



## Prediction and Detection of Virtual Reality induced Cybersickness: A Spiking Neural Network Approach Using Spatiotemporal EEG Brain Data and Heart Rate Variability

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1       **Prediction and Detection of VR induced Cybersickness: A Spiking Neural Network**  
2       **Approach Using Spatiotemporal EEG Brain Data and Heart Rate Variability**

3

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20      neural network, brain, dynamics, spatiotemporal

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26 **Abstract**

27 Virtual Reality (VR) allows users to interact with 3D immersive environments, and will be a  
28 key technology across many domain applications, including access to a future metaverse.  
29 Yet, consumer adoption of VR technology is limited by cybersickness (CS) - a debilitating  
30 sensation accompanied by a cluster of symptoms including nausea, oculomotor issues and  
31 dizziness. A leading problem is the lack of automated objective tools to predict or detect CS  
32 in individuals, which can then be used for resistance training, timely warning systems or  
33 clinical intervention. This paper explores the spatiotemporal brain dynamics and heart rate  
34 variability involved in cybersickness, and uses this information to both predict and detect CS  
35 episodes. The present study applies deep learning of EEG in a spiking neural network (SNN)  
36 architecture to predict CS prior to using VR (85.9%, F7) and detect it (76.6%, FP1, Cz). ECG  
37 derived [sympathetic heart rate variability \(HRV\) parameters can be used for both prediction](#)  
38 [\(74.2%\) and detection \(72.6%\) but at a lower accuracy than EEG](#). Multimodal data [fusion of](#)  
39 [EEG and sympathetic HRV does not change this accuracy compared to ECG alone](#). The  
40 study found that Cz (premotor and supplementary motor cortex) and O2 (primary visual  
41 cortex) are key hubs in functionally connected networks associated with both CS events and  
42 susceptibility to CS. F7 is also suggested as a key area involved in integration of information  
43 and implementation of responses to incongruent environments that induce cybersickness.  
44 Consequently, Cz, O2 and F7 are presented here as promising targets for intervention.

45

46 **Keywords:** Cybersickness; Virtual reality, Spiking neural networks; EEG, ECG, NeuCube.

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**Introduction**

Virtual Reality (VR) technology is becoming prevalent in entertainment, art, education, social and professional settings[1,2]. VR allows for interactive immersion into shared digital environments that can be accessed by many. Despite this, individual experiences in VR remain far from idyllic. Drawbacks exist in the form of cybersickness (CS) - a debilitating sensation accompanied by a cluster of symptoms that include nausea, oculomotor issues and dizziness[3]. It is unfortunate that limitations to human physiology and perception form a barrier to consumer adoption of VR technology; especially since our world continually charges towards a nexus of virtual and real-world interactions. A way to combat CS would be to utilize a tool that predicts or detects it. Yet, these tools must be automated and objective, so that preparations or active responses like training resistance, timely warning systems and clinical intervention can be implemented. Tracking of CS is currently restricted to subjective reports through verbal confirmation or questionnaires. Not only do these methods not allow for future prediction, but they are time inefficient and require manual input. With current technology at our disposal, objective biomarkers correlated with cybersickness can be collected from wearable devices and fed into machine learning algorithms for streamlined, automatic prediction and/or detection of cybersickness events[4]. While various models for prediction and detection of CS severity have been proposed[4,5], there lacks a way to both collect CS data and continue to generate new knowledge about the condition through machine learning assisted approaches. To achieve this, the present study uses a modified version of the brain-inspired NeuCube SNN architecture[6] to both predict and detect CS whilst generating new knowledge about the condition.

76

77 There are several reasons for choosing SNNs for this purpose. SNNs are advanced machine  
78 learning techniques[7] and are considered the third generation of artificial neural networks.  
79 They simulate the behaviour of biological neural networks by creating and updating  
80 connections between spiking neurons (synaptic connections) to learn temporal associations  
81 between them. This architecture and mechanism of learning has several advantages in  
82 temporal information processing[8-13] over that of traditional neural networks. This includes  
83 robustness to noise through the encoding of consecutive time series data, such as EEG, into a  
84 compressed data format known as spikes (binary units) [7]. Encoding procedures such as  
85 threshold-based-spike-generation, produce spikes that represent a change in consecutive  
86 values above a certain threshold, allowing for changes in data to be captured over time .  
87 Additionally, if multiple time series, such as EEG channels, are modelled in a single SNN,  
88 patterns of interactions between the changes in their time series can be detected and analysed.  
89 SNN architectures can further benefit from the usage of brain templates that specify a spatial  
90 distribution in the anatomical shape of a brain. Upon training, these models can be considered  
91 an interpretable spatiotemporal map of the brain activities measured, which assists to better  
92 understand brain dynamics under diverse conditions. Further on, this spatiotemporal map can  
93 be represented as a feature vector, and additional parameters from other biologically relevant  
94 data such as HRV can be added for classification of different brain states.

95

96 Consequently, the present study performs deep learning of integrated EEG and sympathetic  
97 heart rate variability (HRV) data in an interpretable dynamically evolving spiking neural  
98 network (SNN) architecture. This architecture mimics the biological structure and processing  
99 mechanisms of the human brain, and captures spatiotemporal information from EEG signals  
100 to form a dynamically updateable neural map of CS. A machine learning algorithm was

101 developed that can detect CS events (76.6%) and predict it prior to VR usage at resting  
102 baseline (85.9%) using electroencephalogram (EEG) data. F7 alone was the most optimal  
103 input for cybersickness prediction. The algorithm also integrated fusion of electrocardiogram  
104 (ECG) heart rate variability data but it did not improve classification accuracy. The study  
105 found that features related to cybersickness susceptibility are diverse and that highlighted  
106 features change over time. Amongst many important features, Cz (premotor and  
107 supplementary motor cortex) and O2 (primary visual cortex) are key hubs in functionally  
108 connected networks associated with both CS events and susceptibility to CS. According to  
109 accuracy results and analysis of CS related brain hubs, Cz, O2 and F7 present as promising  
110 targets for intervention. The study additionally proceeded with an exhaustive analysis to find  
111 the best time segment during a resting-state EEG baseline and its data length for optimal  
112 prediction accuracy.

113

## 114 **Contributions**

115 In summary, the paper contributes the following:

- 116 - A novel approach to the prediction and detection of cybersickness using interpretable  
117 spiking neural networks (SNN) and weighted K-nearest neighbor (KNN) algorithms  
118 using EEG and ECG data, both separately and in their integration.
- 119 - Optimized SNN architecture based on inherent characteristics of cybersickness
- 120 - Machine learning assisted knowledge discovery and insight into the spatiotemporal  
121 brain dynamics of cybersickness
- 122 - Considerations for feature reduction for diagnostic and predictive CS computational  
123 models.
- 124 - Machine learning extracted clinical biomarkers for the development of intervention  
125 strategies.

126 -

## 127 **Methods**

### 128 *Subjects*

129 Sixty-four participants, male (29) and female (35), age range of 18-33 years (mean 23,  
130 standard deviation  $\pm 4.1$ ). Subjects were recruited from the student and working population.  
131 The exclusion criteria were a previous diagnosis of neurological disorder, cardiovascular  
132 disease, diabetes, gastrointestinal disorder, medications, or smoking. All subjects had either  
133 normal or corrected visual acuity with contact lenses. This study was approved by the  
134 University of Otago Ethics Committee (H20/169) and performed in accordance with relevant  
135 guidelines and regulations. All participants provided signed consent.

136

### 137 *Experimental equipment*

138 A VR video of rotating stars published by previous researchers was played in an HTC Vive  
139 headset (HTC Corporation, Taipei, Taiwan). EEG was recorded using starstim32  
140 (Neuroelectronics). ECG was recorded using Shimmer3 5 lead ECG (Shimmer, Dublin, Ireland)  
141 at a sampling rate of 512 Hz. Five electrodes were placed, two 5 cm above the pelvic girdle,  
142 labelled according to proximity towards the left leg (LL) and right leg (RL), and two 5 cm  
143 below the clavicle, labelled according to proximity towards the left arm (LA) and right arm  
144 (RA), with the fifth electrode at the V3 position relating to the midway point between the 4th  
145 and 5th intercostal space. Data obtained from the LL-RA channel between electrodes was  
146 used for analysis.

147

### 148 *Software*

149 iMotions 8.0 (iMotions, Copenhagen, Denmark) was used to synchronize EEG and ECG  
150 data recordings for a unified collection of measurement time series. Live view of biosensor  
151 data streaming ensured quality data collection and so that markers separating baseline,

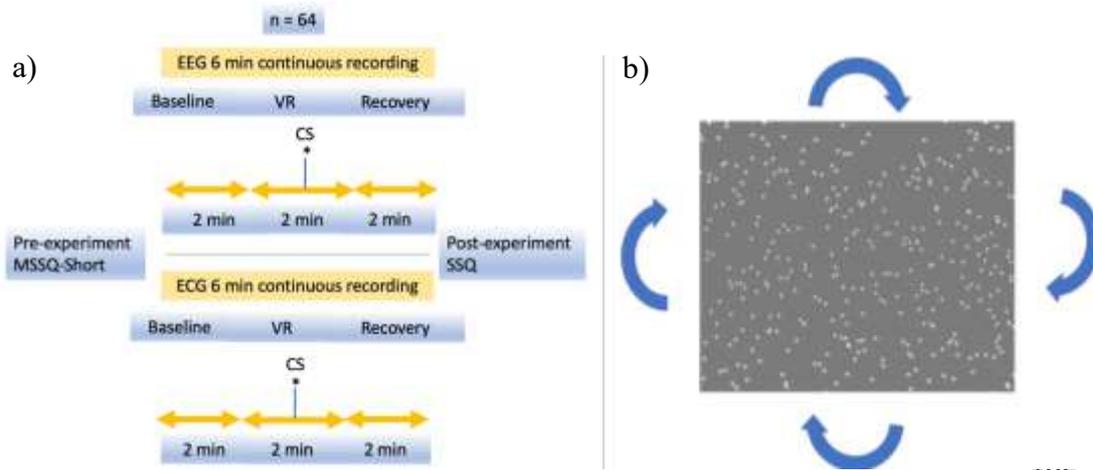
152 stimulation and post stimulation could be placed during the experiment. Neucube was used  
153 for the SNN architecture and feature vector production. Python 3.8.8 was used for the  
154 classification algorithms. HRV was analyzed using Kubios HRV Premium Ver. 3.3  
155 software[14] (Kubios, Kuopio, Eastern Finland). For 10s HRV results, Neurokit2[15] was  
156 used to determine R-peaks and pyHRV[16] was used to calculate RMSSD. The VR video  
157 used in this experiment was developed in previous work by researchers from Stanford  
158 University, and was chosen for its propensity to induce cybersickness in individuals. The VR  
159 video consists of clockwise rotating white dots about the roll axis, dispersed at different  
160 depths through the visual foreground and background[17].

161

#### 162 *Protocol*

163 Participants (n=64) underwent a 2 minute resting state baseline (A) before VR immersion  
164 without HMD usage, then watched a 2 minute VR video of rotating stars (B), followed by  
165 removal of the headset and a 2 minute recovery period. EEG and ECG was recorded  
166 continuously throughout the entire experiment. To mitigate any potential noise, participant  
167 immersion in VR was a passive ordeal where the only requirement was to stare straight ahead  
168 with minimal body and head movement, and all parts the experiment were seated. The  
169 conscious perception of cybersickness was reported via a thumbs up, and was simultaneously  
170 marked on the data stream. Individuals who reported cybersickness and those that did not  
171 (controls) were separated into two groups. A pre-experiment motion sickness susceptibility  
172 questionnaire[18] (MSSQ-Short) was administered to assess motion sickness history and  
173 susceptibility, along with a post-experiment simulator sickness questionnaire[19] (SSQ) to  
174 collect individual sickness ratings.

