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Original Article

Ethanol-Induced Vestibular Dysfunction as a Model for Bilateral Vestibular Syndrome: Similarities in Video Head Impulse Test and Video-Oculography Data

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BACKGROUND: The goal of this study was to compare video head impulse test, video-oculography, and clinical balance test changes induced by ethanol consumption, in order to acquire a model for acute bilateral vestibular syndrome.

METHODS: Four healthy adult men and 5 healthy adult women were recruited as volunteers in the study. Initial video head impulse test, video-oculography, and clinical balance test examinations were made. Participants proceeded to drink standard alcohol doses until a maximum of 1.2‰ breath alcohol concentration was reached. Video head impulse test and clinical balance tests were repeated at every 0.2‰ breath alcohol concentration interval and at the final 1.0-1.2‰ breath alcohol concentration range. Video-oculography examinations were repeated at 1.0-1.2‰ breath alcohol concentration.

RESULTS: Decrease in mean vestibulo-ocular gain at 60 ms between the 0‰ and 1.0-1.2‰ was 0.16 on the left side ($P < .05$) and 0.16 on the right side ($P < .05$). A borderline abnormality (mean 0.79/0.82) (left/right) was observed in vestibulo-ocular gain at the highest breath alcohol concentration. Corrective saccades increased significantly in amplitude and latency. There was a statistically significant, symmetrical decrease in video-oculography smooth pursuit gain. Saccade latency increased but statistically significantly only with right-sided cycles. Saccade accuracy remained constant. Optokinetic reflex gain showed significant decrease. Romberg's test was performed with normal results initially and at 1.0-1-2‰ breath alcohol concentration.

CONCLUSION: Ethanol produces a symmetrical loss in vestibulo-ocular gain measured by video head impulse test. Ethanol also decreases smooth eye pursuit gain and increases pro-saccade latency. Similar findings can be made in vestibular disorders as well as in cerebellar dysfunction. Central pathology should be ruled out in acute bilateral vestibular syndrome.

KEYWORDS: Ethanol, bilateral vestibular syndrome, video head impulse test, video-oculography, smooth pursuit, saccades

INTRODUCTION

Bilateral vestibular syndrome (BVS) is a rare condition caused by loss of vestibular, cochlear, or vestibulo-cochlear function within both inner ears. Terminology is diverse as bilateral vestibulopathy, vestibular dysfunction, and vestibular loss are also used. Symptoms of BVS are oscillopsia and imbalance, which typically get worse when other sensory inputs are dampened (e.g., walking in darkness and on uneven ground).[1,2,3,4] Auditory symptoms are present if the cochlea is also affected. Etiology of BVS is diverse with as much as 50% being idiopathic.[5] Typically diagnosed causes include ototoxic drugs such as aminoglycosides and cisplatin, bilateral Ménière's disease, meningitis, and autoimmune disease. Other causes include bilateral vestibular schwannomas, bilateral vestibular neuritis, neuro-syphilis, vasculitis, neuro-sarcoidosis, congenital malformations, and traumatic inner ear fistula. Bilateral vestibular syndrome is also associated with cerebellar ataxia and CANVAS (Cerebellar Ataxia, Neuropathy and Vestibular Areflexia) syndrome. Kattah et al[6] demonstrated a series of patients with BVS over a 10-year period, with multiple etiologies, some arising primarily from the central nervous system. The Classification Committee of the Barany Society (CCBS) has provided diagnostic criteria for bilateral vestibulopathy[4] and for presbyvestibulopathy.[7]

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Vestibular neuritis (VN) is a common disorder in its typical unilateral form, characterized by an acute loss of vestibular function without cochlear involvement in the affected ear and without central pathology.[8] Although VN may occasionally later affect also the contralateral ear in the same patient,[6,9,10] acute bilateral VN is considered extremely rare. To our knowledge, Yacovino et al[11] are the only ones to report a case of acute BVS, involving the superior branches of the vestibular nerves. Their patient showed bilaterally abnormal VOR gain (video head impulse test (vHIT)) in plane of lateral and superior semicircular canals, abnormal caloric responses, and abnormal utricular function measured by ocular vestibular evoked myogenic potentials (oVEMP) and cervical vestibular evoked myogenic potentials (cVEMP). Ichijo et al[12] demonstrated a case of hypocalcemia-induced acute vestibulopathy with superior and inferior vestibular nerve branch involvement without auditory symptoms.[12] Not only does the uncommon BVS has diverse etiology, but the exact anatomic location also seems diverse, as demonstrated by the studies mentioned earlier.[11,12]

Alcohol is known to have multiple effects on the neural system. Roth et al[13] documented an increasing bilateral vestibular loss of VOR gain after increasing doses of ethanol. Despite the probable cause for the vestibular loss arising predominantly from the central part of the vestibular system as concluded by the authors, the results are nonetheless intriguing: similar VOR gain findings measured with vHIT can be made in predominantly peripheral bilateral vestibulopathy. Martellucci et al[14] demonstrated also a decrease in VOR gain in the context of alcohol binge drinking.[14] Kattah et al[15] studied patients with thiamine deficiency and Wernicke's encephalopathy, known hazards of prolonged alcohol consumption, resulting in a bilateral loss in VOR gain.

