The Use of EEG Biomarkers to Understand Cortical-Vestibular Interactions, in partnership with Vanderbilt University

Presenters: Daniel Romero, AuD, PhD, Tricia Stanley, AuD

Well, hello and thank you everyone for joining us for our webinar today on The Use of EEG Biomarkers to Understand Cortical Vestibular Interactions, which is presented in partnership with Vanderbilt University. It's my pleasure to introduce our presenters today. Dr. Daniel Romero is an audiologist and assistant professor and co director of the Division of Vestibular Sciences in the Department of Hearing and Speech Sciences at Vanderbilt University Medical Center. His primary research interests focus on the intersection of cortical vestibular interactions and their impact on dizziness and balance disorders. Dr. Tricia Stanley is a recent AUD graduate from A.T. still University in Mesa, Arizona.

She is currently completing a vestibular fellowship at Vanderbilt University Medical center. So welcome Dr. Romero and Dr. Stanley. The floor is yours.

Awesome. Thank you very much. Welcome everyone. And we're just so excited to to be here today to present on some of our interesting research. So these are just some of our disclosures we our limitations and risks and just some of our learning outcomes.

So we're going to cover a lot of different topics today. Much of that is going to be new material from the standpoint of normally the areas that we're concerned with within clinical vestibular audiology. And so we're going to just start with an overview of just talking a little bit about the cortic what do we mean by cortical vestibular function beyond just the traditional vestibular ocular reflex or vestibulospinal reflexes? Talk about which key brain areas are involved in processing this afferent vestibular information going up, you know, from the inner ear up to the brain? We're going to talk a little bit about EEG as a tool to how we measure cortical activity and just some of the ways that we're using it.

We're going to talk about just some of the vestibular EEG research, as well as some of our initial investigations and future directions.

So for us to really consider some new concepts, it's really important for us to understand how we currently view our world. Traditionally, we understand the vestibular system for its amazing ability and flexive control of gaze, posture, and gait, which primarily consists of the peripheral and central vestibular pathways.

However, we know that the vestibular system is much more than purely a rudimentary reflex. In fact, we know that the brain utilizes information that it receives from the inner ear much more often than we realize. And now why would we actually want to look at more higher level cortical processing in terms or in the Context of the vestibular system. Well, we know that most clinical assessments of vestibular function are limited to the structural integrity of the VOR or vsr. We also know for many of us doing clinical vestibular testing out there, that many patient populations experience symptoms that are not primarily attributed to peripheral damage.

These can include those with 3 PD, vestibular migraine, Maldeida, barkment, especially the dizziness imbalance symptoms that patients with traumatic brain injury can face. And the research is pretty clear that these specific patient populations, their issues are primarily involving higher order processing of vestibular input within the cortex.

However, additionally, we know that for many clinicians, a diagnosis of a vestibular disorder is really made solely when peripheral involvement is rolled out. And in addition, those with central vestibular impairments are often diagnosed with little to no knowledge of the underlying pathophysiology. It's usually a clinical diagnosis based on meeting certain criteria and then as well as there's really no wellestablished method for quantifying some of the disruptions involved in cortical vestibular processes.

Um, again, we know that the vestibular system is not solely a system of brainstem reflexes with no ability or with no complex processing used by the brain. In fact, once the vestibular, once vestibular information reaches the brain stem, some of that information, a lot of that information actually continues

to travel up through the brain. It passes through the thalamus and is essentially widely distributed across the brain. Some of these major areas include again the thalamus, the parietal insular, vestibular cortex, areas within the temporal parietal areas, posterior parietal cortices, the occipital load, some of the premotor cortexes, as well as the hippocampus and paro hippocampus.

So once signals reach these areas, it passes through the thalamus and then gets widely distributed. And so what we can say for sure is that vestibular signals are widely distributed once it passes through the thalamus and through direct and indirect pathways. And there isn't necessarily one single vestibular cortex, unlike our auditory cortex.

Another couple interesting notes about the vestibular projections. Once they reach these higher level areas, they are primarily bilateral pathways. And however, research has shown that this activation may favor the hemisphere ipsilateral to a stimulated ear or ipsilateral to someone's handedness. However, nevertheless, there's, there seems to be bilateral vestibular projections that are dispersed throughout the, throughout the brain. In fact, there was a very interesting study by Surgeon et al in 2017 that examined 37 studies that primarily focused on examining brain structures involved in balance using FMR or using MRI and the top structures involved in balance.

