NeuroImage: Clinical 39 (2023) 103469

Contents lists available at ScienceDirect

NeuroImage: Clinical

journal homepage: www.elsevier.com/locate/ynicl

Vestibular loss disrupts visual reactivity in the alpha EEG rhythm

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ARTICLE INFO

Keywords:
Vestibular
Alpha rhythm
Reactivity
Vestibulopathy
Visual Dependence

ABSTRACT

The alpha rhythm is a dominant electroencephalographic oscillation relevant to sensory-motor and cognitive function. Alpha oscillations are reactive, being for example enhanced by eye closure, and suppressed following eye opening. The determinants of inter-individual variability in reactivity in the alpha rhythm (e.g. changes with amplitude following eye closure) are not fully understood despite the physiological and clinical applicability of this phenomenon, as indicated by the fact that ageing and neurodegeneration reduce reactivity. Strong interactions between visual and vestibular systems raise the theoretical possibility that the vestibular system plays a role in alpha reactivity. To test this hypothesis, we applied electroencephalography in sitting and standing postures in 15 participants with reduced vestibular function (bilateral vestibulopathy, median age = 70 years, interquartile range = 51–77 years) and 15 age-matched controls. We found participants with reduced vestibular function showed less enhancement of alpha electroencephalography power on eye closure in frontoparietal areas, compared to controls. In participants with reduced vestibular function, video head impulse test gain – as a measure of residual vestibulo-ocular reflex function – correlated with reactivity in alpha power across most of the head. Greater reliance on visual input for spatial orientation ('visual dependence', measured with the rod-and-disc test) correlated with less alpha enhancement on eye closure only in participants with reduced vestibular function, and this was partially moderated by video head impulse test gain. Our results demonstrate for the first time that vestibular function influences alpha reactivity. The results are partly explained by the lack of ascending peripheral vestibular input but also by central reorganisation of processing relevant to visuo-vestibular judgements.

1. Introduction

Electroencephalography (EEG) provides useful insight into brain function (Bazanova and Vernon, 2014). Posterior-predominant 8–13 Hz *alpha* frequency oscillations – the alpha rhythm – were first described by Hans Berger and noted to be 'reactive' (i.e. stimulus-responsive) (Berger, 1929; La Vaque, 1999). Alpha oscillations are generally enhanced by eye closure: alpha oscillations are also suppressed following eye opening, during non-visual sensory stimuli, and with mental effort (Pfurtscheller and Lopes da Silva, 1999). This reactivity is consistent with a general role for alpha oscillations in cortical inhibitory processes (Cook et al., 1998; Goldman et al., 2002; Sauseng et al., 2009), and in attentional processing (Foxe and Snyder, 2011). Reactivity in alpha oscillations to vision, defined as the change in amplitude with vision (eyes closed vs. open) – the so-called *Berger effect* (Barry et al., 2007) – is commonly studied as a neurophysiologic marker of brain health. Less reactivity in alpha oscillations is seen in older compared to younger healthy adults (Barry and De Blasio, 2017; Duffy et al., 1984). Furthermore, less reactivity in alpha oscillations correlates with worse cognition in early and established neurodegenerative disease (Babiloni et al., 2010; Chae et al., 2020; Partanen et al., 1997, 1996; Schumacher et al., 2020; van der Hiele et al., 2008). Besides ageing and neurodegeneration, other determinants of alpha reactivity remain unclear. Known functional cortical interactions between visual and vestibular systems (Angelaki

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https://doi.org/10.1016/j.nicl.2023.103469
Received 20 March 2023; Received in revised form 11 June 2023; Accepted 5 July 2023
Available online 11 July 2023

and Cullen, 2008; Dieterich and Brandt, 2000; Grüsser and Grüsser-Cornehls, 1972), mean that vestibular function may influence the alpha rhythm and contribute to its broader cognitive associations.

Functional interactions between visual and vestibular cortices are important in human health and in vestibular disease (Dieterich and Brandt, 2000). In health, visual and vestibular cortices show reciprocal inhibition, such that visual motion stimuli deactivate the parieto-insular vestibular cortex, and vestibular stimuli deactivate visual cortices (Brandt et al., 1998; Kleinschmidt et al., 2002). Cortical visuo-vestibular interactions likely facilitate the reconciliation of conflicting sensory signals to support coherent perceptual judgements, such as verticality (Dieterich and Brandt, 2000). Given the importance of vestibular signalling to visual cortical responses, vestibular function may *in principle* be an important determinant of the reactivity in alpha oscillations to vision. In this work, we study the effect of vestibular loss (participants with bilateral vestibulopathy) on alpha reactivity to vision.

Acquired vestibular loss provides insight into the function of the balance and spatial orientation systems. Humans with bilateral vestibulopathy become over-reliant on vision for perceptual judgements and postural control (Guerraz et al., 2001), a trait often referred to as 'visual dependence' (Bronstein et al., 1996). Bilateral vestibulopathy patients show less inhibition of visual cortical metabolic activity following vestibular stimulation (Bense et al., 2004), and enhanced visual cortex responses to visual motion (Dieterich et al., 2007), in keeping with weaker visuo-vestibular inhibitory interactions. Weakening of visuo-vestibular interactions in bilateral vestibulopathy likely results from diminished whole brain functional connectivity in parieto-insular vestibular areas (Göttlich et al., 2014). Measuring visual dependence and linking this to alpha reactivity may thus provide insight into the neurophysiologic associations of visuo-vestibular judgements.

We propose that vestibular function facilitates deactivation of cortical areas involved in visual and visuospatial attentional processing. This predicts that the vestibular system supports reactivity in alpha oscillations to vision (hereon termed alpha reactivity), specifically, alpha EEG power enhancement following eye closure. We thus apply EEG in controls and participants with bilaterally reduced vestibular function to test the following hypotheses: (i) long-standing loss of vestibular input is associated with less alpha reactivity; and (ii) levels of vestibular function and visual dependence correlate with alpha reactivity.

2. Materials and methods

2.1. Participants

Fifteen patients (9 female, median age = 70 years, interquartile range = 51——77 years) with chronic (>6 months) reductions in vestibular function (bilateral vestibulopathy) were recruited from a vestibular neurology clinic. The diagnosis of bilateral vestibulopathy included impairment on either caloric (sum of bi-thermal maximum peak slow phase velocity on each side $< 6^{\circ}$/second), video head impulse test gain (bilateral horizontal angular gain < 0.6) or rotational chair testing (reduced horizontal angular vestibulo-ocular reflex gain < 0.1) (Strupp et al., 2017). Aside the clinical signs of bilateral vestibulopathy, there were no other abnormal neurological signs. All had normal audiometry for age. Exclusion criteria were peripheral neuropathy, significant uncorrected visual impairment, or neurological disease. Video head impulse test gain (mean of left and right horizontal canal gains) quantified vestibulo-ocular reflex function (which we refer to as *VOR gain*).

Fifteen age- and sex-matched control participants were recruited (9 female, median age = 70 years, interquartile range = 50–74 years). They had no vestibular or balance symptoms, no pre-existing vestibular disorder, no significant uncorrected visual impairment, and a normal neuro-otological examination. Our study conformed to the standards set by the Declaration of Helsinki. Written informed consent was obtained as approved by the local ethics research committee (reference 17/NE/0133).

2.2. Visual dependence, vestibulo-ocular reflex gain and questionnaires

Visual dependence was measured by a rod-and-disc task on a laptop computer (Cousins et al., 2014) (Fig. 1a). Participants looked at a computer screen through a 30 cm deep viewing cone. The diameter of the cone at eye level was 15 cm and the cone subtended a viewing angle of 39°. A 6 cm white rod was presented on a black background. Around a central 6 cm circle was a field of 220 randomly distributed off-white dots each subtending 1.5° of visual field. The rod was rotated to an angle randomised between −70°, −40°, −20°, 20°, 40° and 70° from vertical. Participants then re-aligned the rod to their subjective visual vertical using keyboard controls. The procedure was then repeated during $30^{\circ}s^{-1}$ clockwise, then $30^{\circ}s^{-1}$ counter-clockwise rotation of the background. Each condition (static, clockwise, counter-clockwise) was repeated 6 times, then results were averaged. Visual dependence was defined as the absolute bias of verticality estimates in the motion conditions compared to the non-motion (static background) condition ($VDEP = \frac{(|R_C|+|R_{CC}|)}{2} - S$, where S = subjective visual vertical in degrees in static background, VDEP = visual dependence in degrees, R_C = subjective visual vertical for clockwise rotation of background in degrees, and R_{CC} = subjective visual vertical for counter-clockwise rotation of background in degrees, Fig. 1a).

Participants completed questionnaires to quantify dizziness and vertigo symptom load. The dizziness handicap inventory (DHI) score quantified the subjective impact of reduced vestibular function on a scale from 0 to 100 (Jacobson and Newman, 1990). The short form vertigo symptom scale (VSS) score defined the burden vestibular symptoms on a scale from 0 to 60 (Yardley et al., 1992).

2.3. EEG acquisition and pre-processing

EEG data were acquired in 32 channels at 1250 Hz (Waveguard™ cap, ANT® Neuro, Enschede, The Netherlands). A 33rd electrode located 10% anterior to Fz (position AFz) was used as the reference. Data were collected for two postural conditions: *sitting* in quiet rest in a comfortable high-backed chair, and *standing* without shoes, with feet placed side by side at a 20 cm distance between the medial malleoli. During both sitting and standing, data were collected under two visual conditions: *eyes open* fixating on a central target at eye level 2 m in front, and *eyes closed*. Two 2-minute recordings were completed for each condition pair (8 trials total). The condition pair order was randomised. Recordings were separated by short breaks for participant comfort.

EEG data were pre-processed using methods described in previous work (Ibitoye et al., 2021), within the EEGLAB toolbox (Delorme and Makeig, 2004). Raw EEG data were detrended and pre-processed using the PREP pipeline (Bigdely-Shamlo et al., 2015). PREP incorporated a 50 Hz line noise removal using CleanLine (an adaptive filter using frequency-domain multi-taper regression to remove sinusoidal artefacts) and a 1 Hz zero-phase high pass filter (Hamming windowed sinc finite impulse response). A 100 Hz zero-phase low-pass 4th order Butterworth filter was applied to eliminate high frequency noise. Further noise reduction was achieved by epoch-based rejection techniques. Data was divided into 1-second epochs. Noisy epochs were rejected using amplitude (*pop_rejspec* function using −100 mV to 30 mV thresholds at 25 Hz to 45 Hz), and joint probability criteria (*pop_jointprob* function using local and global thresholds of 5 standard deviations). Similar proportions of epochs were rejected in control and bilateral vestibulopathy data (controls mean 15.1 % [1.88 % standard deviation], bilateral vestibulopathy 14.8 % [2.81 % standard deviation], $F(1\,78) = 0.777$, $p = .38$).

Independent component analysis (ICA) was applied to denoise the data. Individual participant data was first concatenated across conditions; in so doing this ensures ICA is applied across the data from all conditions (e.g. eyes open, eyes closed) for each participant. This approach minimises bias to condition-specific noise, ensuring the same

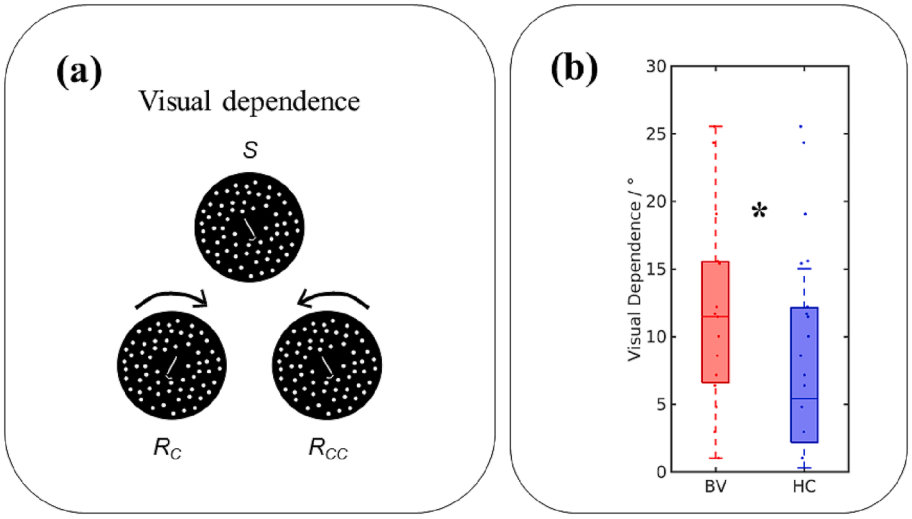

Fig. 1. Visual dependence. (A) Visual dependence as measured by the rod and disk task is illustrated here. A central white rod is shown on a black background overlain with static white dots (*S*). Participants align the central white rod to their perceived vertical; the angle between their judgement and the true vertical is their subjective visual vertical. The task is repeated during clockwise (R_C) and counter-clockwise (R_{CC}) rotation of the visual background. A participant's visual dependence is the difference between the subjective visual vertical during background motion conditions and the static condition. (B) Box plots of visual dependence data for participants with bilateral vestibulopathy (BV) and healthy controls (HC). * = p <.05 for two tailed *t*-test of visual dependence in bilateral vestibulopathy and healthy controls.

components are identified across data. Resultant components were automatically classified using ICLabel – a pre-trained classifier (Pion-Tonachini et al., 2019). Non-brain components were rejected on the basis of thresholds as defined from the ICLabel training dataset (Muscle ≥ 0.18, Eye ≥ 0.13, Heart ≥ 0.33, Line Noise ≥ 0.04, Channel Noise ≥ 0.10, Other ≥ 0.12) (Pion-Tonachini et al., 2019). More components were removed from bilateral vestibulopathy data compared to control data (control mean 9.27 [2.83 standard deviation], bilateral vestibulopathy mean 11.9 [3.04 standard deviation], $F(1\,78) = 37.1$, $p < .001$).