The vHIT is useful in providing diagnostic information regarding bilateral vestibulopathy[4] and is readily available in standard clinical practice. Video head impulse test can distinguish common peripheral vestibular pathology from stroke with high percentage.[16] Other classical, but more time-consuming, otoneurologic examinations include smooth eye pursuit, examination of saccades (by videooculography (VOG)), and caloric testing. Ethanol is known to impair smooth pursuit gain as well as saccades latency, accuracy, and velocity.[17,18,19,20,21] Abnormal VOG findings can also be made in a variety of neurological disorders, especially originating from the cerebellum and brainstem.[22,23] Metabolic, neurodegenerative, inflammatory, ischemic, tumorous, or toxic factors can impair the function of these central areas. Patients with a purely peripheral labyrinth deficit are able to develop normal smooth eye pursuit, unlike the patients with BVS combined with cerebellar degeneration.[24] However, both present with decreased VOR gain.

Table 1. Participant Characteristics

Gender	n (%)
Male	4 (44)
Female	5 (56)
Age mean ± SD	27 ± 2.3

SD, standard deviation.

Table 2. Participant Inclusion Criteria

No previous hazardous alcohol use
Audit score < 8
No history of ear disease or balance disorder
No history of activities including high angular accelerations

AUDIT, Alcohol Use Disorders Identification Test.

The goal of this study was to compare vHIT, VOG, and clinical balance test changes induced by ethanol consumption, in order to acquire a model for acute bilateral vestibulopathy. The secondary goal was to address the potentially diverse etiology of BVS and investigate related central pathology with acute BVS.

MATERIAL AND METHODS

Four healthy adult men and 5 healthy adult women were recruited as volunteers in the study (Table 1). All participants were medical students. Exclusion criteria were previous hazardous alcohol use or an AUDIT[25] score of 8 or more, a medical history with balance problems, or ear disease. The subjects with a history of activities employing high angular accelerations such as figure skating, flying, gymnastics, or other activities consisting of related movements (Table 2) were also excluded. A cohort of young volunteers was chosen as a straightforward strategy to find study subjects who met the inclusion criteria.

The study protocol was approved by the Ethics Committee of The Hospital District of Southwest Finland (ETMK 69/1801/2020), and all participants gave their informed written consent.

Video head impulse test examinations were captured using the EyeSeeCam vHIT (Interacoustics, Assen, Denmark) device performing head turns of 10-20˚ at a peak velocity of 200°/s in the plane the lateral semicircular canals as described in the literature.[26,27] VOR gain was expressed as a mean head-to-eye movements ratio of a

Table 3. Relation Between VOR Gain and BrAC (Mean ± SD)

BrAC	0‰		0.4-0.6‰		0.6-0.8‰		0.8-1.0‰		1.0-1.2‰	
	Left	Right	Left	Right	Left	Right	Left	Right	Left	Right
VOR gain 40 ms	1.19 ± 0.21	1.17 ± 0.26	1.16 ± 0.21	1.20 ± 0.28	1.20 ± 0.19	1.21 ± 0.26	0.86 ± 0.11	0.84 ± 0.04	0.99* ± 0.14	1.05 ± 0.20
VOR gain 60 ms	0.95 ± 0.17	0.98 ± 0.18	0.92 ± 0.16	0.87 ± 0.12	0.98 ± 0.09	0.97 ± 0.12	0.76 ± 0.11	0.80 ± 0.11	0.79* ± 0.09	0.82* ± 0.12
VOR gain 80 ms	0.90 ± 0.10	0.92 ± 0.16	0.85 ± 0.10	0.93 ± 0.04	0.83 ± 0.10	0.84 ± 0.07	0.76 ± 0.17	0.88 ± 0.13	0.73* ± 0.06	0.73* ± 0.10
VOR gain 0-100 ms	1.05 ± 0.10	1.04 ± 0.09	1.01 ± 0.10	1.03 ± 0.07	1.01 ± 0.12	0.98 ± 0.05	0.86 ± 0.10	0.85 ± 0.08	0.87* ± 0.12	0.85* ± 0.06

BrAC, breath alcohol concentration; SD, standard deviation; VOR, vestibulo-ocular reflex.
*Statistically significant change ($P < .05$) between 0‰ BrAC and 1.0-1.2‰ BrAC.

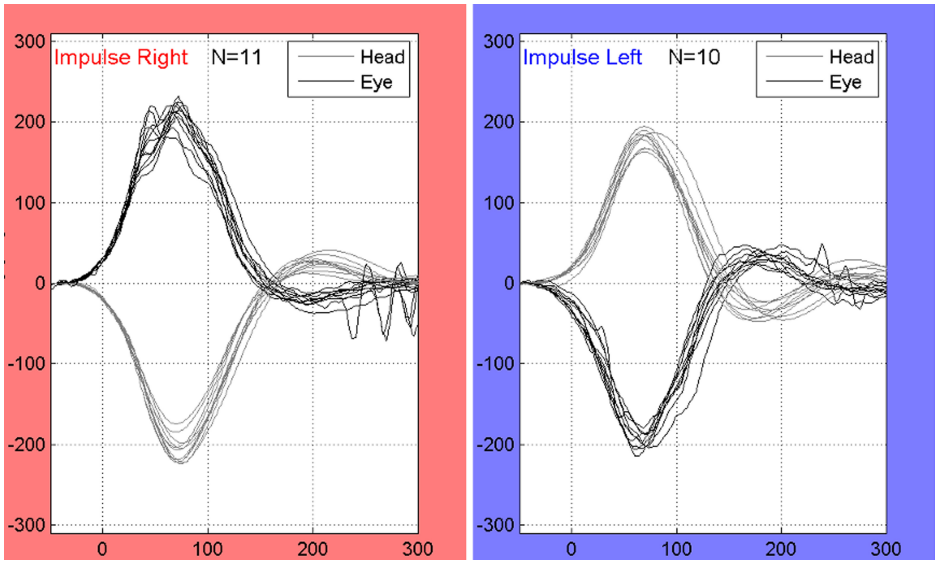

Figure 1. Example of VOR gain and corrective saccades of one study subject measured with vHIT at 0 ‰ BrAC. BrAC, breath alcohol concentration; vHIT, video head impulse test; VOR, vestibulo-ocular reflex.