Not surprising was the cerebellum more than any other brain structure. This should not be surprising to any of us. However, all of the regions beyond the cerebellum were several other cortical or subcortical structures and these included the occipital regions, parietal regions, the corpus callosum as well as the hippocampus and frontal and temporal regions. So there is a lot of solid evidence out there to suggest that that the cerebellum and the brain stem reflexes are sort of supported by higher level centers.

Now there are several cognitive functions that rely on the use of vestibular input input from the periphery. These not this not only includes multi sensory integration but also memory function, self motion perception, postural stability, attention, bodily self consciousness, someone's ability to rotate an

object within their mind has been associated with some type of relationship to the vestibular system. So there's a lot of high complex functions that cognitive functions that use vestibular signals to influence some of these higher level tasks.

Now obviously this is not going to be an exhaustive list, but I will be highlighting a few different areas. So what are just some of the functional clinical manifestations of cortical vestibular deficits?

One manifestation is the absence of self motion perception. This was a paper that was done by Piker and colleagues in 2014 and this was a really interesting study. They had looked at self report symptoms, but differences between younger and older adults experiencing dizziness. And what they ended up observing was that younger adults reported significantly more instances of vertigo and associated nausea and vomiting compared to the older adult populations. Older adults tended to report more symptoms of unsteadiness or falling and less symptoms of actually true vertigo.

And despite the lack of these vertiginous like symptoms in the older adult population, this older adult population actually had more instances of BPPV which produces a true vestibular sensation vertiginous sensation. So even just the the symptoms that patients will report will differ depending on whether they're more younger. And so age seems to have an impact on just someone's ability to report vertigo.

This was Followed up in 2017 by Jacobson ET al. And they ended up looking at the absence of caloric induced vertigo and found that both age and SPV of the caloric response were significant predictors of vertigo perception during the caloric exam. And overall to show evidence suggesting and supporting that age related changes in the central processing of the vestibular system could be relevant or could be present in some cases, especially when we're looking at age related changes in the elderly.

I then joined in for a follow up study, but before I talk about that one, I'm going to talk a little bit about what we mean by the absence of self motion perception. It's been described by a few different, it's gone

by a few different names over the past several years. It's been described as vestibular agnosia, nystagmus sensation, dissociation. However, more recently it's, it's, I think vestibular agnosia is going to, going to stick. That seems to be the most, that seems to be the verbiage that is used most often in the literature now.

But vestibular agnosia simply means the absence of vertigo perception. And so this has been described not only in older adults like I just described, but it's also been described in other clinical populations like acute tbi, white matter disease, those with a history of falls, especially in some cases where patients are experiencing BPPV without the vertigo symptom.

Secondly, self motion perception, based on the available literature, is really thought to be mediated by a functional brain network, by a network of different regions rather than a specific brain network region. And studies have suggested that this network and the connections between specific regions has a, a real big impact on whether or not somebody can perceive their own self motion. And this has been, I think to date been most closely related or associated with a somebody's ability to remain stable on their feet. And so there have been some studies that have examined, okay, what is the effect of absence of self motion perception on someone's postural stability? Well, we took a look at this in 2020 and we looked at the clinic, basically the clinical significance to failing to perceive caloric induced vertigo.

And so we, we looked at 25 older adults, all with normal vestibular function tests. So we ran a comprehensive vestibular function test on them. Every single person enrolled in this study had a normal test. They had normal vamps, vng, rotary chair and vhit. And they were divided not based on their test results, but they were divided based on the presence or absence of whether or not they perceived vertigo during their caloric exam.

And so we had a motion perception group and an absent perception group. And despite no significant differences between the groups on any of the measures of the comprehensive vestibular function test,

the absent perception group showed greater postural instability and greater falls during Condition five of the sensory organization test. Now just to remind you, the sex sensory the condition 5 of the SOT is where eyes are closed and they this the platform is sway referenced. And so primarily within that condition, the individual has to primarily utilize vestibular cues for balance. Well, this is interesting because it tells us that there may be a link in order for somebody to utilize those vestibular cues, there may be a need to perceive those vestibular cues.