The alpha frequency band was defined as 8–13 Hz. For each channel, mean power spectrum density (PSD, microvolts^2/Hz, hereon referred to as power) across each recording was quantified using Welch's estimator as implemented in EEGLAB (Delorme and Makeig, 2004). Individual peak alpha EEG frequencies were also determined for each recording at channel Oz by undertaking a Fourier transform of EEG data then determining the maximum amplitude peak in the frequency range of 7–14 Hz (which is fully inclusive of the alpha EEG frequency range of 8 – 13 Hz). We hereon refer to this measure as the *peak alpha frequency*.

2.4. Sway data acquisition and pre-processing

Sway data were recorded during standing trials. An electromagnetic tracking device (Fastrak; Polhemus, USA) was firmly taped over the occiput to record anteroposterior linear head displacement. The signal was digitised using a custom-built digital-to-analogue converter. This signal was then connected to an additional channel on the ANT® Neuro amplifier. The data were demeaned, detrended, then a 0.1 Hz high-pass 2nd order Butterworth and a 10 Hz zero-phase low-pass 2nd order Butterworth filters were applied to eliminate low and high frequency noise. Total sway – the length of the path traced by anteroposterior sway was determined ($SP = \sum_i^{n-1} s_i = \sum_i^{n-1}(x_{i+1} - x_i)$, where SP = sway path, x = anteroposterior displacement, n = number of data samples, as adapted from Hufschmidt et al. 1980 (Hufschmidt et al., 1980)). One (of two) recordings of sway data were missing for a participant with reduced vestibular function in the standing with eyes open condition, meaning a total sway value was not available for that record.

3. Analysis

3.1. Regression

Alpha oscillations occur across the whole head (Barry and De Blasio, 2017) so we chose to adopt a whole head analysis (in contrast to reporting on arbitrary channels or regions). An advantage of this approach is the provision of statistically robust, data-driven results unbiased by a priori definitions of channels or regions. A potential disadvantage is that spatially restricted effects may be missed. To support a data-driven approach, we applied regression to evaluate statistics at each channel with subsequent correction for multiple comparisons across all channels.

Hypotheses were thus tested using linear mixed-effects regression using a custom MATLABR script. Linear regression was applied with models built and run for each channel. The general approach is outlined below.

Alpha EEG power was the dependent variable. As both age (Duffy et al., 1984) and sex (Cave and Barry, 2021) are known to influence alpha power, across all linear regressions, *age* and *sex* were covariates of no interest (please note the participant groups were also matched for age and sex). A random (participant) intercept was included to account for inter-individual variability in average EEG power. Alpha reactivity was defined by the increase in alpha power with eyes closed compared to eyes open (Barry and De Blasio, 2017; Partanen et al., 1996). By this definition, greater alpha reactivity is equivalent to more suppression of alpha oscillations on eye opening. An independent regression variable representing *visual condition* (eyes open, or eyes closed) captured the effect of eye closure and thus alpha reactivity (Wilkinson notation (Wilkinson and Rogers, 1973): *Alpha EEG Power* ~ *Visual Condition* + *Age* + *Sex* + *(1|Participant)*). Our regression approach to measuring alpha reactivity is similar to recent work in this area (Barry and De Blasio, 2017), with regression being advantageous over simple ratios of power in eyes closed and eyes open conditions in the ability to appropriately take into account both within-participant and between-participant variability.

Specific regressions were defined to address questions of interest. To determine the effect of bilateral vestibulopathy on alpha reactivity, linear regressions were undertaken with *visual condition* and *participant group* (bilateral vestibulopathy or control) as independent variables. The interaction of *participant group* and *visual condition* as a predictor of *alpha EEG power* captured the effect of bilateral vestibulopathy on alpha reactivity (*Alpha EEG Power* ~ *Visual Condition*Participant Group* + *Age* + *Sex* + *(1|Participant)*). Correlation between variables such as visual dependence or VOR gain, and alpha reactivity was determined by separate linear regression models. There, the interaction of the variable with *visual condition* in the prediction of *alpha EEG power* was of interest (e.g. for visual dependence, the following model was used: *Alpha EEG Power* ~ *Visual Condition*Visual Dependence* + *Age* + *Sex* + *(1|Participant)*). To test for the effect of vestibulo-ocular reflex function on relationships, the interaction of *VOR gain* and *visual condition* was added to regressions as a covariate of no interest. In standing data, *total sway* was

an additional covariate of no interest added to minimise any potential contribution from movement-associated EEG artifacts. Sometimes (as discussed in the results), where no effect of postural condition (sitting or standing) was seen, additional exploratory (post-hoc) analyses were run on "pooled" data; in this scenario the postural condition label was removed, and the sitting and standing data combined before the regressions were re-run.

3.2. Threshold-free cluster enhancement

Statistics were evaluated for significance across the whole head adjusting for multiple comparisons (defined as all channels excluding M1 and M2 [mastoids]). We applied threshold-free cluster enhancement (TFCE (Mensen and Khatami, 2013; Smith and Nichols, 2009)), a cluster-based permutation method. TFCE optimally integrates the spatial evidence for clustering across thresholds, increasing sensitivity to spatially contiguous effects in comparison to overly conservative approaches such as Bonferroni correction. TFCE nonetheless provides strict control for family-wise error and thus accounts for multiple comparisons (Smith and Nichols, 2009). Neighbouring channels were defined using methodology described by Mensen & Khatami (Mensen and Khatami, 2013). Though initially developed for the analysis of magnetic resonance imaging data (Smith and Nichols, 2009), TFCE has since been developed for EEG analyses in channel/source space (Mensen and Khatami, 2013) with provision of relevant code through an open repository (https://github.com/Mensen/ept_TFCE-matlab). Although 'threshold-free', TFCE requires two parameters to be defined which weigh the relative influences of spatial extent (E) and the degree of 'activation' (H) in the integration of evidence across thresholds. Empirical and random field theory justifications have been provided for values of E = 2/3 and H = 2 (Smith and Nichols, 2009), and these values have also been shown to be appropriate for EEG data (Mensen and Khatami, 2013). TFCE results are reported as a mean beta co-efficient across significant channels identified, and 95% confidence interval (+/- 1.96 standard errors, based on parametric methods), with a range of p-values (based on the TFCE algorithm) across identified significant channels.

To apply TFCE to linear regression models, the F-statistic for the effect of interest was first determined; the data were then permuted in accord with the principle of exchangeability (Winkler et al., 2014), using Manly's method (Manly, 2018). In contrast to parametric methods, permutation tests make few assumptions about the distribution of data and enable strict control of the false alarm rate (Nichols and Holmes, 2002). Permutation tests for average (e.g. mean alpha EEG power) responses were done by applying the principle of independent and symmetric errors - thus sign-flipping the data (Winkler et al., 2014). Outputs were generated across 10,000 permutations for each regression model. P-values for significance across the whole head were calculated by comparing the initial (unpermuted) regression model's F statistic against the distribution of maximum statistics generated by permutation in each channel. A two-tailed p-value of<0.05 was defined as statistically significant.

3.3. Aperiodic spectrum and periodic alpha power

Further analyses were done to explore background and frequency-specific contributions to alpha reactivity results. It has been shown that EEG power spectra can be modelled as a combination of a background ("aperiodic") baseline, with superimposed narrow-band (periodic) peaks which may have distinct neurophysiologic bases (Donoghue et al., 2020). The Fitting Oscillations and One Over F (FOOOF) algorithm was therefore applied (https://github.com/fooof-tools/fooof, (Donoghue et al., 2020) within a custom MATLAB script). This open-source algorithm models background EEG power spectra density (*PSD*) as a combination of an aperiodic component (*L*), and a sum of Gaussian functions which fit narrow band peaks in the EEG spectra – see Supplementary Fig. 1. The model is defined as: $PSD = L + \sum_{n=0}^{N} G_n$ where $L = b - \log(k + F^{\chi})$ (Donoghue et al., 2020). *L* is a Lorentzian function with offset *b* and exponent χ.

The FOOOF algorithm was run in its *fixed* mode such that $k = 0$. Power spectra and frequencies were generated by the Welch estimator (*pwelch* function in MATLAB). FOOOF settings were: frequency band range = 1 to 40 Hz, peak bandwidth limit = 1 to 12 Hz, maximum number of peaks = infinity, minimum peak height = 0, and peak threshold = 2 standard deviations. The largest matching peak in the frequency range of 7 to 14 Hz defined the periodic component of the alpha rhythm. For reasons of parsimony with our earlier defined – canonical – mean alpha power, periodic power was defined as the peak of the largest matching peak divided by its bandwidth. Should no peak be identified in this range, the FOOOF algorithm was re-run with a narrower frequency band range of 8 to 13 Hz; if no peak was again found, then periodic alpha EEG power was set to zero. An alpha peak was more likely to be found on the first pass in control than bilateral vestibulopathy data (3612/3840 records in controls, 3505/3840 channel records in bilateral vestibulopathy, p <.001, Odds Ratio 0.660 by Fisher Exact Test); groups did not differ in the number of recordings wherein no peak was found (64/3840 channel records in controls, 71/3840 channel records in bilateral vestibulopathy, p = 0.60, Odds Ratio 1.11 by Fisher Exact Test). Peak periodic alpha EEG power (i.e. alpha power not including the aperiodic baseline) – hereon referred to as *periodic alpha EEG power*, the *aperiodic offset* (*b*) and the *aperiodic exponent* (χ) were the measures of interest (see Supplementary Fig. 1).

4. Results

4.1. Participant characteristics

Bilateral vestibulopathy was idiopathic (Kim et al., 2011; Rinne et al., 1998) in most participants with reduced vestibular function (13 of 15, Table 1). Mean VOR gain - a measure of vestibulo-ocular reflex function – was 0.58 (standard deviation 0.24, Table 1, normal values = 1 +/- 0.2). Participants with bilateral vestibulopathy reported varying degrees of disability (mean dizziness handicap inventory score = 12, standard deviation = 7; mean vertigo symptom scale score = 11, standard deviation = 10, Table 1). The burden of dizziness handicap correlated with the frequency and burden of dizziness/unsteadiness (DHI score correlated with VSS score, Pearson r = 0.679, p =.005). VOR gain, however, did not correlate with dizziness handicap or the frequency and burden of vestibular symptoms (VOR gain and DHI score, r = -0.215, p =.44; VOR gain and VSS score, r = 0.513, p =.051). Visual dependence (VDEP) did not correlate with the frequency and burden of dizziness/unsteadiness, or VOR gain (VDEP and DHI score, r = -0.108, p =.70; VDEP and VSS score, r = 0.271, p =.33; VDEP and VOR gain, r = -0.451, p =.09).

In the standing conditions, controls swayed more with eyes closed than open (eyes open: mean = 57.1 cm [over 2-minute trial], standard deviation = 14.8 cm; eyes closed: mean = 83.3 cm, standard deviation = 19.3 cm; F(1,58) = 124, p <.001). Participants with bilateral vestibulopathy also showed an increase in sway with eyes closed (eyes open: mean = 69.6 cm, standard deviation = 38.4 cm; eyes closed: mean = 103 cm, standard deviation = 41.6 cm; F(1,57) = 9.28, p =.004). Sway did not differ between participants with bilateral vestibulopathy and controls with eyes open (F(1,57) = 1.37, p =.24), or with eyes closed (F(1,58) = 2.93, p =.09). Participant group (bilateral vestibulopathy or healthy control) did not significantly moderate the effect of eye closure on sway (F(1,115) = 0.427, p = 0.51), likely due to the wide stance they were requested to adopt (feet apart by 20 cm).

Table 1
Bilateral vestibulopathy participant characteristics. VOR gain = mean of horizontal canal video head impulse test gain. SVV = subjective visual vertical; DHI = dizziness handicap inventory score (range 0 to 100); VSS = vertigo symptom scale score (range 0 to 60). For reference the mean +/- standard deviation for the control group were: age 63 years +/- 20 years, SVV 0.5° +/- 1.2°, visual dependence 6.9° +/- 5.3°, DHI 0 +/- 0 and VSS 1.3 +/- 1.7. * = note that Meniere's disease often spares high-frequency vestibular function (as captured by the video head impulse test); ǂ = vestibular loss was confirmed on caloric testing.