minimum of 10 head impulses on each side. Mean VOR gain was recorded at 40 ms, 60 ms, 80 ms, and 100 ms latency. A VOR gain of 0.79 or less was considered abnormal.[26,28,29] The quantity, latency, and amplitude of overt catch-up saccades and the number of covert saccades was recorded.[30]

Smooth pursuit and pro-saccades were recorded and analyzed with a VOG[31] device (Micromedical VisualEyes by Interacoustics, Assen). Smooth pursuit eye movements were recorded, while the subject followed a target traveling a sinusoidal waveform for 75 seconds. Smooth pursuit gain was expressed as target-to-eye movement ratio. Saccades were recorded as the subject was asked to glance at appearing light targets presenting each 1-3 seconds with no interval in between. The velocity, accuracy, and latency of saccades were measured. Romberg's test and one-legged stance test were performed as clinical balance tests.

Breath alcohol concentration was measured with Dräger Alcotest 6510 (Drägerwerk AG & Co. KGaA, Lübeck, Germany) which is being also used professionally by the traffic police force. Video head impulse test and VOG measurements as well as clinical proprioceptive tests were first made at BrAC of 0‰. The participants were then asked to drink standard doses of alcohol (equivalent of 4 cl of 40% vodka or 12 g of pure ethanol), and BrAC was measured at steady intervals. Video head impulse test measurements were repeated at BrAC of 0.4-0.6‰, 0.6-0.8‰, 0.8-1.0‰, and at the maximum BrAC of 1.2‰. Video-oculography measurements were repeated at BrAC of 1.0-1.2‰. Romberg test and one-legged stance test were repeated at 1.0-1.2‰.

Statistical Analysis

Statistical analyses were performed with the SAS JMP Pro 16 (SAS Institute Inc., Cary, NC, USA) program. The comparison of VOR gain values, number of covert and overt saccades, smooth pursuit gain values, and velocity and latency of rapid eye movement test for each study subject at each level of BrAC were performed with the matched pairs test. *P*-values <.05 were considered statistically significant.

RESULTS

Video Head Impulse Test

Before alcohol intake, mean VOR gain at 60 ms was 0.95 ± 0.17 (mean ± SD) on the left side and 0.98 ± 0.18 (mean ± SD) on the right side and median VOR gain at 60 ms was 0.95/0.97 (left/right). All participants except one had normal VOR gain before drinking alcohol, while one participant presented with a slightly diminished VOR gain of 0.74/0.75 (left/right). Mean VOR gains are presented in Table 3.

Initially only a few overt catch-up saccades were recorded with a percentage of 12.9%/7.6% (left/right) of the head swings having a small saccade. Mean saccade amplitude was 17.8°/s and 15.4°/s (for the left and right, respectively) and mean latency of 51.6 ms/52.6 ms (left/right). No covert saccades were detected before alcohol intake.

All participants reached the 1.0-1.2‰ BrAC range, where mean VOR gain at 60 ms was 0.79 ± 0.09 (mean ± SD) on the left side and 0.82 ± 0.12 (mean ± SD) on the right side. Median VOR gain was 0.78/0.79 (left/ right). Decrease in mean VOR gain at 60 ms between the 0‰ and 1.0-1.2‰ was 0.16 on the left side (95% confidence interval (CI): −0.26 to −0.063, $P < .05$) and 0.16 on the right side (CI: −0.29 to −0.039, $P < .05$). The percentage of head swing with saccadic eye movements increased to 86% on the left side ($P < .0001$) and 95% on the right side ($P < .0001$). Mean saccade amplitude was increased to 117.8 °/s ± 31.1 °/s (mean ± SD) on the left side ($P < .05$) and 119.7 °/s ± 18.2 °/s on the right side ($P < .0001$). Mean saccade latency was increased to 215 ms ± 32.4 ms (mean ± SD) on the left side ($P < .05$), and 214 ms ± 68.7 ms (mean ± SD) on the right side ($P < .05$). A decrease in VOR gain was also found at 40 ms, 80 ms, and a mean 0-100 ms as presented in Table 3. Female participants presented with higher 0-100 ms gain initially and in the 1.0-1.2 ‰ measurement, but no statistical significance was found, however. Figures 1 and 2 illustrate ethanol's effect on VOR gain and corrective saccades measured by vHIT at 0‰ and at 1.0-1-2 ‰ BrAC. Figure 3 illustrates the relation between VOR gain and BrAC.