175



183 Figure 1. a) Experiment flow, b) VR video example

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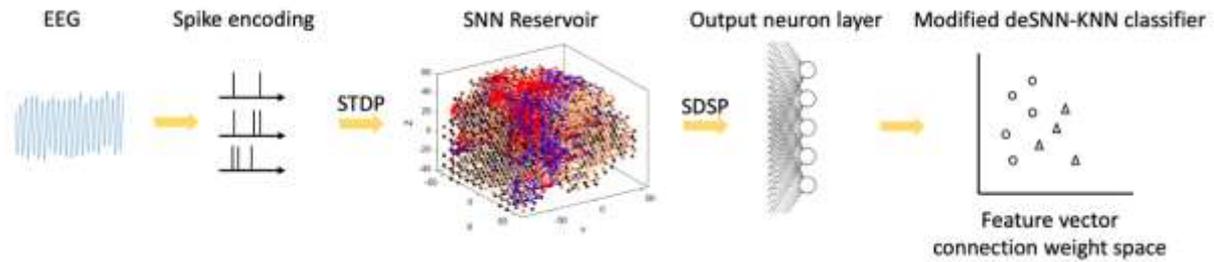
185 *Statistics*

186 A Mann-Whitney U test was run to compare between cybersick and control groups for the  
187 following data: MSSQ-short scores, SSQ scores, spike count and HRV parameters -  
188 Parasympathetic nervous system index (PNS), sympathetic nervous system index (SNS),  
189 stress index (SI), standard deviation of N-N intervals (SDNN), root mean squared of  
190 successive differences of R-R intervals (RMSSD).

191

192 *The NeuCube brain-inspired Spiking Neural Network Architecture [18]*

193 The following sections below describe the general architecture of the model and data pipeline.  
194 This includes initial encoding of the raw EEG data into spikes, training of the SNN reservoir  
195 for knowledge discovery and feature selection, producing a feature vector which represents the  
196 spiking activity in the neural network through connections with an output neuron layer, and  
197 finally classification of this feature vector. A graphical representation of this data pipeline is  
198 shown in Figure 2.



199

200 **Figure 2.** Data pipeline. STDP (spike timing dependent plasticity), SDSP (spike driven  
 201 synaptic plasticity), deSNN-KNN (dynamic evolving spiking neural network-k nearest  
 202 neighbour algorithm)

203

204 *Spike Encoding:*

205 SNNs receive reconstructed input signals as binary waveforms known as spike trains. Thus,  
 206 the raw EEG data must first be transformed into this format. Step Forward (SF) encoding was  
 207 used as a ‘signal to spike encoder’. SF is a threshold-based algorithm that works based on  
 208 updating cutoff values for excitatory and inhibitory spikes, according to a *base* value at time  
 209  $t = 0$  and a user defined threshold value. If a signal’s value is greater than the current  
 210 excitatory cutoff ( $base + threshold$ ) then an excitatory spike is encoded, and the excitatory  
 211 cutoff value is updated as the new *base* value. If the signal’s value at  $t$  is less than the  
 212 current inhibitory cutoff value ( $base + threshold$ ) then an inhibitory spike is encoded and  
 213 the inhibitory cutoff value is updated as the new base value. In some cases, no spike is  
 214 encoded and the base value remains the same[20]. Spike counts for every channel were  
 215 extracted and compared between CS and control groups at baseline (A) and during the CS  
 216 onset event (B).

217

218 *NeuCube Reservoir:*

219 A reservoir of connected neurons were initialized in preparation for spike inputs. A SNN  
 220 reservoir (SNNr) module is in principle scalable in size, and here it is composed of 1471 LIF

221 neurons representing  $1\text{cm}^3$  of the brain, located at the same coordinates as those modelled in  
 222 the Talairach atlas to create a 3D-brain geometry. Defining the spatial location of neurons  
 223 allows spatial-temporal patterns to be elucidated from spike inputs. Connection weights  
 224 between reservoir neurons were randomly initialized using the small world connectivity  
 225 (SWC) approach. The SWC limits connections to only form within a defined radius and the  
 226 random connections creates a diverse set of dynamical states. Connection weights, also  
 227 known as ‘synaptic weights’, modulate any increase or decrease in the membrane potential of  
 228 the post-synaptic neuron. In other words, it is a measure of the contribution of a pre-synaptic  
 229 neuron towards the firing of a post-synaptic neuron. Connections also hold an intrinsic value  
 230 of ‘synaptic delay’, which is the time delay in firing between pre and post synaptic neurons.  
 231 Excitatory and inhibitory synapses within the reservoir are probabilistically determined  
 232 according to the following formula:

233

$$234 \quad P_{i,j} = \begin{cases} C * e^{-(d_{i,j}^{norm}/\lambda)^2} & \text{if } d_{i,j}^{norm} \leq d_{thresh} \\ 0 & \text{otherwise} \end{cases}$$

235

236 Where:  $P_{i,j}$  is the probability of establishing a connection between two neurons  $i$  and  $j$ ;  $C$  is  
 237 the maximum connection probability;  $\lambda$  is the small world connection radius;  $d_{i,j}^{norm}$  is the  
 238 normalized distance between two neurons;  $d_{thresh}$  is the maximum connection distance  
 239 between two neurons. In this way, closer neurons have a higher probability of stronger  
 240 connection weights than neurons further away.

241

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245 *SNNr training:*

246 Training the SNNr involved unsupervised learning of spike trains introduced by ‘input  
 247 neurons’ at 32 EEG channel locations. These locations were gained from the conversion of  
 248 10-10 scalp electrode positions into Talairach coordinates. Input neurons feed spike trains of  
 249 each sample to the SNNr in a temporally synced and spatially distributed manner. Similar to  
 250 the notion of summation at an axon hillock[21,22], an output spike is produced by a post  
 251 synaptic neuron when many input spikes from pre synaptic neurons accumulate over a short  
 252 period of time. As spike trains spread throughout the SNNr, connection weights between  
 253 reservoir neurons are updated according to a rule called ‘Spike Timing Dependent Plasticity’  
 254 (STDP). The sort of learning mimics cellular processes of long-term potentiation and long-  
 255 term depression involved in learning and memory[23].

256

$$257 \quad W(s) = \begin{cases} A_+ \exp[s/t_+] & \text{for } s < 0 \\ A_- \exp[-s/t_+] & \text{for } s > 0 \end{cases}$$

258

259 S is the time delay between presynaptic and post-synaptic firing.  $t_+$  is the pre-synaptic time  
 260 interval.  $t_+$  is the post-synaptic time interval.  $A_+$  is the amplitude of weight increase.  $A_-$   
 261 is the amplitude of weight decrease.

262 The STDP rule implements a form of logical causality, in which connection weights increase  
 263 or decrease proportional to the synaptic delay. If a presynaptic neuron fires before a post-  
 264 synaptic neuron, the connection weight increases between them. Likewise, connection  
 265 weights decrease if a postsynaptic neuron fires before a presynaptic neuron. The end product  
 266 is a trained ‘SNNr cube’ - a neuronal model with connection weights that represent complex  
 267 and dynamic spatiotemporal brain activity.

268

269

270

271 In our study, the training samples were divided into two groups equally, CS (n=32) and  
272 control (n=32). A SNNr cube was trained on all 32 channels of EEG data for each group,  
273 giving two distinct SNNr cubes with different connection weights. The connection weights of  
274 these cubes were subtracted from each other, producing an SNNr cube specific to  
275 cybersickness.

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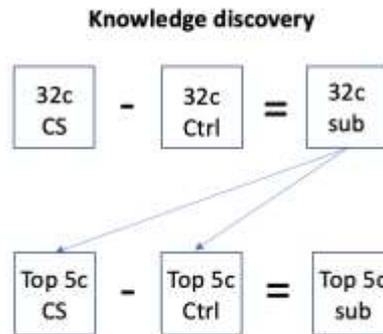
277 *Knowledge Discovery:*

278 Subtracted SNNr cubes were made using data 2 seconds in length selected from time  
279 segments 30-32s and 90-92s at baseline, and from 1 second before the CS event. Since  
280 connections between neurons at SNNr initialization are randomly generated, the same  
281 initialized connections were kept constant for subtractions between cybersick and control  
282 groups. Underpinning this subtraction, was the hypothesis that there would be different brain  
283 information processes and network dynamics in CS versus control subjects. In theory, these  
284 differences would not just appear during the manifestation of CS but also during resting-state  
285 baseline as a precursor to CS or marker of susceptibility. The reason behind selecting two  
286 time points at baseline was to see if these markers might change over time.

287

288 Using the subtracted SNNr cube, clusters of reservoir neurons surrounding each input neuron  
289 were grouped by connection weight. Neuron proportion was calculated as the percentage of  
290 neurons in the cube belonging to each cluster. Total input cluster interactions were compared  
291 to each other in a Feature Interaction Network (FIN) analysis. FIN revealed relative strengths  
292 of functionally connected areas of the brain that discriminate between the two classes. The  
293 top 5 features (channels) by neuron proportion were chosen as input neurons to train a new  
294 SNNr cube, representing only the most informative features that define CS. Data for the

295 control group during VR immersion were selected as the median time of CS induction, which  
 296 was at the 39 second mark. This process is detailed in Figure 3.



297

298 Figure 3. Finding the top five features to create a new SNNr cube with key CS information; c  
 299 = EEG channels, CS = cybersickness, Ctrl = Control, sub = subtracted cube

300 *Producing a Feature Vector:*

301 Default NeuCube processing uses one reservoir cube trained on all data samples for  
 302 classification, with the notion that data of a certain label will have different spike activity and  
 303 spike propagation than data of another label[24]. This study took a different approach by  
 304 subtracting individually trained SNNr cubes, to produce synaptic connection weights within  
 305 the reservoir that form a neural map specific to CS. This map is a template through which  
 306 new data samples are parsed to obtain a feature vector, which is the synaptic connection  
 307 weights between input + reservoir neurons, and output neurons. A dynamic evolving SNN  
 308 algorithm (deSNN) was used to learn the association between class labels and the training  
 309 samples in a supervised manner. deSNN [15] has the advantage over other SNN classification  
 310 models in that it is computationally inexpensive and boosts the importance of the order in  
 311 which input spikes arrives, along with considering all other incoming spikes. Thus, it is  
 312 suitable for on-line learning and early prediction of temporal events. In this algorithm, a new  
 313 output neuron ( $O$ ) for each training sample was created. These output neurons connect to  
 314 every input and reservoir neuron ( $N$ ). The connections have initial weights that are set  
 315 according to the Rank-Order learning rule (RO).

316

317

$$w_{init}(N_n, O_m) = mod^{order(N_n, O_m)}$$

318

319

The RO learning rule boosts the importance of the first incoming spikes on neuronal

320

synapses. The advantage of RO is fast, one-pass learning and asynchronous data entry of

321

synaptic inputs. The value of the *mod* parameter for part 1 of this study was set to a default of

322

0.9. The *O*-*N* connection weights between the SNNr and the output deSNN neurons are then

323

further dynamically tuned by the following spikes via spike driven synaptic plasticity (SDSP)

324

– a modified version of STDP. Due to a bi-stability drift in the SDSP rule, once a weight

325

reaches the defined high value (resulting in LTP) or low value (resulting in LTD), it is fixed

326

for the rest of the training phase. The rate at which a weight reaches LTD or LTP depends on

327

the values of the set *drift* parameter.

