# Exploring the feasibility of mitigating VR-HMD-induced cybersickness using cathodal transcranial direct current stimulation


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***Abstract*— Many head-mounted virtual reality display (VR-HMD) applications that involve moving visual environments (e.g., virtual rollercoaster, car and airplane driving) will trigger cybersickness (CS). Previous research Arshad et al. (2015) has explored the inhibitory effect of cathodal transcranial direct current stimulation (tDCS) on vestibular cortical excitability, applied to traditional motion sickness (MS), however its applicability to CS, as typically experienced in immersive VR, remains unknown. The presented double-blinded 2x2x3 mixed design experiment (independent variables: stimulation condition [cathodal/anodal]; timing of VR stimulus exposure [before/after tDCS]; sickness scenario [slight symptoms onset/moderate symptoms onset/recovery]) aims to investigate whether the tDCS protocol adapted from Arshad et al. (2015) is effective at delaying the onset of CS symptoms and/or accelerating recovery from them in healthy participants. Quantitative analysis revealed that the cathodal tDCS indeed delayed the onset of slight symptoms if compared to that in anodal condition. However, there are no significant differences in delaying the onset of moderate symptoms nor shortening time to recovery between the two stimulation types. Possible reasons for present findings are discussed and suggestions for future studies are proposed.**



***Keywords—HMD-VR, vection, tDCS, inhibition of vestibular cortical excitability, mitigation of cybersickness.***


## I. INTRODUCTION

Virtual reality (VR) environments are designed using three technologies: non-immersive VR [1], semi-immersive VR [2] and immersive VR (IVR) [3]. There are two commonly used forms of IVR [3]: cave automatic virtual environments (CAVEs) and head-mounted displays (HMDs). A CAVE is a specially designed room in which the walls, ceiling, and/or floor are covered with a screen that can project virtual images or videos. An HMD is a VR headset that positions two small screens in front of both eyes, completely blocking out the physical world including the user's body, and allows users to turn their head to examine their surroundings, with the visual presentation moving in the opposition direction of head motion with low latency [3]–[5]. Although a CAVE has many immersive qualities, the current state-of-the-art consumer-friendly HMDs, including PC-powered HMDs (i.e., HTC Vive™), smartphone-based HMDs (i.e., Samsung Gear™), and all-in-one HMDs (i.e., Oculus Quest™), aim to achieve immersive effect in a manner that is both simple and inexpensive.

A serious problem with VR-HMD is that users may develop symptoms similar to motion sickness (MS), a malady called pseudo motion sickness [4], visually-induced MS [5], or cybersickness (CS) [6]. This is unsurprising if the cause of CS can be explained by sensory conflict theory——a widely-accepted theory regarding how MS occurs [7]. That is to say, a sensory mismatch between the vestibular and visual signals results in MS. In the case of VR-HMD, many applications that involve moving visual surroundings (e.g., virtual rollercoaster, car and airplane driving) may elicit an illusory feeling of self-motion, namely vection [8]. Once vection is not supported by corresponding vestibular information (that is, actual physical movement is absent), the visual-vestibular sensory conflict will trigger CS. Regarding the prevention or mitigation of CS, a direct solution is obviously to design a highly immersive VR that combines physical movement (e.g. typical consumer roomscale VR experiences), or utilizes locomotion techniques to avoid vection-inducing visual stimuli. However, such approaches are not always preferable, with physical movement requiring both effort and space, and locomotion techniques impacting immersion. Apart from the VR design itself, two types of indirect methods have been


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proposed, in order to mitigate CS in a simple way: 1) adaptation training; 2) electrostimulation.

Adaptation training requires users repeat the same VR experience over and over [9]. This kind of method is indeed simple, but quite time-consuming. More worryingly, it is likely that once an individual becomes habituated to the virtual world, they might be maladapted to the real world on VR cessation [10]. This phenomenon has something in common with a special case of MS, called 'mal de debarquement', 'land sickness' or 'adaptive aftereffect' [7], [11]. One possible explanation is that a new orientation mechanism replaces the prior stored one with repeated exposure experience so that MS no longer occurs. However, once the newly established mechanism is facing reorientation, symptoms of MS occur once more, such as astronauts after returning to earth from a long trip in space [4].