ID	Age	Sex	Aetiology	VOR gain	SVV / °	Visual Dependence / °	DHI	VSS
1	67	F	Idiopathic	0.28	1.5	10.0	20	2
2	77	M	Idiopathic	0.59	1.0	25.5	0	2
3	54	M	Bilateral Ménière's disease	0.98*ǂ	0.5	6.4	34	3
4	70	M	Idiopathic	0.73	−2.7	7.2	28	9
5	36	F	Idiopathic	0.66	0.7	8.6	28	0
6	68	F	Idiopathic	0.79	1	11.5	58	6
7	74	M	Idiopathic	0.69	−2.9	15.6	42	14
8	48	M	Idiopathic	0.69	1.0	3.0	56	4
9	48	F	Idiopathic	0.2	6.1	11.7	48	19
10	70	F	Idiopathic	0.74	1.9	15.4	48	23
11	72	F	Gentamicin	0.05	1.1	19.1	80	44
12	17	F	Idiopathic	0.72	0.4	1.0	20	9
13	84	F	Idiopathic	0.41	−0.5	24.3	16	8
14	74	F	Idiopathic	0.39	0.0	12.2	26	10
15	71	M	Idiopathic	0.85ǂ	−0.2	4.8	28	13

4.2. Less vestibular function reduces alpha reactivity

In the sitting condition, eye closure led to an increase in alpha EEG power across the head in both controls and bilateral vestibulopathy (TFCE identified 30 significant channels; control mean β co-efficient = 0.942 μV^2Hz^{-1} with 95% confidence interval 0.0937 to 2.75 μV^2Hz^{-1}, p <.001; bilateral vestibulopathy mean β co-efficient = 0.592 μV^2Hz^{-1} with 95% confidence interval 0.204 to 2.10 μV^2Hz^{-1}, p <.001, Fig. 2b). Participants with bilateral vestibulopathy had similar alpha EEG power to controls across the head in each visual condition (TFCE identified no significant channels, p > 0.05 for eyes open, and closed, Fig. 2b). In exploratory results *without* correction for multiple comparisons (Supplementary Fig. 2), channel-level effects were found in the eyes closed condition such that controls had greater alpha EEG power than bilateral vestibulopathy participants at F3, Fz and P8 (uncorrected p <.05, Supplementary Fig. 2b).

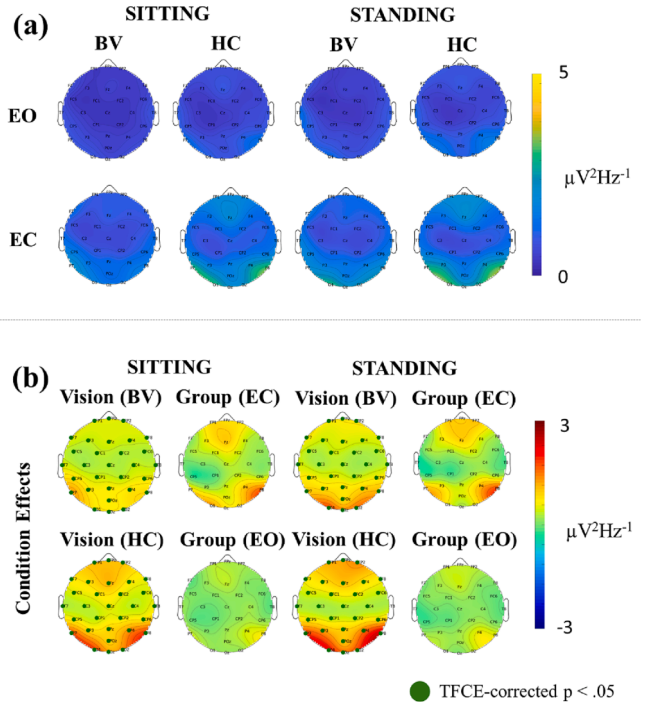

Fig. 2. Alpha EEG power in bilateral vestibulopathy and in controls in sitting and standing postures. Head plots in each of sitting and standing conditions of (a) average alpha EEG power with eyes open (EO) or closed (EC) and (b) effects of group and vision are illustrated, for participants with bilateral vestibulopathy (BV) and healthy controls (HC). The effects of vision are shown for eyes closed relative to eye open, and for group for controls relative to bilateral vestibulopathy. Significant channel effects are highlighted by a green dot (two-tailed p <.05 across all channels by threshold-free cluster enhancement). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

Participant group moderated the effect of eye closure on alpha EEG power in the sitting condition, such that participants with bilateral vestibulopathy showed less alpha EEG power enhancement on eye closure in frontoparietal channels (TFCE identified 8 significant channels: FP1, FPz, FP2, F3, F7, FC1, FC2, P8; mean β co-efficient = -0.592 μV^2Hz^{-1} with 95% confidence interval −1.81 to −0.103 μV^2Hz^{-1}, p range = 0.006 to 0.046, Fig. 4a-b); results *without* correction for multiple comparisons showed effects in other channels (including occipital) not significant in the whole head analysis (uncorrected p <.05 in F4, FC5, Cz, P4, O1, Oz and O2, see Supplementary Fig. 3b).

In the standing condition, eye closure also led to an increase in alpha EEG power across the head in both controls and bilateral vestibulopathy (TFCE identified all 30 channels as significant; control mean β co-efficient = 1.05 μV^2Hz^{-1} with 95% confidence interval 0.140 to 3.63 μV^2Hz^{-1}, p <.001; bilateral vestibulopathy mean β co-efficient = 0.731 μV^2Hz^{-1} with 95% confidence interval 0.136 to 3.07 μV^2Hz^{-1}, p <.001, Fig. 2b). Participants with bilateral vestibulopathy again had similar alpha EEG power to controls across the head in each visual condition (TFCE identified no significant channels, p > 0.05 for eyes open, and eyes closed – Fig. 2b); this was the same *without* correction for multiple comparisons (Supplementary Fig. 2b). The effect of eye closure on alpha EEG power did not depend on participant group, but a trend similar to the sitting condition was observed (TFCE identified no significant channels, p >.05, Fig. 4c-d); results *without* correction for multiple comparisons however showed channel-level interaction effects at Fp1, Fp2, F3, Fz, FC2, P7 and P8 which had not reached significance at the whole head (uncorrected p <.05, Supplementary Fig. 3d).

Postural condition (sitting or standing) did not significantly moderate the relationship between participant group, visual condition or alpha EEG power. We therefore pooled the sitting and standing data to clarify general relationships in a post-hoc analysis. In pooled data, participants with bilateral vestibulopathy showed less alpha EEG power enhancement on eye closure than controls in frontoparietal channels (TFCE identified 10 significant channels: FP1, Fpz, FP2, F3, Fz, F4, FC5, FC1, FC2, P8; mean β co-efficient = -0.523 μV^2Hz^{-1} with 95% confidence interval −1.92 to −0.03 μV^2Hz^{-1}, p range = 0.002 to 0.042, Fig. 4e); results *without* correction for multiple comparisons showed effects at two other channels not reaching significance at the whole head

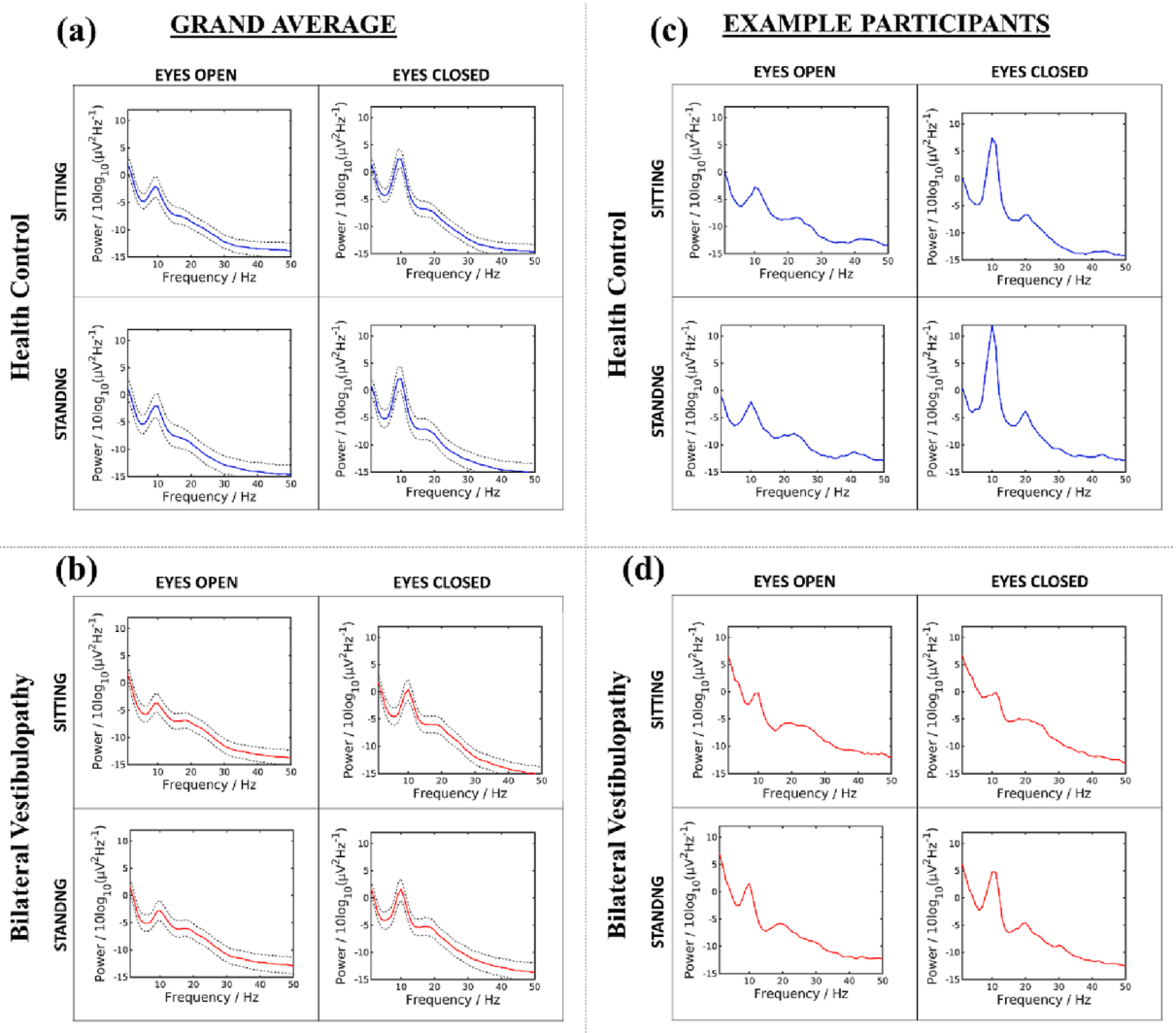

Fig. 3. EEG power spectra in controls and bilateral vestibulopathy. Grand average power spectra across all channels are illustrated for healthy controls (a), and bilateral vestibulopathy (b) in both sitting and standing conditions. Spectra from an example control participant (c) and bilateral vestibulopathy participant (d) are also shown. Note the clear enhancement of alpha EEG power spectral density on eye closure in the control participant but not the bilateral vestibulopathy participant, particularly evident in the sitting data (c-d). Controls plot lines are in blue and bilateral vestibulopathy plot lines in red. Grand average plots (a-b) include dashed lines which represent the upper and lower bounds of the 95% confidence interval of the mean (mean +/- 1.96 standard errors). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

(uncorrected p <.05 in P7 and P4, see Supplementary Fig. 3e).

Average alpha power across the head for each group and condition is illustrated in Fig. 2a. Grand average EEG power spectra for controls and bilateral vestibulopathy patients in each condition are shown in Fig. 3a-b. Example power spectra from a typical control, and bilateral vestibulopathy participant are shown in Fig. 3c-d.

4.3. Vestibular loss affects aperiodic components of the EEG power spectrum

EEG power spectra can be modelled as a combination of narrow-band (periodic peaks) and an "aperiodic" background; periodic and aperiodic components may have distinct neurophysiologic bases (Donoghue et al., 2020). We thus explored the data further after decomposition into periodic and aperiodic elements (offset and exponent; Supplementary Fig. 1 illustrates these measures). In controls, in the sitting condition, eye closure was associated with a significant increase in periodic alpha EEG power (TFCE identified 30 significant channels; mean β co-efficient = 0.134 μV^2Hz^{-1} with 95% confidence interval 0.0230 to 0.0232 μV^2Hz^{-1}, p <.001; Supplementary Fig. 4b); there was no significant effect of eye closure on the aperiodic offset or aperiodic exponent (TFCE p >.05; Supplementary Fig. 5b and 6b respectively). In the standing condition, control periodic alpha EEG power also increased on eye closure (TFCE identified 30 significant channels; mean β co-efficient = 0.146 with 95% confidence interval 0.0177 to 0.264 μV^2Hz^{-1}, p <.001; Supplementary Fig. 4b); there was also a decrease in the aperiodic exponent over posterior channels on eye closure (TFCE identified 5 significant channels: Pz, POz, O1, Oz, O2; mean β co-efficient −0.126 μV^2Hz^{-1} with confidence interval −0.222 to −0.0319 μV^2Hz^{-1}, p-range = 0.023 to 0.044, Supplementary Fig. 6b). Alpha reactivity in controls was thus generally linked to changes in periodic alpha EEG power.

In bilateral vestibulopathy in the sitting condition, eye closure associated with a significant increase in periodic alpha EEG power across the head (TFCE identified 30 significant channels; mean β co-efficient = 0.135 μV^2Hz^{-1} with confidence interval 0.0163 to 0.231 μV^2Hz^{-1}, p-range = 0.002 to 0.007 Supplementary Fig. 4b). The aperiodic offset also increased with eye closure across most channels (TFCE identified 22 significant channels: F7, F3, Fz, F4, F8, FC5, FC1, FC2, FC6, T7, C3, Cz, C4, T8, CP5, CP1, CP2, CP6, P7, P3, P4, P8; mean β co-efficient = 0.139 $\log_{10}(\mu V^2Hz^{-1})$ with confidence interval 0.0148 to

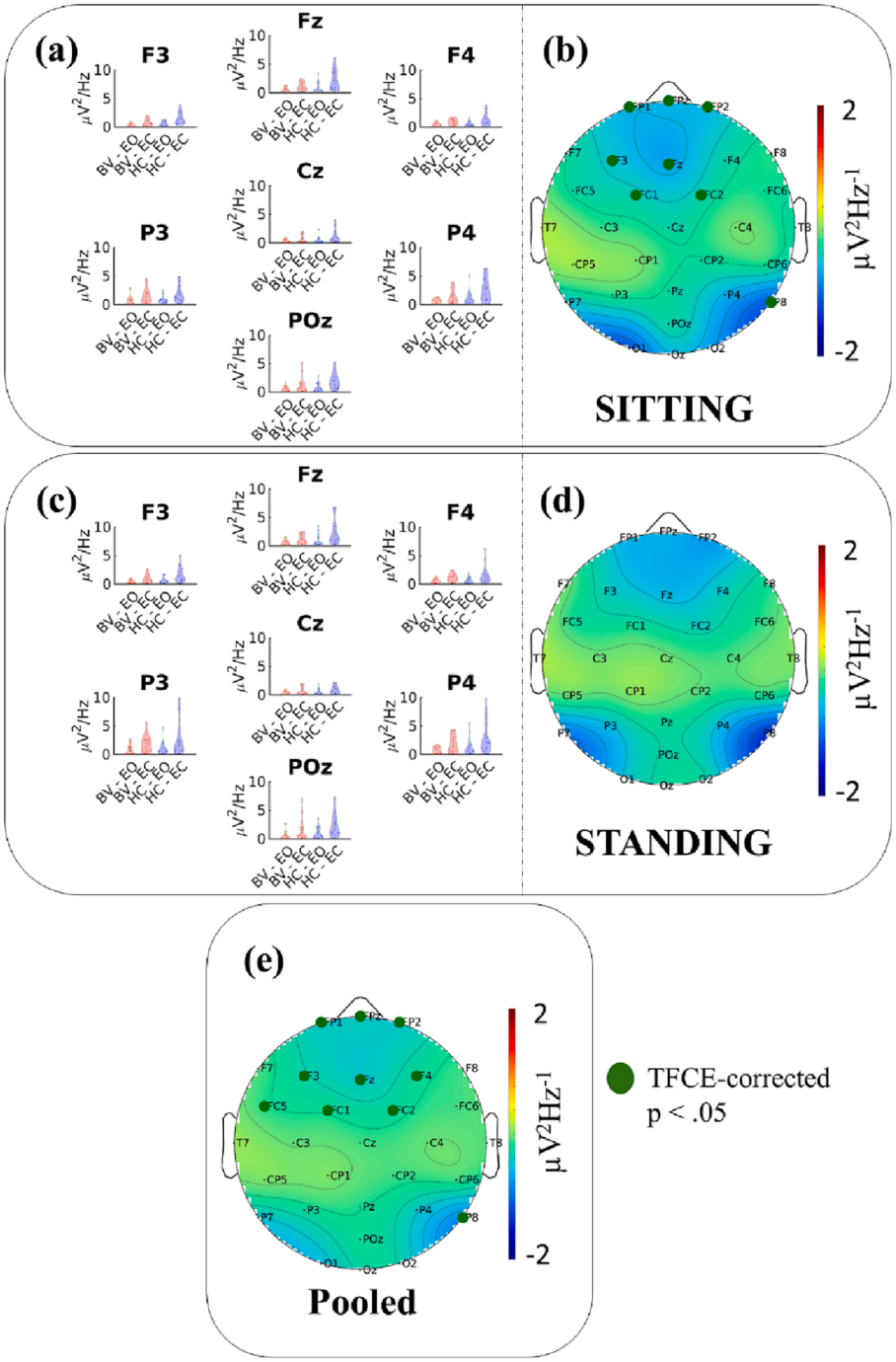

Fig. 4. Interaction of participant group and visual condition in predicting alpha EEG power. Violin plots (a,c) illustrate alpha EEG power data in sitting (a) and standing (c) conditions at a range of channels (F3, Fz, F4, P3, Cz, P4, POz) in bilateral vestibulopathy participants (BV) and healthy controls (HC). Head plots show the interaction effect between participant *group* and *visual condition* in the prediction of alpha EEG power for sitting (b) and standing (d) conditions. The *group* and *visual condition* interaction effect for pooled data (sitting and standing) is shown in (e). Significant channel effects are highlighted by a green dot (plots b, d and e; two-tailed p <.05 across all channels by threshold-free cluster enhancement). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

0.401 $\log_{10}(\mu V^2Hz^{-1})$, p range = 0.002 to 0.009, Supplementary Fig. 5b), as did the aperiodic exponent (TFCE identified 19 significant channels: F7, F3, Fz, F4, F8, FC5, FC1, FC2, FC6, T7, C3, C4, T8, CP5, CP2, CP6, P7, P3, P8; mean β co-efficient = 0.136 with confidence interval 0.00646 to 0.312, p range 0.002 to 0.049; Supplementary Fig. 6b).

In bilateral vestibulopathy, in the standing condition, periodic alpha EEG power also increased on eye closure (TFCE identified 30 significant channels; mean β co-efficient = 0.159 μV^2Hz^{-1} with 95% confidence interval 0.0514 to 0.302 μV^2Hz^{-1}, p range < 0.002, Supplementary Fig. 4b). The aperiodic offset again increased with eye closure across most channels (TFCE identified 22 significant channels: FP1, Fpz, FP2, F7, F3, Fz, F4, F8, FC5, FC1, FC2, FC6, C3, Cz, T8, CP1, CP6, P3, Pz, P4, P8, O1; mean β co-efficient = 0.205 $\log_{10}(\mu V^2Hz^{-1})$ with 95% confidence interval 0.00937 to 0.536 $\log_{10}(\mu V^2Hz^{-1})$, p range 0.002 to 0.38, Supplementary Fig. 5b); the aperiodic exponent also showed an increase with eye closure predominantly in frontal channels on (TFCE identified 9 significant channels: F7, F3, Fz, F4, F8, FC5, FC6, T7, T8; mean β co-efficient = 0.121 with 95% confidence interval 0.0183 to 0.223, p range 0.009 to 0.046, Supplementary Fig. 6b). Alpha reactivity in bilateral vestibulopathy was thus linked to changes in both the periodic alpha and aperiodic components of the power spectrum.

We looked at periodic alpha and aperiodic EEG spectral components of the interaction previously found between participant group and alpha reactivity in sitting data (Fig. 4a-b). We found significant interaction between participant group and visual condition in the prediction of the aperiodic offset (TFCE identified 14 significant channels: F7, Fz, F4, FC5, T7, C4, CP5, CP2, CP6, P7, Pz, P8, POz, O1; mean β co-efficient = -0.180 $\log_{10}(\mu V^2Hz^{-1})$ with 95% confidence interval −0.407 to 0.0191

$\log_{10}(\mu V^2Hz^{-1})$, p range = 0.017 to 0.040; Supplementary Fig. 7b). There was, however, no significant interaction for the aperiodic exponent or periodic alpha EEG power (Supplementary Fig. 7a and 7c respectively). The effect of bilateral vestibulopathy on alpha EEG enhancement was thus seen in the aperiodic part of the spectrum.

4.4. Better vestibulo-ocular reflex function correlates with more alpha reactivity

We undertook further analyses to clarify the factors influencing alpha reactivity in the presence of reduced vestibular input. In the standing condition, higher VOR gain (i.e. less vestibular loss) correlated with more alpha EEG power enhancement on eye closure. TFCE identified 21 significant channels (FP1, FPz, FP2, F3, Fz, F4, FC5, FC1, FC2, C3, Cz, C4, CP1, CP2, P3, Pz, P4, POz, O1, Oz, O2; mean β co-efficient = 1.32 μV^2Hz^{-1} with 95% confidence interval 0.112 to 5.35 μV^2Hz^{-1}, p range = 0.007 to 0.031, Fig. 5c,d); no additional channels showed effects in results *without* correction for multiple comparisons (Supplementary Fig. 8b). No significant correlation was present in the sitting condition but the relationship between VOR gain and alpha EEG power enhancement was generally positive (TFCE p >.05; Fig. 5a,b); results *without* correction for multiple comparisons, however, showed effects in frontocentral areas and occipitally other channels which were not significant at the whole head level (uncorrected p <.05 in F3, Fz, FC1, FC2, Cz, CP2 and Oz, see Supplementary Fig. 8a).

Postural condition (sitting or standing) did not significantly moderate the relationship between VOR gain and alpha EEG power enhancement on eye closure, supporting the null hypothesis of a similar

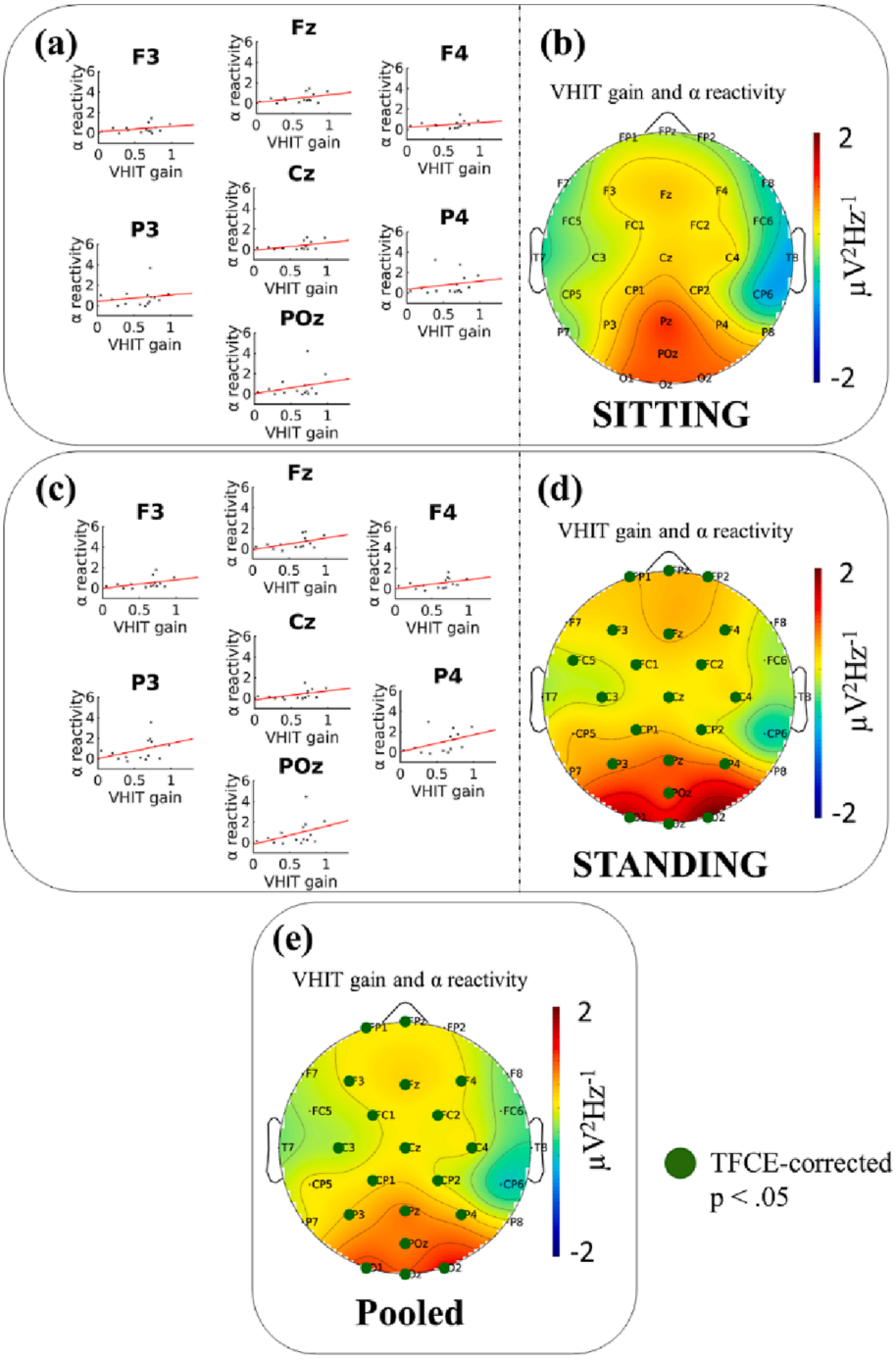

Fig. 5. Video **head impulse test gain and alpha reactivity in bilateral vestibulopathy.** Scatter plots show relationship between alpha EEG power enhancement on eye closure (alpha reactivity) and video head impulse test gain (VHIT gain) for a range of channels in sitting (a) and standing (c) conditions. Head plots show correlation between alpha reactivity and VOR gain across the head in sitting (b) and standing (d) conditions, as well as pooled data (sitting and standing, (e)). Significant channel effects are highlighted by a green dot (plots b, d and e; two-tailed p <.05 across all channels by threshold-free cluster enhancement). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

relationship between these variables within the two postures. We therefore pooled the data across sitting and standing conditions in a post-hoc analysis to reveal the general relationship between alpha EEG enhancement on eye closure, and VOR gain. In pooled data, VOR gain correlated with alpha EEG power enhancement on eye closure. TFCE identified 19 significant channels (FP1, Fpz, F3, Fz, F4, FC1, FC2, C3, Cz, C4, CP1, CP2, P3, Pz, P4, POz, O1, Oz and O2; mean β co-efficient = 1.03 μV^2Hz^{-1} with 95% confidence interval 0.0779 to 3.78 μV^2Hz^{-1}, p range = 0.002 to 0.031, Fig. 5e); two additional channels showed effects in results *without* correction for multiple comparisons (FP2 and FC5; Supplementary Fig. 8c). Better VOR gain was therefore linked to greater alpha reactivity.

We undertook Supplementary (exploratory) analyses to clarify whether vestibulo-ocular reflex function correlated with EEG power in individual visual conditions (eyes open or eyes closed) to explain the reactivity findings. With eyes closed, we found no significant relationship between alpha EEG power and VOR gain in either sitting or standing postures, or pooled data (TFCE-corrected p >.05; Supplementary Fig. 9). In the eyes open condition, there was significant positive correlation between alpha EEG power and VOR gain in sitting data, in a single channel - P4 (TFCE identified 1 significant channel; mean β co-efficient = 1.72 μV^2Hz^{-1} with 95% confidence interval 0.981 to 2.46 $\mu V^2Hz^{-}1$, p = 0.019, Supplementary Fig. 10). In an analysis pooling sitting and standing data, only channel P4 was again significant (TFCE identified 1 significant channel; mean β co-efficient = 1.85 μV^2Hz^{-1} with 95% confidence interval 0.960 to 2.74 $\mu V^2Hz^{-}1$, p = 0.010). The correlation between alpha reactivity and vestibulo-ocular reflex function was thus generally not explained by relationships within individual visual conditions.

Next, we looked for a relationship between alpha reactivity and symptom load in participants with bilateral vestibulopathy. No relationship was found between either dizziness handicap inventory or vertigo symptom scale score, and alpha EEG enhancement on eye closure in either sitting or standing data.

4.5. More visually dependent participants with reduced vestibular function have less alpha reactivity

Visual dependence was defined as the deviation of the subjective visual vertical by background visual motion (Cousins et al., 2014). As expected, participants with bilateral vestibulopathy were more visually dependent than controls during the rod and disk task (Fig. 1b, mean [standard deviation] visual motion-induced tilt of the visual vertical: bilateral vestibulopathy 11° [7°], controls 7° [5°]; two-tailed *t*-test: t (28) = 2.09, p =.045, Cohen d = 0.76). We investigated for a relationship between visual dependence and alpha EEG enhancement on eye closure. In patients, in the sitting condition, more visual dependence correlated with less alpha EEG enhancement on eye closure in frontocentral areas (TFCE identified 6 significant channels: F3, Fz, FC5, FC1, Cz, CP2; mean β co-efficient = -0.0241 $\mu V^2(Hz^{\circ})^{-1}$ with 95% confidence interval −0.0461 to −0.00591 $\mu V^2(Hz^{\circ})^{-1}$, p range = 0.021 to 0.033, Fig. 6a,b). Controls instead showed positive correlation between visual dependence and alpha EEG power enhancement on eye closure in right frontal and left centroparietal channels (TFCE identified 3 significant channels: F4, FC2, CP5; mean β co-efficient 0.0742 $\mu V^2(Hz^{\circ})^{-1}$ with 95% confidence interval 0.0291 to 0.126 $\mu V^2(Hz^{\circ})^{-1}$, p range = 0.009 to 0.025, Fig. 6e,f). Participant group moderated the relationship between visual dependence and alpha EEG power enhancement on eye closure in frontocentral channels (TFCE identified 6 significant channels: Fz, F4, FC2, FC6, Cz, C4; mean β co-efficient = 0.0710 $\mu V^2(Hz^{\circ})^{-1}$ with 95% confidence interval 0.0145 to 0.156 $\mu V^2(Hz^{\circ})^{-1}$, p range = 0.002 to 0.022, Fig. 6d). In summary, more visual dependence correlated with less alpha reactivity in patients but the opposite was seen in controls.

Results in the standing condition were similar to sitting (Fig. 7a-f). In

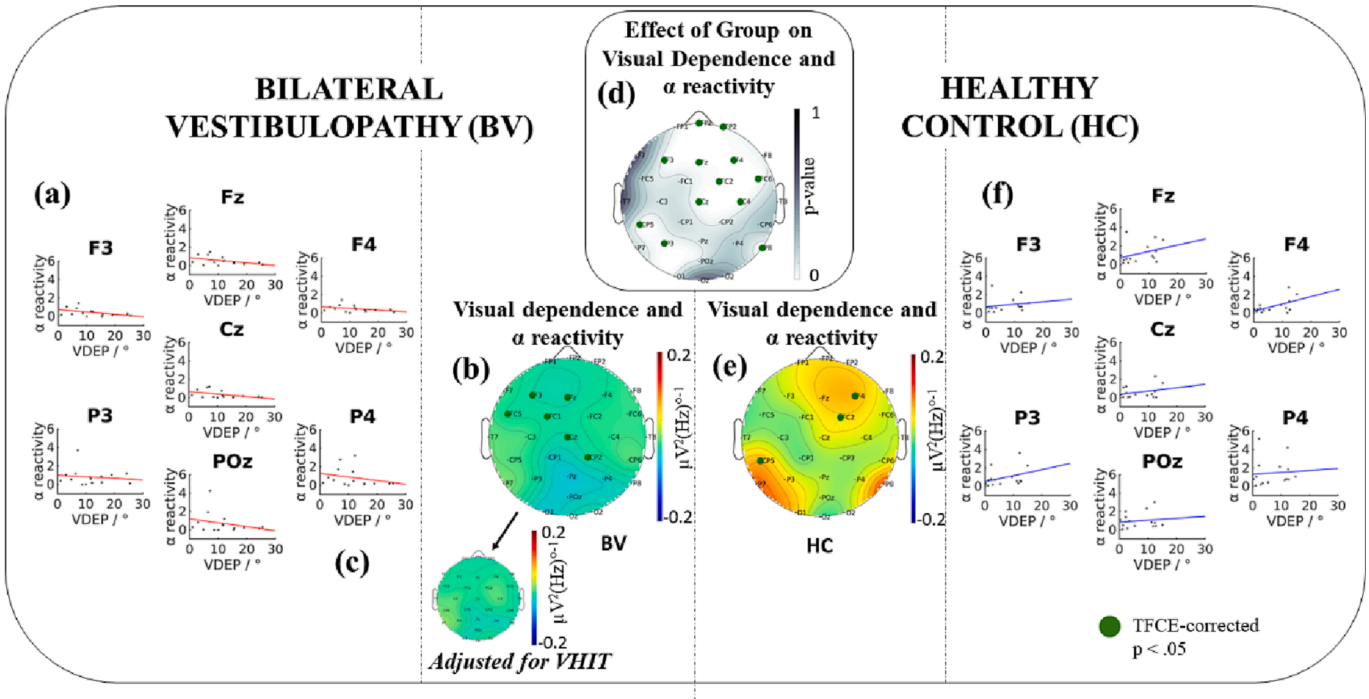

Fig. 6. Visual dependence and alpha reactivity in the sitting condition. Head plots show the correlation between visual dependence and alpha reactivity (effect of eye closure on alpha EEG power) in bilateral vestibulopathy (b), and in health (e) in the sitting condition. In bilateral vestibulopathy, the effect of adjusting this relationship for the effect of video head impulse test gain (VHIT) on alpha reactivity is shown in plot (c). Scatter plots show the underlying data for visual dependence and alpha reactivity at a range of channels for bilateral vestibulopathy (a) and controls (f). The interaction between group (patients, controls), visual dependence and visual condition (alpha reactivity) in the prediction of alpha EEG power is illustrated by head plot of p-values (d). Green dots show channels of significant correlation between alpha reactivity and VOR gain (two-tailed p <.05 across all channels by threshold-free cluster enhancement). BV = bilateral vestibulopathy; HC = healthy control. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

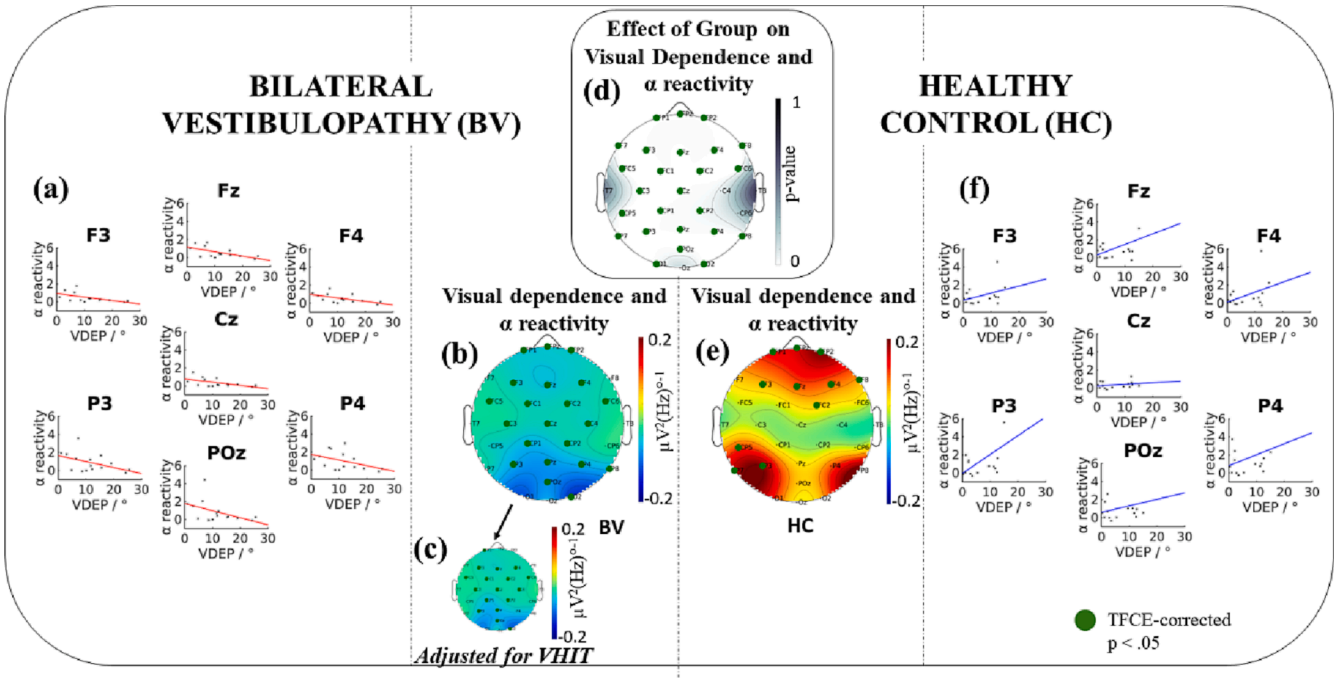

Fig. 7. Visual dependence and alpha reactivity in the standing condition. Head plots show the correlation between visual dependence and alpha reactivity (effect of eye closure on alpha EEG power) in bilateral vestibulopathy (b), and in health (e) in the standing condition. In bilateral vestibulopathy, the effect of adjusting this relationship for the effect of video head impulse test gain (VHIT) on alpha reactivity is shown in plot (c). Scatter plots show the underlying data for visual dependence and alpha reactivity at a range of channels for bilateral vestibulopathy (a) and controls (f). The interaction between group (patients, controls), visual dependence and visual condition (alpha reactivity) in the prediction of alpha EEG power is illustrated by head plot of p-values (d). Green dots show channels of significant correlation between alpha reactivity and VOR gain (two-tailed p <.05 across all channels by threshold-free cluster enhancement). BV = bilateral vestibulopathy; HC = healthy control. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

patients with bilateral vestibulopathy, more visual dependence again correlated with less alpha EEG enhancement on eye closure. TFCE identified 21 significant channels (mean β co-efficient = -0.0506 $\mu V^2(Hz^\circ)^{-1}$ with 95% confidence interval −0.227 to −0.0072 $\mu V^2(Hz^\circ)^{-1}$, p range = 0.002 to 0.037, Fig. 7a,b). Controls again showed positive correlation between visual dependence and visual alpha EEG enhancement on eye closure, but over more frontal and left parietal channels (TFCE identified 11 significant channels: FP1, FPz, FP2, F3, Fz, F4, F8, FC2, CP5, P3, P7; mean β co-efficient = 0.138 $\mu V^2(Hz^\circ)^{-1}$ with confidence interval 0.0131 to 0.321 $\mu V^2(Hz^\circ)^{-1}$, p =.006 to 0.049, Fig. 7e,f). Participant group moderated the relationship between visual dependence and alpha reactivity as before (TFCE identified 25 significant channels: FP1, FPz, FP2, F7, F3, Fz, F4, F8, FC5, FC1, FC2, FC6, C3, Cz, CP5, CP1, CP2, P7, P3, Pz, P4, P8, POz, O1 and O2; mean β co-efficient = 0.150 with 95% confidence interval 0.00511 to 0.490, p range = 0.002 to 0.038, Fig. 7d).

Next, we investigated the influence of vestibulo-ocular reflex function on the relationship between visual dependence and alpha reactivity in bilateral vestibulopathy. In the sitting condition, adjusting for the interaction of VOR gain with visual condition abolished the relationship in patients between visual dependence and alpha EEG power enhancement on eye closure (p >.05 for all channels, Fig. 6b and 6c). In the standing data, adjusting for VOR gain attenuated but did not abolish this relationship, which remained significant in the majority of channels (Fig. 7b and 7c). TFCE identified 17 significant channels (previously 21 channels without adjustment in Fig. 7b; mean β co-efficient = -0.0407 $\mu V^2(Hz^\circ)^{-1}$ with confidence interval −0.185 to −0.00320 $\mu V^2(Hz^\circ)^{-1}$, p range = 0.006 to 0.043, Fig. 7c). The influence of visual dependence on alpha reactivity was thus in part explainable by vestibulo-ocular reflex function.

We undertook Supplementary analyses to clarify whether visual dependence correlated with EEG power in individual visual conditions (eyes open or eyes closed) to explain the visual reactivity findings. With eyes open there was no significant relationship between alpha EEG power and visual dependence in sitting (TFCE p >.05; Supplementary Fig. 11), or standing conditions (TFCE p > 0.05; Supplementary Fig. 12), in either controls or bilateral vestibulopathy; participant group also did not moderate the relationship between alpha EEG power and visual dependence (TFCE p >.05; Supplementary Fig. 11c and 12c). In the eyes closed condition, there was again no significant relationship between alpha EEG power and visual dependence in sitting (TFCE p >.05; Supplementary Fig. 13), or standing conditions (TFCE p >.05; Supplementary Fig. 14), in either controls or bilateral vestibulopathy. Participant group however moderated the relationship between eyes closed alpha EEG power and visual dependence in the standing data within frontal channels (TFCE identified 6 significant channels: FP1, FPz, FP2, Fz, F4, FC2; mean β co-efficient = 0.188 with 95% confidence interval 0.0198 to 0.381, p range = 0.028 to 0.049, Supplementary Fig. 14c). The relationship between visual dependence and alpha reactivity was thus generally not explained by data in individual visual conditions.

4.6. Peak alpha EEG frequency is similar in participants with reduced vestibular function and controls

We measured peak alpha frequency for its potential influence on alpha EEG power (Bazanova, 2012), or as a variable affected by the loss of vestibular input (Table 2). In sitting data, peak alpha frequency was similar in patients and controls in eyes open, and eyes closed conditions (p >.05, Table 2). Peak alpha frequency also did not differ between groups in standing data, in either eyes open, or eyes closed conditions (p >.05, Supplementary Table 1).

5. Discussion

We investigated the relationship between alpha reactivity, and vestibular function. We defined alpha reactivity as the increase in alpha

power with eyes closed compared to eyes open. We studied acquired chronic bilateral vestibulopathy as a model of reduced vestibular function. We found less alpha EEG power enhancement on eye closure in patients with chronic bilateral vestibulopathy compared to controls. In vestibular loss, more alpha reactivity correlated with greater residual vestibulo-ocular reflex function. Greater visual dependence (i.e., larger influence of background visual rotation on visual verticality perception) correlated with less alpha reactivity in bilateral vestibulopathy, and this relationship was partly explainable by vestibulo-ocular reflex function. Our reactivity findings were not explained by relationships between alpha EEG power in the individual (eyes open or eyes closed) visual conditions. Our results demonstrate that vestibular function influences alpha reactivity. They also establish a link between visual dependence, and alpha reactivity.

5.1. Alpha oscillations

Alpha frequency EEG oscillations are involved in the control of cortical activity and excitability. Alpha oscillations propagate from higher to lower order sensory cortices (Halgren et al., 2019), in keeping with a role in the top-down control of sensory processing (Haegens et al., 2011). Local increases in alpha activity lead to reductions in cortical excitability (Sauseng et al., 2009), metabolic activity (Cook et al., 1998), and blood flow (Goldman et al., 2002). Through these changes in cortical function, alpha oscillations likely mediate the suppression of task-irrelevant processing (Foxe and Snyder, 2011). The suppression of task-irrelevant processing is important to performance and cognition, as revealed by studies showing correlation between more alpha activity in task-irrelevant brain areas and better cognitive performance (Haegens et al., 2010; Meeuwissen et al., 2011). Changes in alpha oscillatory activity – including alpha reactivity – thus reflect neurophysiologic processes relevant to performance and cognition. This may be of particular relevance to cortical excitatory levels which are known to be mediated by alpha activity. Indeed, using transcranial magnetic stimulation as a direct probe, it has been shown that cortical excitability is adapted in a clinically dependent manner in bilateral vestibular loss patients (Ahmad et al., 2017).

We obtained data in sitting and standing body postures and found no significant effect of posture on alpha reactivity in bilateral vestibulopathy and controls. Previous work has reported on the effects of body position/posture on EEG – and specifically alpha oscillations. Much of this previous work has compared supine/prone in comparison to sitting postures (Lifshitz et al., 2017; Rice et al., 2013; Spironelli et al., 2016; Spironelli and Angrilli, 2017). A main finding has been more high frequency and less low frequency oscillatory activity in seated compared to supine postures (Lifshitz et al., 2017; Spironelli et al., 2016), which is suggested to reflect cortical inhibition in supine postures (Spironelli et al., 2016). Work in prone and supine postures has revealed some of the change in oscillatory power at the scalp relates to shifts of cerebrospinal fluid (Rice et al., 2013). Other postural work has looked at EEG with a focus on balance-related activity in a single visual condition – providing no information on reactivity (Edwards et al., 2018; Ibitoye et al., 2021). We identified two studies of healthy controls looking at EEG power in both eyes open and eyes closed conditions, which focused on sitting and upright postures, as in our work (Thibault et al., 2014; Zhavoronkova et al., 2012). The first reported an increase in alpha power when standing compared to sitting with the eyes closed, but not with the eyes open (Zhavoronkova et al., 2012). The authors also found a decrease in alpha power with the eyes open when comparing standing on a slightly unstable surface to standing on solid ground (Zhavoronkova et al., 2012). The second study compared supported standing (with the back against a wall) to sitting (Thibault et al., 2014); they reported no effect of upright posture on alpha oscillatory power across eyes open, and eyes closed visual conditions (Thibault et al., 2014). The results from these previous studies suggest alpha power is similar in upright and sitting postures but can be influenced by the upright posture's balance control demands. Neither study specifically reported on alpha reactivity (Thibault et al., 2014; Zhavoronkova et al., 2012) – the focus of our present work, precluding a direct comparison with our findings. Our results of similarity in alpha reactivity in sitting, and standing in a non-challenging setting nonetheless broadly align with this prior evidence.

5.2. Vestibular function and alpha reactivity

We found no significant differences in average alpha EEG power between patients with bilateral vestibulopathy and controls in both eyes open and eyes closed conditions. To our knowledge, our study is the second to investigate EEG measures in this patient group. The first study to do so assessed the effect of rotation on EEG in patients and controls (Gale et al., 2016). At baseline (while seated without motion with eyes open in the dark) they found no significant differences in alpha power between the groups (Gale et al., 2016). Our results are consistent with this previous work, and argue against large systematic differences in average, baseline cortical activation between patients and controls.

We found less enhancement of alpha EEG power on eye closure in patients with bilateral vestibulopathy compared to controls. In agreement, we also found a correlation between better vestibulo-ocular reflex function and more visual alpha EEG enhancement on eye closure in patients with bilateral vestibulopathy. These results together provide clear support not only for a general effect of vestibular function on alpha reactivity, but suggest this effect depends quantitatively on residual vestibular function. In decomposing the EEG power spectra into periodic and aperiodic components (Supplementary Fig. 1), we found that periodic alpha power was relevant to reactivity findings in controls and in bilateral vestibulopathy. This result suggests alpha reactivity in both populations relates to changes in a true narrow-band oscillatory signal, linked to thalamocortical and cortico-cortical interactions (Halgren et al., 2019).

In bilateral vestibulopathy, but not controls, we additionally found the aperiodic exponent and offset increased with eye closure (Supplementary Fig. 4b and 5b). Others have reported visual reactivity in aperiodic components of the power spectrum in health, albeit with a larger sample size and in a paediatric population (Hill et al., 2022); the lack of visual reactivity in the aperiodic offset and exponent in our control data may thus simply reflect our relatively small sample size. The exact meaning of, and underlying mechanisms involved in, the aperiodic EEG component are far from understood (Tröndle et al., 2023). There is neuronal and modelling data (Gao et al., 2017; Manning et al., 2009) suggesting aperiodic components of the EEG spectrum are implicated in the modulation of background excitatory excitability during visuo-spatial attentional processing (Pietrelli et al., 2022). Additionally, aperiodic components have been found to correlate with periodic alpha power (Kosciessa et al., 2020; Tröndle et al., 2023), suggesting shared neurophysiological bases. A role for aperiodic activity in controlling neuronal excitability relevant to visuospatial attention would generally agree with reported changes in functional connectivity (Göttlich et al., 2014; Helmchen et al., 2020) and cortical excitability in bilateral vestibulopathy (Ahmad et al., 2017; Seemungal et al., 2013). These neurophysiological effects relate to the profound changes in visual function which develop following vestibular loss (Morland et al., 1995).

Our finding that participants with reduced vestibular function differed from controls particularly in frontoparietal (but not occipital) areas is of potential interest. An absence of a difference between the groups in alpha reactivity over occipital channels was unexpected. Indeed, increases in alpha oscillations following eye closure – though generally a whole head finding – are most marked posteriorly over occipital channels (Barry et al., 2007). Why then might the effect of bilateral vestibulopathy on alpha reactivity have not been statistically significant in posterior areas? Our recovery of a typical posterior-predominant *Berger* effect (alpha power increases with eye closure) in both control participants and those with reduced vestibular function

argues against variability/noise in posterior channels as a simple explanation for this finding.

A potential explanation for finding no difference between our two groups in alpha reactivity in posterior channels, is that the effect on vestibular function on alpha reactivity might be spatially restricted. Previous work has indeed shown that alpha oscillations likely consist of spatially separable subcomponents. A multidimensional (space, frequency, time) factor analysis of task-free EEG data reported at least two spatially separable (occipitoparietal, and occipitotemporal predominant) components in the alpha frequency range which varied across individuals (Barzegaran et al., 2017; Knyazeva et al., 2018). This previous work does not however provide sufficient detail to explain our finding of largely frontoparietal effects. Another potential explanation might be that our results arise from inadequate statistical power, particularly in the context of our data-driven approach which lacks a spatial (channels of expected effect) hypothesis. If so, uncorrected analyses might be informative. Our findings without multiple comparisons correction, did show a non-significant channel-level effect of vestibulopathy on alpha reactivity in occipital channels in the sitting condition (Supplementary Fig. 3b). This provides some support for the possibility that our data-driven whole-head approach in the context of our sample size masked spatially restricted effects.

A third possibility is that the effect of vestibulopathy might be predominantly on aperiodic components of the EEG power spectrum with different topography to periodic alpha power. Studies detailing periodic alpha and aperiodic components of EEG spectra in health have indeed shown that that the aperiodic exponent has a central-predominant distribution, the aperiodic offset has a fronto-occipital-predominant distribution (Hill et al., 2022; Merkin et al., 2023), and periodic alpha EEG power has an occipital-predominant distribution (Tröndle et al., 2023). Both the aperiodic offset and exponent are known to increase with eye closure (Hill et al., 2022). We found that in bilateral vestibulopathy (but not in controls), the aperiodic exponent and offset increased on eye closure and that this increase generally spared occipital channels (Supplementary Fig. 5 and Supplementary Fig. 6), consistent with their spectra having a different topography to periodic alpha reactivity. We are also mindful that we found an effect of vestibulopathy on visual reactivity in the aperiodic offset of the EEG power spectrum (Supplementary Fig. 7). The topography of aperiodic components of the EEG power spectrum affected by vestibular loss may thus provide a partial explanation for our finding of an effect of vestibulopathy on alpha reactivity in frontoparietal but not occipital areas. Irrespective of the exact basis of the topography of the effect of vestibulopathy at the group level, our other finding in bilateral vestibulopathy of widespread correlation between alpha reactivity and residual vestibulo-ocular reflex function (Fig. 5 and Supplementary Fig. 8) nonetheless suggests a general effect of vestibular loss on alpha reactivity.