328

329

$$w_{final}(N_n, O_m) = w_{init}(N_n, O_m) + drift_{up} * n_{spikes} - drift_{down} * n_{spikes}$$

330

331

*drift<sub>up</sub>* is the value increase in synaptic weight after pre-synaptic firing. *drift<sub>down</sub>* is the

332

value decrease in synaptic weight with no pre-synaptic firing. *drift<sub>up</sub>* is set to 0.08 and

333

*drift<sub>down</sub>* is set to 0.08 for part 1 of the study. SDSP works similar to STDP except that the

334

post-synaptic membrane potential is assumed to always reach above threshold when the pre-

335

synaptic neuron fires, leading to an increase in connection weight of the synapse between two

336

neurons. At the same time, if no firing occurs from the pre-synaptic neuron, the connection

337

weight of the synapse is decreased.

338 Altogether, the deSNN algorithm provided brain-inspired feature vectors for every sample,  
339 consisting of both input-output neuron connections, and reservoir-output neuron connections  
340 that can be classified.

341

342 The following connection strategies between the SNNr neurons and the deSNN classifier  
343 neurons were explored in this paper while searching for an optimal model:

- 344 - SNNr cube trained on all data of 32 input neurons; 1471 SNNr neurons connected to  
345 each output neuron in the evolved deSNN classifier;
- 346 - SNNr cube trained on all data of 32 input neurons; only the 32 input neurons are  
347 connected to the output neurons;
- 348 - SNNr cube trained on 5 channel data; 1471 SNNr neurons connected to each output  
349 neuron in the evolved deSNN classifier
- 350 - SNNr cube trained on all data using all combinations of 5 top input neurons (e.g. top  
351 channels); only the 5 input neurons are connected to each output neuron;

352

353 *ECG*

354 The following heart rate variability parameters were computed: PNS, SNS, SI, SDNN,  
355 RMSSD. The selected time segments were 2 minutes, 30s and 10s in length. Only RMSSD  
356 was analysed for the 10s time segments, due to the statistical unreliability of the other  
357 parameters for this length of data. RMSSD is considered a reliable indicator for  
358 parasympathetic cardiac activity robust to the signal noise of respiration. Meanwhile, SI is an  
359 index for sympathetic activity Both parasympathetic and sympathetic activity contribute to  
360 SDNN. PNS and SNS are validated indicators of parasympathetic and sympathetic  
361 activity[25,14].

362

363 *Classification*

364 Three different algorithms were used to classify the feature vectors, with leave-one-out cross  
365 validation (LOOCV):

366

367 *Modified KNN:*

368 A distance-based algorithm between data points. The study employed a modified version of  
369 KNN, in which the following parameters were optimized using an exhaustive grid search:

- 370 1)  $k$  is for all neighbours or restricted by class label;
- 371 2) Using Manhattan distance or Euclidean distance;
- 372 3) Distance initially weighted uniformly or by signal-to-noise ratio (SNR) that identifies  
373 the importance of the features (see the wwkNN method [37]);
- 374 4) Neighbours weighted during voting –
- 375 a. Uniform (equally)
- 376 b. By the inverse of their distance
- 377 c. By the function:  $\frac{\text{max distance} - (\text{neighbour} - \text{minimum distance})}{\text{max distance}}$
- 378 5) Feature weights weighted during voting –
- 379 a. Uniform for each feature
- 380 b. SNR for each feature

381

382 *Linear Discriminant Analysis (LDA):*

383 An algorithm that finds linear combinations of features that separate classes along a  
384 hyperplane. Least squares solution was used with optimized shrinkage.

385

386 *Light Gradient Boosting Machine (LightGBM):*

387 LightGBM is a gradient boosting framework that uses tree-based learning algorithms.

388 Optimized for number of trees, learning rate, boosting type (gradient boosting decision tree,  
389 GBDT), gradient-based one-side sampling (goss), dropouts meet multiple additive regression  
390 trees (dart)).

391

392 This study approached data fusion by combining feature vectors representing synaptic  
393 connection weights with the output layer of NeuCube and HRV variables that yielded the  
394 best accuracies. These include the best combination of parasympathetic or sympathetic  
395 features which would be added on to the final feature vector.

396

397 Part 2 of the study used high capacity computing provided by New Zealand eScience  
398 Infrastructure (NeSI) to extend the previous analysis using the modified KNN algorithm. The  
399 goal was to find the best time segment for prediction out of the 2 minute EEG resting-state  
400 baseline, partitioned into varying data lengths (2s, 5s, 10s). The analysis similarly scans  
401 through all types of model training, and feature vector type in terms of connections to the  
402 output neurons as in Part 1. The difference is that *mod* and *drift* parameters for SDSP were  
403 optimized for classification of the best time segment for prediction and also for detection to  
404 see if this would improve accuracy. Additionally, the value of *drift<sub>up</sub>* was set to always be  
405 more than *drift<sub>down</sub>*, which implements stronger I-O connection increases compared to  
406 decreases. This type of SDSP also maintains stronger I-O connections for input neurons that  
407 fire more compared to those that fire less, thereby boosting their importance further. Varying  
408 data lengths serve to explore whether capturing more EEG data improves prediction  
409 accuracy, whereas optimizing for SDSP improves the transformation of the EEG data into the  
410 feature vector for classification.

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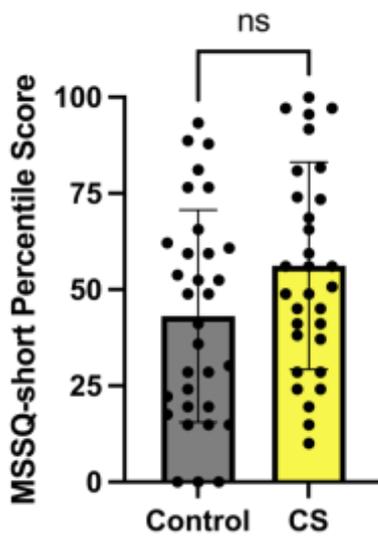
412 **Results**

413 **Part 1**

414 *MSSQ-short and SSQ Scores*

415 MSSQ-short scores did not differ significantly ( $P>0.05$ ) between CS and Control groups  
416 (Figure 4). SSQ scores differed significantly between CS and control ( $P>0.0001$ ) (Figure 5).  
417 CS groups had significantly higher SSQ scores than controls, showing that MSSQ-short  
418 percentile scores were not a good indicator of sickness in VR usage.

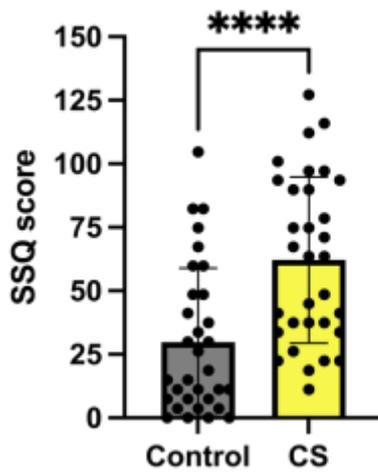
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422 **Figure 4.** MSSQ-short scores  $P>0.05$ . Error bars show  $\pm$  SEM.

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427 **Figure 5.** SSQ scores, \*\*\*\* =  $p < 0.0001$ . Error bars show  $\pm$  SEM.

428

429 *EEG*

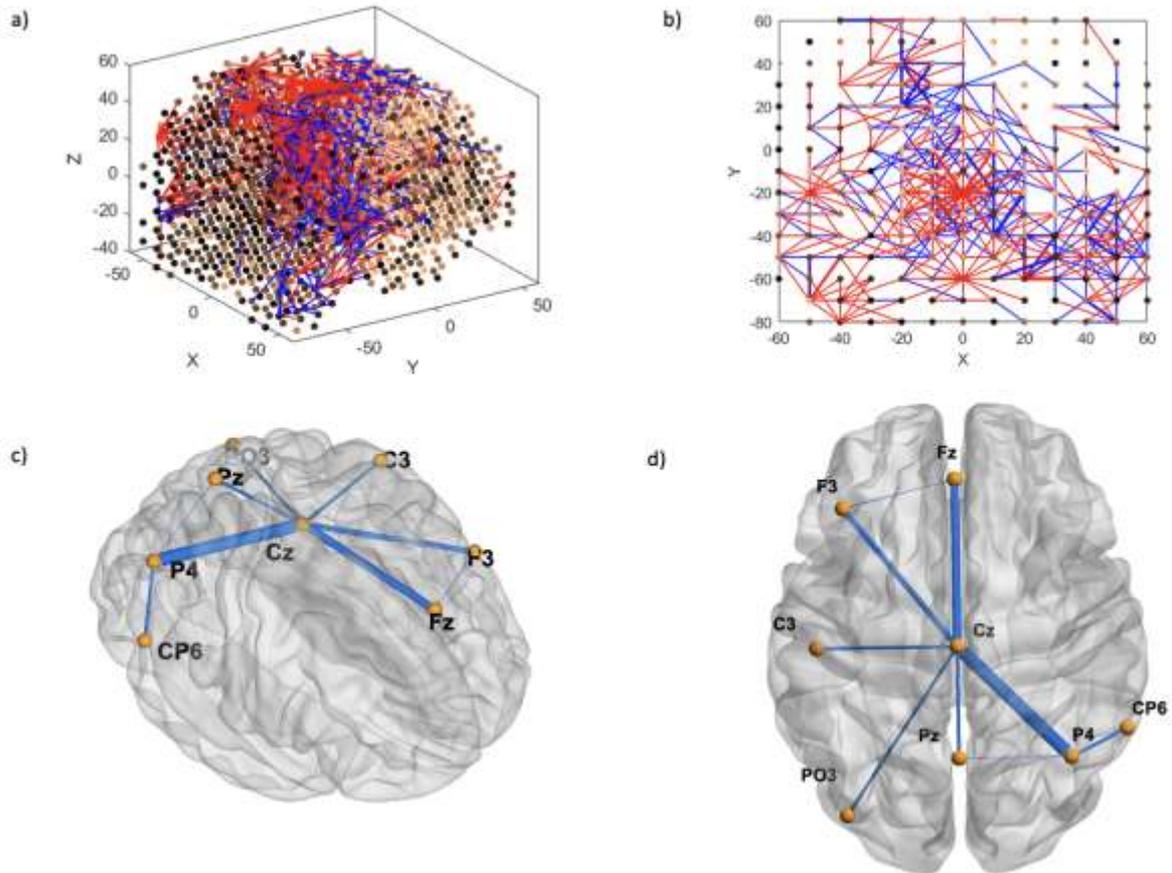
430 Functional connectivity analysis at resting baseline EEG (30-32s) shows that CS prone  
431 individuals have more concentrated negative connections in the Cz area, interspersed with  
432 surrounding positive connections (Figures 6a and 6b), when compared to controls. Feature  
433 interaction analysis (FIN) revealed that Cz is likely a hub for brain activity processing in this  
434 time segment, either collecting or sending out this information to all the other key channels  
435 located in the left and interhemispheric frontal and bilateral parietal areas (Figure 6c and 6d).  
436 The top 5 features according to neuron proportion were P4, Fz, Cz, PO3 and F3, with Cz  
437 being the highest (Figure 7). A second time segment further on in the baseline (90-92s) was  
438 analysed, which showed that other important features (T8, CP6, Fz, FC5, T7) can appear at  
439 different time segments (Figure 8 and Figure 9). During the CS event, the high functional  
440 connectivity seen at baseline in CZ changes to interspersed positive and negative  
441 connections. Meanwhile there is a shift towards O2 positive connection dominance. FIN  
442 analysis (Figure 10) showed that both O2 followed by Cz are most likely hubs for  
443 cybersickness processing, where both have the highest neuron proportion (Figure 11). The  
444 top 5 features according to neuron proportion were FC6, FP2, FP1, Cz and O2. These results  
445 indicate that important areas identified in the baseline that are also found during the  
446 manifestation of cybersickness could be important biomarkers of susceptibility to  
447 cybersickness.

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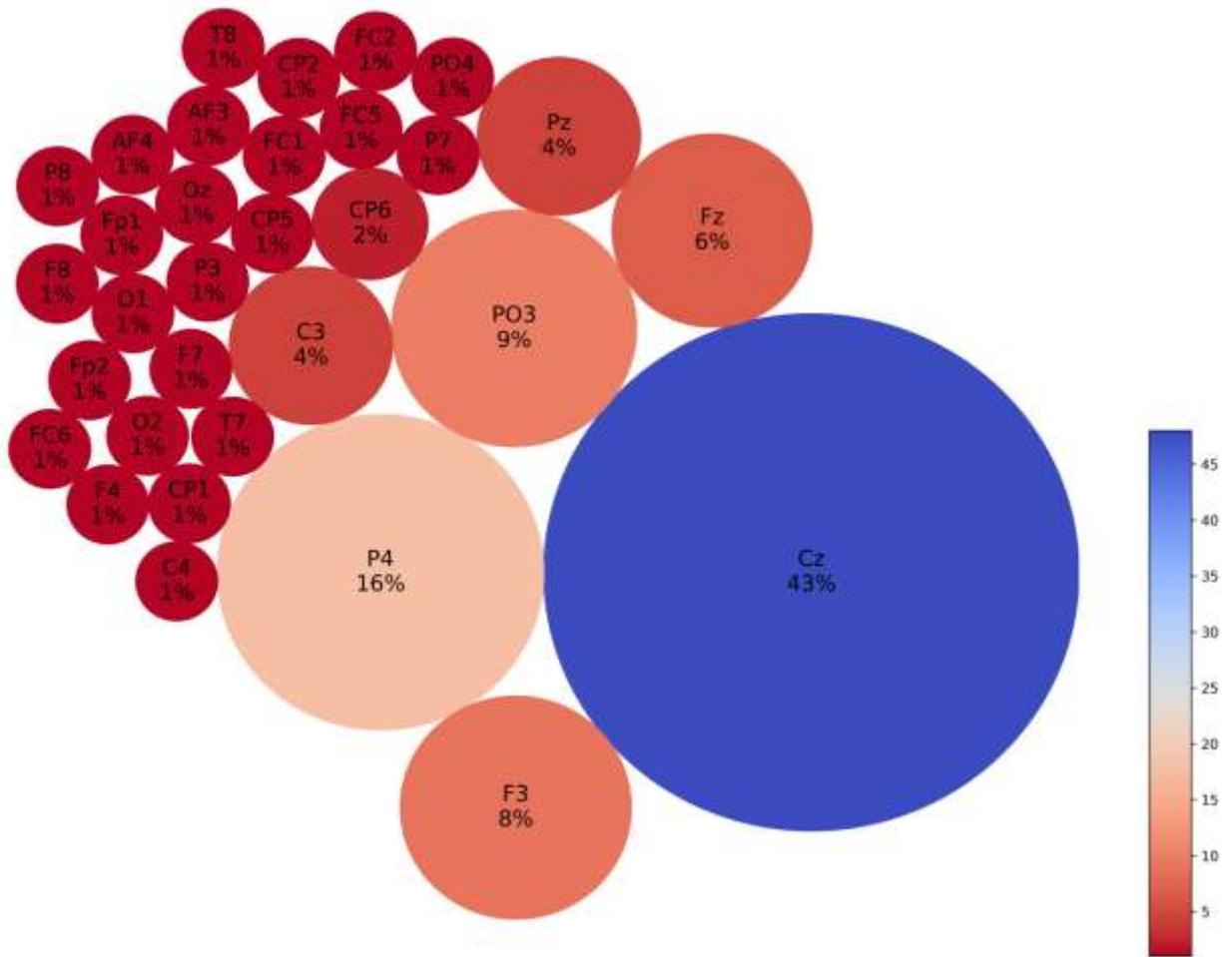
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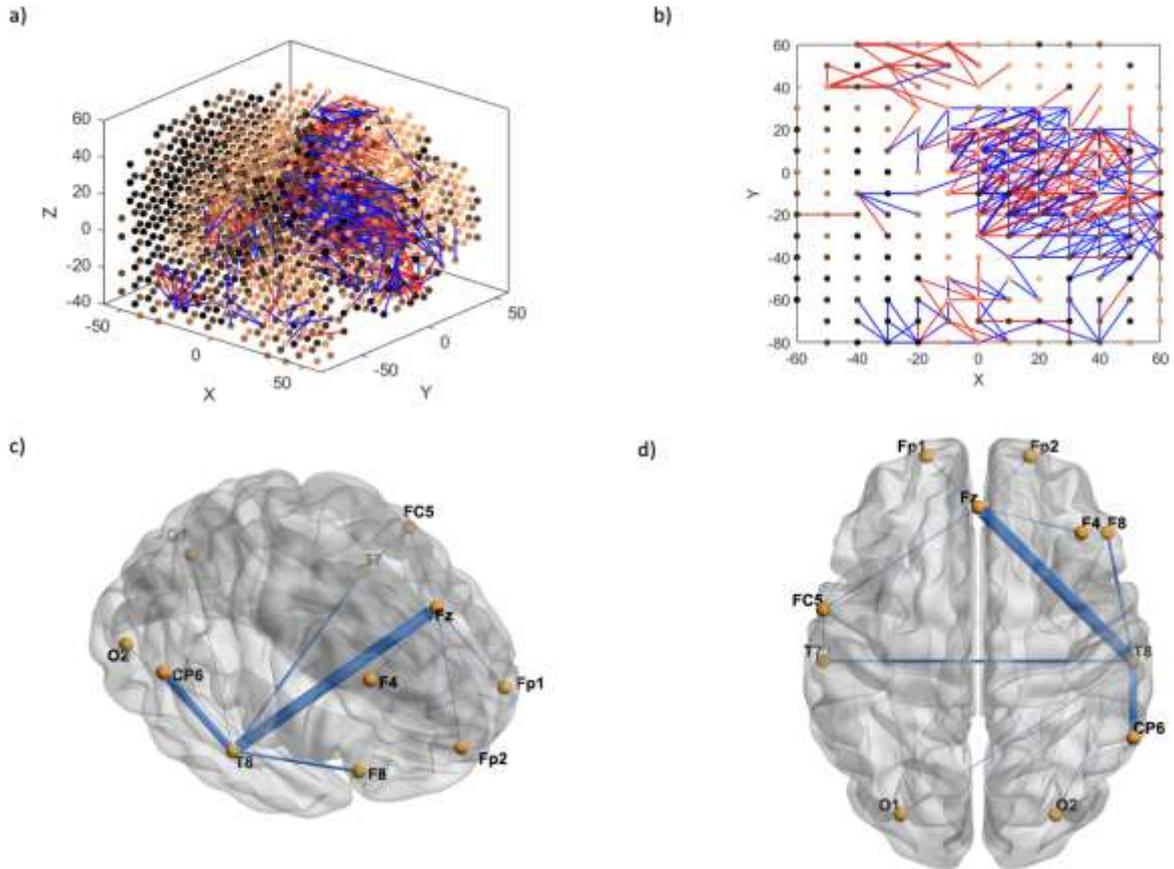


452 **Figure 6.** Resting-state baseline 30-32s subtracted network dynamics. Functional  
 453 connectivity of neurons in the SNNr is represented by **a)** right hemisphere medial view of the  
 454 SNNr and **b)** axial view. Blue lines are positive connections, red lines are negative  
 455 connections. Brighter neurons have stronger connections. Feature interaction networks  
 456 between channels are represented by **c)** right hemisphere medial view and **d)** **axial view**.  
 457 Thicker lines indicate stronger interaction whether they be positive or negative. *These*  
 458 *interactions confirm our hypothesis that even at baseline of 30-32s, there is a significant*  
 459 *difference between the brain information processes of CS versus control subjects.*



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**Figure 7.** Neuron proportion clustered by connection weights in the subtracted SNNr = SNNr/control – SNNr/cs at resting-state baseline 30-32s. Blue indicates higher proportion, red indicates less neuron proportion. It shows a larger difference between the CS and control subjects in the brain areas Cz, F3, P4, PO3, Fz, Pz and C3, with a dominant factor of Cz.



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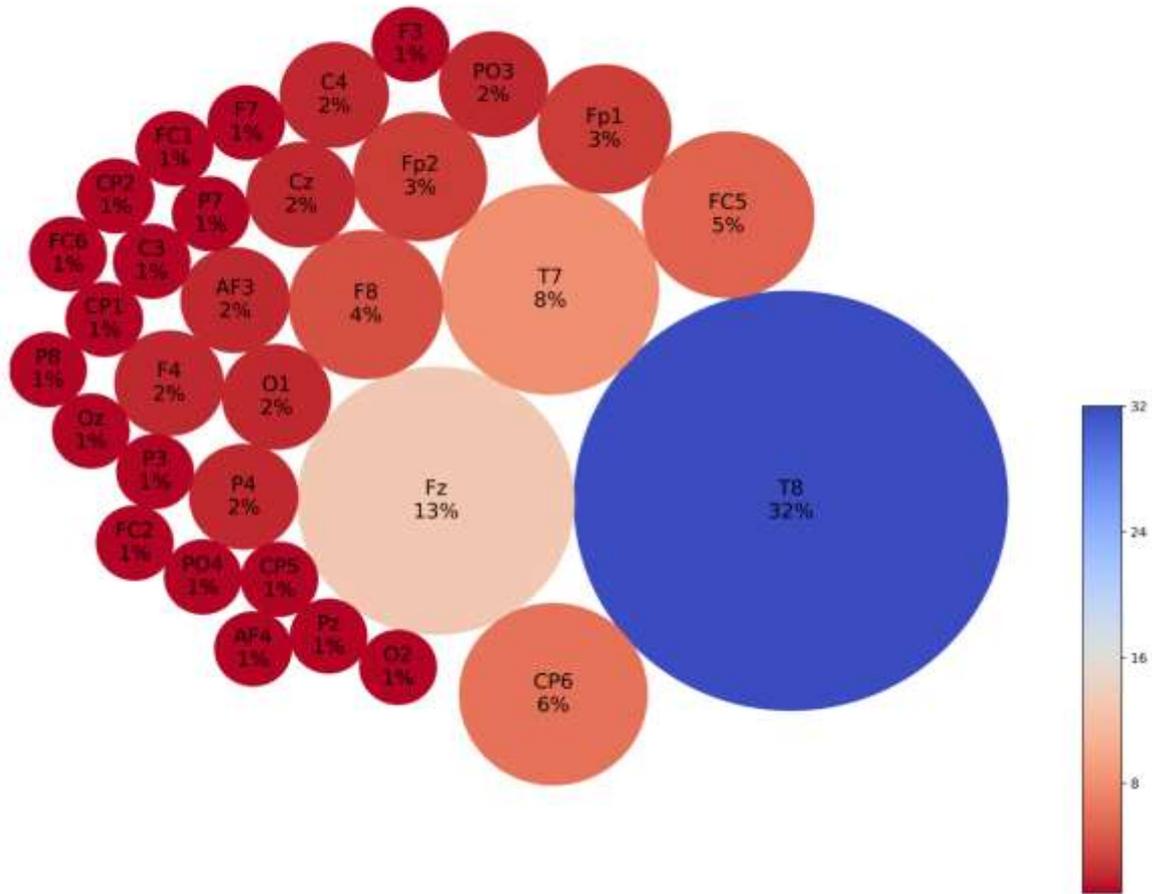
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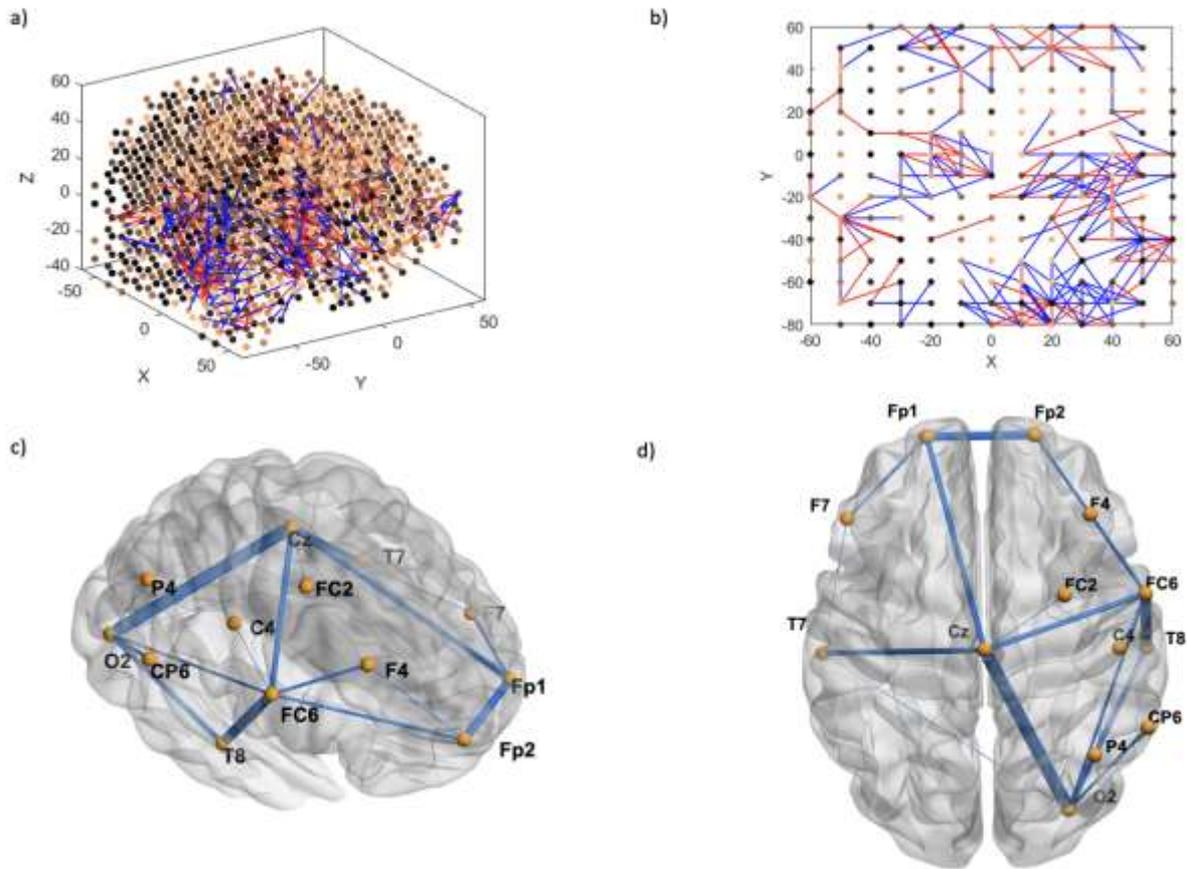
**Figure 8.** Resting-state baseline 90-92s subtracted network dynamics. Functional connectivity of neurons is represented by **a)** right hemisphere medial view and **b)** axial view. Blue lines are positive connections, red lines are negative connections. Brighter neurons have stronger connections. Feature interaction networks between channels are represented by **c)** right hemisphere medial view and **d) axial view**. Thicker lines indicate a stronger interaction whether they be positive or negative. *These interactions confirm our hypothesis that even at the 90-92 baseline, there is a significant difference between the brain information processes of CS versus control subjects.*