Electrostimulation is quite the opposite. This kind of method is, to some extent, just like motion sickness pills, which can mitigate symptoms with a faster effect than adaptation training [8], [12]. These electrostimulation methods are based on the hypothesis of dynamic multisensory reweighting. That is to say, our brain makes decisions by optimizing multisensory signals. In the context of CS, the weights of visual and vestibular signals can be adjusted (reweighted) based on the signal certainty and reliability [6]. The clearer and more reliable the input sensory signal is, the more weight it will be given (that is, up-weighting); on the contrary, less weight will be given when a cue isn't reliable (that is, down-weighting). Obviously, the principles behind current electrostimulation approaches are to down-weight vestibular [8] and up-weight visual signals [12].

Given that the vestibular system consists of vestibular sensory organs (e.g., otoliths and semi-circular canals) and vestibular cortices (e.g., parieto-insular vestibular cortex) [13], [14], there should be two ways to down-weight vestibular signals: 1) adding noise signals into vestibular sensory organs to affect the certainty of the sensory inputs; 2) inhibiting vestibular cortical excitability to affect the reliability of sensory inputs. However, currently, only the former has been studied, using galvanic vestibular stimulation (GVS) and bone-conducted vibration [8], [15]. It remains unknown whether inhibiting the vestibular cortical excitability could mitigate CS or not.

Transcranial direct current stimulation (tDCS) is a non-invasive electrical brain stimulation technique that modulates underlying cortical excitability [16]. Depending on whether anodal or cathodal stimulation is applied, tDCS increases or decreases cortical excitability, respectively [16]. The most prototypical brain region for showing increases and decreases in cortical excitability is motor cortex [17], [18]. However, for cognitive areas it is common to observe excitatory effects following anodal tDCS, but rarely to observe inhibitory effects following cathodal tDCS [19]. Similarly, for vestibular areas, it is also common to observe anodal tDCS-induced effects (such as in temporoparietal junction (TPJ)) [12], [20], but rarely to find cathodal tDCS-based studies.

Although a previous study, conducted by Arshad et. al., has shown encouraging results by using 1.5mA and 15-min cathodal tDCS at left parietal cortex [21], they focused on traditional off-axis vertical rotation (OAVR)-induced MS, rather than HMD-VR-induced CS. Note that the parietal cortex used here is believed to be part of the pure vestibular cortical area (PIVC), which is different from TPJ which is a visual-vestibular multisensory brain area related to body position and orientation. More details about the neural pathways of the vestibular cortical areas can be found in Frank and Greenlee's review paper [14]. Therefore, the goal of this study was to replicate Arshad et. al.' s tDCS solution [21] to assess its feasibility of mitigating CS. The significance of this study is twofold: 1) If Arshad et. al.'s approach [21] is able to mitigate CS, then it will lay the foundation of the development of combined sensory organ and cortex based multimodal neurostimulation for mitigation of CS; 2) If it does not work, then Arshad et. al.'s approach [21] might possibly be not strong enough to inhibit vestibular cortical activities, nor might inhibiting the vestibular cortex alone be enough to mitigate CS. Thus, future studies can be planned e.g. with further optimized tDCS parameters or advanced neuroimaging and neurostimulation technologies.

## II. Methods and Materials

### A. Participants and Screening measures

Potential participants were recruited through social media and the <redacted for review> subject pool. Exclusion criteria were abnormal vision, neurological disorders, contra-indications of tDCS, and MS susceptibility questionnaire (MSSQ) score <10 or MSSQ score >36, or MSB score =0. Here, MSSQ score is a sum of total sickness score during childhood (<12 years of age, MSA) and during adulthood over the last 10 years (MSB) [22]. As the name implies, MSSQ is designed for MS. It is still unclear whether MSSQ is a good predictor for CS, although it has already been used in some VR studies [23]. Thus, the MSSQ and MSB thresholds shown above were specifically determined by our pilot studies, where a total of seventeen participants involved in either VR videos (including car driving, motorbike riding, and rollercoasters) or customized VR scenes (see Section tDCS and VR stimuli). For VR videos, we found that people who scored under 10 in the MSSQ were very unlikely to experience symptoms, therefore the MSSQ score screening criteria was set to a threshold between 10 and 36. Regarding the customized VR scenes, we found that giving more importance to the adult section of the MSSQ (that is MSB) could better select participants. This works under the assumption that adult susceptibility to MS is more recent and therefore more representative of the participants' current state; this approach was adopted by Arshad et. al. [21]. Finally, twenty-three participants who successfully passed the screening criteria were recruited. Written informed consent was obtained from each participant according to procedures approved by the ethics panel of the University of Glasgow (No. 300160027), College of Science and Engineering. All participants were randomly assigned into either the cathodal (age=22.8, MSSQ= 15.97, MSB=6.7) or anodal (age= 20.3, MSSQ=19.48, MSB=8.99) groups. The data from three male participants had to be discarded as two of them did not reach the required moderate symptoms and another one admitted to not having understood the experiment instructions at the end of his session. The remaining twenty were ten males and ten females. Every participant was paid £6 per hour for taking part in the experiment. This study was carried out from March 2018 to September 2019, which was before the Covid-19 outbreak.