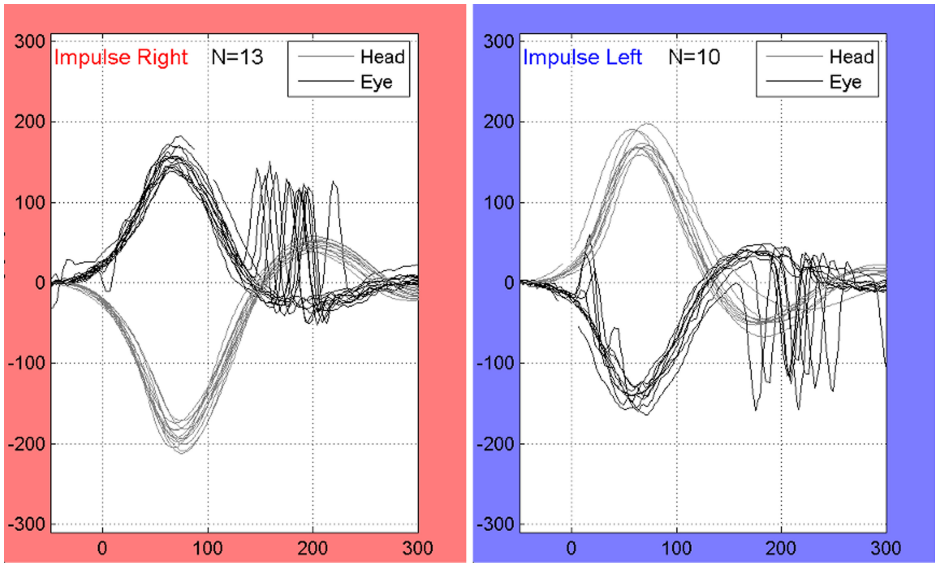

Figure 2. Example of VOR gain and corrective saccades of the same study subject (as in Figure 1) measured with vHIT at 1.0-1.2 ‰ BrAC. BrAC, breath alcohol concentration; vHIT, video head impulse test.

Video-oculography

Initially, all participants had normal smooth eye pursuit, normal pro-saccades, and normal findings in the optokinetic examination. Results for smooth eye pursuit gain, pro-saccade latency, pro-saccade accuracy, and optokinetic examination gain are presented in Tables 4-7.

After alcohol consumption, VOG measurements were repeated at 1.0-1-2‰ BrAC. Smooth eye pursuit gain showed a statistically significant decrease in both directions ($P < .05$ in both eyes and both sided movements); however, decrease of smooth pursuit gain to clinically abnormal range (gain below 62% as described by Intercoustics) was infrequent. Saccade latency increased in both eyes and both directions, but statistical significance was only found in eye movements to the right side in both eyes ($P < .05$). Mean saccade accuracy remained constant and did not decrease after alcohol consumption. Optokinetic testing showed statistically significant decrease in pursuit gain in both directions in both eyes. Figures 4 and 5 illustrate smooth pursuit at 0‰ and at 1.0-1-2‰ BrAC. Figures 6 and 7 illustrate pro-saccades at 0‰ and at 1.0-1-2‰ BrAC.

Clinical Proprioceptive Tests

All participants performed a normal Romberg test before alcohol consumption, and all repeated a normal test at 1.0-1.2‰ BrAC, respectively. Single-legged stance test was normal initially, but at 0.8‰ BrAC, only 22% of participants could perform the test with a normal result. A statistically significant decrease in one leg stance results was found between 0.4 and 0.6‰ BrAC and 1.0 and 1.2‰ BrAC tests ($P < .05$).

DISCUSSION

In this study, we have demonstrated an ethanol-induced bilateral and symmetrical loss of vestibular function described by a loss in vestibulo-ocular reflex gain measured by vHIT. This is the first study to demonstrate a symmetrical loss in smooth eye pursuit gain, an

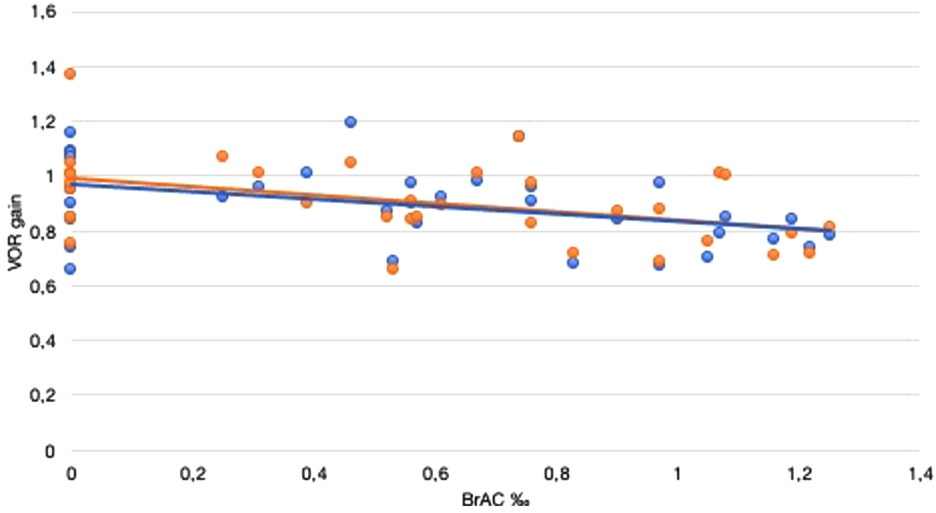

Figure 3. Relation between VOR gain at 60 ms and BrAC. BrAC, breath alcohol concentration; VOR, vestibulo-ocular reflex. Blue dot represent left-sided head swings; orange dots represent right-sided head swings.

Table 4. Video-oculography Smooth Eye Pursuit Gain in Relation to BrAC

Smooth Eye Pursuit Gain (%) (mean ± SD)	0‰ BrAC	1.0-1.2‰ BrAC
Left eye, left cycle	105.1 ± 7.5	89.6* ± 11.5
Left eye, right cycle	107.3 ± 8.3	89.7* ± 15.2
Right eye, left cycle	110.4 ± 5.8	99.9* ± 10.9
Right eye, right cycle	111.2 ± 7.3	96.3* ± 14.4

BrAC, breath alcohol concentration; SD, standard deviation.
*Statistically significant change ($P < .05$) between 0‰ BrAC and 1.0-1.2 ‰ BrAC.

