

Ocular Motor Function in Patients with Bilateral Vestibular Weakness

Seyyed Amir Hossein Ghazizadeh Hashemi¹, *Sadegh Jafarzadeh², Majid Haddadi Aval², Reza Hosseinabadi³

Abstract

Introduction:

Patients with bilateral weakness (BW) have many difficulties in gaze stability that interfere with their normal function. The aim of this study was to evaluate ocular motor functions in patients with BW to better understand the problem of gaze instability in these patients.

Materials and Methods:

Patients were referred from the Otolaryngology Department for Vestibular Assessment to our clinic between November 2014 and March 2015. We assessed ocular motor function (gaze, saccade, and smooth pursuit) in patients over the age of 18 years with BW, as verified by a caloric test.

Results:

Seventy-eight patients completed all the tests. The mean age of patients was $51.9 (\pm 15.9)$ years, and 47 (60%) were female. Abnormal results were found in five (6.4%), 32 (41%), and seven (9%) patients with respect to gaze, smooth pursuit, and saccade, respectively. There were positive but relatively weak relationships between age and ocular motor results.

Conclusion:

Patients with BW suffer from dizziness and unsteadiness. These patients have abnormal function in ocular motor (especially smooth pursuit) tests. The ocular motor dysfunction is responsible for gaze instability in static positions such as standing.

Keywords:

Gaze stability, Ocular motor, Vertigo.

Received date: 12 Jul 2015 Accepted date: 12 Nov 2015

¹Department of ENT, School of Medicine, Shahid Beheshti University of Medical Sciences, Tehran, Iran.

²Department of Audiology, School of Paramedical Sciences, Mashhad University of Medical Sciences, Mashhad, Iran. ³Department of Audiology, Tehran University of Medical Sciences, Tehran, Iran.

^{*}Corresponding Author:

Tel: 09125583372, E-mail: jafarzadehs@mums.ac.ir

Bilateral weakness (BW) refers to bilateral and abnormally low function of the horizontal semicircular canals, superior vestibular nerve, and vestibular nuclei in a caloric test. It is a common sequela of vestibular disorders such as Meniere's disease, vascular insufficiency, vestibular ototoxicity, and autoimmune disease. Patients with BW experience many problems such as vertigo, imbalance, and dynamic symptoms such as vestibular ocular reflex (VOR) dysfunction, gaze instability, and visual blurring due to oscillopsia. Visual blurring occurs because of deficient VOR during head movement such that the patient relies on compensatory catch-up saccade (1). This situation may occur during reading, walking (2), driving, or any other situation that requires head movement (3).

However, some patients with BW have difficulties in gaze stability even without movement. The ocular head motor abnormality (gaze, saccade, and smooth pursuit) represents abnormal function of the and gaze central vestibular system instability for static and moving objects (4). These tests have a major role in detecting vestibular disorders such as acute vestibular syndrome and differential diagnosis between central and peripheral lesions (5), as well as abnormal results of ocular motor function observed in many vestibular disorders (6).

The aim of this study was to evaluate ocular motor function in patients with bilateral weakness in order to better understand gaze instability problems in these patients, and to use this information for vestibular rehabilitation.

Additionally, because of the dependency of ocular motor function on the visual system and patient's age (7-9), we also measured the relationship between the aging process and ocular motor function that was not related to visual problems.

Materials and Methods

Subjects were referred from the

Otolaryngology Department for Vestibular Assessment to our clinic between November 2014 and March 2015. All subjects had vertigo and/or unsteadiness and vestibular disorders including Meniere's disease, vestibular neuritis, and head trauma, as diagnosed by an otolaryngologist. We performed routine vestibular assessment using electronystagmography (Hortmann, Otometrics, Denmark) for each patient.

The inclusion criteria were age above 18 years and BW in the caloric test (slow phase velocity lower than 12°/s for summation of warm and cold irrigation on each side) (10,11). We excluded all patients with middle-ear abnormalities such as tympanic membrane perforation (confirmed by otoscopy, tympanometry and audiometry), visual disorders (such as cataract) and history of ear or eye abnormality or surgery. All patients were informed about the study process and signed an informed consent form prior to any tests.

Procedure

Otoscopy (Reister, Germany), audiometry (Madsen, Denmark), and 226-Hz probe tone tympanometry (Madsen, Denmark) were performed to rule out middle-ear abnormalities. In the audiometry assessment, patients responded to air conduction and bone conduction stimuli in a sound-treated room using a modified Hughson-Westlake procedure.

Patients were requested not to eat for 6 hours prior to electronystagmography (ENG) testing and to suspend using vestibular suppressant drugs such as Betaserc or any other medicine prescribed for vertigo for 2 days prior to the tests.

