



Original Article

Bilateral Vestibular Hypofunction in Quantitative Head Impulse Test: Clinical Characteristics in 23 Patients

Hilla Levo, Heikki Aalto, Timo P. Hirvonen

University of Helsinki and Helsinki University Hospital, Otolaryngology & Head and Neck Surgery, Helsinki, Finland

Cite this article as: Levo H, Aalto H, Hirvonen TP. Bilateral Vestibular Hypofunction in Quantitative Head Impulse Test: Clinical Characteristics in 23 Patients. J Int Adv Otol 2017; 13: 354-7.

OBJECTIVE: To explore clinical features of patients with bilateral vestibular hypofunction (BVH) verified in motorized head impulse test (MHIT).

MATERIALS and METHODS: We examined clinical records of 23 adult patients (10 males and 13 females), whose gain of the vestibulo-ocular reflex in the MHIT was bilaterally lowered. Fifteen of 62 unilateral cochlear implant (CI) recipients routinely tested both pre- and postoperatively with the MHIT had BVH. Eight of 198 vestibular outpatients selected to the MHIT due to clinical causes had BVH. Clinical characteristics and a questionnaire regarding current sensations were analyzed.

RESULTS: The mean gain \pm SD in the MHIT was 0.26 \pm 0.17 on the right and 0.26 \pm 0.14 on the left side. The mean gain in the CI recipients did not differ from that of vestibular outpatients ($p>0.05$). All outpatients with BVH suffered from oscillopsia, whereas only 46% of CI recipients experienced oscillopsia ($p=0.048$). Instability was more prominent ($p=0.004$) and quality of life further decreased ($p=0.012$) among vestibular outpatients compared with CI patients. Most common etiology for the BVH was meningitis. Other causes were either sudden or progressive loss of labyrinthine function, bilateral Menière's disease, and ototoxicity.

CONCLUSION: BVH is rare even in a specialized clinic. Vestibular outpatients were more disabled than CI recipients with the BVH.

KEYWORDS: Labyrinthine dysfunction, chronic instability, vestibulo-ocular reflex, vestibulopathy

INTRODUCTION

Vestibular dysfunction is a common problem causing vertigo and imbalance due to disturbances in gaze and postural stability. Neuhauser and Lempert ^[1] reported prevalence of 5% and incidence of 1.4% in general population in Germany. Agrawal et al. ^[2] estimated the overall prevalence of vestibular dysfunction in United States population aged ≥ 40 years being 35% with significant increase with age.

Unilateral vestibular hypofunction can be largely compensated, since balance is maintained not only by means of vestibular organ, but also of vision and proprioception. When both vestibular organs are engaged, compensation becomes difficult. Prevalence of bilateral vestibular hypofunction (BVH) is low, and it has varied from 28 to 120 per 100,000 ^[3-5].

The BVH may be hard to identify, when the symptoms begin gradually and the patient has other complaints, such as profound hearing loss. Vestibular loss may vary from partial to total affecting variably low or high frequencies ^[6]. Almost 50% of the patients receiving cochlear implant (CI) have had vertigo and dizziness in their history, although additional vestibular symptoms or dysfunction after implantation is rare ^[7]. Symptoms of BVH may consist of oscillopsia, unsteadiness, and moving difficulties especially in the dark and on uneven surfaces.

Traditionally, the diagnosis of the BVH has relied on the absence of caloric responses. The aim of the present study was to characterize our consecutive patients with BVH verified in quantitative head impulse test. We also wanted to explore whether the possible causes behind this rare condition would be the same with this approach.

MATERIALS and METHODS

Patients

Altogether 260 patients were referred to the motorized head impulse test (MHIT) in our vestibular laboratory. We examined clinical records of 23 adult patients (10 males and 13 females), whose gain of the vestibulo-ocular reflex (VOR) in the MHIT was

Corresponding Address: Timo P. Hirvonen E-mail: timo.hirvonen@hus.fi

Submitted: 21.06.2017 • **Revision Received:** 06.07.2017 • **Accepted:** 23.07.2017 • **Available Online Date:** 02.11.2017

©Copyright 2017 by The European Academy of Otolaryngology and Neurology and The Politzer Society - Available online at www.advancedotology.org

bilaterally lowered. Their mean age was 59 (range, 41-80) years. Fifteen of 62 unilateral CI recipients, all of whom were routinely tested both pre- and postoperatively with the MHIT had BVH, and their hard-of-hearing had lasted on average 31 (range, 3-73) years. Their results did not change significantly when retested on average 1.5 years after the operation. Eight vestibular outpatients referred to the MHIT on clinical basis had BVH. Their vestibular symptoms had lasted on average 5 (range, 1-28) years. Simultaneous caloric areflexia was encountered in all patients tested, and all patients underwent magnetic resonance imaging.