And so those with the absent perception group, this really I think kickstarted a lot of our interest in some of these higher level cortical processes involved in everyday vestibular like tasks. And so all of this really to say is that there seems to be a disconnect between in some patients between what we measure and what patients report. And so how do we essentially move beyond the brainstem to understand how the brain is utilizing or interacting with vestibular signal signals that are arriving from the periphery.

And that kind of brings me into eeg. Now there are a ton of methods to use to measure brain activity and some of them have advantages over others. For example, EEG is, usually has excellent temporal resolution. So it can measure very fast moment to moment changes happening in the brain. However, you don't necessarily have a great way of localizing where these changes are happening within specific regions.

So EEG has great temporal resolution and poor spatial resolution. This is sort of the opposite to an MRI or functional mri. An FMRI has excellent spatial resolution. It can identify specific structures within the brain with incredible accuracy. However, it has very poor temporal resolution.

And so it doesn't have the ability to measure these fast moment to moment changes that are happening. And so I'm going to be focusing more on these moment to moment changes utilizing eeg. And so the EEG signal, what are we actually measuring here? Well, it is a direct measure of brain

activity. You place them electrodes on the scalp and you are measuring pyramidal cells is in fact the discharge and the electrochemical signals that these cells kind of give off.

They create an, they create this potential that travels, that travels through the scalp and is able to be detected by the electrode. And so these pyramidal, pyramidal cells are made up of dendrites. And what happens is when the charges positive charge within the extracellular space, it creates a negative potentials that allows. And if all of these pyramidal cells are synchronized, you're talking about thousands or hundreds of thousands of pyramidal cells synchronizing their potentials that is large enough to be picked up by the scalp electrode.

And so Again, thousands, even sometimes millions of these cells, which are active synchronously and within the same orientation at the same time, can travel through the scalp and get picked up by the electrode, which is essentially all EEG is. And EEG itself has long been utilized to measure brain activity. And it can really be broken up five main frequency bands. You may have heard these, their names in, in the past, but it essentially is based off of their frequency. And so we have the delta rhythm, theta rhythm, the alpha rhythm, the beta rhythm, and the gamma rhythm.

These are all these oscillations that the brain naturally produces are all make up the EEG signal. And so you can find each frequency band kind superimposed on one another within the raw eeg. And EEG itself has been, has been used for a long time to assess neural dynamics in both normal and abnormal clinical populations with different diseases or disorders affecting the brain. For example, it's been most used in patients with epilepsy, dementia, those with attention deficits, perception, communication, or even those disorders affecting state of mind. Most of the time, EEG is looked at by measuring some type of baseline within a certain frequency band and then looking at some type of experimental condition and then comparing how that frequency band changes in response to the manipulation.

And so it's often characterized by either a decrease or increase in this oscillatory activity happening, which can be referred to as either a suppression or enhancement, respectively. And so I'll talk a little bit more about primarily the alpha rhythm as we, as we move forward, but this is sort of a higher level view of how these frequency bands are not only represented across the time domain, but also across the frequency domain, which is the figure on the bottom right.

And typically when you're, when you're talking about EEG recordings, very similar to, to as far as the, the uses utilizes the 1020 system. So very similar to how we would measure an ABR in terms of where we place our electrodes and the name that it goes by. And again, this system relies on the anatomical points from the back of the, from the inion to the tip of the nose. And usually they're assigned letters. We have F for frontal, P for parietal, C for central, T for temporal, O for occipital, and A for usually the earlobes.

And these recordings can be analyzed across both time and frequency domains. So a lot of this, especially given our familiarity with evoke potentials, auditory and vestibular evoked potentials, a lot of this is actually very much overlapped. We're actually collecting EEG information and this, this oscillatory information. When we're measuring any of a potential that is coming from the scalp, we just consider it noise and we actually just filter it out. So at the top is, is some of this, this is I believe the alpha rhythm that's represented across the time domain.

And then in the middle plot it's represented across the frequency domain. And then you have your traditional time frequency domain plot.

These data can also be. These same data can also be represented spatially. Again I mentioned earlier that EEG has poor spatial resolution, but nevertheless it's, it can also. Multiple studies have been, have tried to plot at least regional activity, not necessarily a specific brain structure. And so typically you'll sort of see these types of plots within certain EEG studies which are known as scalp topography plots

where it will show basically the differences in voltage across the scalp.