The ENG test was performed according to standard protocols for a two-canal recording of horizontal and vertical eye movements. The skin was cleaned and the standard cup electrodes placed on the outer canthus of the eyes for the horizontal canal and up and down of the eye for the vertical canal. Ocular motor tests (gaze, smooth pursuit and saccade) were performed using standard ENG computerized settings, according to the standard protocol (Hortmann, Otometrics, Denmark). Evaluation of optokinetic function requires 90% filling of visual field with stimulus, which is not possible using the ENG/VNG setting. Therefore, in many clinical settings (including our study), this test is not performed.

Data analysis

Data were analyzed using SPSS 19.0 software. Descriptive analysis was used to define the proportion of ocular motor dysfunction in BW patients and the mean (and standard deviation) patient age. Pearson correlation was used to determine the relationship between ocular motor results and age.

Results

Patients

In total, 105 people entered the study, although 27 were subsequently excluded. The mean age of the remaining 78 participants was 51.9 (\pm 15.9) years, and 47 (60%) were female. Abnormal results were found in five (6.4%), 32 (41%) and seven (9%) patients with respect to gaze, smooth pursuit and saccade, respectively. This shows that BW patients have greater difficulty in following objects. Table 1 shows the correlation between age with gaze, smooth pursuit and saccade results.

Table1: the relationship between age and abnormalocular motor responses.

	*	Gaze	Smooth	Saccade
			pursuit	
All patients	Correlation	0.272^*	0.519^{*}	0.307^{*}
	P value	0.016	0.000	0.006
Up to 40 years old.	Correlation	0.109	0.160	0.569^{*}
	P value	0.597	0.435	0.002
Older than 40 years.	Correlation	0.366*	0.469^{*}	0.408^{*}
	P value	0.008	0.000	0.003

*- correlation is significant at the 0.05 level.

The abnormal ocular motor function results had a positive but weak relationship with patients' age. However, this finding is not sufficient to assume that the aging process is a cause of ocular motor abnormalities.

Discussion

The dynamic visual acuity test usually emphasizes gaze stability in dynamic situations such as the turning of the head (12). These tests show gaze instability in different patients with vestibular disorders; however, our study shows that patients with BW have gaze instability even in static positions and when performing simple tasks such as following an object. Patients with BW tend to have more abnormal results in smooth pursuit rather than gaze or saccade, thus the greater difficulty in following a moving object. This may have serious consequences because in everyday activities, rather than a non-laboratory situation, smooth pursuit has a greater role in gaze stability. especially in self-generated movements (13). Ocular motor function and especially smooth pursuit results tend to be abnormal in chronic dizziness (6). We also observed positive, but relatively weak, relationships between ocular motor abnormalities and age. The aging process causes more abnormal results in ocular motor testing. Other studies have also shown similar results (7-9,14). This process usually begins in the fourth decade of life and results in reduced gain for smooth pursuit (7), increased saccade latencies (8), decreased saccade velocity (9), and even reduced saccadic function in the real word (14). However, the aging process is not responsible for all abnormal results in smooth pursuit, especially among younger individuals.

Different areas in the brain stem, central nervous system, and visual and vestibular pathways are responsible for normal ocular motor functions (15,16). Therefore, the abnormality in ocular motor function may stem from the visual or vestibular system control and Postural (17). Postural development are related to ocular motor function (18), and older patients usually develop imbalance and gait performance related to vestibular abnormality and gaze instability (19). By ruling out visual abnormality, we showed that ocular motor dysfunction, and particularly abnormal results of smooth pursuit, coexist with bilateral vestibular weakness; therefore, for diagnosis of vestibular disorders, a complete history and multiple examinations of eye movement are required (20).

BW affects gaze stability. Gaze instability in a static situation such as standing could result in an imbalance, and has serious adverse effects on patients' quality of life (21).

Conclusion

Patients with BW suffer from dizziness and unsteadiness. These patients have abnormal function in ocular motor tests. Ocular motor dysfunction is responsible for gaze instability in static positions such as standing.

Acknowledgements

The authors are grateful to the central clinic of vertigo and balance rehabilitation for their instrumental support.

References

1. Ramaioli C, Colagiorgio P, Saglam M, Heuser F, Schneider E, Ramat S, et al. The effect of vestibulo-ocular reflex deficits and covert saccades on dynamic vision in opioid-induced vestibular dysfunction. PloS one. 2014;9(10):e110322.

2. Guinand N, Pijnenburg M, Janssen M, Kingma H. Visual acuity while walking and oscillopsia severity in healthy subjects and patients with unilateral and bilateral vestibular function loss. Archives of otolaryngology head and neck surgery. 2012;138(3):301-6.

3. Ward BK, Agrawal Y, Hoffman HJ, Carey JP, Della Santina CC. Prevalence and impact of bilateral vestibular hypofunction: results from the 2008 US National Health Interview Survey. JAMA otolaryngology-head & neck surgery. 2013; 139(8):803-10.

4. Huh YE, Kim JS. Bedside evaluation of dizzy patients. Journal of clinical neurology (Seoul, