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Gaze stabilization deficits during locomotion and their impact on dynamic visual acuity in bilateral vestibulopathy

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31 **Abstract**

32 **Background:** Bilateral vestibulopathy (BVP) is characterized by postural imbalance, gait
33 instability, and impaired gaze stabilization, often causing oscillopsia during walking.
34 Whereas postural and gait deficits affect nearly all patients, visual symptoms are less
35 common and vary in severity. To better understand the determinants of locomotion-induced
36 visual deficits in BVP, we simultaneously quantified head-trunk kinematics, vestibular-
37 mediated gaze stabilization, retinal slip, and dynamic visual acuity (DVA) during walking.

38 **Methods:** Ten patients with BVP and ten healthy controls (HC) walked on a treadmill at 0.4,
39 0.8, 1.2, and 1.6 m/s while fixating a target. We recorded eye movements, head and trunk
40 kinematics, and DVA. Eye and head kinematics were used to assess angular vestibulo-
41 ocular reflex (aVOR), linear gaze stabilization (IGS), and retinal slip in the horizontal (yaw)
42 and vertical (pitch) planes. Subjective visual symptoms were assessed using the Oscillopsia
43 Functional Impact questionnaire (OFI).

44 **Results:** Patients showed worse DVA than HC across locomotion speeds. Head and trunk
45 kinematics were altered, particularly with stronger head-on-trunk stabilization in the pitch
46 plane, which was associated with better DVA. Patients generated compensatory eye and
47 gaze responses during locomotion, but these were compromised in a plane-specific manner.
48 In the yaw plane, aVOR remained largely compensatory, and less compensatory yaw aVOR
49 responses were associated with worse DVA. In the pitch plane, deficits were more
50 pronounced, with more impaired aVOR, overshooting IGS, and increased retinal slip
51 exceeding the threshold for stable vision already at slow walking speeds. These alterations
52 were related to both vestibular dysfunction in the high-frequency domain and reported
53 oscillopsia severity.

54 **Conclusions:** Locomotion-induced visual acuity deficits and oscillopsia in BVP are linked
55 to altered head kinematics and plane-specific vestibular gaze stabilization deficits that scale
56 with the severity of peripheral vestibular dysfunction. Increased head-on-trunk coupling may
57 represent a beneficial compensatory strategy associated with better DVA and could be
58 targeted in rehabilitation.

59 1. Background

60 Bilateral vestibulopathy (BVP) is characterized by bilaterally impaired or absent vestibular
61 function, leading to oscillopsia, postural imbalance and unsteady gait, often worsening in
62 darkness and/or on unsteady ground [1, 2]. These deficits impair everyday activities such
63 as walking, cycling, or driving, are associated with an increased risk of falls [3-5], and have
64 been shown to reduce patients' quality of life [6, 7]. While nearly all patients with chronic
65 BVP suffer from persistent postural and gait instability, only a subset of them (depending on
66 the epidemiological study 30–70% [8-10]) experience clinically relevant visual symptoms,
67 most prominently oscillopsia during walking. The determinants of these locomotion-induced
68 visual symptoms, as well as potential coping strategies that may account for inter-individual
69 variability in symptom severity, remain insufficiently understood.

70 In contrast to impaired balance control during locomotion, which has been studied
71 extensively in BVP [4, 11, 12], gaze stabilization deficits, classically attributed to the
72 vestibulo-ocular reflex (VOR), are typically assessed in isolation from walking [13], either at
73 a mechanistic level using passive head rotations such as the head impulse test [2, 14] or at
74 a functional level using dynamic visual acuity (DVA) testing during sitting [15-17]. Only one
75 prior study has examined head–eye coordination during locomotion in BVP and reported
76 that patients were still able to generate appropriately timed compensatory eye movements
77 that were, however, insufficiently scaled to prevent disturbing retinal slip [18]. Similarly, the
78 few studies that have assessed DVA during walking, have consistently demonstrated
79 degraded performance in patients with BVP that worsens with increasing locomotion speed
80 [19, 20]. However, because gaze stabilization mechanisms and functional visual acuity have
81 rarely been studied in parallel, the determinants of locomotion-induced visual symptoms and
82 potential compensatory strategies that might mitigate oscillopsia in BVP remain unresolved.

83 Several mechanisms may account for the marked inter-individual variability in symptom
84 severity. At the mechanistic level, while the angular vestibulo-ocular reflex (aVOR) and
85 vestibular-mediated linear gaze stabilization (IGS) represent the primary mechanisms of
86 gaze stabilization [21-25], optokinetic and proprioceptive mechanisms may partially
87 compensate for vestibular deficits during slow locomotion but are likely too slow to be
88 effective at higher walking speeds. In addition, locomotion provides an additional source of
89 gaze stabilization control that is not engaged during passive head movements. Accordingly,
90 studies in animal models have demonstrated that locomotor commands arising from spinal
91 central pattern generator circuits can support gaze stabilization via feedforward
92 mechanisms, even in the absence of other afferent sensory inputs [26, 27]. This predictive

93 mechanism is most effective when trunk–neck motion is tightly coupled during locomotion,
94 a condition that may be accentuated in patients with BVP. Consistent with this, a previous
95 study reported increased head-on-trunk stabilization during walking in BVP [28], and the
96 degree to which this strategy is adopted may contribute to inter-individual differences in
97 symptom severity. Finally, at the perceptual level, inter-individual differences in the ability to
98 adapt to retinal slip may contribute to variability in the subjective burden of visual instability.

99 The aim of this study was to elucidate the determinants of locomotion-induced visual
100 symptoms in patients with BVP. To this end, we simultaneously quantified gaze stabilization
101 mechanisms, specifically eye–head coordination compensating for angular head rotations
102 (aVOR) and linear head translations (IGS), together with DVA across a wide range of
103 locomotion speeds, from slow to fast walking, in patients with BVP and healthy controls
104 (HC). Based on these simultaneously acquired measures, we first examined the relationship
105 between gaze stabilization mechanisms and DVA to identify potential mechanistic
106 contributors to locomotion-induced visual impairment. We then related these measures to
107 (1) the clinical measures of peripheral vestibular impairment, (2) patterns of trunk–head
108 coordination that may facilitate predictive gaze stabilization, and (3) questionnaire-based
109 ratings of subjective visual symptoms, thereby linking objective measures of gaze
110 stabilization and visual function to the subjective burden of visual instability.

111

112 **2. Methods**

113 *2.1 Participants*

114 Ten patients with BVP (4 females, mean age 53.9 ± 15.6 years, mean height 177.6 ± 7.0
115 cm, and mean weight 75.7 ± 10.2 kg) and 10 matched HC (4 females, mean age 51.4 ± 17.6
116 years, mean height 173.7 ± 9.0 cm, and mean weight 68.2 ± 9.8 kg) participated in this
117 study. All patients underwent three-dimensional video head impulse testing (3D vHIT) of the
118 horizontal, anterior, and posterior semicircular canals, as well as bithermal caloric irrigation.
119 Bilateral vestibular deficit was clinically confirmed based on a pathological vHIT, defined as
120 a horizontal canal gain < 0.6 , calculated as the median VOR gain 60 ms after head
121 movement onset, and/or bilaterally reduced or absent caloric responses, defined as the sum
122 of maximal slow-phase velocities during warm and cold irrigation $< 6^\circ/\text{s}$ [2]. Detailed clinical
123 characteristics are provided in Table 1.

124

125 2.2 Experimental Procedures

126 Participants walked on a pressure-sensitive treadmill (h/p/cosmos®, Nussdorf-Traunstein,
127 Germany) while fixating a visual target positioned straight ahead at eye level. The target
128 was displayed on a 50-inch LCD monitor (LG 50NANO766QA; 3840 × 2160 pixels; 60 Hz)
129 located 2.0 m in front of the treadmill and consisted of a central fixation point subtending
130 0.15° of visual angle (Figure 1a).

131 Visual acuity was assessed during quiet standing (see DVA assessment below) and
132 repeated at the end of the experiment to account for potential learning or fatigue effects.
133 Participants subsequently performed four locomotion conditions (0.4, 0.8, 1.2, 1.6 m/s),
134 presented in pseudo-randomized order, while maintaining fixation on the target. During
135 locomotion, eye, head, trunk, and gait kinematics were recorded (see below), and DVA was
136 assessed concurrently. Data acquisition started only after participants had reached steady-
137 state locomotion and ended before deceleration, thereby avoiding transitional phases.

138 To ensure a constant viewing distance, deviations from the target distance of 2.0 m were
139 continuously monitored. An elastic tether attached to the participant's lower back provided
140 haptic feedback to assist in maintaining a stable position relative to the display. Between
141 conditions, participants rested for at least 2 minutes or longer if required. Each trial continued
142 until DVA assessment was completed and a minimum of 100 gait cycles had been recorded.

143 After completion of the treadmill recordings, all patients completed the Oscillopsia Functional
144 Impact (OFI) scale, a 43-item questionnaire assessing the functional impact of oscillopsia
145 during everyday activities.[29] For further analysis, we considered the total score and four
146 items specifically related to oscillopsia during walking: item 3 (visual world moving,
147 bouncing, or jumping while walking), item 9 (missing a turn in an unfamiliar environment
148 because a sign could not be read), item 10 (difficulty recognizing familiar faces while
149 approaching a group of people), and item 31 (increased visual motion when walking on
150 grass or sand).

151

152 2.3 Dynamic Visual Acuity Assessment

153 DVA was assessed using Landolt-C optotypes presented on the same display as the fixation
154 target [30]. Stimulus presentation was controlled by custom-written MATLAB software using
155 Psychtoolbox [31]. During testing, the fixation target was transiently replaced by a Landolt-
156 C optotype presented for 500 ms. The orientation of the optotype gap was randomized

157 across eight possible directions, and participants reported the perceived orientation verbally.
158 Responses were entered by the experimenter, triggering the subsequent trial.
159 Optotype size was adaptively adjusted in logMAR units using a best-PEST procedure [30]
160 implemented in the Palamedes toolbox [32]. After each response, a logistic psychometric
161 function was updated online, and the next stimulus level was selected to maximize
162 information around the current threshold estimate. The acuity threshold for each condition
163 was defined as the optotype size corresponding to the midpoint of the fitted psychometric
164 function, i.e., performance halfway between chance and perfect accuracy. A fixed number
165 of 28 optotype presentations was used to estimate the threshold for each condition [33].
166 Visual acuity was expressed in logMAR units, with a lower bound of -0.3 logMAR reflecting
167 high acuity. For each locomotion condition, DVA was calculated as the difference between
168 standing visual acuity (averaged across pre- and post-locomotion assessments) and visual
169 acuity during locomotion.

170

171 *2.4 Eye, head, trunk, and locomotion recordings*

172 Eye movements were recorded using a head-mounted video-oculography system
173 (EyeSeeCam Sci2; EyeSeeTec GmbH, Munich, Germany) at a sampling rate of 120 Hz.
174 Although binocular recordings were obtained, only the right eye was included in the analysis,
175 yielding eye position in the horizontal (yaw) and vertical (pitch) planes. Eye movements were
176 calibrated using a five-point calibration procedure with targets spanning 8.5° of visual angle
177 on the display.

178 Head, trunk, and lower limb kinematics were captured using a six-camera motion capture
179 system (Qualisys AB, Göteborg, Sweden) at 120 Hz. Eye-tracking and motion capture data
180 were temporally synchronized via the Lab Streaming Layer (LSL) framework [34]. Four
181 reflective markers were attached to the head, mounted on the eye-tracker goggles, while
182 three markers were placed on the right scapula, enabling reconstruction of six degrees-of-
183 freedom head and upper trunk motion. From these recordings, head and trunk rotations in
184 the pitch and yaw planes, as well as head translations in the vertical and horizontal
185 directions, were derived.

186 For gait analysis, three reflective markers were placed on each heel to track foot motion in
187 three-dimensional space. Gait cycles were identified from the anterior–posterior heel

trajectories (aligned with the direction of treadmill progression) by detecting heel-strike events as local maxima.

2.5 Data Analysis

For analysis, we considered eye rotation and head rotation in the horizontal (yaw) and vertical (pitch) planes. In addition, linear head motion was converted into an angular equivalent to quantify gaze stabilization against translational head movements [22, 35]. Specifically, linear head translations in the vertical and lateral directions were converted into the angular shift of the fixation target induced by translation using basic trigonometry, i.e., $\theta = \arctan\left(\frac{z}{x}\right)$, where z denotes vertical or horizontal head displacement and x the instantaneous target distance. Because anterior-posterior head movements alter viewing distance, the effective target distance was updated dynamically based on anterior-posterior head position relative to the nominal 2 m viewing distance.

Eye and head signals were segmented into individual gait cycles and time-normalized by resampling each cycle to 100 samples. Occasional eye blinks shorter than 250 ms were interpolated using cubic spline interpolation, whereas gait cycles containing longer blink periods were excluded. Eye and head position signals were differentiated to obtain velocity traces and subsequently low-pass filtered using a fourth-order Butterworth filter with a cutoff frequency of 10 Hz [22, 36]. Head kinematics were calculated from the recorded head motion signals. Peak linear and angular head velocities in vertical (pitch) and horizontal (yaw) planes were obtained for each gait cycle and subsequently averaged across all gait cycles.