These rhythms are, are actually very stable across species. And so these, for example, the alpha rhythm can not only be measured within a human being but also multiple other species. And so it really, these rhythms actually favor good translational EEG research, both those that are utilizing human participants, but also those using animal models. Obviously some of the advantages I talked about, direct measure of brain activity, it can really match the speed of cognition as far as EEG and its moment to moment changes capabilities. You get a lot of rich data with eeg.

Sometimes it is overwhelming, but obviously there are some disadvantages. The potentials that you're measuring from these pyramidal cells have to really be large enough to travel through the scalp. So there is definitely some loss of information that you are not necessarily able to measure via eeg. So there are some things happening obviously within the brain that are invisible to eeg. I talked about the uncertainties in the anatomical localization and then obviously very time consuming in just some of the pre processing and some of the data analysis that occurs.

One of the other things that I mentioned, the poor spatial resolution, this is like largely due to a phenomenon know known as volume conduction. And what I mean is, you know, whatever. Let's say you have an electrode right there, the one that's highlighted in orange. Let's say that that is at the central, central parietal region, whatever that electrode picks up. Yes, probably includes pyramidal cells directly within that region, but it may also include large scale potentials from distant regions as you can see here.

And that's what I mean by, that's one of the main drivers and reasons why EEG has poor spatial res.

Other instances or events happening within the brain, if they're large enough, they can travel, travel and spread across multiple electrodes which may not reflect the region that that electrode is assigned. So

this is just again to show you why EEG has poor spatial resolution.

I mentioned some of the pre processing that's involved with some EEG data.

Obviously it's very time consuming, especially if you're kind of working with some of the raw data. It can often be tedious, not, not the greatest, not fun. But if you do it well and do it right, you can do it, you can do it the first time and you don't really have to worry about it from that point. But, but prior to analyzing any EEG signal, it does need to go undergo some processing steps to reduce noise and artifacts, just like with anything else. And so even with some of our evoked potentials that we tend to use, so similar to like an abr, EEG may be affected very heavily by muscle and eye blink artifact.

And so certainly we're trying to filter a lot of that out, projecting those bad channels, interpolate, interpolating those channels where necessary to, and then kind of referencing our data, which data segments are we trying to extract. And then once all of that pre processing happens, we're able to then sort of look at those different frequency bands and have some confidence in what we're, what we're actually looking at.

So this is an example of just some raw and some raw EEG data that we've kind of worked with at some point. And so what you can see is a bunch of waveforms there. These are all different, different scalp electrodes. So each, each waveform represents a different location on the scalp or different scalp region. So you can see it goes from the temporal parietal.

If you see an odd number, that usually means on the left side of the scalp. An even number represents an electrode on the right side of the scalp. So all the way from temporal parietal down to parietal occipital. And what I'm highlighting here with the red arrow is a moment is just an example of how we might clean some of these data. You can see that the temporal parietal electrode on the right side, the participant moved and created some muscle artifact there.

So we usually apply some type of algorithm to isolate and reduce those artifacts as much as you possibly can while trying to preserve the oscillatory activity.

And you can see the full clean version underneath the raw. So not exactly as messy, but completely not taken away either. So you sort of have to kind of go through this, this nice balance of, of what to of of your pre processing.

So now I want to kind of shift gears a little bit just now that I've given you sort of a, some more information about what we mean by EEG and how that may be different from our standard clinical evoked potentials. We're going to focus on the alpha band because it is by far the most studied and is easiest to calculate and identify. This band was first described by Hans Berger in 1929. So they've known, we've known about it for a long time. And it's been traditionally understood as an idling of the brain.

So if you're kind of in a relaxed state, you're not, your eyes are closed, you're not really doing much, the alpha rhythm is present. And as soon as you start to open up your eyes, as soon as you start to interact with your world, the alpha rhythm starts to diminish. And so it's, it's been always kind of understood as this passive idling that the brain undergoes, however, and its precise physiology has historically been poorly understood. We didn't necessarily know exactly where this rhythm was being generated. They knew it was primarily being generated somewhere within parietal and occipital regions, but still within as far as certain structures not really able to directly identify.

So the alpha rhythm, like I mentioned before, really dominates the EEG signal during quiet wakefulness, especially when the eyes are closed. And over the years the alpha rhythm has been attributed to more and more functions. It's really been, it is not uniform, it doesn't necessarily serve one purpose. It's been, studies have shown that it's, that this alpha rhythm can vary based on someone's

age, someone's mental state, during, with and without a cognitive task. More recent literature has suggested that there is, is the alpha rhythm has an intimate role in interacting with the thalamus, interacting with processing sensory information from the thalamus to the cortex, but also from the cortex to the thalamus.

And so it's really been, I think more recently been suggested that it plays some important role in the thalamocortical loop.

And so I think more recent literature has shown that rather than a passive idling or noise that is happening within the brain, the alpha band represents an active and dynamic signal processor. In fact, there seems to be a network of regional alpha generators. So the alpha is not necessarily being generated by one specific area, but rather a network of alpha generators used for different aspects of sensory, cognitive and multi sensory integration. And you can see the alpha rhythm here on the right side. Again Each row represents, you can just see that beautiful oscillatory activity.

And, yeah, you can kind of see it there. But if I take a little segment of that, that kind of, that, that oscillatory activity that's occurring is the alpha rhythm that is purely visible within the time domain.

And so how has the alpha rhythm been used to understand higher level vestibular stimulation or vestibular processing? Well, I'm going to talk about a couple of different studies here, but this was a study by Gail in Gail and colleagues in 2016. And they looked at the alpha rhythm, different frequency bands too, but mainly the alpha rhythm evoked by natural vestibular stimulation in humans. And they looked at 22 young healthy adults and nine patients with bilateral vestibular loss. And what they observed was that in the healthy group, participants demonstrated a suppression of the alpha band, primarily in bilateral, temporal, parietal and occipital regions.

So they would measure their baseline at resting and then they would give them the stimulation and see how the differences in the alpha rhythm changed. And they saw suppression of that alpha band within certain regions happening in a young, healthy group. However, in patients with bilateral vestibular loss, that suppression that occurred was significantly smaller.

Other studies have looked at and, and this finding of alpha suppression, I'm gonna, I'm gonna focus on this because this kind of directly feeds in some of the, the current work that we're doing. But alpha suppression in and of itself has been replicated in at least two different labs and one in Europe, where they looked at eight healthy individuals and they performed real rotation and static rotation. So there were the static rotation served sort of as this, as the sham trial. And they also demonstrated a suppression of the alpha band during the real trials, but not necessarily in the static. So there seems to be, within the last five to 10 years, an understanding that the alpha rhythm suppresses during sensory stimulation.

This was a study in 2023 that looked at 20 young healthy adults and looked at how their alpha rhythm changed during the caloric test. And what this study observed and reported was that there were changes within the alpha rhythm during caloric stimulation. And they also demonstrated a possible correlation or association between their, an individual's alpha rhythm and their peak slow phase velocity.

And so interest in EEG to study higher order vestibular processing has really grown over the years and primarily driven by advances not only in technology, but also techniques and in an understanding of what some of these rhythms actually mean. Newer evidence does show distinct changes within the alpha band as well as some other bands. But again, I'm going to focus, we're going to focus primarily on the alpha rhythm during controlled physiologic stimulation. And so some of these studies have a TR has attributed that EEG alpha suppression suppression during vestibular stimulation could reflect

cortical engagement within these multisensory vestibular areas that are happening within, that's happening within the brain. And so Dr. Stanley is going to talk about just some of our initial investigations into some of this work because we really wanted to not only rep.

Replicate some of these things, we, we know, we've, we've read across, we've read about some of these studies, but we wanted to see it ourselves. And so we wanted to not only replicate it, but also try to isolate, you know, some of these bottom up versus top down processes happening. And so I will go ahead and turn it over to Dr. Stanley.

Thank you, Dr. Romero. So I will be discussing two initial investigations that have been completed at our lab here at Vanderbilt. Next slide please.

Dr. Romero, can you change? Perfect. So the first study was completed in fall 2024. The focus of this study was to kind of take an exploratory approach in developing clinically practicable methods for measuring EEG during caloric testing and then also examining its relationship to the specific lower order peak SPV response that we see with calorics and also higher order or self motion perception during caloric testing. The goal of this study was to be kind of an exploratory study while also motivating future research in this area.