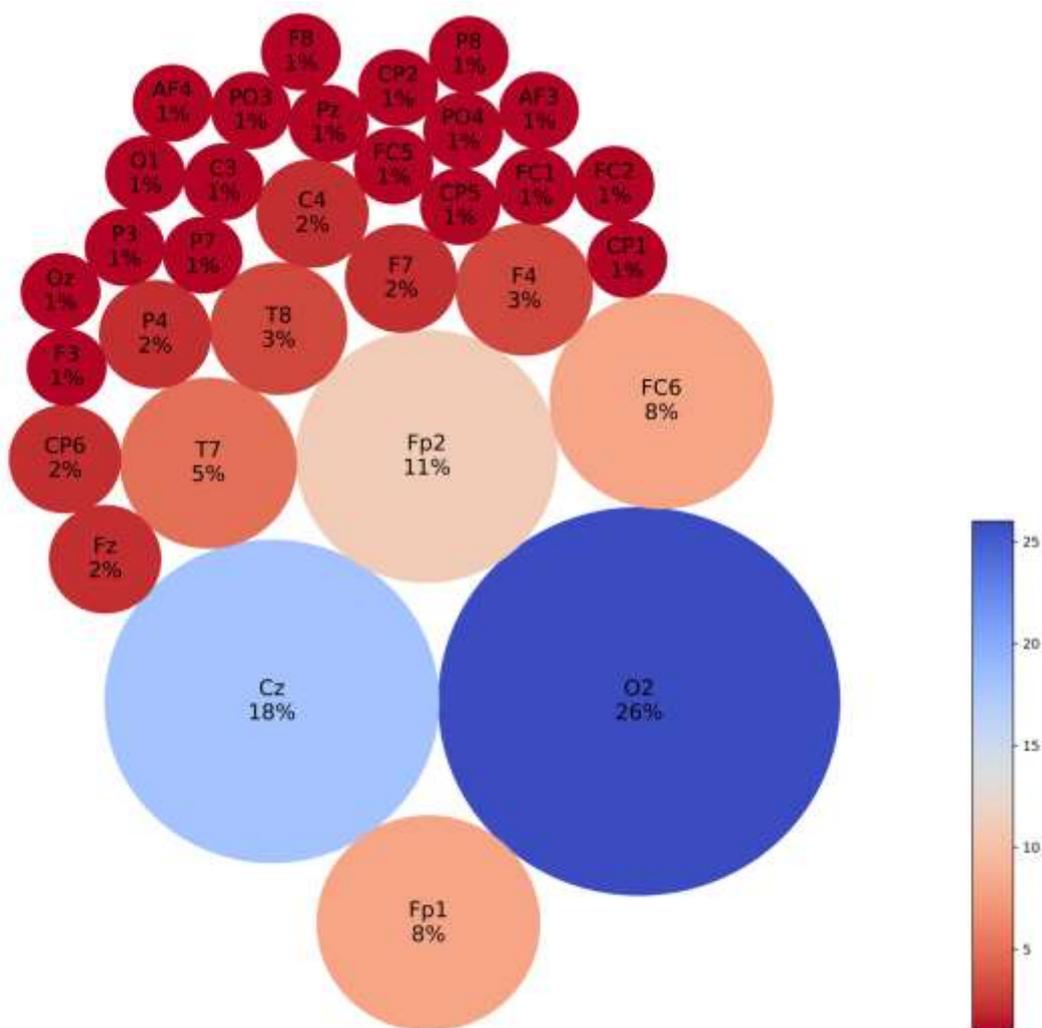
489

490 **Figure 9.** Neuron proportion clustered by connection weight at 90-92s resting-state baseline.  
491 Blue indicates higher proportion, red indicates less neuron proportion. It shows a larger  
492 difference between the CS and control subjects in the brain areas T8, CP6, Fz, T7, FC5 with a  
493 dominant factor of T8.

494



495 **Figure 10.** Cybersickness network dynamics of the  $SNNr=SNNr/control - SNNr/cs$ , in VR.  
 496 Functional connectivity of neurons is represented by **a)** medial view) and **b)** (axial view).  
 497 Blue lines are positive connections, red lines are negative connections in **a) and b)**. Brighter  
 498 neurons have stronger connections. Feature interaction networks between channels are  
 499 represented by **c)** and **d)**. Thicker lines indicate stronger interaction whether they be positive  
 500 or negative. *These interactions confirm our hypothesis that there is a significant difference*  
 501 *between the brain information processes of CS versus control subjects during the CS*  
 502 *manifestation when subjects are exposed to VR. Some of these interactions have been*  
 503 *captured already at baseline (see Fig.6 and Fig.8).*  
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**Figure 11.** Neuron proportion in  $SNNr = SNNr/control - SNNr/cs$ , clustered by connection weights during a VR experiment. Blue indicates a higher proportion, and red indicates a lower proportion of neurons. *The difference in the connectivity confirms our hypothesis that there is a significant difference between the brain information processes of CS versus control subjects during the CS manifestation, when subjects are exposed to VR, with dominating brain areas being O2, Cz, Fp2, Fp1 and Fc6. Some of these areas have been captured already at baseline (see Fig.7).*

522 *Classification results*

523 Overall, our modified KNN algorithm was the best for both prediction (EEG 76.6%, ECG  
 524 74.2%) and detection (EEG 75%, ECG 72.6%) of CS (Tables 1-5 and Figure 12). Both EEG  
 525 and ECG had similar classification accuracies, although EEG alone was slightly better.  
 526 Although data fusion of both EEG and ECG could increase accuracy to 77.4% for prediction,  
 527 it reduced the accuracy for detection to 70.9% (Figure 12).

528

<b>Prediction 30-32s</b>						
<b>I-O connectio n</b>	32 Channel Trained			5 Channel Trained		
	LDA	KNN	LGBM	LDA	KNN	LGBM
<b>1471 Reservoir + I-O</b>	59.4%	65.60%	68.8%	53.1%	67.20%	53.1%
<b>32</b>	62.5%	67.20%	62.5%	N/A	N/A	N/A
<b>5</b>	54.7%	60.90%	54.7%	59.4%	73.40%	59.4%
<b>P4</b>	48.4%	65.60%	57.8%	46.9%	51.60%	51.6%
<b>Fz</b>	50.0%	59.40%	46.9%	57.8%	70.30%	70.3%
<b>Cz</b>	43.8%	60.90%	57.8%	39.1%	60.90%	54.7%
<b>PO3</b>	0.00%	57.80%	48.4%	0.00%	57.80%	54.7%
<b>F3</b>	53.1%	62.50%	62.5%	53.1%	64.10%	64.1%
<b>Best combo out of 5</b>	Cz+F3 56.3%	P4, Fz, Cz 75.00%	P4, PO3 64%	Cz+F3 62.5%	Fz, Cz <b>76.6%</b>	Cz 70.3%

529

530 Table 1. Prediction accuracies of LDA, modified KNN and LGBM classification algorithms  
 531 at baseline 30-32s for all subtracted SNNr Cubes.

532

<b>Prediction 90-92s</b>						
<b>I-O connection</b>	32 Channel Trained			5 Channel Trained		
	LDA	KNN	LGBM	LDA	KNN	LGBM

<b>1471 Reservoir + I-O</b>	53.1%	67.20%	60.9%	50.0%	65.60%	73.4%
<b>32</b>	57.8%	70.30%	64.1%	N/A	N/A	N/A
<b>5</b>	51.6%	68.80%	70.3%	54.7%	64.10%	68.8%
<b>T8</b>	48.4%	71.90%	60.9%	50.0%	62.50%	56.3%
<b>CP6</b>	59.4%	54.70%	56.3%	56.3%	64.10%	67.2%
<b>Fz</b>	50.0%	60.90%	59.4%	0.00%	64.10%	54.7%
<b>FC5</b>	18.8%	60.90%	56.3%	42.2%	60.90%	54.7%
<b>T7</b>	23.4%	65.60%	53.1%	42.2%	57.80%	54.7%
<b>Best combo out of 5</b>	T8+CP6+Fz 59.4%	T8 73.40%	T8 61.3%	T8,CP6,Fz, FC5 57.8%	T8, CP6 <b>75%</b>	T8,CP6 66.1%

533

534 Table 2. Prediction accuracies of LDA, modified KNN and LGBM classification algorithms

535 at baseline 90-92s for all subtracted SNNr Cubes.

