong a sense of motion. So perception's a funny thing. When we look at this in terms of clinical testing, I'm interested in the.

The nystagmus and the time decay. How long is that patient able to hold onto the response? But the patient's perception is also really quite interesting in that they, at one point will. So in this case, around 50 or so seconds into the tracing will actually perceive that they've stopped moving. They may consciously know that they're moving, but physiologically they feel that the chair has stopped.

And in some cases the fluid will start actually shifting opposite, and you can get a rebound of the nystagmus. So you can see that here with the nystagmus coming on now in the left beating. Now, the, the velocity storage mechanism is what's going to prolong that nystagmus. And so anything between 10 and 30 seconds after that peak is, is a typical velocity storage response or time decay. So the velocity storage is where we think part of the, if not part of, but most of the problem is for some of our vestibular patients.

So the velocity storage mechanism is supposed to be there. It's there because our vestibular system is really not great at understanding accelerations less than 1 hertz. Remember, our best response for the vestibular system in the VOR is around 1 and so below that, we're getting a lot of just inability of that cupula to perform and be as sensitive to that input. So the central system is trying to enhance that lower

performance. And so what it does is it creates this feedback loop between the nucleus and the cerebellum to Integrate some signals coming from the vestibular system, but also our visual system, somatosensory everything all together in order to sustain that activity.

So that is the difference between the cupula's response peaking at around 10, and our actual step velocity or time decay, which maybe doesn't decline until 20 to 30 seconds after the onset. So the reason I bring this up and kind of harp on it is really related to when this goes wrong. And what we find with many of our studies looking at the. These abnormalities in, in terms of velocity storage, because on the surface, physiologically, it makes sense that this should be where probably the problem is. We do find that there have been studies showing that abnormalities in velocity storage are there for people with severe motion sensitivity.

We see this related particularly to offloading the otolith organs. So as I said, in some of the aerospace or NASA studies, many of these were done with parabol. So you can get on YouTube and pull up some of the parabolic flight videos. And some of the data that they've pulled there is really quite interesting. But they are seeing these patterns associated with velocity storage and motion sickness.

But on the flip side, there hasn't really been much evidence to show that the actual VOR itself relates to motion sickness severity. So it's not necessarily that the peripheral system is not working the way it's supposed to, but it's that central integration, starting at the nuclei to the cerebellum, where this becomes challenging. And so, you know, in theory, should we expect prolonged time constants if we're doing step testing in these patients? I mean, maybe that's, it's possible. It's also possible that those are going to look completely normal, unfortunately.

But physiologically, this is where the problem is, is, okay, you know, I'm a diagnostician. I like numbers, I like data. Motion sickness is challenging in this regard, but the idea would be, can we find a clinical test or some sort of finding that can help us predict patients who may be more at risk for motion

sickness? And unfortunately, that's probably not the case. And this is, is, you know, nothing to be said about our diagnostic skills or even our tests.

It's just that motion sickness is incredibly complicated. And these are, you know, we, we know that there are environmental factors. So your anxiety level, you may be in a place that's really kind of smelly. You may have just eaten. And now we introduce emotion stimuli that's triggering this cascade.

So the flow of our motion sickness cascade is going to be triggered by numerous different components. And it may not be that These components trigger you every time, which is somewhat frustrating for people. But I think you can understand then the complexity and why this becomes a bit more challenging to understand and not quite as clear when we start looking at these patients clinically. So I, you know, I, I don't want to, you know, disparage. Maybe at some point we'll have a better tool that can identify or at least give us a better understanding of who may be most at risk or perhaps how this interplays with some of our other diagnoses.

But the, the multitude of players that are involved in this sort of cascade is really quite daunting. So the, there's, you know, there's not much particularly, I think that you're going to find on your, your diagnostics, aside from higher rates of dizziness with stimuli or perhaps increased nausea, particularly on your vor tasks. All right, so when we think about, you know, how do we quantify these, these patients and, you know, you never have to quantify motion sickness. We do quite often for research studies when we're looking at high versus low susceptibility for motion sensitivity. And this is typically going to be done using what's called the Motion Sickness Susceptibility Questionnaire or the mssq.

You'll see that quite often in the literature. Most of the time, though, you're not necessarily going to get a patient specifically just because they have motion sickness. It's typically they have migraines and subsequently they also have motion sickness. But there are quite a few different diagnoses that can present similar ish to motion sickness or maybe associated with symptoms that are like motion

sickness. So, you know, for people who have abrupt onset of motion sensitivity, that's always a little bit more concerning than people who have, of course, had it their whole life.