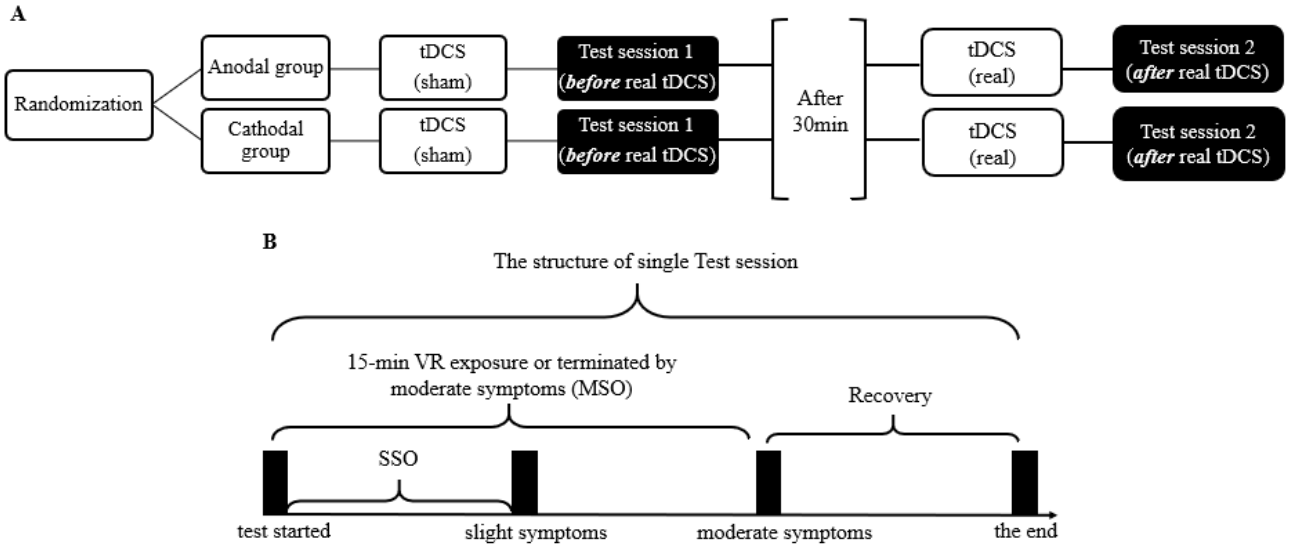


Fig 1. Experimental procedure. A) Group and session design of the study; B) Procedure for each testing session.

### *B. Procedure*

Before the date of the experiment participants were given information and told what their participation would entail. They were instructed not to consume recreational drugs or more than 3 units of alcohol the day before the experiment, and coffee or any amount of alcohol the same day. At the time of their respective sessions, participants were taken to the room where the experiment would take place. Participants were first given the consent form, and once signed, an updated version of the tDCS safety questionnaire to make sure they met the safety conditions.

The procedure for each group consisted of two test sessions: before and after the active tDCS, as shown in the two black rectangles in Figure 1A. Each session was structured as shown in Figure 1B. The tDCS was operated and the electrodes were fitted on participants' heads by an experienced tDCS operator. The researcher leading the experiment did not know if each participant was experiencing cathodal or anodal stimulation, only the tDCS operator knew (double-blind design). After each stimulation, participants were given a questionnaire to make sure they were not able to differentiate between the sham and the real stimulation conditions. Before the visual stimulation began, participants had the subjective sickness scale (see *Section C. Study design*) explained to them and were instructed to report every time that their symptom severity changed. They were then told to put on the HMD and to not move their heads while the stimuli lasted. Once they were ready, the VR stimuli started. Every time participants reported a change in their symptom severity a note of the time was recorded. Once participants reported moderate symptoms, the HMD was removed from their heads, at which point the recovery period started which would last until they reported being back at "1" in terms of symptom severity. Note that each participant was given a 30-min break from the moment the first test session ended to ensure an equal resting period for subjects while keeping a heightened susceptibility state [22]. After the break the real stimulation started, and then the second test session was carried out.

### *C. Study Design*

We used a double-blinded and 2×2×3 mixed-subject study design, which was modelled after Arshad et. al.' work [21]. Data were both collected and analysed in an anonymized manner. The between-subjects condition is the type of tDCS stimulation (cathodal or anodal). While the hypothesis is based around cathodal stimulation, having an anodal condition permits the exclusion of the possibility of nonspecific tDCS effects. The first within-subjects variable is the timing of VR exposure (before or after active tDCS, as shown in Figure 1A). The second within-subjects variable is the sickness scenario (slight symptoms onset (SSO)/moderate symptoms onset (MSO)/recovery, as shown in Figure 1B). The SSO refers to the moment where participants experience any slight CS symptoms, the MSO being the point at which participants report moderate symptoms, which leads to the end of VR exposure. Lastly, Recovery is the point at which participants report the disappearance of their symptoms after VR exposure is terminated. Here, the severity of their symptoms was collected using subjective verbal report where 1 = "no symptoms", 2 = "slight symptoms", 3 = "moderate symptoms" (This subjective scale is adapted from Arshad et. al.'s work [21]). The experimental hypothesis was that the timing of VR exposure would interact with sickness scenario in the cathodal stimulation group in the form of an increased time for SSO and MSO, and reduced recovery time between the before and after stimulation conditions. This difference should be exclusive to the cathodal group, and therefore not be present in anodal stimulation.

### *D. Statistical Analysis*

All statistical analyses were done using SPSS 19.0 with a 0.05 alpha level. The Greenhouse–Geisser correction was used when assumptions of sphericity were not met.

#### *1) Exploring the feasibility of tDCS.*

To examine the feasibility of cathodal tDCS in mitigating CS, we focused on comparisons of time on the three sickness scenarios in cathodal group. This analysis involved a repeated measures ANOVA with two within-subject factors: timing of

VR stimulus exposure (before and after cathodal tDCS) and sickness scenarios (SSO/MSO/Recovery).

*2) Exploring the specificity of tDCS.*

To examine the specificity of cathodal tDCS, we also focused on the comparison of time on the three sickness scenarios in the anodal group. Similarly, this analysis involved a repeated measures ANOVA with two within-subject factors: timing of VR stimulus exlosure (before and after anodal tDCS) and sickness scenarios (SSO/MSO/Recovery).

*3) Exploring the polarity-dependent effect.*

To examine the polarity-dependent effect, we further analyzed the tDCS-related differences between the cathodal and anodal groups. This involved a mixed ANOVA with a between-subject factor (cathodal and anodal) and a within-subject factor (normalized time on the three sickness scenarios, quantified as shown in Equation 1).

$$\text{Normalized time}=\frac{time_{after}-time_{before}}{time_{before}} \times 100\%, \quad \text{time} \in \{\text{SSO, MSO, Recovery}\} \tag{1}$$

## E. tDCS and VR Stimulus

tDCS was delivered by a battery-driven, constant current stimulator (NeuroConn, Germany) via a pair of rubber electrodes (35cm$^2$) with conductive paste. A constant current of 1.5 mA intensity was delivered for 15 min (30-sec ramp up and 30-sec ramp down). The active stimulation (anodal or cathodal) electrode was placed on the left parietal cortex (at P3 of the 10/20 system; see Fig. 2) and the reference electrode was located on the ipsilateral shoulder on the deltoid muscle. For sham stimulation, the electrodes were placed at the same positions as active stimulation, but the stimulator was turned off after a 30-sec ramp up and ramp down period. Since the onset of tDCS often generates a tingling or itching sensation over the first minute of the stimulation, this sham procedure blinded the participants from differentiating between active and sham conditions.

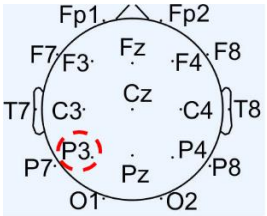


Fig 2. The active tDCS electrode placed on the left parietal cortex (at P3 according to international electroencephalography (EEG) 10/20 system which is an internationally recognised method that allows EEG electrode placement to be standardised [24]).