Gain and phase were then computed for the two gaze stabilization mechanisms (aVOR and IGS) in both planes using the following signal pairs (Figure 1b). For aVOR, eye angular velocity was related to angular head velocity. For IGS, gaze velocity (defined as the sum of eye and angular head velocities) was related to the angular equivalent of translational head motion [22]. Gains were calculated for each gait cycle as the slope of a least-squares regression (the MATLAB in-built function *robustfit*) between the respective velocity signals [37]. Positive gain values indicate compensatory behavior, whereas negative values indicate in-phase or synergistic behavior. Phase was calculated for each gait cycle by determining the phase shift between sine fits of the corresponding velocity traces constrained to the stride and stepping frequency for yaw and pitch rotations, respectively. By convention, an ideal compensatory response corresponds to a phase of 0° , whereas perfectly in-phase

221 behavior yields -180° . Gain and phase values were subsequently averaged across all gait
222 cycles.

223 Finally, overall gaze stability was assessed using image slip velocity, computed by
224 combining gaze (eye + angular head) and the angular equivalent of translational head
225 motion. Image slip velocities $< 4^\circ/\text{s}$ were considered indicative of stable vision [21]. For each
226 trial, the mean image slip velocity magnitude was quantified.

227 To quantify trunk–head coordination, a head anchoring index (HAI) was computed for pitch
228 and yaw planes based on the variability of head orientation in space and relative to the trunk,
229 as described previously [28, 38]. Specifically, the HAI was computed based on the standard
230 deviation of absolute head orientation in the global coordinate system (σ_a) and the standard
231 deviation of the head orientation relative to the trunk (σ_r):

232
$$HAI = \frac{\sigma_r^2 - \sigma_a^2}{\sigma_r^2 + \sigma_a^2}$$

233 Higher HAI values indicate stabilization of the head in space (head and trunk moving
234 relatively independently), whereas lower values reflect a trunk-referenced strategy (head
235 moving en bloc with the trunk).

236 All data processing and analyses were performed using custom MATLAB scripts.

237

238 *2.6 Statistical Analysis*

239 Statistical analyses were performed in R. Descriptive data are reported as mean $\pm$ standard
240 deviation (SD) for continuous variables and as median (range) for ordinal OFI scores. Effects
241 of locomotion speed (0.4, 0.8, 1.2, and 1.6 m/s), group (HC vs. BVP), and their interaction
242 on head kinematics, DVA, aVOR gain and phase, IGS gain and phase, retinal slip, and HAI
243 were evaluated using linear mixed-effects models with a random intercept for participant.
244 Models were fitted using lmerTest, and fixed effects were evaluated with Type III tests using
245 Satterthwaite’s approximation for denominator degrees of freedom. For the exploratory
246 association analyses, we focused on patients at 1.2 m/s, a walking speed close to a
247 commonly preferred gait pace and therefore considered functionally most relevant.
248 Associations between vestibular function, gaze stabilization, head kinematics, subjective
249 complaints, compensatory strategies, and DVA were assessed using Spearman rank
250 correlations. Given the small sample size, these analyses were considered exploratory.
251 Statistical significance was set at $\alpha = 0.05$.

252

253 **3. Results**

254 *3.1 Patient-reported visual symptoms*

255 Oscillopsia was reported by almost all patients (9/10). Overall symptom severity, quantified
256 by the OFI complete score, varied markedly across patients (median 64.5, range 24-126).
257 Single OFI items that specifically capture oscillopsia during walking showed substantial
258 inter-individual variability for OFI 3 (median 3.0, range 2-4), OFI 9 (median 0.0, range 0-3),
259 OFI 10 (median 1.5, range 0-4), and OFI 31 (median 3.0, range 1-4) (Figure 1d).

260 Subjectively reported oscillopsia symptoms were directly linked to clinical vestibular function
261 measures. Worse horizontal vHIT function corresponded to higher OFI complete scores (ρ
262 = -0.70, $p = 0.025$) and higher OFI 3 scores ($\rho = -0.90$, $p < 0.001$). Worse anterior vHIT
263 function corresponded to higher OFI 31 scores ($\rho = -0.69$, $p = 0.029$), and worse posterior
264 vHIT function to higher OFI 3 ($\rho = -0.67$, $p = 0.033$) and OFI 10 scores ($\rho = -0.66$, $p =$
265 0.039). No clear relationships were observed for caloric function.

266

267 *3.2 Effects of locomotion speed on head kinematics, visual acuity and gaze stabilization* 268 *mechanisms*

269 *3.2.1 Head kinematics*

270 We first examined whether head kinematics in patients differed from HC and how they were
271 modulated by locomotion speed. Peak angular head velocities (Figure 2a) in the yaw plane
272 were relatively stable across locomotion speeds and were generally higher in patients than
273 in HC ($F(1, 18.3) = 6.68$, $p = 0.019$). In contrast, peak angular head velocities in the pitch
274 plane increased with locomotion speed ($F(3, 44.5) = 9.16$, $p < 0.001$) and were comparable
275 between patients and HC. Peak linear head velocities (Figure 2b) in the yaw plane were
276 likewise comparable between patients and HC and remained largely unchanged across
277 walking speeds. In the pitch plane, however, peak linear head velocities increased markedly
278 with locomotion speed ($F(3, 46.5) = 105.58$, $p < 0.001$) but were overall lower in patients
279 than in HC ($F(1, 18.3) = 9.66$, $p = 0.006$).

280 Finally, we examined how the head was stabilized with respect to locomotion-induced trunk
281 motion using the HAI. In the yaw plane, HAI increased with locomotion speed ($F(3, 51.3) =$
282 12.47 , $p < 0.001$) and was comparable between patients and HC. In the pitch plane, HAI
283 was significantly lower in patients than in HC ($F(1, 17.6) = 5.03$, $p = 0.038$), indicating that

284 patients preferentially adopted a more head-on-trunk stabilization strategy rather than
285 stabilizing the head in space.

286

287 3.2.2 *Dynamic visual acuity*

288 We next examined visual acuity during standing and walking. Visual acuity assessed during
289 standing showed excellent test-retest reliability across repeated measurements obtained
290 before and after the locomotion trials in both patients ($ICC[1,1] = 0.91$) and HC ($ICC[1,1] =$
291 0.96). DVA was significantly reduced in patients compared to HC ($F(1, 18.1) = 9.50$, $p =$
292 0.006), while remaining largely stable across the tested range of walking speeds.
293 Descriptively, however, HC showed a trend toward reduced DVA at higher walking speeds
294 (Figure 1c).

295

296 3.2.3 *Angular vestibulo-ocular reflex (aVOR)*

297 We then assessed the mechanisms contributing to gaze stabilization, starting with the aVOR
298 (Figure 3). Angular eye movements that compensate for head rotations via the aVOR
299 showed plane-specific patterns across locomotion speeds. In the yaw plane, compensatory
300 aVOR responses dominated across all speeds and remained comparable between patients
301 and HC.