So in those cases where I tend to see it more often are in post concussion and acute migraine. You know, the rest of them are incredibly rare, and I hope you never see them in terms of showing up on your doorstep for testing. But anytime there's an acute onset, we want to at least make sure there's nothing more centrally mediated that we need to be concerned about. But those are patients who, you know, we want to get those over to their medical provider for evaluation if that's the case. But typically when you see this in acute patients, you're going to see it associated with concussion or migraine.

All right, so in terms of what some of the questionnaires you can do if you're so inclined, the motion sickness, the MSSQ is done in two parts. So I have it listed here. And again, this is one we. I typically am only doing this when we're doing a research study. And research studies just need to quantify some of these.

These metrics. So, you know, which bin are you going to put this patient in? Is this something that is, you know, pretty mild or. Or is this someone who is incredibly motion sick? Clinically, though, I mostly just ask them, do you get motion sick?

And. And people are really good at understanding what that means. If they're not quite as clear, I typically will then come back with, can you read in the car without getting sick? Because sometimes what I find with patients is that they've learned how to avoid it. And so if I ask an adult if you get motion sick, they may tell me no, and then quantify it as long as I sit in the front seat and don't read.

So I find that if someone comes at that response with no, then. And we come back with, let's give you a little bit more specific and get at that. Because many of these cases where you're dealing with more centrally central vestibulopathy, like migraine especially, there's a incredibly high prevalence of motion

sickness in this group. So I expect it to be there. All right, so the clinical presentation in your lab is.

Is it's highly variable for the most part. When you think about motion sickness, think about triggers that are going to integrate the visual and vestibular system. Most commonly, you're going to notice abnormalities in smooth pursuit. That's going to be your biggest one. And it may just look a bit more psychotic than you would expect.

OPK may also be something that's a bit more reduced. But another component of this is not necessarily the abnormality of the response, but the perception that that patient has of that stimuli. So they may be able to do smooth pursuit perfectly fine. They may just hate you for it. It's triggering that same sort of VOR pathway that is associated with their motion sickness.

Dynamic visual acuity is also one that you may find is helpful. It may or may not be abnor, but again, you're integrating a visual and vestibular task. In a patient who has abnormalities in this integration, that's likely going to also trigger symptoms. Now, some studies have shown that patients with motion sickness give higher caloric responses, but in general, they're still within our range of expectations. So it's not necessarily something you should expect to see.

There are occasions where you will see higher rates of velocity, but it's. It's not a Reliable metric and, and vemps really haven't been particularly helpful specifically when looking at motion sickness. There are other conditions, particularly migraine, where vemps are a bit more interesting. And so that may play a role there, but not necessarily in identifying motion sickness only. So there are a few other conditions that we associate with with motion sensitivity, the most common being vestibular migraine.

And in our migraine group, most of them are going to be prone to motion sickness. This is assumed to be part of this mechanism that's triggering the underlying vestibular migraine component. The dizziness tends to be very consistent. It's associated with abnormalities and integration, visual and vestibular

information. And it does tend to be something that, again, is more common in women.

There are numerous overlapping mechanisms. Most of them we associate with the shared pathways between the brainstem and cerebellum associated with migraine. And also perhaps an underlying overall hypersensitivity of the central vestibular system.

Meniere's is another one that can be associated with higher rates of motion sensitivity. They tend to be kind of in the middle. So they have more motion sensitivity than non Meniere's patients, but not as much as our vestibular migraine patients. And what's a little bit different about them is they don't typically describe pediatric onset. This is something that happens a bit more after the Meniere starts.

You know, the challenge, though, is that there is this relationship between Meniere's and migraine. And so maybe we're really just looking at that emotion sensitivity issue there.

In terms of other peripheral vestibulopathies, there really hasn't been a relationship noted. So think like labyrinthitis or bppv. Those tend to be fairly neutral in terms of prediction. And then the bilateral areflexia, I wouldn't expect you to see motion sensitivity there. All right, so in terms of counseling, we know that our patients are highly likely to demonstrate motion sickness.

So what information can we give them? You know, we're not giving medical advice, but there are plenty of other things that people need to be doing in order to manage. And the absolute most important piece is we need to prevent it. Prevention is going to be much easier than trying to cure it. We can give them some ideas about things to avoid.

Most of these should be fairly straightforward. You probably shouldn't eat something heavy. You know, you probably shouldn't be drinking alcohol if you're prone to motion sickness and then, you know, getting on a boat. Some, you know, other people don't realize these things and may still be trying to

scroll through their phone while they're in the car. So there, there are some things that we'll typically advise, most of which people are usually pretty good at picking up.