Given that the vestibular sensory organs, otoliths and semi-circular canals are sensitive to linear (e.g., gravity) and angular (e.g., rotation) acceleration stimuli respectively [6], [13], the nature of the VR stimuli is an outdoor rollercoaster application which aimed to induce at least two kinds of visual-vestibular sensory mismatch through virtual linear and angular acceleration: 1) visual-otoliths mismatch; 2) visual-semi-circular canals mismatch. Here, the virtual linear and angular acceleration was achieved by changing the speed of rotations and movements of participant's point of view. Specifically, the participants' point of view is a camera that follows a programmable route that is created by placing a number of "waypoints" in the virtual space. When the camera passes through any of these waypoints, they can execute code that changes the rotation of the camera, the speed of these rotations, or the speed of movement of the camera through the route. The final version of the route (see Fig 3) was developed through testing several participants in pilot studies. Even though the route is restarted after roughly 1 minute, the nature of the rotations leads to changes in the direction of the camera, making each lap slightly different than the rest thus avoiding predictability. This VR stimuli was developed by Unity 3D version 2017.3 as well as its terrain-making tool. The presentation delivery device was the Oculus Rift Development Kit 2.

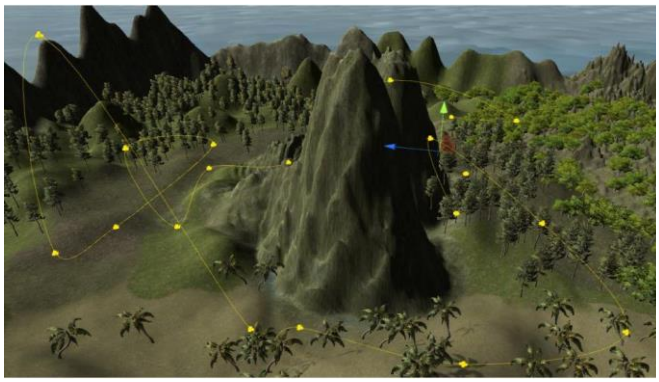

Fig 3. The route followed by the camera. The yellow dots are the points at which code is executed to change the rotation of the camera, the speed of these rotations, or the speed of movement of the camera.

# III. Results

## A. tDCS safety and Sham condition

None of the participants reported adverse effects from tDCS stimulation apart from slight tingling sensations. Participants' accuracy rate for identifying the sham condition when given a choice between the two sessions was roughly at chance-level (50%) and every participant described it as "a guess". This suggests that the sham condition was a success and participants could not differentiate it from active stimulation.

## B. The feasibility of tDCS on mitigating CS

First of all, by using boxplots of the raw data, the datasets of one participant in Onset scenario and three participants in Recovery scenario were removed, according to the mild and extreme outliers. Also, one participant forgot to report in Onset scenario.

For the remaining datasets, we found a main effect of test session ($F(1, 6) = 9.507$, $p = 0.022$) and sickness scenario ($F(2, 12) = 8.520$, $p = 0.022$), but there was no test session × sickness scenario interaction ($F(2, 12) = 1.021$, $p = 0.390$). Although further paired t-test shows that there was a significant difference in Recovery between test session 1 and 2, it is contrary to our hypothesis, with the Recovery in test session 2 (M=29.333 sec, SD=4.553 sec) being significantly longer than that in test session 1 (M=12.609 sec, SD=3.396 sec, as shown in Fig. 4A ($t(6)=-3.988$, $p=0.007$)). A possible