5.3. Visual dependence and alpha reactivity

We found that the relationship between visual dependence and alpha reactivity differed between patients and controls. Widespread negative correlation between the two variables was found in individuals with vestibular loss, whilst a positive correlation in frontal areas was instead found in healthy controls. Our results raise the possibility that different mechanisms underpin the relationship between visual dependence and alpha reactivity in the presence or absence/reduction of vestibular input. In normal subjects, greater alpha reactivity is associated with more visual dependence. Indeed, intuitively, one might propose that alpha reactivity is itself a measure of the visual system's stimulus-dependent excitability, and this is supported by the correspondence between alpha reactivity and relative overweighting of visual motion information in perceptual judgements (visual dependence).

What processes could be acting, in the presence of bilateral vestibular dysfunction, that reverse the normal positive correlation between alpha reactivity and visual dependence? We propose that the primary, or trigger, process is the loss of ascending vestibular signals – otherwise necessary for normal inhibition of visual cortices (Brandt et al., 1998). Restated, we argue the loss of vestibular inputs leads directly to less visual alpha EEG enhancement on eye closure (i.e. equivalent to less inhibition of visual cortical activity (Goldman et al., 2002)). In parallel, less ascending vestibular information leads to secondary, perhaps compensatory, processes of re-weighting of multisensory perceptual judgements away from vestibular, and towards visual information. The latter is further supported by the finding that, whilst standing, statistically adjusting for VOR gain attenuated but did not abolish the relationship between alpha reactivity and visual dependence. As this was not the case for seating data, the finding indicates that during the more posturally demanding task the multisensory processes that underpin visual dependence become more prominent and influence alpha reactivity over and above the severity of the peripheral vestibular loss.

We found correlation between visual dependence and less alpha reactivity in patients with bilateral vestibulopathy, but this was only partially explained by vestibulo-ocular reflex function. Our results (Fig. 6 a and b) suggest visual dependence and vestibulo-ocular reflex function independently influence (or are influenced by) alpha reactivity. Indeed, statistical independence between measures of visual dependence and vestibulo-ocular reflex function has previously been shown in the context of vestibular neuritis (Cousins et al., 2014), supporting a more general proposition that higher (e.g. multisensory judgements such as visual dependence) and lower order vestibular functions (e.g. vestibulo-ocular reflex function) are dissociable (Kanayama et al., 1995; Seemungal, 2014). Compensatory processes are suggested to underpin this dissociation in acting on cortical systems to re-enable normal behaviour despite persistently abnormal brainstem and peripheral vestibular signalling (Seemungal, 2014; Yip and Strupp, 2018). Such processes are likely important in the emergence of the negative correlation between visual dependence and alpha reactivity in our chronic bilateral vestibulopathy patients.

Though it is well known that visual dependence increases following vestibulopathy (Bronstein et al., 1996), the underlying mechanisms have remained elusive (Roberts et al., 2018). Disrupted functional connectivity in the thalamocortical vestibular network is likely important (Ibitoye et al., 2022). A potential role for alpha oscillations in the link between vestibulopathy and visual dependence is raised by their established role in thalamocortical signalling (Klimesch et al., 2007), and in influencing perception and attention (Van Diepen et al., 2019). Indeed, it is worth reflecting that in a rod and disc task, a less biased (and thus more correct) decision on the subjective visual vertical (the rod's alignment) depends critically on selective attention to the rod while ignoring distracting background visual motion. Alpha oscillations play a specific role in selective attention as they mediate the suppression of attention to task-irrelevant signals (Snyder and Foxe, 2010). In so doing, alpha oscillations generally shape perception and behaviour (Zhou et al., 2021). Our finding of a relationship between less visual alpha activity and more visual dependence in patients could thus be a consequence of an impairment in selective attention. Within this narrative, vestibular loss impairs alpha reactivity; less alpha reactivity then leads to an impairment of selective visuospatial attention (otherwise supported by usual reactivity); impaired selective attention then leads to underperformance in the rod and disc task due to greater distraction by moving visual stimuli, inducing 'visual' bias (dependence) in verticality judgements. To further explore this proposition, we suggest the question of whether attentional impairments, vestibular function and alpha reactivity are associated or interdependent should be a focus of future work.

5.4. Further implications

Visuospatial processing and attention are also known to depend on vestibular function. Work in rodents established the existence of thalamic and cortical neurones which integrate visual, somatosensory

and vestibular information to convey orientation and heading information necessary for visuospatial tasks (Taube, 2007). In humans, vestibular signals are known to project extensively to visual areas within a larger thalamocortical network (Lopez and Blanke, 2011). Connectivity with visuospatial and memory areas likely explains the association between bilateral vestibulopathy, hippocampal atrophy (Göttlich et al., 2016) and underperformance in visuospatial tasks (Brandt et al., 2005). Altered visuospatial processing accounts for biased judgements in bilateral vestibulopathy patients, for example in the subjective vertical (Bisdorff et al., 1996), and 'visual dependence' in verticality decisions (Bronstein et al., 1996) – a finding replicated in the current study. Given the established links between vestibulopathy, visuospatial processing and attention, and between attention and alpha reactivity, our finding of a relationship between vestibular functioning and alpha reactivity may be important. Our results raise the tentative possibility of changes in alpha reactivity (which reflects cortical excitability (Sauseng et al., 2009)) as a neurophysiologic mechanism through which vestibular loss impairs visuospatial processing and attention.

6. Summary

Overall, our results show that alpha reactivity depends on vestibular function. The lesser ability to enhance the alpha rhythm on eye closure (and relatedly suppress alpha rhythm by eye opening) may be mechanistically connected to cortical excitability changes observed when vestibular input is pathologically reduced. Furthermore, we establish a relationship between vestibulo-ocular reflex gain, visual dependence and alpha reactivity. This is in part directly related to the amount of residual vestibular function but also to central multi-sensory processes triggered by, but to some extent independent of, the peripheral vestibular loss.

Data and code availability statement

Data supporting the results of this work are available at an Open Science Framework repository.

Custom MATLAB scripts used for this work can be made available on reasonable request to the corresponding author.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

Data will be made available on request.

Acknowledgements

We thank the participants for their support of this work. This work was supported by funding of the Dunhill Medical Trust (R481/0516) to A.M.B. and by the Imperial NIHR Biomedical Research Centre. R.T.I. is supported by a Guarantors of Brain Postdoctoral Clinical Fellowship. T. J.E. is supported by a Wellcome Trust Sir Henry Wellcome Postdoctoral Fellowship (Grant Number: 222747/Z/21/Z). D.K. Is supported by the National Institute for Health and Care Research UCLH Biomedical Research Council.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.nicl.2023.103469.

References

Ahmad, H., Roberts, R.E., Patel, M., Lobo, R., Seemungal, B., Arshad, Q., Bronstein, A., 2017. Downregulation of early visual cortex excitability mediates oscillopsia suppression. Neurology 89 (11), 1179–1185.
Angelaki, D.E., Cullen, K.E., 2008. Vestibular system: the many facets of a multimodal sense. Annu. Rev. Neurosci. 31 (1), 125–150.
Babiloni, C., Lizio, R., Vecchio, F., Frisoni, G.B., Pievani, M., Geroldi, C., Claudia, F., Ferri, R., Lanuzza, B., Rossini, P.M., 2010. Reactivity of cortical alpha rhythms to eye opening in mild cognitive impairment and Alzheimer's disease: an EEG study. J. Alzheimers. Dis. 22 (4), 1047–1064.
Barry, R.J., Clarke, A.R., Johnstone, S.J., Magee, C.A., Rushby, J.A., 2007. EEG differences between eyes-closed and eyes-open resting conditions. Clin. Neurophysiol. 118 (12), 2765–2773.
Barry, R.J., De Blasio, F.M., 2017. EEG differences between eyes-closed and eyes-open resting remain in healthy ageing. Biol. Psychol. 129, 293–304.
Barzegaran, E., Vildavski, V.Y., Knyazeva, M.G., 2017. Fine Structure of Posterior Alpha Rhythm in Human EEG: Frequency Components, Their Cortical Sources, and Temporal Behavior. Sci. Rep. 7, 8249.
Bazanova, O.M., 2012. Alpha EEG Activity Depends on the Individual Dominant Rhythm Frequency. J. Neurother. 16 (4), 270–284.
Bazanova, O.M., Vernon, D., 2014. Interpreting EEG alpha activity. Neurosci. Biobehav. Rev. 44, 94–110.
Bense, S., Deutschlander, A., Stephan, T.h., Bartenstein, P., Schwaiger, M., Brandt, T.h., Dieterich, M., 2004. Preserved visual–vestibular interaction in patients with bilateral vestibular failure. Neurology 63 (1), 122–128.
Berger, H., 1929. Über das Elektrenkephalogramm des Menschen. Archiv für Psychiatrie und Nervenkrankheiten 87 (1), 527–570.
Bigdely-Shamlo, N., Mullen, T., Kothe, C., Su, K.-M., Robbins, K.A., 2015. The PREP pipeline: standardized preprocessing for large-scale EEG analysis. Front. Neuroinform. 9, 16.
Bisdorff, A.R., Wolsley, C.J., Anastasopoulos, D., Bronstein, A.M., Gresty, M.A., 1996. The perception of body verticality (subjective postural vertical) in peripheral and central vestibular disorders. Brain 119 (Pt 5), 1523–1534.
Brandt, T., Bartenstein, P., Janek, A., Dieterich, M., 1998. Reciprocal inhibitory visual-vestibular interaction. Visual motion stimulation deactivates the parieto-insular vestibular cortex. Brain 121, 1749–1758.
Brandt, T., Schautzer, F., Hamilton, D.A., Brüning, R., Markowitsch, H.J., Kalla, R., Darlington, C., Smith, P., Strupp, M., 2005. Vestibular loss causes hippocampal atrophy and impaired spatial memory in humans. Brain 128, 2732–2741.
Bronstein, A.M., Yardley, L., Moore, A.P., Cleeves, L., 1996. Visually and posturally mediated tilt illusion in Parkinson's disease and in labyrinthine defective subjects. Neurology 47 (3), 651–656.
Cave, A.E., Barry, R.J., 2021. Sex differences in resting EEG in healthy young adults. Int. J. Psychophysiol. 161, 35–43.
Chae, S., Park, J., Byun, M.S., Yi, D., Lee, J.H., Byeon, G.H., Suk, H.W., Choi, H., Park, J. E., Lee, D.Y., 2020. Decreased Alpha Reactivity from Eyes-Closed to Eyes-Open in Non-Demented Older Adults with Alzheimer's Disease: A Combined EEG and [18F] florbetaben PET Study. J. Alzheimers. Dis. 77 (4), 1681–1692.
Cook, I.A., O'Hara, R., Uijtdehaage, S.H.J., Mandelkern, M., Leuchter, A.F., 1998. Assessing the accuracy of topographic EEG mapping for determining local brain function. Electroencephalogr. Clin. Neurophysiol. 107 (6), 408–414.
Cousins, S., Cutfield, N.J., Kaski, D., Palla, A., Seemungal, B.M., Golding, J.F., Staab, J.P., Bronstein, A.M., Barton, J.S., 2014. Visual dependency and dizziness after vestibular neuritis. PLoS One 9 (9), e105426.
Delorme, A., Makeig, S., 2004. EEGLAB: an open source toolbox for analysis of single-trial EEG dynamics including independent component analysis. J. Neurosci. Methods 134 (1), 9–21.
Dieterich, M., Bauermann, T., Best, C., Stoeter, P., Schlindwein, P., 2007. Evidence for cortical visual substitution of chronic bilateral vestibular failure (an fMRI study). Brain 130 (8), 2108–2116.
Dieterich, M., Brandt, T., 2000. Brain activation studies on visual-vestibular and ocular motor interaction. Curr. Opin. Neurol. 13 (1), 13–18.
Donoghue, T., Haller, M., Peterson, E.J., Varma, P., Sebastian, P., Gao, R., Noto, T., Lara, A.H., Wallis, J.D., Knight, R.T., Shestyuk, A., Voytek, B., 2020. Parameterizing neural power spectra into periodic and aperiodic components. Nat. Neurosci. 23 (12), 1655–1665.
Duffy, F.H., Albert, M.S., McAnulty, G., Garvey, A.J., 1984. Age-related differences in brain electrical activity of healthy subjects. Ann. Neurol. 16 (4), 430–438.
Edwards, A.E., Guven, O., Furman, M.D., Arshad, Q., Bronstein, A.M., 2018. Electroencephalographic Correlates of Continuous Postural Tasks of Increasing Difficulty. Neuroscience 395, 35–48.
Foxe, J.J., Snyder, A.C., 2011. The Role of Alpha-Band Brain Oscillations as a Sensory Suppression Mechanism during Selective Attention. Front. Psychol. 2, 154.
Gale, S., Prsa, M., Schurger, A., Gay, A., Paillard, A., Herbelin, B., Guyot, J.-P., Lopez, C., Blanke, O., 2016. Oscillatory neural responses evoked by natural vestibular stimuli in humans. J. Neurophysiol. 115 (3), 1228–1242.
Gao, R., Peterson, E.J., Voytek, B., 2017. Inferring synaptic excitation/inhibition balance from field potentials. Neuroimage 158, 70–78.
Goldman, R.I., Stern, J.M., Engel, J., Cohen, M.S., 2002. Simultaneous EEG and fMRI of the alpha rhythm. Neuroreport 13 (18), 2487–2492.
Göttlich, M., Jandl, N.M., Wojak, J.F., Sprenger, A., von der Gablentz, J., Münte, T.F., Krämer, U.M., Helmchen, C., 2014. Altered resting-state functional connectivity in patients with chronic bilateral vestibular failure. Neuroimage Clin 4, 488–499.