302 In contrast, pitch aVOR performance was more impaired in patients than in HC, as reflected
303 by lower gains ($F(1, 17.0) = 5.05$, $p = 0.038$) and more negative phase values ($F(1, 18.2) =$
304 6.54 , $p = 0.020$). In both groups, pitch aVOR responses showed a marked dependence on
305 locomotion speed. Pitch aVOR gain progressively decreased and became negative at faster
306 walking speeds ($F(3, 51.2) = 45.90$, $p < 0.001$), indicating a shift toward more synergistic
307 eye-head behavior. Similarly, pitch aVOR phase shifted toward more negative values with
308 increasing speed ($F(3, 51.6) = 45.98$, $p < 0.001$) (Figure 3b).

309

310 3.2.4 *Linear gaze stabilization (IGS)*

311 We next examined how combined head–eye rotations (i.e., gaze) contribute to the
312 stabilization of translational head motion (Figure 4a,b). IGS responses showed significant
313 speed-dependent modulation in both planes. In the yaw plane, IGS gain decreased with
314 increasing locomotion speed ($F(3, 51.4) = 14.51$, $p < 0.001$), progressively moving away

315 from the ideal compensatory range. Yaw IGS phase likewise became more negative with
316 increasing speed ($F(3, 50.9) = 17.89, p < 0.001$), indicating a marked attenuation of
317 compensatory IGS at higher walking speeds. Patients showed less negative yaw IGS phase
318 values than HC ($F(1, 17.6) = 5.06, p = 0.037$), whereas yaw IGS gain was otherwise broadly
319 comparable between groups (Figure 4b).

320 In the pitch plane, patient performance diverged more clearly from that of HC. Pitch IGS gain
321 depended significantly on locomotion speed ($F(3, 52.4) = 5.61, p = 0.002$) and showed a
322 significant speed-by-group interaction ($F(3, 52.4) = 3.47, p = 0.023$). With increasing speed,
323 pitch IGS gain increased in patients and exceeded 1 at higher walking speeds, indicating an
324 overshooting response, while HC remained close to the compensatory optimum across
325 speeds. Pitch IGS phase also changed significantly with locomotion speed ($F(3, 51.7) =$
326 $4.01, p = 0.012$) and showed a significant speed-by-group interaction ($F(3, 51.7) = 2.81, p =$
327 0.048), with patients shifting toward less negative phase values at higher walking speeds,
328 while HC remained comparatively stable and closer to the compensatory optimum (Figure
329 4b).

330

331 3.2.5 Resultant image slip

332 Finally, to quantify the functional consequences of the observed head–eye coordination
333 patterns, retinal slip magnitude was computed as the residual retinal motion remaining after
334 compensatory gaze responses (see Methods; Figure 4c). As a physiological measure of
335 gaze stabilization performance during locomotion, retinal slip magnitude should remain
336 below $4^\circ/\text{s}$ to ensure stable vision. In the yaw plane, slip magnitude remained broadly stable
337 across walking speeds and was comparable between groups, with descriptively higher
338 values in patients that approached or exceeded the threshold for stable vision, whereas
339 values in HC remained within the functional range across speeds (Figure 4c).

340 In the pitch plane, retinal image slip in patients was even more pronounced. Pitch slip
341 magnitude increased significantly with locomotion speed ($F(3, 51.5) = 22.03, p < 0.001$) and
342 was overall higher in patients than in HC ($F(1, 18.1) = 6.90, p = 0.017$). Whereas HC
343 remained within or close to the functional range during slower walking, pitch slip in patients
344 was already above the threshold for stable vision at slow walking speeds and became
345 markedly elevated at higher speeds (Figure 4c).

346

3.3 Associations between vestibular function, gaze stabilization, subjective complaints and compensatory strategies in patients

Finally, we evaluated potential associations between vestibular function, gaze stabilization, subjective complaints and compensatory strategies in patients. To focus on a functionally relevant locomotor condition, analyses were restricted to 1.2 m/s, a walking speed close to a commonly preferred gait pace. We first examined whether clinical vestibular measures were associated with plane-specific head kinematics and gaze stabilization mechanisms. In the yaw plane, better horizontal vHIT function was associated with lower peak angular and linear head velocities ($\rho = -0.77$, $p = 0.009$; $\rho = -0.84$, $p = 0.002$, respectively). In the pitch plane, better anterior vHIT function was associated with lower retinal slip magnitude ($\rho = -0.68$, $p = 0.042$).

We next examined whether plane-specific head kinematics and gaze stabilization mechanisms were associated with DVA. In the yaw plane, worse DVA was strongly associated with lower aVOR gain ($\rho = -0.90$, $p < 0.001$) and more negative aVOR phase values ($\rho = -0.95$, $p < 0.001$). By contrast, in the pitch plane, no clear associations were observed between DVA and gaze stabilization mechanisms or head kinematics.

We further evaluated whether gaze stabilization mechanisms and DVA outcomes were related to a previously reported compensatory strategy in BVP, namely increased head-on-trunk stabilization during walking. In the pitch plane, lower HAI values were associated with lower DVA values ($\rho = 0.68$, $p = 0.042$), indicating that a stronger trunk-referenced, head-on-trunk stabilization strategy was associated with better DVA. No clear associations were observed in the yaw plane. Finally, we examined whether DVA outcomes were related to subjective oscillopsia complaints as assessed by the OFI. Higher OFI item 31 scores ("oscillopsia complaints while walking on uneven ground") were associated with worse DVA ($\rho = 0.71$, $p = 0.032$).

4. Discussion

The aim of this study was to elucidate the determinants of locomotion-induced visual symptoms in patients with BVP. In line with previous work, our data confirm that visual symptoms, particularly oscillopsia during walking, represent a major clinical problem in BVP, but also show that their severity varies markedly across patients [8-10]. In our cohort, almost all patients reported oscillopsia, yet questionnaire-based oscillopsia severity scores (i.e.,

OFI) spanned a wide range, indicating substantial inter-individual variability in the subjective burden of visual instability. This variability was at least partly related to the clinical severity of vestibular dysfunction, particularly to measures of vHIT gain rather than to outcomes of caloric testing, suggesting that high-frequency vestibular deficits are more relevant for gaze stabilization during walking [13, 39]. Conversely, subjective symptom severity was also related to functional visual acuity, as higher OFI item 31 scores, reflecting oscillopsia while walking on uneven ground, were associated with worse DVA. By combining quantitative measures of locomotion-induced head motion, vestibular-mediated gaze stabilization mechanisms, and functional visual acuity, our study further suggests that the extent of visual impairment in BVP cannot be explained by a single factor alone, but rather reflects the interaction of altered head kinematics, impaired compensatory eye and gaze responses, and potentially adaptive compensatory strategies such as increased head-on-trunk stabilization.