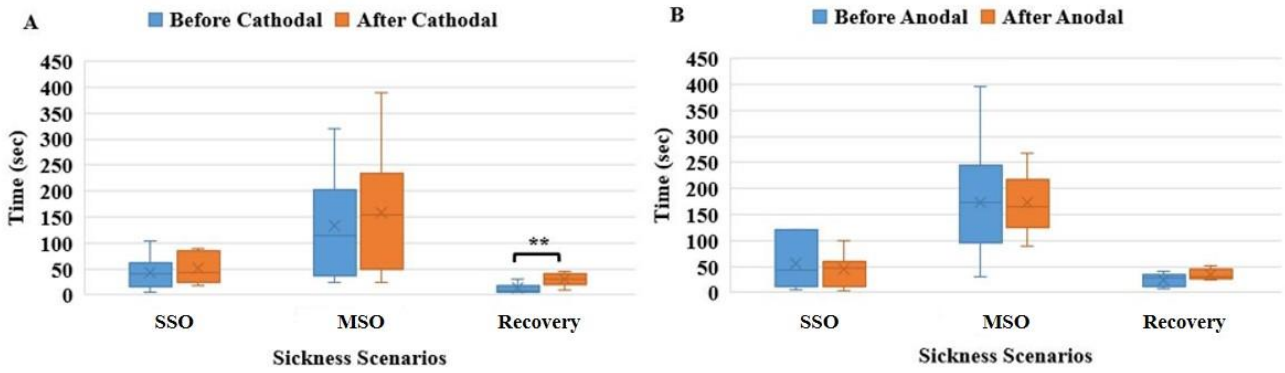


Fig 4. The boxplots for three sickness scenarios: A) between before and after Cathodal tDCS. **p<0.01; B) between before and after active Anodal tDCS.

reasoning behind this phenomenon might be that participants experienced more CS symptoms than those in test session 1, so needed more time to recover (note that the same trend was obtained with the anodal group as shown in Fig. 4B). For the time of VR exposure, although the median time after tDCS was reduced, there was no significant difference with p=0.181 for SSO and p=0.282 for MSO. These results reveal that the tDCS mitigation protocol modelled after Arshad et. al.'s work [21] were not confirmed for HMD-VR-induced CS.

### *C. The specificity of tDCS on mitigating CS*

Similarly, by using boxplots of the raw data, the datasets of three participants in SSO scenario and four participants in Recovery scenario were removed, according to the mild and extreme outliers. For the remaining datasets, we found a main effect of sickness scenario ($F(2, 10) = 16.078$, $p = 0.001$), but there was no significant main effect of test session ($F(1, 5) = 0.084$, $p = 0.784$) and test session × sickness scenario interaction ($F(2, 10) = 0.362$, $p = 0.581$). As shown in Fig. 3B, further paired t-test shows that the mean value of SSO (M=54.639 sec, SD=17.853 for before Anodal and M=44.040, SD=12.267 for after Anodal) and MSO was reduced (M=172.836 sec, SD=33.650 for before Anodal and M=172.731, SD=18.029 for after Anodal), but no statistical significance (t(6)=1.193, p=0.278 for SSO and t(9)=0.004, p=0.997 for MSO). Regarding the mean value of Recovery, an increasing trend can be seen with M=24.885 sec and SD=4.889 for before Anodal and M=34.662 sec and SD=4.470 for after Anodal, respectively. These results reveal that anodal tDCS at left parietal cortex was unable to mitigate CS.

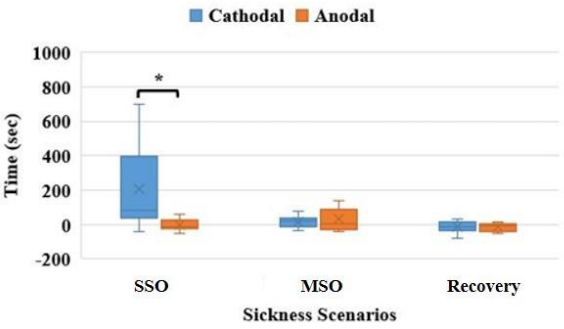


Fig 5. The polarity-dependent tDCS differences in CS. *p<0.05.

### *D. Exploring the polarity-dependent tDCS effect*

For anodal group, according to mild and extreme outliers in boxplots, the normalized datasets of two participants in SSO, one participant in MSO and Recovery respectively were removed. Similarly, for the cathodal group, one participant in MSO scenario was removed.

Based on the remaining datasets, the mixed ANOVA shows a significant main effect of stimulation type ($F(1, 7) = 6.430$, $p = 0.039$), sickness scenarios ($F(2, 14) = 6.958$, $p = 0.025$) and a trend towards significant stimulation type × sickness scenarios interaction ($F(2, 14) = 5.271$, $p = 0.051$). Further paired t-test shows that there is a significant difference in SSO scenario (t(7)=2.460, p=0.041) between cathodal (M=225.495 sec, SD=89.335 sec) and anodal group (M=-2.490 sec, SD=13.245 sec, as shown in Fig 5). These results reveal that the cathodal tDCS indeed somehow delayed the onset of slight symptoms if compared to that in anodal condition. However, there are no significant differences in delaying the onset of moderate symptoms and reducing time to recovery between the two stimulation types.