536

<b>Detection CS onset</b>						
<b>I-O connectio n</b>	32 Channel Trained			5 Channel Trained		
	LDA	KNN	LGBM	LDA	KNN	LGBM
<b>1471 Reservoir + I-O</b>	57.8%	70.30%	75.0%	56.3%	57.80%	65.6%
<b>32</b>	65.6%	<b>75.00%</b>	70.3%	N/A	N/A	N/A
<b>5</b>	50.0%	62.50%	67.2%	53.1%	62.50%	60.1%
<b>FC6</b>	59.4%	65.60%	62.5%	53.1%	67.20%	65.6%
<b>Fp2</b>	42.2%	56.30%	59.4%	43.8%	59.40%	59.4%
<b>Fp1</b>	46.9%	60.90%	57.8%	0.00%	64.10%	59.4%
<b>Cz</b>	25.0%	56.30%	54.7%	53.1%	56.0%	64.1%
<b>O2</b>	51.6%	53.10%	45.3%	0.00%	50.0%	59.4%
<b>Best combo out of 5</b>	FC6 59.4%	Fp2, Cz 68.80%	Fp1,Cz 68.8%	FC6+Fp2+C z 57.8%	Fp2, Cz 68.80%	FC6,Fp1,Cz 68.8%

537

538 Table 3. Detection accuracies of LDA, modified KNN and LGBM classification algorithms

539 at the time of the CS event for all subtracted SNNr Cubes.

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541

ECG Prediction			
Time Segment	ML Algorithm		
	LDA	KNN	LGBM
<b>2 Min Baseline</b>	56.5% SI+SDNN	<b>74.2%</b> PNS+SNS	69.4% SNS
<b>15-45s</b>	61.3% PNS + SNS +SDNN + RMSSD	67.7% SI	67.7% SNS + SI + RMSSD
<b>75-105s</b>	62.9% PNS + SNS	<b>74.2%</b> SNS	71.0% SNS +SDNN
<b>25-35s</b>	6.5% RMSSD	67.7% RMSSD	61.3% RMSSD
<b>85-95s</b>	16.1% RMSSD	59.7% RMSSD	51.6% RMSSD

542

543 Table 4. Prediction accuracies of LDA, modified KNN and LGBM classification algorithms  
544 at different time segments for the best combination of HRV parameters.

545

ECG Detection			
Time Segment	ML Algorithm		
	LDA	KNN	LGBM
<b>2 Min VR</b>	61.3% ( PNS or SDNN) + SNS + Mean HR	<b>72.6%</b> SNS+SI	69.4% SI + SDNN
<b>30s VR</b>	66.1% PNS + SNS +SI	69.4% PNS + SNS + SI/ PNS + SI + RMSSD	67.7% SI + SDNN + Mean HR
<b>VR 10s</b>	56.5% RMSSD	58.1% RMSSD	54.8% RMSSD

546

547 Table 5. Detection accuracies of LDA, modified KNN and LGBM classification algorithms  
548 at different time segments for the best combination of HRV parameters.

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550

551 **Figure 12.** Best KNN classification accuracies for EEG and ECG (HRV) in multiple time

552 segment analyses.

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554 *EEG considerations*

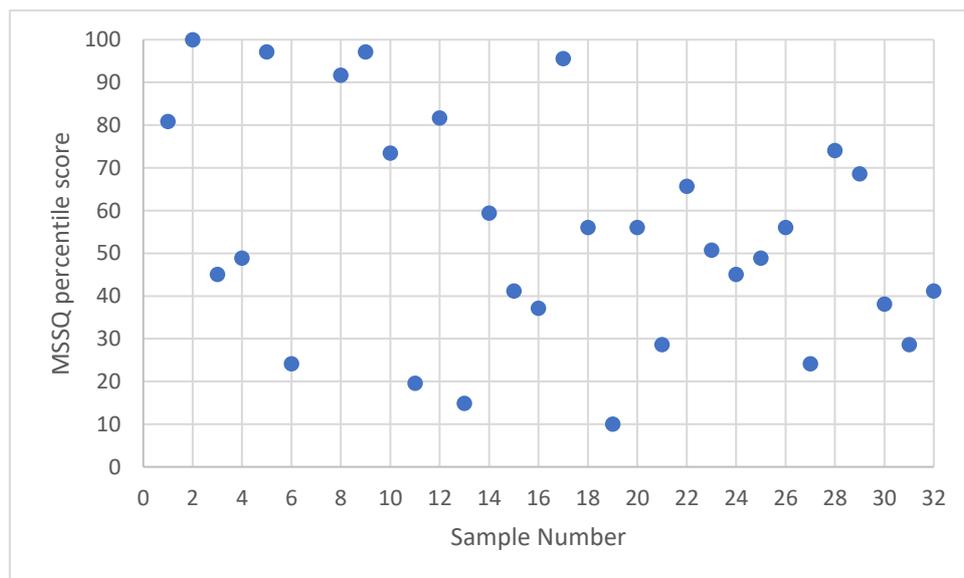
555 Some participants were predicted at 30-32s (samples 3, 4, 18, 24, 27) but not at 90-92s and

556 vice versa (samples 9, 22 and 23). A hypothesis was that spike count, MSSQ-short percentile

557 scores, SSQ total scores, or CS onset times could explain why some participants were

558 predicted in one baseline segment but not the other. This was not the case, as none of the

559 above showed any deviation from the norm when graphed (appendix 1-6).

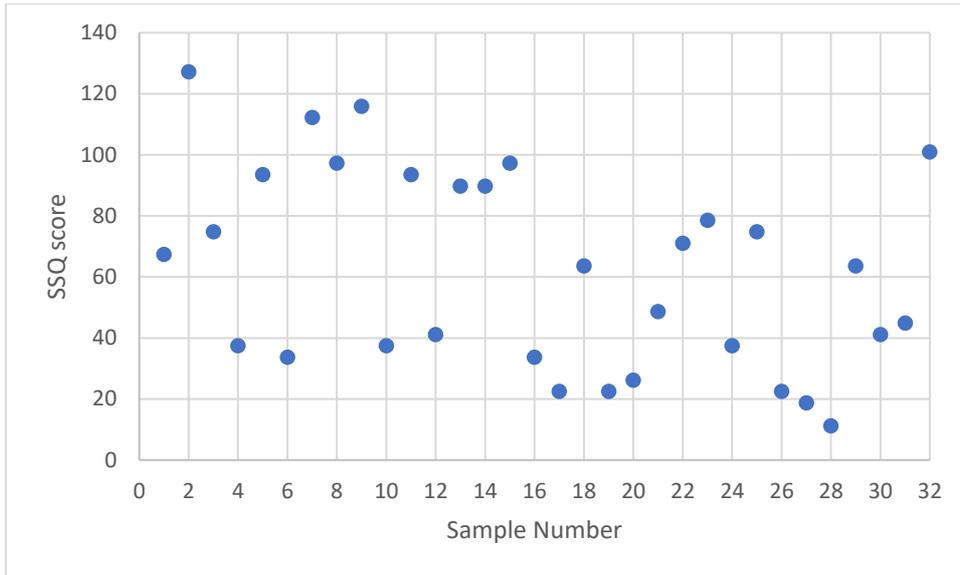


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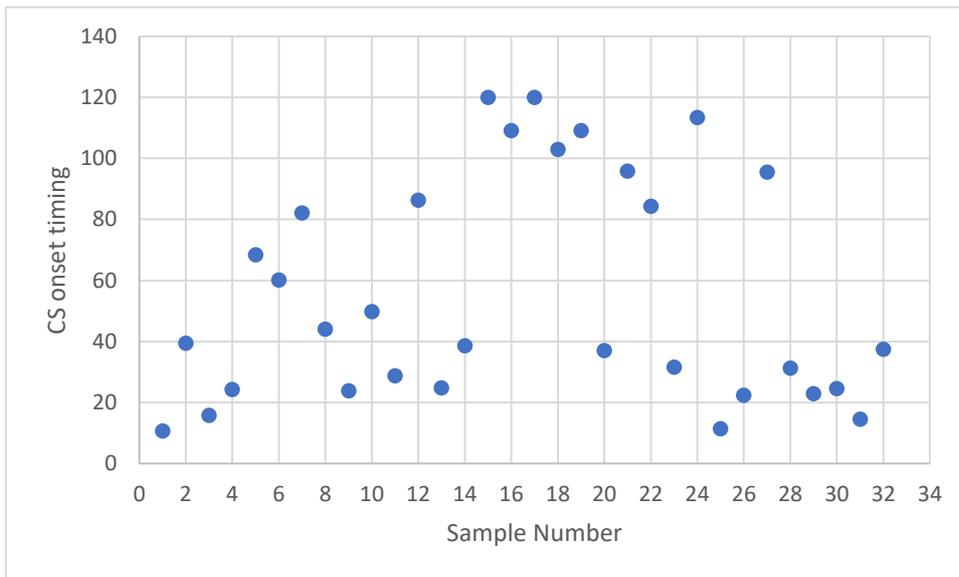
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562 Appendix 1. MSSQ percentile score breakdown for the CS group.

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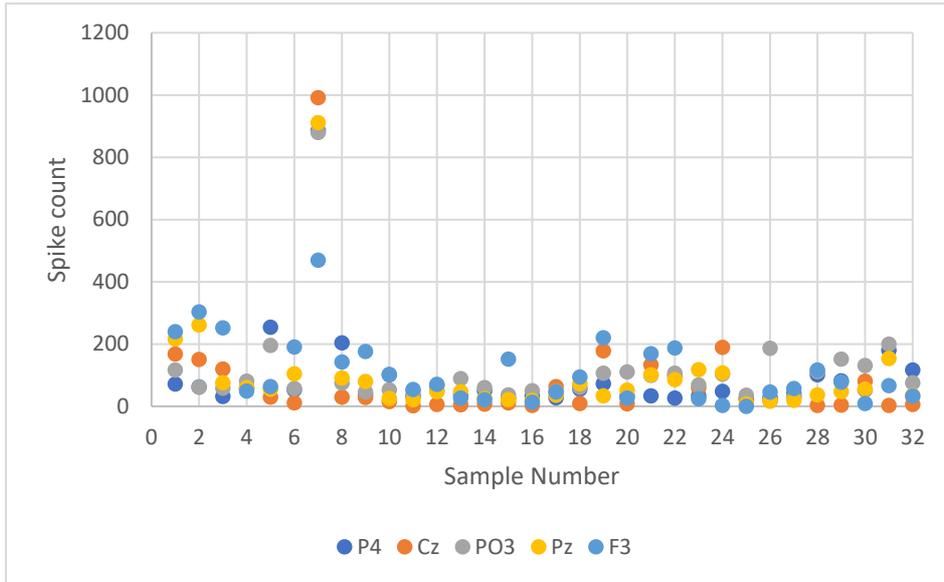