Table 5. Video-oculography Pro-saccade Latency in Relation to BrAC

Pro-saccade Latency in ms (Mean ± SD)	0‰ BrAC	1.0-1.2‰ BrAC
Left eye, left cycle	184.7 ± 26.0	197.1** ± 21.6
Left eye, right cycle	183.2 ± 29.5	205.3* ± 19.4
Right eye, left cycle	172.3 ± 31.0	187.0** ± 19.4
Right eye, right cycle	171.3 ± 31.5	198.7* ± 24.9

BrAC, breath alcohol concentration; SD, standard deviation.
*Statistically significant change ($P < .05$) between 0‰ BrAC and 1.0-1.2 ‰ BrAC; **no statistically significant change ($P > .05$) between 0‰ BrAC and 1.0-1.2‰ BrAC.

increase in pro-saccade eye movement latency, and a loss in opto-kinetic reflex gain under the same circumstances and in the same cohort. Both biological genders were included in this study.

Ethanol has broad effects on the nervous system. As demonstrated by our study, it affects the vestibulo-ocular reflex consisting of a

Table 6. Video-oculography Pro-saccade Accuracy in Relation to BrAC

Pro-saccade Accuracy (%) (Mean ± SD)	0‰ BrAC	1.0-1.2‰ BrAC
Left eye, left cycle	90.3 ± 5.0	95.4** ± 6.7
Left eye, right cycle	95.1 ± 5.7	94.4** ± 6.1
Right eye, left cycle	88.6 ± 7.9	88.8** ± 7.3
Right eye, right cycle	91.8 ± 5.3	91.9** ± 8.0

BrAC, breath alcohol concentration; SD, standard deviation.
**No statistically significant change ($P > .05$) between 0‰ BrAC and 1.0-1.2 ‰ BrAC.

Table 7. Optokinetic Eye Movement Gain in Relation to BrAC

Optokinetic Eye Movement Gain (%) (Mean ± SD)	0‰ BrAC	1.0-1.2‰ BrAC
20 dps		
Left eye, left cycle	87.2 ± 18.8	45.3* ± 17.5
Left eye, right cycle	83.4 ± 15.5	42.0* ± 18.5
Right eye, left cycle	83.4 ± 19.2	45.9* ± 17.9
Right eye, right cycle	86.5 ± 14.9	40.3* ± 18.3
40 dps		
Left eye, left cycle	58.1 ± 27.8	20.7* ± 9.9
Left eye, right cycle	66.6 ± 28.6	21.9* ± 16.8
Right eye, left cycle	60.3 ± 27.6	20.2* ± 10.2
Right eye, right cycle	68.4 ± 26.9	21.1* ± 15.6

BrAC, breath alcohol concentration; SD, standard deviation.
*Statistically significant change ($P < .05$) between 0‰ BrAC and 1.0-1.2‰ BrAC.