Methods

Vestibular function was determined with motorized, custom-made head impulse rotator, in which horizontal head and eye movements were simultaneously measured during 23 rapid, randomized horizontal head impulses toward each side. The gain of the horizontal VOR was determined as the mean of ratio between eye velocity and inverted head velocity of individual impulses calculated during the 30-ms period before the peak head velocity to the right and left. A detailed description of the MHIT has been published earlier^[8]. In the MHIT, the gain of VOR is ≥ 0.8 among normal population.

All the BVH patients were examined clinically before MHIT. A separate questionnaire was sent on average 3.5 (range, 1-7) years after the diagnosis about their present balance difficulties, dizziness, frequency of unsteadiness, and influence on the quality of life. Each symptom was graded in a five-grade scale (from 0=no to 4=severe).

Patients gave informed consent for the study through a protocol approved by the local ethics committee.

Statistical Analysis

The differences between the patient groups were analyzed using Independent Samples Mann-Whitney U-test running in Statistical Package for the Social Sciences statistics, version 22 (IBM Corp.; Armonk, NY, USA).

RESULTS

An example of the MHIT of a patient, who was suffering from bilateral Menière's disease, is shown in Figure 1. His gain was bilaterally partly lowered to 0.40/0.35. Another patient is presented in Figure 2. She had had streptomycin treatment for tuberculosis in early 70s. Later, she suffered from sudden partial loss of hearing on the left. Her gain was profoundly decreased to 0.03/0.19.

The mean gain \pm SD in the MHIT of 23 patients with the BVH was 0.26 ± 0.17 on the right and 0.26 ± 0.14 on the left side. The mean gain in the CI recipients did not differ significantly from that of vestibular outpatients, and the prevalence of oscillopsia was not related to the lower gain ($p>0.05$).

Etiology

Likely causes and main findings of the patients are shown in Table 1. Most common etiology for BVH was meningitis in seven patients. Five patients suffered from either sudden or progressive loss of labyrinthine function, four had bilateral Menière's disease, and two patients had bilateral ototoxic damage. Miscellaneous causes were seen in three patients, and in two patients, the cause remained unknown.

Subjective Sensations

Most patients had simultaneously several types of balance symptoms. Seven patients had vertigo (35%), seven felt like falling (35%), 16 also had unsteadiness (80%), three patients (15%) had unspecific dizziness, and one patient had no symptoms. Three patients experienced dizziness and moving difficulties constantly, the remaining had symptoms intermittently. Dizziness or balance difficulties were considered mild in six patients (30%), moderate in eight (40%), or severe in three patients (15%). Three (15%) patients had no difficulties.

Three (15%) patients considered their symptoms not decreasing the quality of life, five (25%) slightly decreasing the quality of life, and the remaining 12 (60%) found their symptoms severely decreasing the quality of life.