With respect to head kinematics, our findings show that head movements in patients with BVP were altered in a plane-specific manner during walking [28, 40]. Patients showed higher angular head velocities in the yaw plane, whereas linear head motion in the pitch plane was reduced. Such changes may, on the one hand, reflect impaired head stabilization due to reduced vestibular feedback during locomotion [40]. On the other hand, they may also indicate adaptive strategies that adjust head motion to the constraints of impaired gaze stabilization. This interpretation is particularly supported in the pitch plane, where reduced linear head motion was accompanied by lower HAI values, indicating stronger head-on-trunk rather than head-in-space stabilization [28]. In the yaw plane, patients with more severely impaired horizontal vHIT function showed higher angular and linear head velocities. This may reflect poorer dynamic head stabilization, but could also suggest an attempt to increase vestibular drive and thereby exploit residual horizontal VOR function, although evidence for a direct functional benefit remains limited.

In line with previous research, patients showed considerably worse DVA across locomotion speeds [19, 20], indicating that they were less able than HC to compensate for locomotion-induced head movements so as to maintain stable vision during walking. Our mechanistic findings further help to specify how this reduced compensation was expressed across different planes and gaze stabilization mechanisms. In general, patients with BVP were still able to generate compensatory eye and gaze movements during locomotion, but these responses were compromised in a plane-specific manner [18]. In the horizontal plane, patients were still able to generate largely compensatory aVOR responses across

413 locomotion speeds, indicating at least partly preserved angular gaze stabilization during
414 walking [22, 25]. Within the BVP group, however, worse DVA was strongly associated with
415 weaker yaw aVOR compensation, reflected by lower gain and more negative phase values,
416 suggesting that residual horizontal angular VOR function is an important determinant of DVA
417 in this plane. By contrast, horizontal IGS, i.e., gaze stabilization of linear head translations
418 by combined eye-head movements, was comparatively weak and became less
419 compensatory with increasing speed in both groups [22, 25]. Despite these only moderate
420 horizontal deficits, retinal slip in the yaw plane was moderately increased in patients and
421 approached or exceeded the threshold for stable vision.

422 In the pitch plane, gaze stabilization deficits in patients were more pronounced. In HC, pitch
423 aVOR is known to undergo a considerable speed-dependent shift from compensatory to
424 more synergistic behavior, with head and eyes moving increasingly in concert with faster
425 locomotion speeds [22, 25, 41]. Although this general pattern was preserved in patients,
426 pitch aVOR was clearly more compromised in BVP, as reflected by overall lower gains and
427 more negative phase values. Notably, however, these pitch-plane aVOR abnormalities in
428 patients were not clearly related to DVA. Pitch IGS was likewise more impaired in patients
429 than in HC. Whereas pitch IGS in HC remained closer to the compensatory optimum [25],
430 patients showed increasingly elevated pitch IGS gain together with less negative phase
431 values, resulting in overshooting responses. These deficits were directly reflected in the
432 retinal slip data: Pitch retinal slip magnitudes were substantially higher in patients, exceeded
433 the threshold for stable vision already during slow walking, and increased further with
434 locomotion speed. Moreover, higher pitch retinal slip magnitudes were associated with
435 worse anterior vHIT function, linking the vertical gaze stabilization deficit to the clinical
436 severity of vestibular dysfunction in BVP.

437 Beyond the clinical severity of vestibular dysfunction, adaptive compensatory movement
438 strategies may also contribute to the degree of visual impairment in BVP [42]. In line with
439 previous work, our data indicate that patients preferentially stabilized the head en bloc with
440 the trunk, particularly in the pitch plane [28]. Importantly, the stronger this strategy was
441 expressed, the better DVA was preserved. This association is plausible in light of animal
442 studies showing that compensatory eye movements during locomotion can be supported by
443 internal feedforward signals derived from locomotor spinal pattern generator commands [27,
444 43]. Such a mechanism has been primarily described in tadpoles, in which trunk and head
445 form a largely unified mechanical segment [44]. In humans, a comparable feedforward

446 contribution to gaze stabilization would likely depend on locomotion-induced head motion
447 being sufficiently predictable [45, 46], which may be facilitated by increased head-on-trunk
448 stabilization and reduced neck motion, as observed in our patients. While tentative, our
449 findings are compatible with the idea that trunk-referenced head stabilization may represent
450 an adaptive strategy to support gaze control in BVP. This interpretation may also be clinically
451 relevant, as it raises the possibility that such compensatory behavior could be specifically
452 targeted or reinforced within vestibular rehabilitation to improve visual symptoms during
453 walking.

454 Several limitations should be considered when interpreting these findings. First, the sample
455 size was small, particularly for the patient-specific association analyses, which were
456 exploratory and restricted to a single, functionally relevant walking speed and should
457 therefore be interpreted with caution. Future studies in larger patient cohorts will be needed
458 to confirm the robustness of the reported associations. Finally, locomotion was assessed
459 under controlled treadmill conditions, which may differ from overground walking in terms of
460 gait dynamics and head motion patterns [47]. Although treadmill paradigms allow precise
461 control of locomotion speed, it remains unclear whether the observed BVP-related deficits
462 in head–eye coordination generalize to natural environments, particularly under challenging
463 conditions such as uneven ground or reduced lighting. Future studies using mobile gait and
464 gaze-tracking technologies will therefore be important to test whether the relationships
465 identified here also hold in more ecological, patient-relevant settings [48, 49].

466

467 **5. Conclusions**

468 By combining quantitative measures of head kinematics, gaze stabilization mechanisms,
469 retinal slip, and functional visual performance during locomotion, this study provides a more
470 comprehensive account of locomotion-induced visual impairment in BVP. We found that
471 patients with BVP are still able to generate compensatory eye and gaze responses during
472 walking, but that these responses are compromised in a plane-specific manner and
473 insufficient to stabilize vision to the same extent as in healthy individuals. The extent of this
474 deficit is linked to the marked inter-individual variability in patient-reported symptom severity.
475 At the same time, our findings indicate that compensatory movement strategies, particularly
476 increased head-on-trunk stabilization, are an important determinant of functional outcome
477 and likely support gaze stabilization by facilitating predictive, feedforward control of head–
478 eye coordination during locomotion.

479

480 6. Declarations

481 *Ethics approval and consent to participate:* The study protocol (20–1137) was approved by
482 the local Ethics Committee of the University of Munich. The study was conducted in
483 accordance with the Declaration of Helsinki. Prior to participation, all participants gave
484 written informed consent.

485 *Consent for publication:* Not applicable.