Next slide please.

So this first study involved 10 participants younger than 40 years of age. Nine female participants and one male participant were in the study. All participants were right handed. And then they had to not report a history of otologic symptoms, migraine headache or other neurologic issues. They were hooked up to a 128 channel wireless EEG system and net station 5.4 software.

Some of that you can see in the photo on this slide. Participants were placed in a supine position with the head elevated between 20 and 30 degrees. And then they were under video goggles for this, this time period. So there was a resting period that was recorded while they were laying there. The EEG cap was recording and then there also was a caloric stimulation period.

Each of those were two minutes long. And I'll go into that a little bit more on the next slide. During the caloric stimulation period, the irrigated ear was chosen based on the handedness of the participant. So in this case it was always in the right ear. Because all participants were right handed, the stimulus was delivered at 44 degrees Celsius for 30 seconds at a flow rate of about 500 milliliters per minute.

After this, participants were given a vestibular relevant task. They were asked to rate the intensity of their sense of motion from a scale of 0 meaning no perception to 100 with maximum perception. Next slide please.

So after this, after the participants underwent the procedures, an EEG analysis was completed. I want to first turn your attention to the picture on the right side, top right, where I just wanted to point out that there was the first 30 seconds of the 2 minutes recording was taken out just to try to eliminate some of any non vestibular effects on the EEG signal. So the remaining minute and 30 seconds was what was analyzed. For the purpose of our study and these results, it was used a 200 Hz sampling rate. Only high quality or good channels were included in the analysis using the criteria listed below.

These good channels were used to calculate the relative changes in spectral power in response to the caloric stimulation or an event related change. The equation that was used for that can be found on the lower right side of the slide. The event related change was meant to show suppression and enhancement in cortical activity during the caloric stimulation. And then the EEG power for each frequency band was calculated using Welch's method for spectral density power spectral density estimation. And then the analysis was performed using a custom program in matlab.

Next slide please.

A Shapiro Wilk test was done and showed that data from the individual EEG channels that was used to calculate that event related change was not normally distributed in any of these four frequency bands that were looked at, as you can see in the figure on the right side as well. Next slide.

Perfect. And then from there a Wilcoxon signed rank test revealed that the greatest and most consistent change in EEG power in response to caloric stimulation was observed in the alpha band. So you can see that in the chart on the bottom of the page in that red circle and then also in the chart kind of where the alpha band is next to that red arrow.

This difference in alpha power between resting and during the caloric periods was significant. All participants in this study showed caloric induced suppression within the alpha band. And the relative and of note, the relative difference in power for delta, theta and beta were not significant. So the only significant change that we saw was in the alpha band. Next slide please.

And this is just a really nice visual. You can see that in this figure. This is an example from one of the patients or One of the subjects of this study you can definitely see on the left is the original kind of alpha oscillatory pattern and then you can see right after that vestibular stimulus it really does suppress quite significantly. Next slide.

In summary, in this first study we develop or the lab developed a novel method of analyzing the EEG signal relative to the caloric test. While the use of EEG during vestibular stimulation exists in current literature, as Dr. Romero pointed out, advancements in both stimulation techniques and analytical methods have clarified its utility with modern studies providing robust evidence of distinct EEG changes in response to vestibular activation. So in this study we observed group level suppression of the alpha

band. However, it was still unknown whether these observations were being primarily driven by a bottom up process or due to the caloric stimulation, or whether it was more due to a top down or cognitive task process that was causing the suppression. Next slide.

Second study happened over the summer of this year. The goal of this second study was to expand on the findings of the first study where alpha suppression occurred in response to caloric testing. The lab essentially wanted to investigate if this alpha suppression was because of the caloric stimulus or if it was something else that was causing it. This study had two aims. The first one was to evaluate the vestibular and cognitive contributions to alpha suppression by comparing real and sham caloric stimulations with and without mental tasking.

We predicted that real caloric stimulation without tasking would suppress alpha more than a sham, reflecting that vestibular input and caloric stimulation with tasking would produce greater alpha suppression than caloric stimulation without tasking reflecting additional cognitive demand.