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566 Appendix 2. SSQ score breakdown for the CS group  
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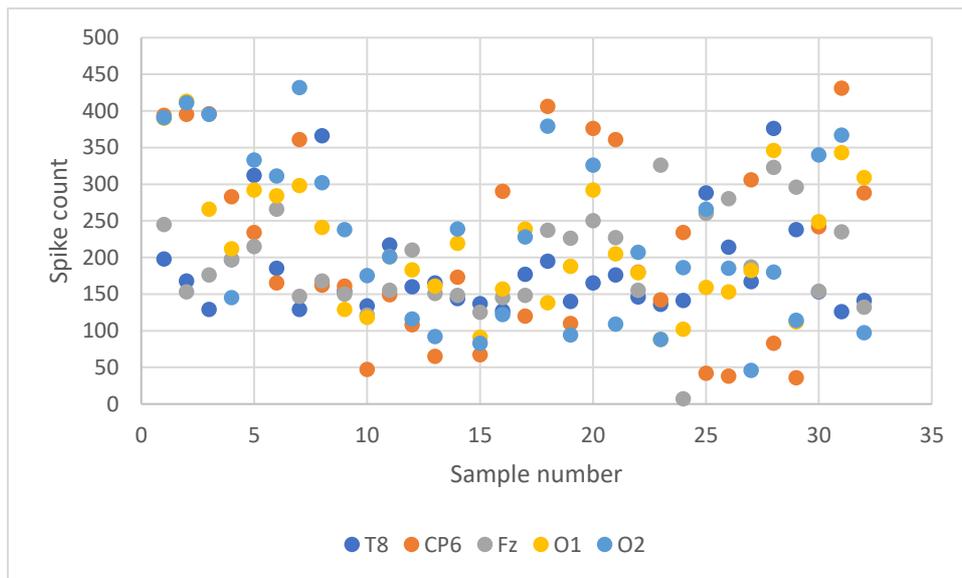


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586 Appendix 3. CS onset timings  
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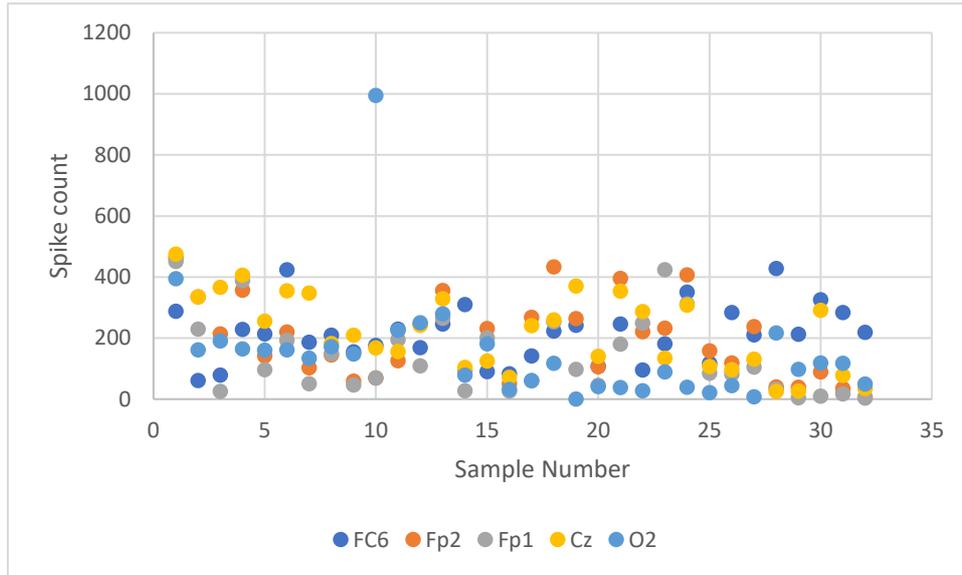


Appendix 4. Spike count of 30-32s time segment for all CS participants. Sample number 3, 4, 18, 24 and 27 were predicted at 30-32s but not at 90-92s.



Appendix 5. Spike count of 90-92s time segment for all CS participants. Sample number 9, 22, 23 were predicted at 90-92s but not at 30-32s.

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Appendix 6. Spike count for all CS participants during the CS onset.

650 It was hypothesized that the spike count at each channel would be different in the CS groups  
651 compared to controls at all time segments. We found that the spike count was significantly  
652 lower in the CS group than in the controls at the 30-32s baseline segment (for P4, Fz, Cz  
653 PO3, F3) and during VR immersion (O2) ( $P < 0.0001$ ), but not at 90-92s. Sample 7 in 30-32s  
654 has a high spike count compared to others, as does sample 10 during VR immersion, but  
655 removal of these samples does not change the statistical differences (30-32s  $P < 0.0001$ , VR  
656 immersion  $P < 0.001$ ) between CS and controls spike count. Classification accuracy, however,  
657 remains similar for 30-32s and 90-92s analysis (76.6% and 75.0%, respectively).

658

659 *ECG and HRV considerations*

660 Sympathetic indexes (SNS + SI) outclassed other HRV parameters in terms of classification  
661 accuracy. A normal control baseline may be easy to predict or detect, but small changes in  
662 these HRV values may not always equate to cybersickness. The differences between CS and  
663 Ctrl groups must be more complicated than just their means, medians and distributions,  
664 because as a whole, there are no significant differences ( $p > 0.05$ ) between any of the HRV

665 parameters used here according to a Mann Whitney u rank test. Furthermore, both of the  
 666 KNN algorithms employed here employ a min/max voting type on the importance of K-  
 667 neighbours, which takes into account a weighted Euclidian distance via signal to noise ratio  
 668 (SNR) between sample data points. These weightings between all data points and between K-  
 669 neighbours are still influenced by the sample sizes and distribution of the data. Therefore, it  
 670 may also be possible that a larger sample size is needed to more accurately represent  
 671 cybersickness.

672

673 **Part 2**

674 After extensive analysis of different time segments and data lengths, the 2 second time  
 675 segment relating to 110 - 112 seconds, with only one of I-O connection (F7 channel), yielded  
 676 the best results of 85.9% accuracy. Overlapping time segments for data lengths 5s and 10s  
 677 did not reach the same performance, achieving a max 75-76% using the best combination of  
 678 I-O connections. In addition, detection performance was boosted to 76.6% after SDSP  
 679 optimization.

680

	EEG	EEG + ECG Fusion
Prediction	85.9% (F7)	74.2% (75-105s, SNS)
Detection	76.6% (FP1, Cz)	72.6% (2 mins, SNS + SI)

681

682 Table 6. Improved accuracies for CS prediction and detection using EEG. Analysis included  
 683 time segment and data length optimization (prediction only), and SDSP optimization of the  
 684 *mod*, *driftup* and *driftdown* parameter. Fusion accuracies increased for detection but not for  
 685 prediction.

686

687

688 **Discussion**

689 *Classification*

690 This paper presents a proof of concept for on-the-spot prediction of cybersickness at resting  
691 state baseline and near-instant detection of cybersickness during its onset. The algorithms are  
692 based on brain inspired SNN architectures and HRV classification. Another study has also  
693 demonstrated the predictive capacity of their algorithm for CS at resting baseline with a  
694 smaller sample size of n=19[26]. Near-instant detection was demonstrated by Nam et al. [27]  
695 but required PCA preprocessing, power spectral analysis for EEG and 7 other biosignals. The  
696 present study shows that only 2 seconds of EEG data and 30 seconds of ECG data are  
697 required, and both biosignals can be used individually or together to predict and detect CS.  
698 The modified deSNN-KNN classification algorithm produced the best results in terms of  
699 accuracy, over LDA and light-GBM. It was found that similar classification accuracies can be  
700 obtained by using either earlier (30-32s, 76.6%) or later time segments (90-92s, 75%) at  
701 baseline. Upon further investigation, the study found that time segment optimization was still  
702 important (85.9%). Simplifying feature vectors by removing reservoir – output neuron  
703 connections, and leaving the direct connections of input neurons to output neurons increases  
704 accuracies (Table 1-3). In addition, reducing redundancy in training data by focusing on key  
705 cybersickness relevant areas also has the same positive effect on accuracy. However, in the  
706 case where a model is trained on all 32 features, but only the top 5 features are considered, a  
707 reduction leads to a decrease in accuracy (75.00% to 68.80%) (Table 3). This highlights that  
708 in idealistic scenarios, not just a few but all features a model is trained on should be  
709 considered when eliminating redundancy. However, it is important to note that there is a  
710 trade-off in considering all features, as computational cost increases when conducting  
711 exhaustive searches.

712

713 Our analysis did not reveal why some participants were predicted in one baseline segment but  
714 not the other. An explanation is that this could be due to differences in the temporal  
715 characteristics of the spiking activity of neurons captured by the connection weights between  
716 input clusters and between individual reservoir neurons. Another explanation could be due to  
717 the nature of clinical studies, where there is interindividual variation between participants.

718

719 Fusion of EEG and ECG did not yield much improvement in accuracy, and in the case of  
720 detection it worsened accuracies in part 1. Multi-modal data fusion was investigated to  
721 explore if information from two organs would lead to increased accuracy, especially because  
722 they are biologically linked through the nervous system both in anatomy and also in  
723 association to nausea[28]. Because of the disparity in classification performances between  
724 EEG and ECG, it is likely that the classification algorithm's ability to differentiate strongly  
725 between labels is 'drowned' out by the ECG HRV features, which is why the EEG now adds  
726 no useful information for classification beyond what is already there. It is also possible that  
727 KNN being a distance based algorithm, gets worse with higher dimensional feature vectors, a  
728 trend shown as well in the improved classification performances the less features there are in  
729 the feature vector.

730

731 *MSSQ and SSQ scores*

732 In our experiment, the MSSQ-short was not a good predictor of cybersickness induction or  
733 sickness ratings. This points towards the need for questionnaires more targeted at visually  
734 induced motion sickness[29] to assess susceptibility. SSQ scores were a good adjunct to the  
735 subjective cybersickness reports in the separation of cybersick and control groups.

736

737 *Related spatiotemporal brain dynamics were discovered in the following areas:*

738 Fz Brodmann 8 visual attention and eye movements

739 T8, T7: Auditory processing

740 CP6: Auditory processing, speech comprehension

741 O1, O2: Retinotopic mapping of visual scene, edge detection

742 P4: Angular gyrus attention, memory retrieval, language number processing, spatial

743 cognition

744 PO3: Associative visual cortex (V3, V4, V5).

745 F3: Frontal eye fields, visual attention and eye movements.

746 FC5, FC6: Brocas speech production and articulation (primarily left hemisphere),

747 language processing.

748 FP1, FP2: Executive function, decision making

749 F7, F8: Active maintenance of stimulus information, interoceptive, limbic emotion-

750 motivational, and sensory input integration

751 CS is a complex condition with many brain areas involved[30,31]. Presented in this study is

752 functional connectivity of the brain that predicts future CS, meaning that an individual with

753 similar neural maps may be susceptible to cybersickness, and connectivity that marks the CS

754 event. In the present study, a high neuron proportion grouped by connection weight of frontal

755 (FC6, FP1, FP2) regions during the CS event, and temporal regions (T8) during resting

756 baseline are consistent with another study showing changes in these areas well into the CS

757 event. In addition, areas involved in CS include those for visual + attention processing and

758 executive function (CP6, O2, PO3, F3, F4, FP1, FP2). Liu et al. [30] found reduced

759 gravitational frequency means (transition of EEG power spectral density, temporal changes

760 within a frequency band), and gravitational frequency standard deviation (dispersion of brain

761 signal) at FP1, FP2, TP9 and TP1. Power spectral entropy (disorder of time sequence signals  
762 and irregularity of multi-frequency component signals) and Kolmogorov complexity (time  
763 domain complexity) were all reduced at FP1 and FP2 during VIMS[30]. However, it was  
764 noted that these changes may be related to other factors, such as alertness level or various  
765 mental conditions, and not limited or specific to VIMS. Our finding of an increase in O2's  
766 interaction with other areas during cybersickness highlights that visual processing is altered  
767 beyond just the demands of normal visual processing in VR. O2 has been selected as an  
768 important feature in other machine learning studies as well [27,32,33], but the possible  
769 differences in results compared to the discussed brain analysis and imaging studies may be in  
770 the temporal specificity (2 seconds long) of our analysis compared to longer data lengths  
771 analysed.