We'll also talk a little bit about breathing activities. We know that breathing can help to regulate the parasympathetic, sympathic, parasympathetic nervous system which can reduce our, our need to vomit. So that can be useful and you know, just to also give them information about habituation. We know that people can habituate. It's, it's just you're only going to be able to habituate to that very specific stimulus.

So if you know that I need to be on this tiny boat, you know, five days out of the week, every week for the rest of my life, well, you can usually habituate to that. There's going to be a smaller subset set of patients who can't habituate. And that's just, unfortunately they're just wired to perceive motion a bit more. Some additional things that you can recommend or their medical provider can recommend is kind of the usual suspects. Meclizine or other first generation antihistamines.

This helps to block the receptors in our vomiting center, which is helpful. These are safe to use during pregnancy, so they're kind of a go to. Unfortunately they are sedating so patients will sometimes not like to do them. One of the things that I think people mess up on, not mess up, but they don't realize. You have to take this before you have to pre game with these medications.

Again, if you're trying to stop motion sickness after it started, you're not going to be effective. So if you know you're getting in the car to go on that road trip trip, you need to pop the meclizine a couple hours before.

So additionally you're going to see this with other medications like scopolamine. This is something you actually need to do a little bit earlier. So four to eight hours before the exposure, but it does last a little bit longer. And this one tends to be a bit more beneficial for otolith mediated problems. So think like

boats and rocking sorts of sensations versus spinning.

But again, scopolamine is another common one that you'll see.

All right, so we know that our dizzy patients are likely going to get motion sick, particularly if you're working in migraine. There are loads of things that people can do to help reduce these effects. But knowing those environmental triggers is really quite important and understanding the environment that our patients are in. So I know that my, my high school concussion patients are likely going to be gamers. So we have to have those talks about what's going to be a trigger for them.

And then looking ahead as we start thinking about increased access to high speed trains, autonomous cars, as well as commercial space flight.

So in terms of kind of take homes here, this is incredibly common. Most of you have probably felt this and are at least maybe at risk for feeling this, but motion sickness is going to require the peripheral vestibular system in order to trigger this cascade. And just keep in mind, once you trigger it, it's likely not going to stop until you stop the offending stimulus. So prevention is your key here. And trying to reduce those symptoms and reduce the onset of that cascade will improve our outcomes and hopefully then reduce your sensitivity to that motion.

All right, so that's all I have for you. I'm sorry for going over a little bit.

So my email's there if you have questions that come up as well. And if you have questions now, I'm happy to take gum.

Thank you so much. Dr. Bogle, we do have a question related to the exam, which is what abnormality is commonly expected on vestibular laboratory testing in patients with motion sickness? Very good. Yes.

So the most common one that you will see will be related to the smooth pursuit pathway. So I apologize if I didn't mark that one on the slides, but that would be the most common, common one. Great, thank you so much. Sure. Yeah.

And there is one more question just about that Paul proposed about prolonged symptoms. So several days after exposure and it can be, it can be common. We think about this in terms of something like mild to debarkment, where you can have these prolonged symptoms. Now for everyone. You get off a boat, you're going to feel that sense of motion as you reacclimate some people just takes a little bit longer.

So it wouldn't surprise me a couple of days wouldn't be outside of the range of normal.

Another question came in about, you know, would you expect someone who has vestibular migraines to become more susceptible to motion sickness over time as they have more migraines? Maybe what we do find is that as the patient's migraine changes, perhaps to include more vestibular symptoms or perhaps the migraine severity increases. We do often see patients describe an increase in susceptibility to motion sickness. It's not something that I expect is damaging, but it is something that we do see. Patient's report.

All right, great questions. Another Alicia asked, can we expect PT to help with motion sickness, possibly in terms of habituation exercises. We'll often send patients to PT to help with some of those interaction components. And that's a piece that they can be really quite helpful with this.

All right, and it looks like final question Kimberly asks about neural mismatch or sensory mismatch being the, the, the common cause. And it's going to be sensory mismatch. So sensory system, just meaning your, your visual and vestibular system. I think I got them all. Wonderful.

Thank you so much. Dr. Vogel, would you mind advancing to the last slide, please?

Thank you, Dr. Bogle, for your time and expertise today and thanks to everyone who participated in today's course. As a reminder, for those seeking CEUs, your exam will be available in the next 10 minutes. And be sure to complete your exam within the next seven days for live credit. We look forward to your feedback on the course evaluations and hope to see you again in future courses on audiology online.

This concludes today's course. Have a great rest of your day.