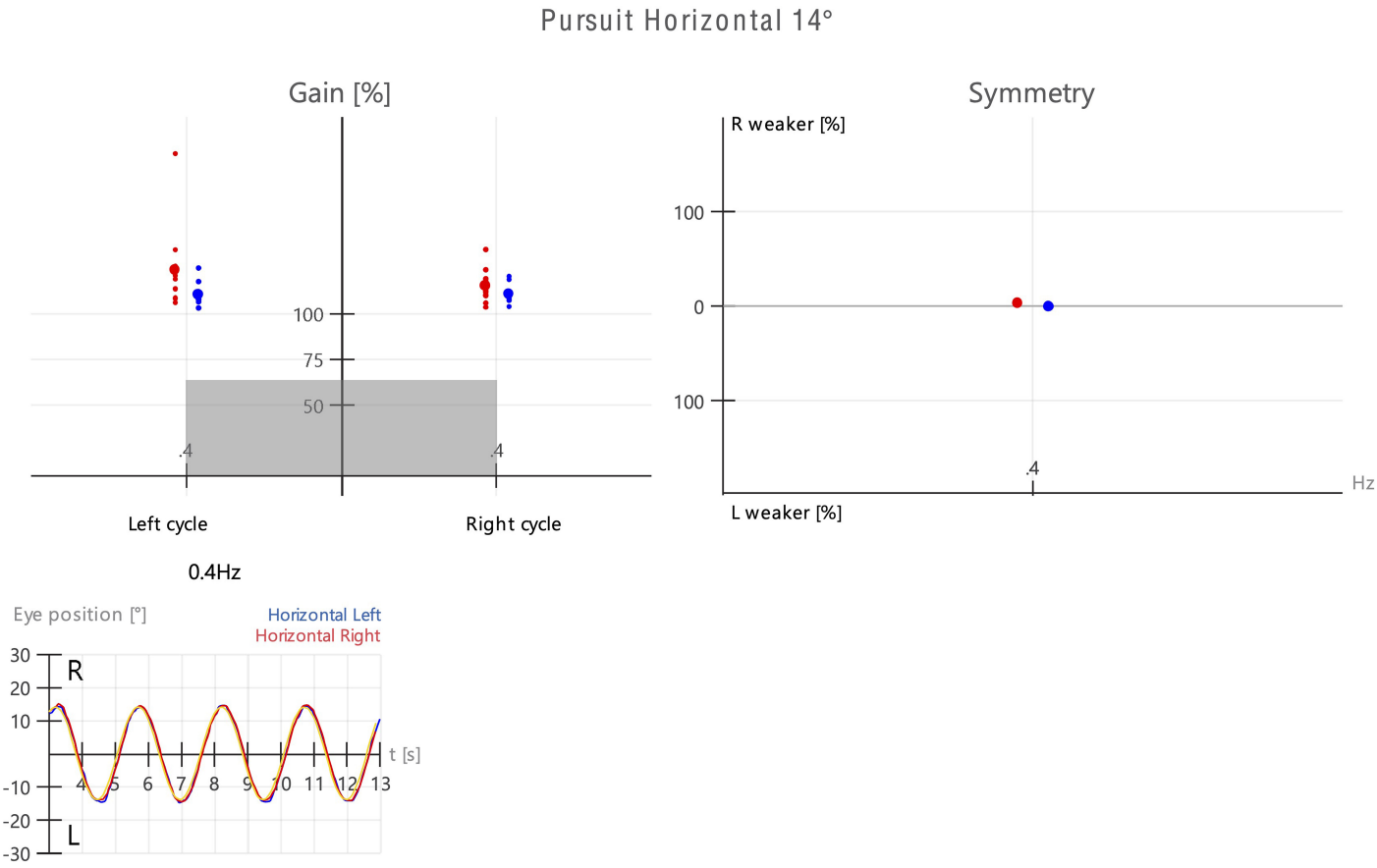

Figure 4. Example of smooth pursuit eye movement of one study subject at 0 ‰ BrAC. BrAC, breath alcohol concentration.

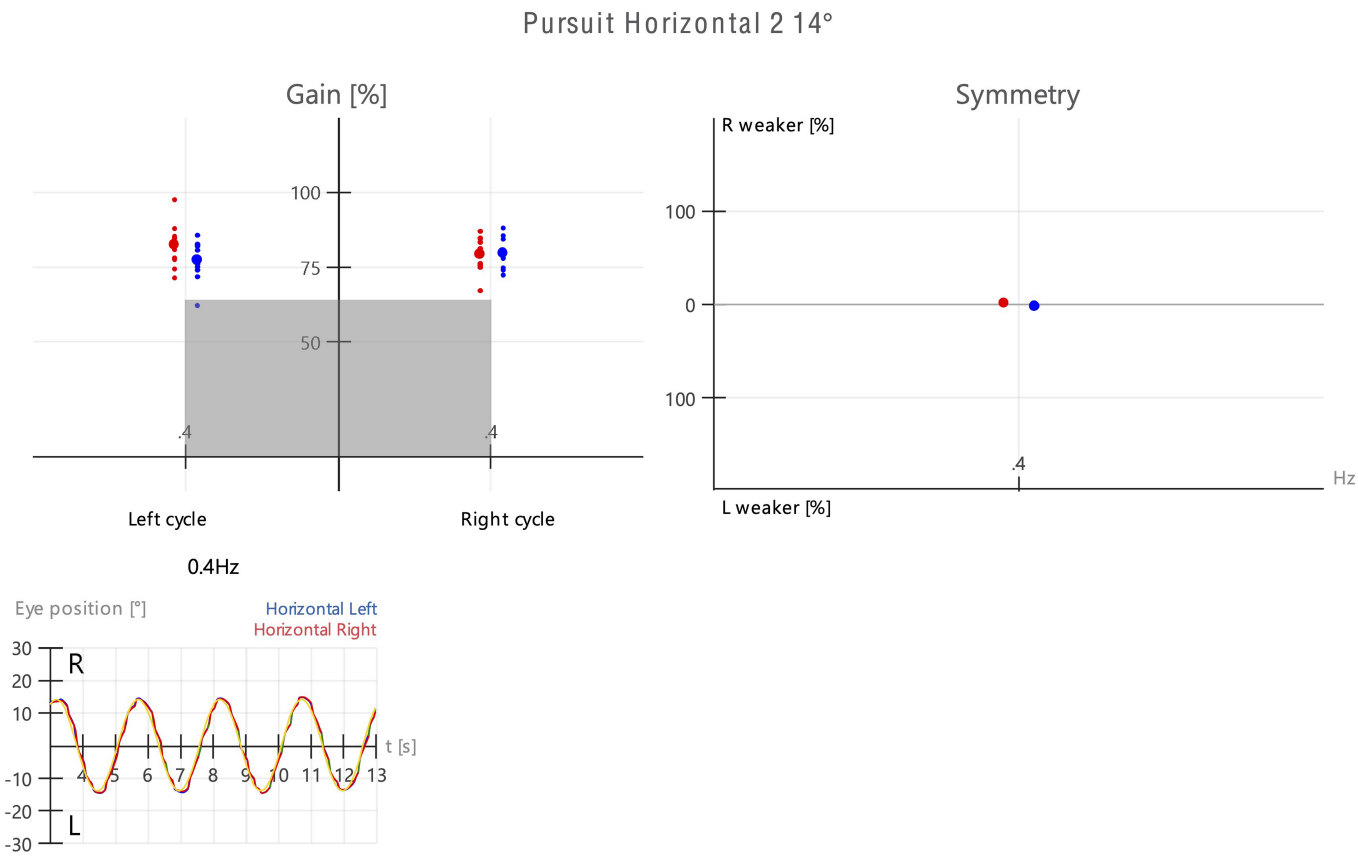

Figure 5. Example of smooth pursuit eye movement of the same study subject (as in Figure 4) at 1.0-1.2‰ BrAC. BrAC, breath alcohol concentration.