486 *Availability of data and materials:* De-identified data reported in this article will be shared
487 with appropriately qualified investigators upon reasonable request for a period of 5 years
488 after publication.

489 *Competing interests:* The authors declare that they have no competing interests.

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492 *Author contributions:* Sandra Kollmansperger: Conceptualization, Methodology,
493 Investigation, Formal analysis, Visualization, Writing – review & editing. Solome Kabtimer:
494 Investigation, Formal analysis, Writing – original draft. Julian Decker: Methodology,
495 Investigation, Formal analysis, Writing – review & editing. Klaus Jahn: Methodology,
496 Funding acquisition, Writing – review & editing. Ken Möhwald: Methodology, Investigation,
497 Writing – review & editing. Max Wuehr: Conceptualization, Methodology, Investigation,
498 Formal analysis, Visualization, Supervision, Funding acquisition, Writing – original draft.

499 *Use of generative AI in manuscript preparation:* ChatGPT (OpenAI, GPT-5.5) was used
500 solely for language editing. It was not used to generate or modify data, analyses, figures,
501 scientific interpretations, or conclusions. All output was reviewed by the authors.

502

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649 **Tables & Figures**

Patient	Age	Sex	Etiology	Caloric L/R		vHIT horizontal L/R		vHIT anterior L/R		vHIT posterior L/R		OFI
01	34	F	idiopathic	0.6	0.6	0.1	0.11	0.53	0.40	0.13	0.30	70
P02	63	M	idiopathic	13.6	9.7	0.26	0.27	0.27	0.34	0.31	0.46	64
P03	59	F	genetic/neurodegenerative	3	2.8	0.17	0.15	0.33	0.17	0.38	0.14	93
P04	67	F	idiopathic	5.2	5.2	0.38	0.47	0.53	0.65	0.43	0.55	78
P05	46	M	idiopathic	0	0	0.37	0.53	0.28	0.33	0.32	0.39	24
P06	75	M	ototoxic	4	11	0.46	0.55	0.33	0.58	0.34	0.53	46
P07	29	M	idiopathic	1	5	0.74	0.81	0.95	0.45	0.28	0.50	50
P08	44	M	audiovestibular	1.2	1.3	0.13	0.08	0.30	0.24	0.17	0.17	126
P09	46	F	audiovestibular	2.4	3.8	0.6	0.77	0.39	0.25	0.66	0.57	26
P10	76	M	genetic/neurodegenerative	14.5	11.9	0.46	0.35	0.53	0.44	0.33	0.37	65

650 **Table 1: Patient characteristics**

651 Abbreviations: F: female; M: male; L/R: left/right; Caloric: sum of maximum slow-phase velocity during
652 bithermal caloric testing (°/s); vHIT: vHIT: video head impulse test, horizontal gain at 60 ms;
653 horizontal/anterior/posterior: semicircular canal planes; OFI: Oscillopsia Functional Impact scale.

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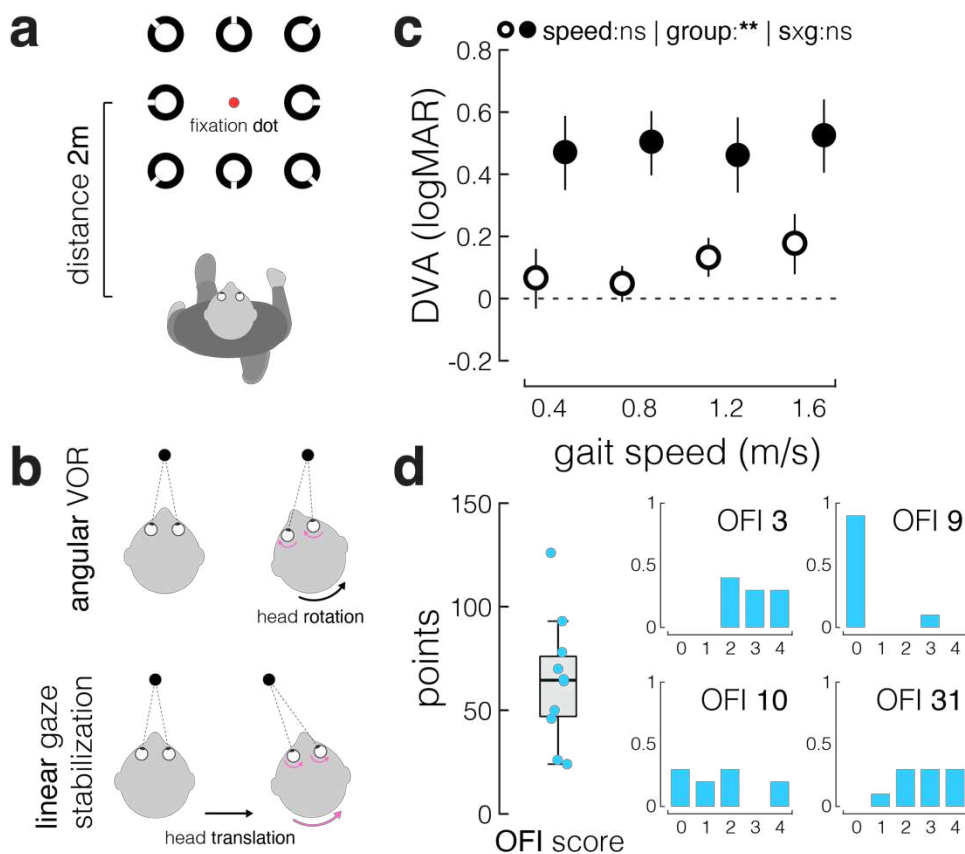
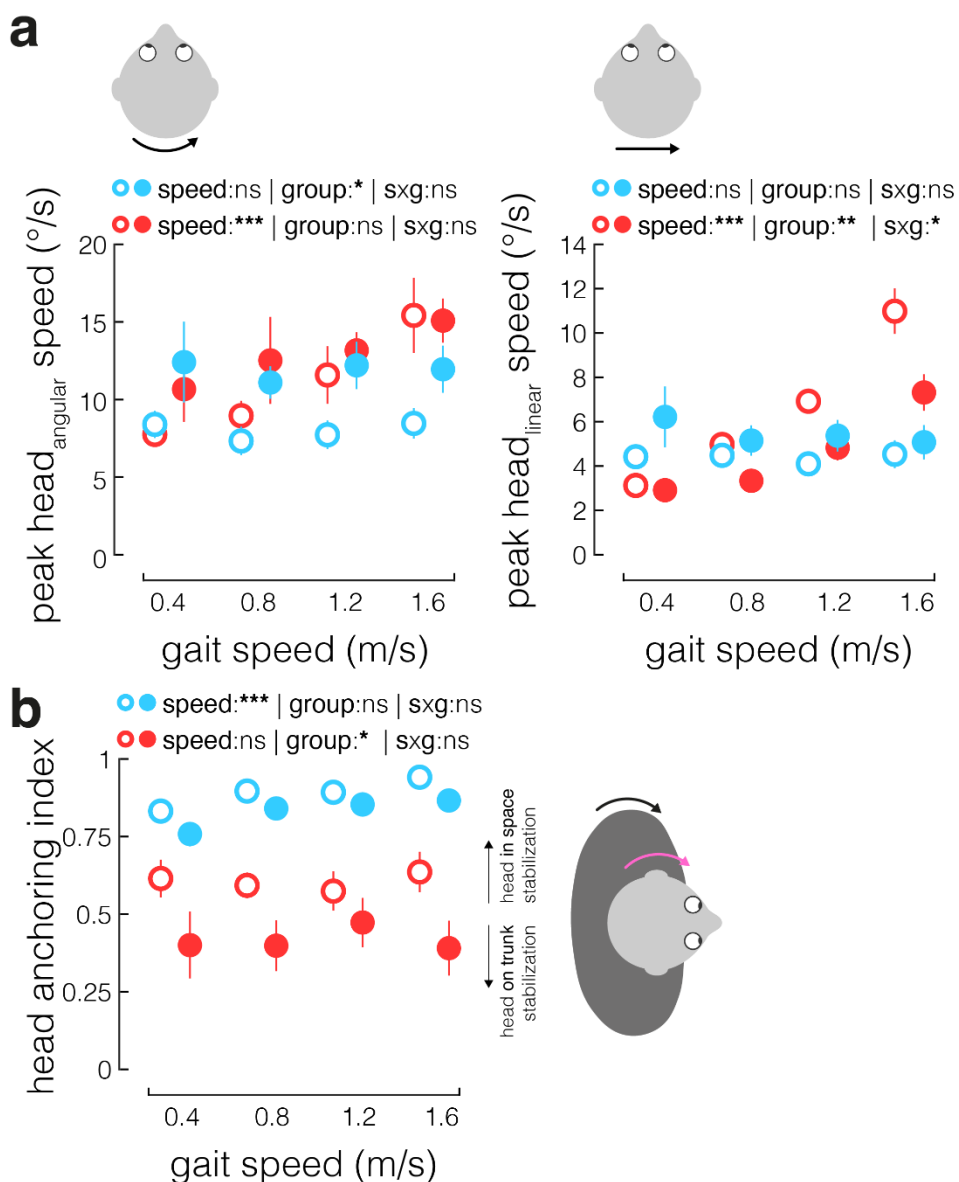