We also wanted to see if there the degree of suppression observed during caloric stimulation was influenced by baseline cortical excitability. Research has shown that resting alpha state offers a stable index of baseline cortical excitability, which is why we were interested in that. Specifically, the second objective of this study was to examine whether individual differences in resting alpha power can predict vestibular responsiveness as which was measured by peak slow phase velocity from caloric stimulation. In the study we hypothesized that there would be no relationship between these factors since the caloric test is clinically considered a test of end organ and brainstem reactivity and not something more cortical. Next slide please.

In this study there were 20 healthy adults ages 18 to 39 years. This time there were 15 female participants, 15 male participants. Again, they were required to have no history of hearing loss, neurologic disorders, dizziness concerns or vestibular migraine history EEG was recorded on each of

these participants while they underwent caloric testing and also during resting period. It was recorded using a 64 channel cap as pictured in these diagram in the pictures below. And it was this peak SPV was derived via electronystagmography or ENG during the 90 seconds after the 32nd caloric irrigation data was the data was down sampled to 200 hertz.

It was band pass filtered accordingly and re referenced and linked to mastoids. So participants underwent three different experimental conditions. One of them being a sham caloric which I'll go into more detail about on the next slide and two being traditional calorics. But one of them they were given a mental task, the other one they were not given a mental task. Next slide please.

So just going into each of these conditions that the patients underwent so resting condition first this was to establish baseline eeg, that baseline alpha rhythm that we were interested in that lasted for about a minute and a half. Patients were just asked to lay with their eyes closed in the narc. The sham caloric involved body temperature air being delivered into the earth. It again aligned with their handedness with a flow of upgrade of about 8 liters per minute. Again they were tasked with keeping their eyes closed just laying there in the dark.

This was not a condition where we expected to see a caloric response just because when just because it's the same temperature as the body. And as we all know, calorics do elicit a response because of the changes in temperature that they caused. So when it came to a real caloric without a task, warm air was delivered again at aligned with the handedness, same flow rate. Participants had their eyes closed in the dark and then a vestibular response was measured. That's it.

They were not given anything else to talk about after the irrigation. And then same kind of general procedure in the caloric with mental tasking, except for the patient was asked to count out loud by threes while they had their eyes closed and were laying in the dark. And then again a vestibular response was measured in all of these as peak spv. Next slide please.

So once all of this data was collected, a one way ANOVA test was run and it showed a significant main effect. So post hoc analysis confirmed that the real caloric with tasking was the only condition that resulted in a significant suppression of the alpha band. And that's highlighted really well in the figures that are on this slide as well. As you can see, there was no difference between the sham and real calorics without tasking but alpha suppression was significantly greater when participants were tasking or they were counting up by threes. This suggests that their active engagement with the task was likely driving the more and suppressing the alpha rhythm.

Therefore, it suggested that this event related change was not primarily driven by vestibular stimulation or part of a bottom up process, but it is more likely influenced by the mental task which supports the idea of more of a top down process process. Next slide please.

So looking back into AIM two, the resting state alpha power was analyzed to assess its relationship with the peak SPV at the individual level. As you can see, the resting state alpha power negatively correlates with peak spv, which is what the graph on the right is showing you.

In the study that was completed, resting state alpha power accounted for 34% of the variability within the total peak SPD. Therefore, greater resting state alpha power was associated with a smaller total caloric response, which was an interesting finding. Next slide please.

So in expanding on the original study, we were able to determine that the alpha rhythm only suppressed significantly when there is a mental task added and not during a real or sham caloric stimulation without a mental task. These results suggest that a caloric induced alpha suppression is primarily top down and not really driven by the caloric stimulus itself. Further, an inverse relationship between the resting baseline alpha power and the strength of the vestibular response could indicate the role of alpha oscillation in top down suppression of the vor. Next slide please.

So there are some limitations to the studies that were completed and these are definitely some important things to consider. The first being that both samples were very young and very healthy. We also wanted to point out that the there are some limitations of the caloric stimulus. It's not a precise stimulus and it's technically kind of more artificial and it's not. It's not really present in real world scenarios.

So. And then it also kind of limits ecological validity since the activation of the semicircular canals does normally occur in pairs and in caloric testing. As we know, we're activating one lateral semicircular canal at a time. And then we also need to consider the complex interactions occurring simultaneously. As Dr. Romero talked about during the beginning of this lecture.