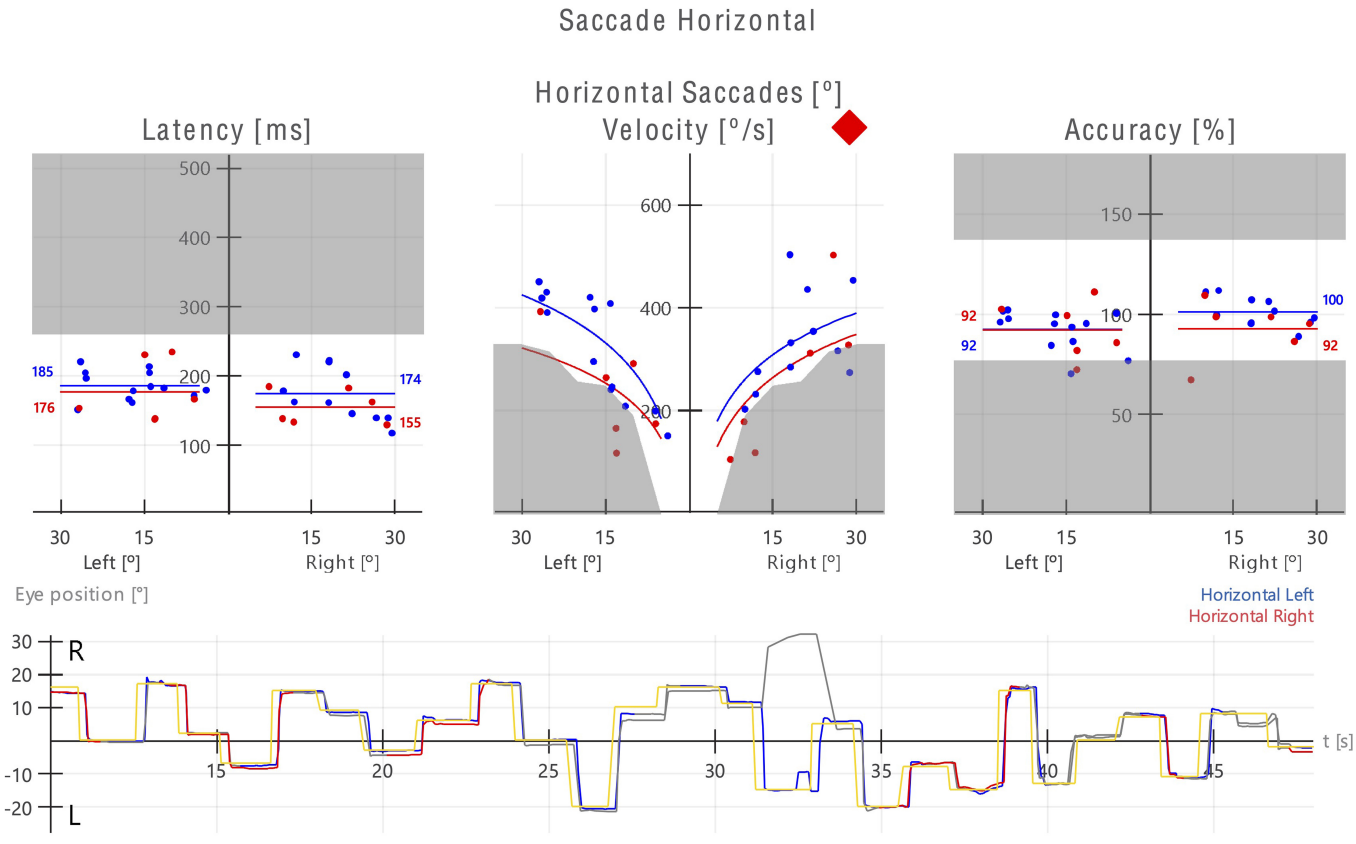

Figure 6. Example of pro-saccade eye movement of one study subject at 0‰ BrAC. BrAC, breath alcohol concentration.