Figure 1: Experimental paradigm, dynamic visual acuity, and oscillopsia-related symptom burden. (a) Schematic of the experimental setup. Participants walked on a treadmill while Landolt-C optotypes were presented on a screen positioned 2 m in front of them. Eye movements, head and trunk movements, and gait events were recorded simultaneously. (b) Schematic illustration of the gaze stabilization mechanisms analyzed, including the angular vestibulo-ocular reflex (aVOR) and linear gaze stabilization (IGS). (c) Dynamic visual acuity (DVA) during locomotion in healthy controls (HC; open circles) and patients with bilateral vestibulopathy (BVP; filled circles). Significant speed, group, and speed-by-group interaction (sxg) effects are indicated where applicable. (d) Oscillopsia Functional Impact scale (OFI) total score and normalized response distributions for selected OFI items related to oscillopsia during walking. * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$; ns: not significant.



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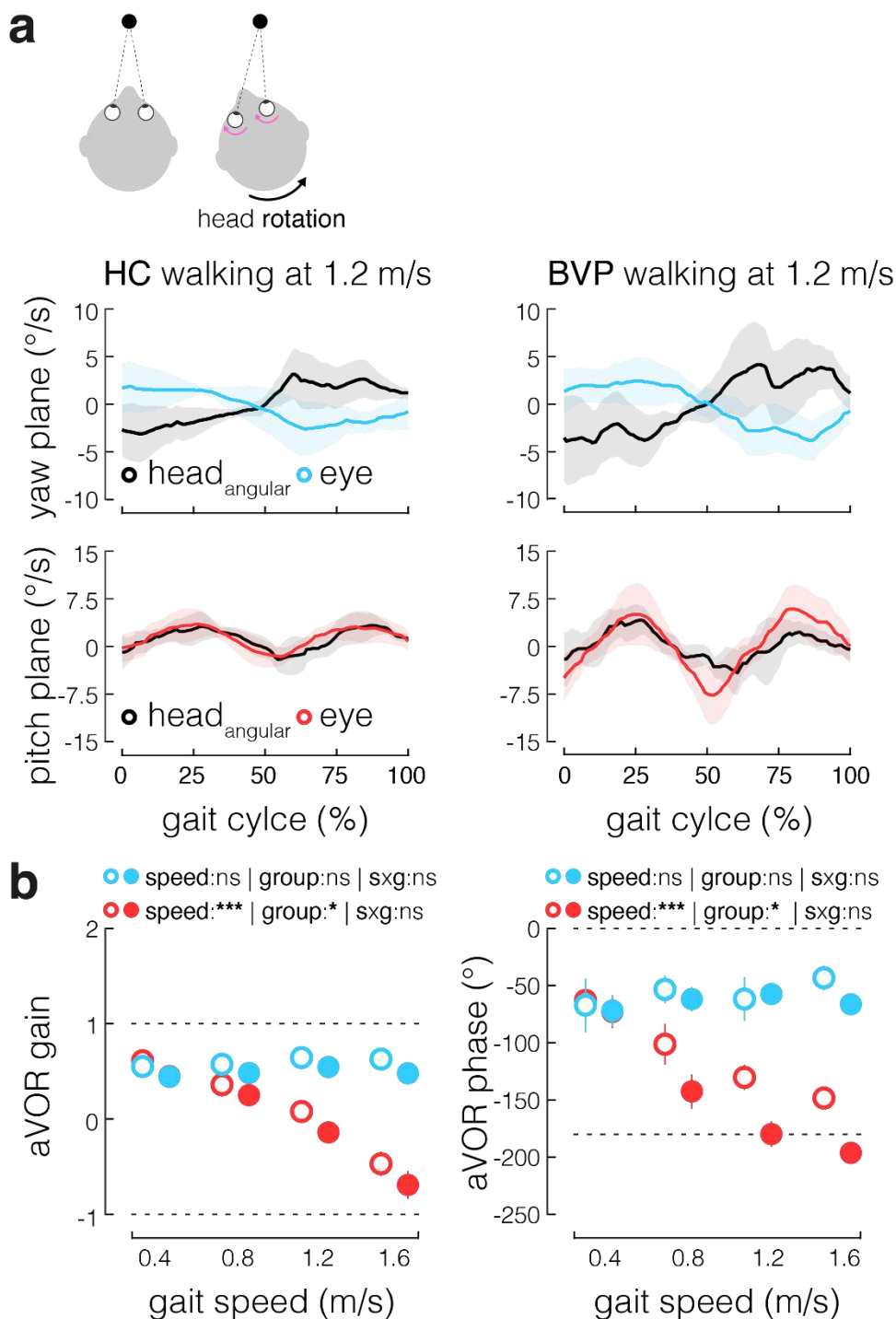
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Figure 2: Head and trunk kinematics across locomotion speeds. (a) Peak angular and peak linear head velocity in the yaw (blue) and pitch (red) planes during treadmill walking in healthy controls (HC; open circles) and patients with bilateral vestibulopathy (BVP; filled circles). Linear head translations were converted to angular equivalents for direct comparison with angular head motion. (b) Head anchoring index in the yaw (blue) and pitch (red) planes for HC and patients with BVP, with higher values indicating stronger head-in-space stabilization and lower values indicating stronger head-on-trunk stabilization. Significant speed, group, and speed-by-group interaction (sxg) effects are indicated where applicable. * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$; ns: not significant.