We also need to think about things like novelty to stimulus multimodal effects on alpha suppression, including tactile and auditory stimulation, as well as generalized attention and anxiety. So we attempted to minimize some non vestibular effects on the EEG by removing the first 30 seconds, as I mentioned from the analysis. But it is still possible that non vestibular effects could be contributing to what we found. Next slide, please.

All right, so it is clear that vestibular information reaches these cortex. Reaches the cortex. And these findings do have real world implications seen on patients that are in our clinic.

And then some of these populations that we think this can be expanded to are patients that have traumatic brain injury. So there are clinical implications on cognition and postural stability in a variety of clinical populations, including these traumatic brain injury patients. And so I wanted to talk about that a little bit further and talk about next steps. Next slide.

So while clinical evidence outside of experimental studies show very high like results with high variation, which is largely associated with the heterogeneity of a patient with a tbi, for those of you who have seen patients with TBI in clinic, we all know that no two patients with traumatic brain injury are the same. However, there is a very consistent finding across studies, and that is that patients with a traumatic brain injury tend to have difficulty maintaining balance that are during tasks that require a high use of vestibular input. So as Dr. Romero was talking about condition five on the sensory organization test, or when they have the eyes closed and the platforms kind of moving, that can be very, very difficult for them because it relies mostly on their vestibular system. Even though a large portion of these patients have normally clinical vestibular exams, they still have difficulty with this maintaining balance and when they need to rely on their vestibular system. Next slide, please.

So these are two figures from two different papers. The one on the left is from aiken et al. 2022. This basically shows that equal the difference between equilibrium scores in patients with a TBI, as indicated by the yellow circles, compared to control patients that do not have a history of tbi. And the blue triangles.

And then the second figure on the right is from a paper by Taylor and colleagues in 2021 which looked at all aspects of vestibular function testing. They show that there was a lot more difficulty in maintaining postural stability using vestibular cues in patients who have TBI when compared to other abnormalities from other vestibular tests. So again, this is kind of just re emphasizing the fact that despite having normal kind of more practice, more common vestibular function test findings, there are still a lot of difficulties in maintaining postural equilibrium in patients with tbi. So this is another study by Taylor et Al in 2021. It looks at equilibrium scores across the different sensory conditions in the sensory organization test.

As you can see, the scores when the patients had to rely on vestibular input are significantly lower than when they relied on vision and somatosensory information, despite the results of a normal standard vestibular function test. Again, this suggests that the balance issues in patients with TBI are not peripheral in nature and likely come from somewhere higher up in the system. Next slide.

So what we want to determine is if EEG can provide information on how the brain is processing information during these higher based vestibular tasks. So we want to see if the data that we find on EEG can be better correlated with patient symptoms, self motion perception and functional performance. So these questions are still outstanding. Next slide.

Looking forward, we have determined that EEG offers a non invasive window into looking into the cortex supporting cortical vestibular processing, postural control and self motion perception.

There is potential that bio EEG biomarkers could offer insights into vestibular function, and these EEG biomarkers could also hold promise for advancing the diagnosis, prognosis and rehabilitation of central vestibular disorders. Thank you. Next slide.

All right, thank you very much.

Thank you so much. And we'll go ahead and take any questions.

We do still have a few minutes for questions, so if anybody has a question, please feel free to type it into the Q and A pod. I found this very fascinating. It's been a long time since I've done vestibular testing clinically, but so this brought back a lot of memories and was really kind of fascinating to see how you can get into kind of the upper level cortical processing of, of the vestibular vestibular signals. That's pretty amazing.

Interesting to see where this work goes moving forward.

So I would like to thank Both of you, Dr. Romero and Dr. Stanley, for sharing your time and expertise with us today. And I'd also like to thank everyone who participated in the course. I'd like to remind you that the quiz will be available on your pending courses page in the next 10 or 15 minutes. And please thank you for forwarding. Thank you so much for advancing the slide.

Yes, please remember to log into your account and look for that exam in the next 10 or 15 minutes. Also remember to complete it in the next seven days so that you can earn the CEU credit. We also look forward to getting your feedback on the course evaluations. This helps us to improve our courses and bring you topics of interest in the future. So that concludes our webinar today.

Thank you again for attending and we hope to see you in the future on courses. Have a great rest of your day.