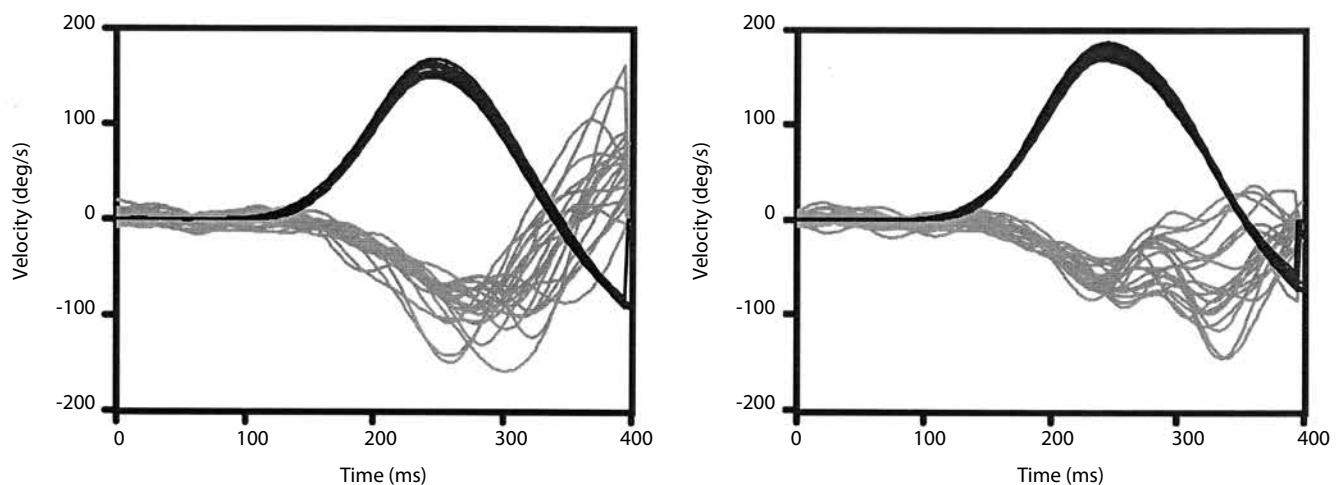


Figure 1. The MHIT results of the patient with bilateral Menière's disease. The panel of the left shows head impulses to the left in black and the compensatory eye movements to the right in gray. The panel on the right shows head impulses to the right and eye movements to the left, respectively. The gain is lowered bilaterally to 0.40/0.35, and the patient has some residual vestibular function on both sides.

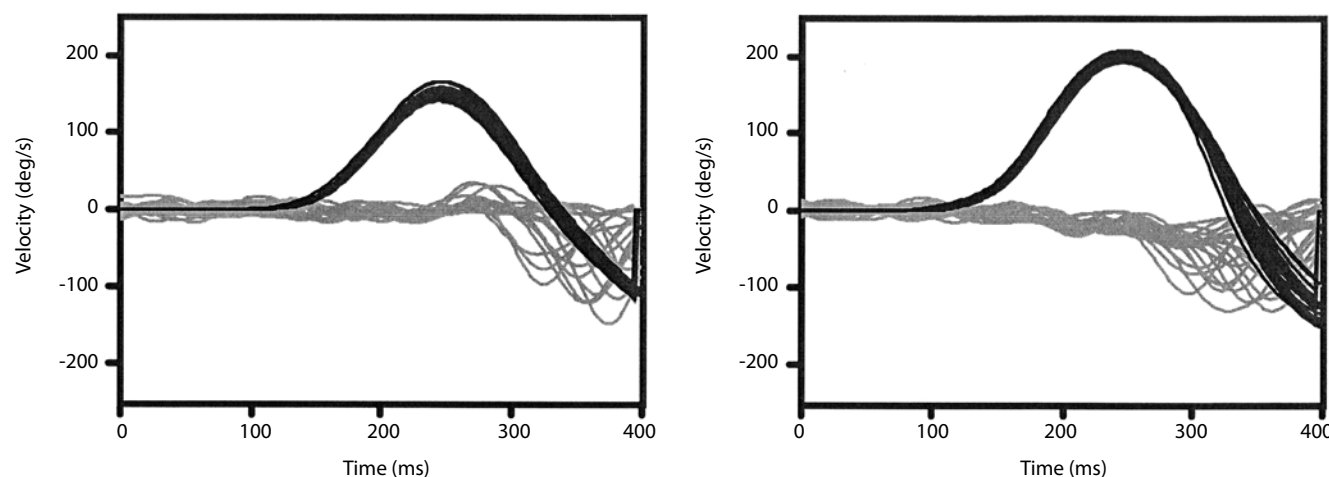


Figure 2. The MHIT result of the patient who had been treated with streptomycin for tuberculosis in the 70s. She later suffered from sudden partial loss of hearing on the left ear. The patient has a profound bilateral vestibular loss with negligible gain, and late refixation saccades are seen toward both direction.

Table 1. Characteristics of patients with bilateral vestibular hypofunction

Patient Number	Sex	Age (years)	PTA (dB) (right/left)	Gain (right/left)	Etiology
1	female	49	112/100	0.20/0.33	Menière's disease
2	female	77	95/85	0.43/0.60	SLLF
3	male	44	7/2	0.31/0.48	Unknown
4	female	65	10/75	0.03/0.19	Ototoxic
5†*	female	49	10/7	0.44/0.31	Ototoxic
6	male	44	115/98	0.55/0.31	Meningitis
7	male	55	deaf/deaf	0.09/0.12	PLLF
8	male	71	deaf/112	0.21/0.24	Menière's disease/VS
9	female	61	12/8	0.14/0.13	Meningitis
10	male	74	93/deaf	0.66/0.35	Menière's disease
11 †*	female	41	deaf/deaf	0.06/0.12	Meningitis
12	male	50	93/98	0.37/0.37	Meningitis
13 †*	female	71	deaf/112	0.04/0.24	SLLF
14	male	80	97/82	0.27/0.34	PLLF
15	female	62	100/deaf	0.10/0.02	MELAS
16	male	67	25/42	0.37/0.18	Meningitis
17	male	72	100/deaf	0.40/0.35	Menière's disease
18	female	43	113/deaf	0.16/0.20	Meningitis
19	female	68	100/108	0.32/0.44	Meningitis
20	male	69	deaf/87	0.28/0.20	PLLF
21	female	43	23/30	0.16/0.24	Unknown
22	female	46	55/3	0.21/0.00	VS/VN
23	female	42	deaf/deaf	0.12/0.23	Menière's disease