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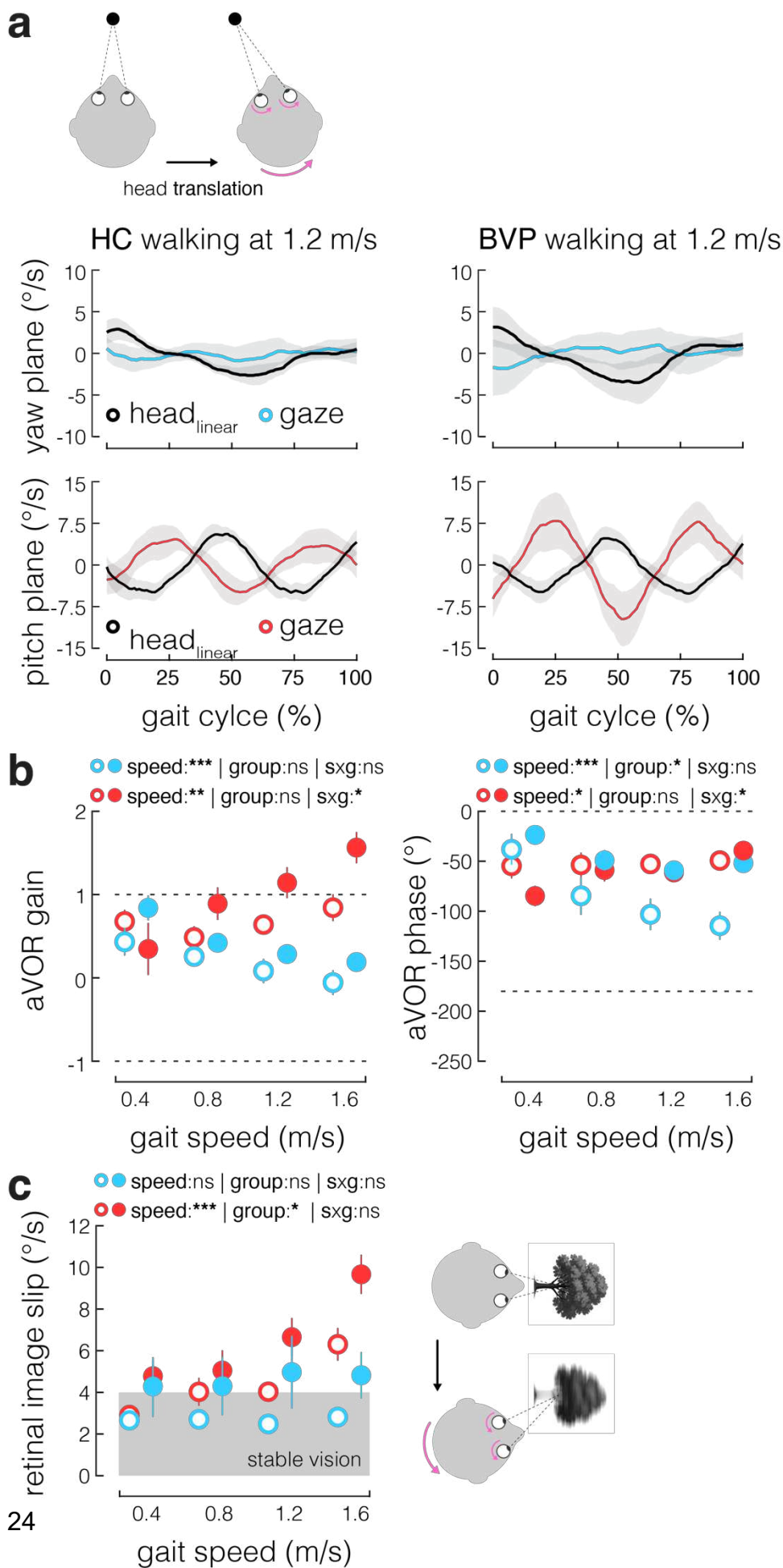
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Figure 3: Angular gaze stabilization across locomotion speeds. (a) Mean eye and head angular velocity profiles (deg/s) over the gait cycle for healthy controls (HC) and patients with bilateral vestibulopathy (BVP) walking at 1.2 m/s. Eye movements are shown in yaw (blue) and pitch (red), and head angular velocity in black. Shaded areas indicate between-participant variability. (b) Corresponding angular vestibulo-ocular reflex (aVOR) gain and phase in the yaw (blue) and pitch (red) planes across locomotion speeds for HC (open circles) and patients with BVP (filled circles). Significant speed, group, and speed-by-group interaction (sxg) effects are indicated where applicable. * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$; ns: not significant.



687 **Figure 4: Linear gaze stabilization and resultant retinal image slip.** (a) Mean gaze and linear head velocity
688 profiles (deg/s) over the gait cycle for healthy controls (HC) and patients with bilateral vestibulopathy (BVP)
689 walking at 1.2 m/s. Gaze movements are shown in yaw (blue) and pitch (red), and linear head translations
690 were converted to angular equivalents for direct comparison with gaze motion (black). Shaded areas indicate
691 between-participant variability. (b) Corresponding linear gaze stabilization (IGS) gain and phase in the yaw
692 (blue) and pitch (red) planes across locomotion speeds for HC (open circles) and patients with BVP (filled
693 circles). (c) Resultant retinal image slip in the yaw (blue) and pitch (red) planes across locomotion speeds for
694 HC (open circles) and patients with BVP (filled circles). Significant speed, group, and speed-by-group
695 interaction (sxn) effects are indicated where applicable. *p < 0.05; **p < 0.01; ***p < 0.001; ns: not significant.