Göttlich, M., Jandl, N.M., Sprenger, A., Wojak, J.F., Münte, T.F., Krämer, U.M., Helmchen, C., 2016. Hippocampal gray matter volume in bilateral vestibular failure. Hum. Brain Mapp. 37 (5), 1998–2006.
Grüsser, O.J., Grüsser-Cornehls, U., 1972. Interaction of vestibular and visual inputs in the visual system. Prog. Brain Res. 37, 573–583.
Guerraz, M., Yardley, L., Bertholon, P., Pollak, L., Rudge, P., Gresty, M.A., Bronstein, A. M., 2001. Visual vertigo: symptom assessment, spatial orientation and postural control. Brain 124, 1646–1656.
Haegens, S., Osipova, D., Oostenveld, R., Jensen, O., 2010. Somatosensory working memory performance in humans depends on both engagement and disengagement of regions in a distributed network. Hum. Brain Mapp. 31, 26–35.
Haegens, S., Händel, B.F., Jensen, O., 2011. Top-down controlled alpha band activity in somatosensory areas determines behavioral performance in a discrimination task. J. Neurosci. 31 (14), 5197–5204.
Halgren, M., Ulbert, I., Bastuji, H., Fabó, D., Erőss, L., Rey, M., Devinsky, O., Doyle, W.K., Mak-McCully, R., Halgren, E., Wittner, L., Chauvel, P., Heit, G., Eskandar, E., Mandell, A., Cash, S.S., 2019. The generation and propagation of the human alpha rhythm. Proc. Natl. Acad. Sci. U.S.A. 116 (47), 23772–23782.
Helmchen, C., Machner, B., Rother, M., Spliethoff, P., Göttlich, M., Sprenger, A., 2020. Effects of galvanic vestibular stimulation on resting state brain activity in patients with bilateral vestibulopathy. Hum. Brain Mapp. 41 (9), 2527–2547.
Hill, A.T., Clark, G.M., Bigelow, F.J., Lum, J.A.G., Enticott, P.G., 2022. Periodic and aperiodic neural activity displays age-dependent changes across early-to-middle childhood. Dev. Cogn. Neurosci. 54, 101076.
Hufschmidt, A., Dichgans, J., Mauritz, K.-H., Hufschmidt, M., 1980. Some methods and parameters of body sway quantification and their neurological applications. Arch. Psychiatr. Nervenkr. 228 (2), 135–150.
Ibitoye, R., Castro, P., Desowska, A., Cooke, J., Edwards, A., Guven, O., Arshad, Q., Murdin, L., Kaski, D., Bronstein, A., 2021. Small vessel disease disrupts EEG postural brain networks in 'unexplained dizziness in the elderly'. Clin. Neurophysiol. 429, 118505.
Ibitoye, R.T., Mallas, E.-J., Bourke, N.J., Kaski, D., Bronstein, A.M., Sharp, D.J., 2022. The human vestibular cortex: functional anatomy of OP2, its connectivity and the effect of vestibular disease. Cereb. Cortex. https://doi.org/10.1093/cercor/bhac085.
Jacobson, G.P., Newman, C.W., 1990. The development of the Dizziness Handicap Inventory. Arch. Otolaryngol. Head. Neck Surg. 116 (4), 424–427.
Kanayama, R., Bronstein, A.M., Gresty, M.A., Brookes, G.B., Faldon, M.E., Nakamura, T., 1995. Perceptual studies in patients with vestibular neurectomy. Acta Otolaryngol. Suppl. 520 (Pt 2), 408–411.
Kim, S., Oh, Y.-M., Koo, J.-W., Kim, J.S., 2011. Bilateral vestibulopathy: clinical characteristics and diagnostic criteria. Otol. Neurotol. 32, 812–817.
Kleinschmidt, A., Thilo, K.V., Büchel, C., Gresty, M.A., Bronstein, A.M., Frackowiak, R.S. J., 2002. Neural correlates of visual-motion perception as object- or self-motion. Neuroimage 16, 873–882.
Klimesch, W., Sauseng, P., Hanslmayr, S., 2007. EEG alpha oscillations: The inhibition–timing hypothesis. Brain Res. Rev. 53 (1), 63–88.
Knyazeva, M.G., Barzegaran, E., Vildavski, V.Y., Demonet, J.-F., 2018. Aging of human alpha rhythm. Neurobiol. Aging 69, 261–273.
Kosciessa, J.Q., Grandy, T.H., Garrett, D.D., Werkle-Bergner, M., 2020. Single-trial characterization of neural rhythms: Potential and challenges. Neuroimage 206, 116331.
La Vaque, T.J., 1999. The history of EEG Hans Berger. J. Neurother. 3 (2), 1–9.
Lifshitz, M., Thibault, R.T., Roth, R.R., Raz, A., 2017. Source Localization of Brain States Associated with Canonical Neuroimaging Postures. J. Cogn. Neurosci. 29, 1292–1301.
Lopez, C., Blanke, O., 2011. The thalamocortical vestibular system in animals and humans. Brain Res. Rev. 67 (1-2), 119–146.
Manly, B.F.J., 2018. Randomization, bootstrap and Monte Carlo methods in biology. chapman and hall/CRC.
Manning, J.R., Jacobs, J., Fried, I., Kahana, M.J., 2009. Broadband shifts in local field potential power spectra are correlated with single-neuron spiking in humans. J. Neurosci. 29 (43), 13613–13620.
Meeuwissen, E.B., Takashima, A., Fernández, G., Jensen, O., 2011. Increase in posterior alpha activity during rehearsal predicts successful long-term memory formation of word sequences. Hum. Brain Mapp. 32 (12), 2045–2053.
Mensen, A., Khatami, R., 2013. Advanced EEG analysis using threshold-free cluster-enhancement and non-parametric statistics. Neuroimage 67, 111–118.
Merkin, A., Sghirripa, S., Graetz, L., Smith, A.E., Hordacre, B., Harris, R., Pitcher, J., Semmler, J., Rogasch, N.C., Goldsworthy, M., 2023. Do age-related differences in aperiodic neural activity explain differences in resting EEG alpha? Neurobiol. Aging 121, 78–87.
Morland, A.B., Bronstein, A.M., Ruddock, K.H., 1995. Vision during motion in patients with absent vestibular function. Acta Otolaryngol 115 (sup520), 338–342.
Nichols, T.E., Holmes, A.P., 2002. Nonparametric permutation tests for functional neuroimaging: a primer with examples. Hum. Brain Mapp. 15 (1), 1–25.
Partanen, J., Soininen, H., Könönen, M., Kilpeläinen, R., Helkala, E.L., Riekkinen Sr, P., 1996. EEG reactivity correlates with neuropsychological test scores in Down's syndrome. Acta Neurol. Scand. 94, 242–246.
Partanen, J., Soininen, H., Helkala, E.-L., Knnen, M., Kilpelinen, R., Riekkinen, P., 1997. Relationship between EEG reactivity and neuropsychological tests in vascular dementia. J. Neural Transm. 104 (8-9), 905–912.
Pfurtscheller, G., Lopes da Silva, F.H., 1999. Event-related EEG/MEG synchronization and desynchronization: basic principles. Clin. Neurophysiol. 110 (11), 1842–1857.
Pietrelli, M., Samaha, J., Postle, B.R., 2022. Spectral Distribution Dynamics across Different Attentional Priority States. J. Neurosci. 42 (19), 4026–4041.
Pion-Tonachini, L., Kreutz-Delgado, K., Makeig, S., 2019. ICLabel: An automated electroencephalographic independent component classifier, dataset, and website. Neuroimage 198, 181–197.
Rice, J.K., Rorden, C., Little, J.S., Parra, L.C., 2013. Subject position affects EEG magnitudes. Neuroimage 64, 476–484.
Rinne, T., Bronstein, A.M., Rudge, P., Gresty, M.A., Luxon, L.M., 1998. Bilateral loss of vestibular function: clinical findings in 53 patients. J. Neurol. 245 (6-7), 314–321.
Roberts, R.E., Ahmad, H., Patel, M., Dima, D., Ibitoye, R., Sharif, M., Leech, R., Arshad, Q., Bronstein, A.M., 2018. An fMRI study of visuo-vestibular interactions following vestibular neuritis. Neuroimage Clin 20, 1010–1017.
Sauseng, P., Klimesch, W., Gerloff, C., Hummel, F.C., 2009. Spontaneous locally restricted EEG alpha activity determines cortical excitability in the motor cortex. Neuropsychologia 47 (1), 284–288.
Schumacher, J., Thomas, A.J., Peraza, L.R., Firbank, M., Cromarty, R., Hamilton, C.A., Donaghy, P.C., O'Brien, J.T., Taylor, J.-P., 2020. EEG alpha reactivity and cholinergic system integrity in Lewy body dementia and Alzheimer's disease. Alzheimers. Res. Ther. 12, 46.
Seemungal, B.M., 2014. The cognitive neurology of the vestibular system. Curr. Opin. Neurol. 27 (1), 125–132.
Seemungal, B.M., Guzman-Lopez, J., Arshad, Q., Schultz, S.R., Walsh, V., Yousif, N., 2013. Vestibular activation differentially modulates human early visual cortex and V5/MT excitability and response entropy. Cereb. Cortex 23 (1), 12–19.
Smith, S., Nichols, T., 2009. Threshold-free cluster enhancement: addressing problems of smoothing, threshold dependence and localisation in cluster inference. Neuroimage 44 (1), 83–98.
Snyder, A.C., Foxe, J.J., 2010. Anticipatory attentional suppression of visual features indexed by oscillatory alpha-band power increases: a high-density electrical mapping study. J. Neurosci. 30 (11), 4024–4032.
Spironelli, C., Angrilli, A., 2017. Posture Used in fMRI-PET Elicits Reduced Cortical Activity and Altered Hemispheric Asymmetry with Respect to Sitting Position: An EEG Resting State Study. Front. Hum. Neurosci. 11, 621.
Spironelli, C., Busenello, J., Angrilli, A., 2016. Supine posture inhibits cortical activity: Evidence from Delta and Alpha EEG bands. Neuropsychologia 89, 125–131.
Strupp, M., Kim, J.-S., Murofushi, T., Straumann, D., Jen, J.C., Rosengren, S.M., Della Santina, C.C., Kingma, H., 2017. Bilateral vestibulopathy: Diagnostic criteria Consensus document of the Classification Committee of the Bárány Society. J. Vestib. Res. 27, 177–189.
Taube, J.S., 2007. The head direction signal: origins and sensory-motor integration. Annu. Rev. Neurosci. 30 (1), 181–207.
Thibault, R.T., Lifshitz, M., Jones, J.M., Raz, A., 2014. Posture alters human resting-state. Cortex 58, 199–205.
Tröndle, M., Popov, T., Pedroni, A., Pfeiffer, C., Barańczuk-Turska, Z., Langer, N., 2023. Decomposing age effects in EEG alpha power. Cortex 161, 116–144.
van der Hiele, K., Bollen, E.L.E.M., Vein, A.A., Reijntjes, R.H.A.M., Westendorp, R.G.J., van Buchem, M.A., Middelkoop, H.A.M., van Dijk, J.G., 2008. EEG markers of future cognitive performance in the elderly. J. Clin. Neurophysiol. 25, 83–89.
Van Diepen, R.M., Foxe, J.J., Mazaheri, A., 2019. The functional role of alpha-band activity in attentional processing: the current zeitgeist and future outlook. Curr Opin Psychol 29, 229–238.
Wilkinson, G.N., Rogers, C.E., 1973. Symbolic description of factorial models for analysis of variance. J. R. Stat. Soc. Ser. C. Appl. Stat. 22 (3), 392.
Winkler, A.M., Ridgway, G.R., Webster, M.A., Smith, S.M., Nichols, T.E., 2014. Permutation inference for the general linear model. Neuroimage 92, 381–397.
Yardley, L., Masson, E., Verschuur, C., Haacke, N., Luxon, L., 1992. Symptoms, anxiety and handicap in dizzy patients: development of the vertigo symptom scale. J. Psychosom. Res. 36 (8), 731–741.
Yip, C.W., Strupp, M., 2018. The Dizziness Handicap Inventory does not correlate with vestibular function tests: a prospective study. J. Neurol. 265 (5), 1210–1218.
Zhavoronkova, L.A., Zharikova, A.V., Kushnir, E.M., Mikhalkova, A.A., 2012. EEG markers of upright posture in healthy individuals. Hum. Physiol. 38 (6), 604–612.
Zhou, Y.J., Iemi, L., Schoffelen, J.-M., de Lange, F.P., Haegens, S., 2021. Alpha Oscillations Shape Sensory Representation and Perceptual Sensitivity. J. Neurosci. 41 (46), 9581–9592.