†*: the patient is dead; VS: vestibular schwannoma; VN: vestibular neuritis; SLLF: sudden loss of labyrinthine function; PLLF: progressive loss of labyrinthine function; MELAS: mitochondrial encephalopathy with lactic acidosis and stroke-like episodes; PTA: pure-tone average

Vestibular outpatients experienced significantly more oscillopsia compared with that of only 46% in CI recipients ($p=0.048$). The vestibular outpatients experienced also more instability ($p=0.004$), and

their symptoms had more impact on the quality of life ($p=0.012$) than CI recipients.

DISCUSSION

We found 23 patients with BVH confirmed with the MHIT in our vestibular laboratory. During these years, we measured approximately 200 ambulatory vestibular patients with the MHIT, and the BVH was diagnosed in eight patients. The majority consisted of 15 CI patients. The vestibular outpatients were more disabled with their BVH than the patients receiving CI. Most common causes of the BVH were sudden or progressive loss of labyrinthine function including Menière's disease, meningitis, and ototoxicity.

According to earlier studies [3-5], the prevalence of BVH is varying from 28 to 120 per 100,000. Our hospital district has a population of approximately one and a half million, which indicates that at least 400 to 1,800 patients with BVH would reside in this region, if the prevalence would be similar. As the BVH patients do not usually suffer from vertigo, but unsteadiness [9], it is probable that many patients were examined primarily by general practitioner or neurologist and not remitted to us. Therefore, the majority of the patients with BVH in our hospital district may be undiagnosed.

Caloric testing measures low frequencies of vestibular function. It is useful for identifying unilateral loss of vestibular function, but even bilateral loss of caloric responses does not necessarily indicate a complete absence of vestibular function. Head impulse test measures high-frequency band of vestibular function [10]. The MHIT is motorized version of quantitative head impulse tests, which are capable of detecting dysfunction of individual semicircular canal [8]. When both sides of vestibular organs are defective in head impulse test, the eyes are unable to fixate to the target during fast head motion, and refixation saccades are triggered to catch up the target. This phenomenon is reflected directly on the MHIT reports (Figure 1, 2), and it may be experienced as oscillopsia. However, as subjective sensations do not seem to concur with measured level of vestibular loss, rehabilitation, or other interventions in the BVH have to be individualized.

According to Jen [11], in 50% of the cases, the cause of BVH remains unknown. Strupp and Brandt [12] considered that degenerative cere-

bellar diseases would be responsible for 20% of the cases classified as idiopathic. In only two of our patients (8%), the etiology remained unknown, and they had no signs of cerebellar disease. Jen^[11] found ototoxic aminoglycosides, bilateral Menière's disease, and bacterial meningitis being the most common causes of BVH. This finding was close to our findings. Bacterial meningitis can completely abolish vestibular function^[13]. Hearing loss is generally explained by labyrinthitis, and the possible vestibular loss may be missed because of poor general condition. Wiener-Vacher et al.^[13] found bilateral vestibular impairment in 5.7% of their children after bacterial meningitis. In our study, meningitis was also common and encountered in seven patients.

Aminoglycosides have both cochlear and vestibular toxicity with varying clinical appearance. However, vestibular damage may be missed, if vestibular symptoms are not severe, and in addition, hearing loss may dominate as presenting symptom^[14]. Both of our patients with ototoxicity had received systemic aminoglycoside treatment for their severe infections years ago, and their hearing was not decreased during the treatment.

When vestibular function was studied in families with inherited autosomal dominant hearing loss, vestibular symptoms were not a major complaint, although vestibular loss could be detected in clinical evaluations. The lack of self-reported vestibular symptoms may be due to an adaptation to a lack of vestibular information from an early age^[15]. Correspondingly, it is probable that in our adult CI patients, the BVH has progressed quietly, and they were mainly troubled with their hearing loss instead of imbalance. When the vestibular loss develops slowly, the individual learns to manage without proper vestibular input. Instead, older patients with rapidly progressed BVH symptoms have more difficulties, since they have less time and capacity to habituate to the condition.