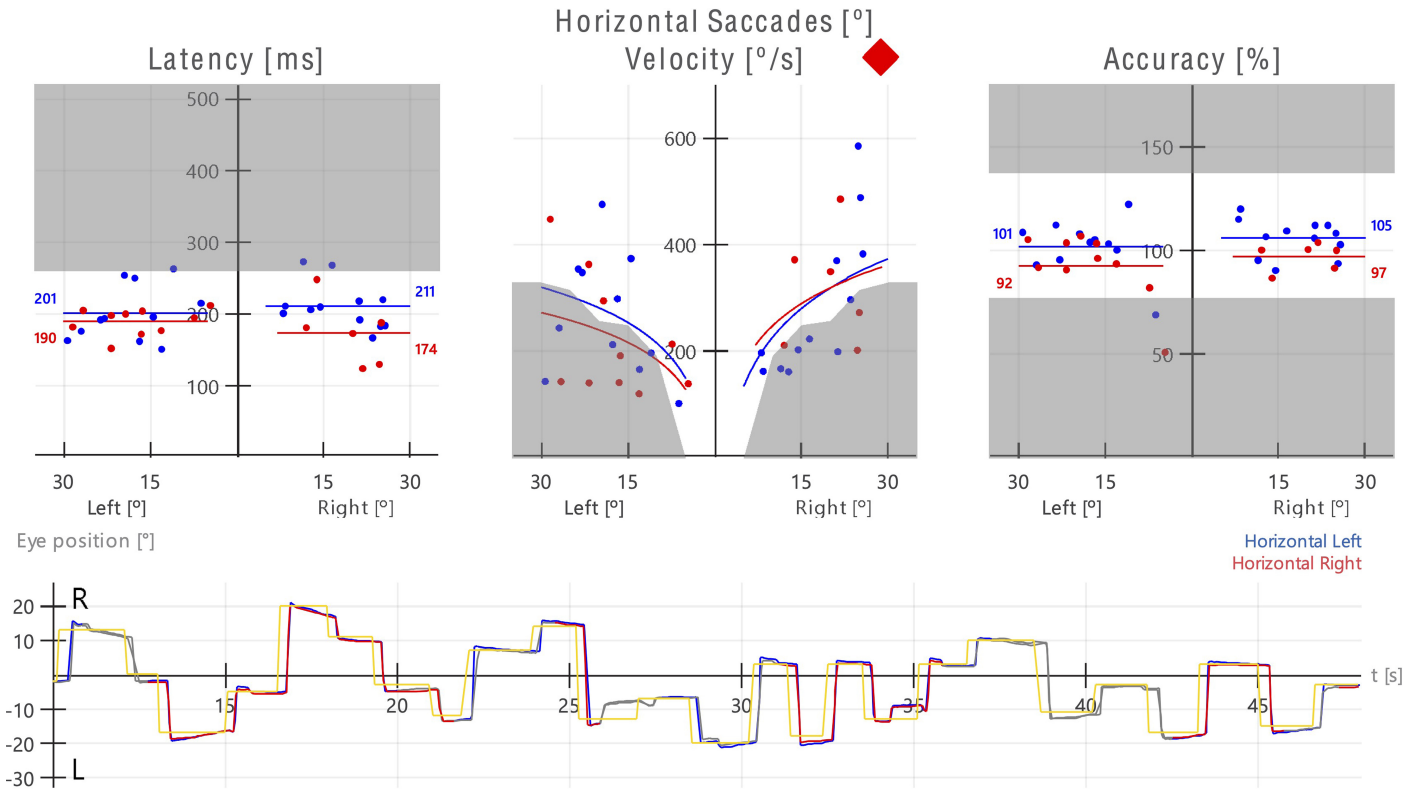

Figure 7. Example of pro-saccade eye movement of the same study subject (as in Figure 6) at 1.0-1.2‰ BrAC. BrAC, breath alcohol concentration.

3-neuron arc from scarpa's ganglion in the internal acoustic meatus to the vestibular nucleus in pons and the nuclei of cranial nerves III, IV, and VI.[32] Horizontal and vertical saccadic eye movements have their supranuclear origin in the frontal eye field. The horizontal saccade pathway proceeds through paramedian pontine reticular formation and medial longitudinal fasciculus for synchronized commands for cranial nerves VI and III. The vertical saccade pathway proceeds through mesencephalic reticular formation in the midbrain to superior and inferior colliculi for cranial nerves III and IV.[33] Smooth pursuit movements are more delicate and require the adequate functioning of the temporal-occipital-parietal-pontine pathway, resolving in the respective common pontine or mesencephalic pathways described for the saccades. The cerebellum plays a major part in fine tuning smooth pursuit and saccadic eye movements.[33,34] Normal performance in optokinetic nystagmus requires the appropriate functioning of saccade and smooth pursuit movement pathways, which both were affected by ethanol consumption in the present study. Romberg's test was performed with good results by participants also after alcohol consumption suggesting sufficient functioning of the posterior column and proprioceptics. Cerebellar control of movement is essential in single-legged stance test, and a decline in performance toward higher BrAC was, hence, expected.

At highest BrAC, mean VOR gain reached 0.79/0.82 (left/right) in vHIT. Although we could not clearly demonstrate a model for bilateral vestibulopathy, a borderline abnormality was, however, observed. One could argue that by extrapolating the results and increasing BrAC to levels not approved by the ethics committee, at some point, VOR gain might decrease symmetrically under 0.6. VOR gain is expected to continue to decrease in a linear fashion. Findings in the present study are in line with previous studies on ethanol's effects on VOR gain measured with vHIT.[13,14] Findings on ethanol's effects on smooth pursuit and pro-saccade latency are also in line with previous research.[17] However, our study did not show a decrease in saccade accuracy. We assume that saccade accuracy would decrease with higher BrAC. Our inclusion of optokinetic testing highlights symmetricity in smooth pursuit and saccadic eye movement losses. Limitations of this study include a small size cohort due to decision by the ethics committee (n = 9, after one initial study subject was unable to attend) and moderate levels of maximum BrAC. Also, due to the data measurement time restraints for the whole study cohort, we were not able to build a repeated measures model, as vHIT measurements could not be obtained for every BrAC value for every study subject. The number of examinations feasible in the given time window was also limited. As we focused in vHIT, VOG, and clinical proprioceptive examinations, other alcohol-related vestibular findings such as the persistent positional nystagmus were neglected.

The cause of BVS remains unknown in as much as 50% of cases, while the most commonly known cause is ototoxicity.[5,35] However, the majority of BVS patients with an underlying cerebellar or neuroinflammatory cause, in conjunction with bilaterally decreased VOR gain, show abnormalities in smooth pursuit and saccadic eye movements, similar to our study findings. Acute bilateral VN remains extremely rare, and thorough investigations should be made to rule out other pathology. Likewise, patients with chronic BVS require careful exclusion of central causes.

Ethanol produces a symmetrical loss in VOR gain measured by vHIT. Ethanol also decreases smooth eye pursuit gain and pro-saccade latency. Similar findings can be made in vestibular disorders as well as in cerebellar dysfunction. Central pathology should be ruled out in acute BVS.

Ethics Committee Approval: This study was approved by Ethics Committee of The Hospital District of Southwest Finland (Approval No: ETMK 69/1801/2020).

Informed Consent: Written informed consent was obtained from the patients who agreed to take part in the study.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – O.A.I.O., J.N.S., J.S.; Design – O.A.I.O., J.N.S., J.S.; Supervision – J.N.S., J.S.; Resources – N/A; Materials – O.A.I.O., J.N.S., L.H., J.S.; Data Collection and/or Processing – O.A.I.O., J.N.S., L.H.; Analysis and/or Interpretation – O.A.I.O., J.N.S., L.H., J.S.; Literature Search – O.A.I.O., J.S.; Writing – O.A.I.O., J.N.S., L.H., J.S.; Critical Review - J.N.S., L.H., J.S.

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