772

773 Of interest is the brain activity hub found at Cz, which had altered connectivity at resting-  
774 state baseline as well as during the onset of cybersickness when compared with controls.  
775 Reduced spike count at Cz before VR immersion may indicate that there is less frequency of  
776 communication from this area to other connected areas. Cz interacts with three cortices  
777 simultaneously, the somatosensory, motor and also is positioned over the mid cingulate,  
778 which has increased functional connectivity with the left V5/MT during cybersickness[34].  
779 Krokos, Varshney [35] found high activity power in the central regions similar to the location  
780 of Cz, of average scalp maps according to independent component analysis. Brodmann area 5  
781 corresponds to Cz, which is part of the superior parietal lobule and post central gyrus. It is  
782 located immediately posterior to the primary somatosensory cortex. Neuroimaging evidence  
783 suggests that this area contributes to movement planning. Furthermore, one study showed a  
784 correlation between the activity of area 5 neurons and the starting or final coordinates of limb  
785 movement. This suggested that BA5 is involved in processing spatial information for limb

786 movement. Emerging evidence suggests that BA5 is also involved in the inhibition of  
787 movement[36]. A transcranial magnetic stimulation study found a causal role for BA5 in the  
788 regulation of corticospinal output during preparation that differentiates between whether a  
789 movement is withheld or executed[37]. One may think that Cz's role in movement and also  
790 as a marker of future cybersickness at resting baseline lends possible credence to the postural  
791 instability theory of motion sickness, which postulates that postural instability is both a  
792 marker and a predictor of motion sickness, likely extending as well to cybersickness in virtual  
793 reality[38]. Our results, however, suggest that although processes related to motor control are  
794 altered during the event, we cannot speak for postural instability itself. Furthermore, a recent  
795 study shows that postural instability itself is not a good predictor of cybersickness. For purely  
796 visually induced motion sickness (VIMS), increases in functional connectivity were also  
797 found between the right MT/V5 and anterior insula. Decreased functional connectivity was  
798 also found between the left and right V1[34]. The left MT/V5 in particular is an area  
799 important for processing of "what" but not "where", in priming for motion direction but not  
800 spatial position[39]. Nonetheless, cortical areas that control movement and visual processing  
801 are clearly involved in cybersickness.

802 Interestingly, cortical areas for visually induced cybersickness also overlap with areas  
803 involved in vestibular processing: Cz and FC6 – premotor and supplementary motor  
804 (movement processing, planning and inhibition) and P4 – medial superior temporal (motion  
805 detection). In this study, it can be observed that the size of the nodal cluster and strength of  
806 connectivity shift to right hemispheric dominance during CS, a preference also observed in  
807 vestibular processing. Overall, there appears to be an alteration of activity and connection in  
808 areas related to motor control and planning, as well as visual processing. These areas may  
809 become targets of intervention for future studies. [40]

810

811 F7 was highlighted as an area of interest after its correlation as an input to produce high  
812 accuracies in part 2 of the analysis. F7 relates to Brodmann area 45, the inferior frontal gyrus  
813 (IFG) [41]. The IFG and also anterior insular (AI), which also has associations with V5/MT  
814 as described above, is part of the ventrolateral prefrontal cortex (VLPFC). The VLPFC is  
815 involved in a host of functions related to active maintenance of stimulus information,  
816 including being both a control and integrative node in the brain and an interface between  
817 sensory and motor areas[42,43]. Not only does it handle awareness of the immediate  
818 moment but also implementation of reactions to it. Furthermore, it is involved in forming  
819 immediate connections between sensory processing and action control[42]. In addition, F7  
820 integrates interoceptive, limbic emotion-motivational (from orbitofrontal and subcortical  
821 areas), and sensory input (object identity from the ventral visual pathway) [42,44-46]. In  
822 particular, visual information of behavioural significance travels from the ventral pathway to  
823 the VLPFC, and later to the dorsolateral prefrontal cortex (DLPFC) and arcuate area. From  
824 here additional information from the dorsal pathway is then integrated to form a precursor of  
825 motor command[43]. In a transcranial magnetic stimulation (TMS) study, it was found that  
826 the left VLPFC had a role in the regulation of negative emotions using positive reappraisal,  
827 which is the ability to reinterpret the meaning of an emotional event or stimulus into a more  
828 positive light. The VLPFC further produces a top-down biasing effect[47] that drives  
829 selection and retrieval dynamics in the posterior cortex[42]. There also exists underlying  
830 asymmetry in the activation of the IFG/AI. F7 refers to the left IFG, and it has been found  
831 that incongruency in a flanker task activates IFG/AI, whereas the right IFG/AI (F8 was also a  
832 top 5 feature along with F7 in the best time segment) is activated more by errors [48]. The  
833 IFG/AI is also involved in post error slowing, where performance is slowed down due to  
834 making an error[42]. The IFG/AI-anterior cingulate cortex network is also thought to be  
835 involved in incongruency detection and resolving, and the ability to inhibit inappropriate

836 responses[42]. All together, it is not too far a stretch to imagine that a brain area involved in  
837 immediate recognition, regulation, resolution and action on the incongruity and error in the  
838 environment could be one of the key role players in susceptibility to cybersickness, and this  
839 is reflected in its superior performance for prediction amongst all other features. The  
840 additional discovery of F7 in part 2 of the analysis has led to a comprehensive picture of  
841 cybersickness, in which there is now a node specific in function for integration and control in  
842 response to incongruent environmental information commonly found in VR stimuli that  
843 induce cybersickness[49] , in addition to areas mention above involved with visual  
844 processing (O2) and motor planning (Cz).

845

#### 846 *ECG*

847 This study tried to use ultra-short-term RMSSD recordings in an attempt to classify  
848 cybersickness without having to capture more than 10 seconds of ECG data. Ultra-short-term  
849 RMSSD recordings (30s and 10s) have been statistically reliable in previous studies, but this  
850 parameter alone does not yield high accuracies (Table 4 and 5). Although reductions in  
851 RMSSD have been associated with cybersickness intensity, more evidence is needed to explore  
852 the role of parasympathetic cardiac indicators in cybersickness[50]. Conversely, nausea and  
853 visually induced motion sickness have been found to be mediated by the brain with links to  
854 sympathetic cardiac responses [28,51-53,34]. Although statistical differences between HRV  
855 parameters were not found, it was found that classification algorithms for cybersickness using  
856 sympathetic HRV indexes are still viable. This finding is shared with other studies where HRV  
857 has shown promise for cybersickness classification[4]. This suggests that the differences in  
858 sympathetic parameters of HRV in cybersick people versus control are more complex and  
859 simpler types of statistical analysis may not pick up on it.

860

861 Future suggestions and limitations:

862 Given that HRV is computed using R-R intervals of an ECG wave, it may be the case that  
863 other parameters, arising also from other aspects of the ECG wave could be helpful as  
864 features, such as those used in detecting other pathologies like atrial fibrillation [54-56].

865 Further research could elucidate on this matter.

866

867 The NeuCube SNNr has some similarities to a liquid state machine (LSM)[57]. In a LSM,  
868 both reservoir computing[58] and a spiking neural network is used to learn dynamical  
869 systems. Spike inputs cause a propagation of spike activity throughout the reservoir, which  
870 are like ‘ripples’ caused by a ‘stone falling into liquid’. However, NeuCube differs in that the  
871 structure is brain-inspired with stationary spatial mapping of inputs, and in that it uses  
872 unsupervised and supervised learning[59]. This application of SNNr allowed for new  
873 knowledge generation about CS and directed feature selection, and even revealed promising  
874 targets for intervention. Still, the reservoir and output layer connections were detrimental to  
875 classification performance. It is likely that these connections served as noise to the classified  
876 feature vector. However, the information synthesized and stored within the SNNr is still  
877 meaningful and valuable. Other training parameters of the cube could be optimized such as  
878 the leak rate in membrane potential, the learning rate, refractory time for neuron firing and  
879 number of training iterations[6]. Nonetheless this points towards the need for future research  
880 on how to maximize interpretability and knowledge discovery alongside classification  
881 performance. Moreover, given that SNNr activity is primarily influenced by its initial  
882 connections, a careful consideration on how to initialize neurons within the SNNr is needed.  
883 In this study the neurons within the SNNr have no distant connections because of the limited  
884 radius set by the small world connectivity approach. However, in an actual brain, there are  
885 both distant and local connections between neurons[60]. NeuCube allows for long distance

886 connections to be created through a probability [6], but these connections are not currently  
887 biologically informed. Future research can expand on how to generate a more biologically  
888 plausible SNNr and on how to use the information generated within it to enhance model  
889 performance.

890

891 Some additional points also require consideration. This study used machine learning to  
892 extract information about the spatiotemporal processes within the cybersick brain but future  
893 studies could explore the role of the interplay between motor control, motor planning and  
894 visual processing in VR on CS. The feature interaction network analysis only showed  
895 interactions between cortical areas, but not whether they were increasing or decreasing  
896 connections. Future studies could shed light on how key cybersickness centers in the brain act  
897 to control the flow of information between cortical areas. Furthermore, the finding that  
898 different features can be found at different time segments, but still give similar accuracies,  
899 points towards the complexity of the cybersickness condition within the brain. It may  
900 therefore be of interest to look at the change in features over time, rather than the features at  
901 snapshots in time to understand cybersickness in more detail. Finally, it is not yet known if  
902 the multimodal data fusion shown in this study could be improved by other biosignals and  
903 this could be valuable research to conduct moving forwards.

904

905 Conclusion:

906 The paper proposes and demonstrates that a brain-inspired spiking neural network (SNN)  
907 model can be created and used for on-the-spot prediction of cybersickness at resting state  
908 baseline and near-instant detection of cybersickness during its onset. Using this SNN model  
909 means that instead of storing raw data, each sample can be stored as a feature vector  
910 representing brain activity, which means less memory storage and processing requirements.

911 The model can be dynamically updated on new data, modifying both the weighted template  
912 neural map and the feature vectors to produce new insights. HRV alone or data fusion with  
913 EEG are useful biosignals for the prediction and detection. Motor processing areas under Cz  
914 and visual processing areas at O2 are key sites containing biomarkers as a precursor and  
915 detector of cybersickness and could be useful target areas for clinical intervention.

916

917 **List of abbreviations**

918 VR – Virtual reality

919 CS – Cybersickness

920 ECG – Electrocardiogram

921 EEG – Electroencephalogram

922 SNN – Spiking neural network

923 HRV – Heart rate variability

924 KNN – K-nearest neighbor

925 LL – Left leg

926 RL – Right leg

927 LA – Left arm

928 RA – Right arm

929 MSSQ – Motion sickness susceptibility questionnaire

930 SSQ – Simulator sickness questionnaire

931 PNS – Parasympathetic nervous system index

932 SNS – Sympathetic nervous system index

933 SI – Stress index

934 SDNN – Standard deviation of N-N intervals

935 RMSSD – Root mean squared of successive differences

936 STDP – Spike timing dependent plasticity

- 937 SDSP – Spike driven synaptic plasticity
- 938 deSNN – Dynamic evolving spiking neural network
- 939 SF – Step forward
- 940 LSM – Liquid State Machine
- 941 SNNr – Spiking neural network reservoir
- 942 FIN – Feature interaction network
- 943 RO – Rank-order learning rule
- 944 LOOCV – Leave-one-out cross validation
- 945 SNR – Signal to noise ratio
- 946 LDA – Linear discriminant analysis
- 947 LightGBM – Light gradient boosting machine
- 948 GBDT – Gradient boosting decision tree
- 949 Goss – Gradient-based one-side sampling
- 950 Dart – dropouts meet multiple additive regression trees
- 951 VIMS – Visually induced motion sickness
- 952 VLPFC – Ventrolateral prefrontal cortex
- 953 DLPFC – Dorsolateral prefrontal cortex

954

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956 available from the corresponding author on reasonable request.

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966

967

968

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