## IV. Discussion

Based on the between-subject analysis, the presented findings reveal that cathodal tDCS delayed the onset of slight CS symptoms if compared to anodal tDCS, suggesting cathodal tDCS's inhibitory effect on vestibular cortical excitability. However, this inhibitory effect was not observed in delaying moderate symptoms and reducing the time to recovery. More importantly, we did not find this inhibitory effect using within-subject analysis, which is the fundamental difference between the current work and that of Arshad and colleagues [21]. The results suggest that Arshad's tDCS approach may not be strong enough to inhibit vestibular cortical activities during cybersickness, or may be ineffective due to inhibiting the vestibular cortex alone. Here, we discuss possible reasons for these findings in the context of stimuli approaches and tDCS procedure.

### *A. Stimuli Approaches*

Arshad and colleagues [21] adopted traditional OAVR as their stimuli approach to induce MS. Since their speed of rotation was constant, and the direction of rotation was fixed (rightwards), their MS was purely induced by the visual-otoliths mismatch. However, in current work, we are using rollercoaster as the stimuli which was designed to induce both visual-otolith and visual-semi-circular canals mismatch; thus, we surmise that our magnitude of sensory mismatch was higher than Arshad's case. In this context, the same current intensity (1.5mA) and dose (15 min) were possibly not sufficient.

Therefore, on the one hand, future studies can be planned with better optimized tDCS parameters. On the other hand, to provide more objective measures of participants internal state, physiological signals or advanced neuroimaging technologies can be used to verify the differences in the induced magnitude of sensory mismatch.

### *B. tDCS Procedure*

We finished tDCS immediately prior to CS induction, but Arshad et. al.'s work [21] carried out tDCS during MS induction. That is, we were actually investigating the aftereffect of tDCS, while they focused on the online effect. Based on the self-reported severity of CS, our within-subject results did not show any statistically significant differences in the aftereffect on CS. However, in a recent study about GVS, the authors found an encouraging aftereffect on CS, although it was as short as 3 minutes, based on every 3-minute post-GVS observation [15]. This may indicate that the suppression on vestibular sensory organs is more effective than that on vestibular cortical areas.

The reason that we did not use tDCS during CS induction is that cognitive activities during tDCS may interfere with or abolish tDCS effects [25]. Specifically, as mentioned earlier, vection is a trigger of CS. Previous research has found that the determinant of vection perception is attention [26], which is a fundamental capability for perception and cognition [27]. Therefore, the perception of vection is a kind of attention-demanding cognitive activity so that CS induction during tDCS may indeed affect the tDCS effect according to [25]. In addition, from the perspective of user experience, asking participants to simultaneously wear VR-HMD and tDCS device was difficult due to the large size of tDCS sponge electrodes (35 $cm^2$ area in current study instead of 25 $cm^2$ in Arshad et. al.'s work [21]).

Therefore, on the one hand, it is worthy to comply with [22]'s suggestion and study how to maintain the aftereffect of tDCS longer. On the other hand, given the success of Arshad et. al.' work [21], future study should be done with smaller electrodes-based tDCS to explore the online impact of tDCS on VR-HMD-induced CS. Particularly, an interesting research question is that is it possible that the tDCS during CS induction to some extent can distract brain attentional network so that the awareness of vection is reduced and CS is mitigated?

## V. Limitation and Conclusion

This study did not use objective measurements (such as physiological data or postural data [28]) to assess the mitigation effect of tDCS, therefore we might miss finding a statistically significant and technically quantizable mitigation effect. Thus, a multimodal objective measurement can be planned for future study. This is especially pertinent given the progress on AI-based physiological feature learning for VR application [29] and highly-integrated research-grade [30] or commercial [31] VR-biosensing platforms.

Also, given that cognitive activities during tDCS may interfere or abolish tDCS effects, a potential limitation for tDCS-based CS mitigations is that they are perhaps not suited to real-life VR applications, where the users have to perform cognitive activities. However, it is indeed an option for prophylactic treatment which can be implemented prior to experience VR scenes.

Overall, based on between-subject analysis, we indeed found the delayed onset of slight CS symptoms, suggesting the possible inhibitory effect following cathodal tDCS. However, this inhibitory effect was not observed in the other two sickness scenarios, MSO and Recovery. Regarding the within-subject analysis, we did not find any subjective measurements-based significant results that could validate our hypothesis that cathodal tDCS stimulation over left parietal cortex would mitigate cybersickness.

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