CONCLUSION

We found 23 patients with the BVH confirmed using MHIT in our clinic. The BVH was found in eight of 198 ambulatory vestibular patients. Approximately, every fourth of CI recipients tested had BVH. The main causes for the BVH were sudden or progressive loss of labyrinthine function including Menière's disease, meningitis, and ototoxic damage. The CI patients experienced less disability from their BVH than the vestibular outpatients.

Ethics Committee Approval: Ethics committee approval was received for this study from the ethics committee of Helsinki University Hospital.

Informed Consent: Written informed consent was obtained from patients who participated in this study.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept - T.P.H.; Design - T.P.H.; Supervision - T.P.H.; Resources - T.P.H.; Materials - H.A., T.P.H.; Data Collection and/or Processing - H.A.,

T.P.H.; Analysis and/or Interpretation - H.L, T.P.H.; Literature Search - H.L, T.P.H.; Writing Manuscript - H.L, T.P.H.; Critical Review - H.L, H.A, T.P.H.

Acknowledgements: We thank M.D, Ph.D. Topi Jutila for preparing the figures.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study has received no financial support.

REFERENCES

1. Neuhauser HK, Lempert T. Vertigo: epidemiologic aspects. *Semin Neurol* 2009; 29: 473-81. [\[CrossRef\]](#)
2. Agrawal Y, Carey J, Della Santina C, Schubert M, Minor L. Disorders of Balance and Vestibular Function in US Adults. *Arch Intern Med* 2009; 169: 938-44. [\[CrossRef\]](#)
3. Guinand N, Boselie F, Guyot JP, Kingma H. Quality of Life of Patients With Bilateral Vestibulopathy. *Ann Otol Rhinol Laryngol* 2012; 121: 471-7. [\[CrossRef\]](#)
4. Della Santina C, Migliaccio A, Hayden R, Melvin TA, Fridman G, Davidovics N, et al. Current and Future Management of Bilateral Loss of Vestibular Sensation - An update on the John Hopkins Multichannel Vestibular Prosthesis Project. *Cochlear Implants Int* 2010; 11: 2-11. [\[CrossRef\]](#)
5. Ward B, Agrawal Y, Hoffman H, Carey J, Della Santina C. Prevalence and Impact of Bilateral Vestibular Hypofunction Results From the 2008 US National Health Interview Survey. *JAMA Otolaryngol Head Neck Surg* 2013; 139: 803-10. [\[CrossRef\]](#)
6. Sprenger A, Wojak JF, Jandl NM, Hertel S, Helmchen C. Predictive mechanisms improve the vestibulo-ocular reflex in patients with bilateral vestibular failure. *J Neurol* 2014; 261: 628-31. [\[CrossRef\]](#)
7. Jutila T, Aalto H, Hirvonen TP. Cochlear Implantation Rarely Alters Horizontal Vestibulo-ocular Reflex in Motorized head impulse Test. *Otol Neurotol* 2012; 34: 48-52. [\[CrossRef\]](#)
8. Hirvonen M, Aalto H, Migliaccio A, Hirvonen TP. Motorized Head Impulse Rotator for Horizontal Vestibulo-ocular Reflex. *Arch Otolaryngol Head Neck Surg* 2007; 133: 157-61. [\[CrossRef\]](#)
9. Kim S, Oh YM, Koo JW, Kim JS. Bilateral Vestibulopathy: Clinical Characteristics and Diagnostic Criteria. *Otol Neurotol* 2011; 32: 812-7. [\[CrossRef\]](#)
10. MacDougall H, McGarvie L, Halmagyi M, Curthoys I, Weber K. Application of the Video Impulse Test to Detect Vertical Semicircular Canal Dysfunction. *Otol Neurotol* 2013; 34: 974-9. [\[CrossRef\]](#)
11. Jen JC. Bilateral vestibulopathy: Clinical, Diagnostic, and Genetic Considerations. *Semin Neurol* 2009; 29: 528-533. [\[CrossRef\]](#)
12. Strupp M, Brandt T. Peripheral vestibular disorders. *Curr Opin Neurol* 2013; 26: 81-9. [\[CrossRef\]](#)
13. Wiener-Vacher S, Obeid R, Abou-Elew M. Vestibular Impairment after Bacterial Meningitis Delays Infant Posturo-motor Development. *J Pediatr* 2012; 161: 246-51. [\[CrossRef\]](#)
14. Selimoglu E. Aminoglycoside-Induced Ototoxicity. *Curr Pharmaceut Des* 2007; 13: 119-26. [\[CrossRef\]](#)
15. Street V, Kallman J, Strombom P, Bramhall N, Phillips J. Vestibular function in families with inherited autosomal dominant hearing loss. *J Vest Res* 2008; 18: 51-8.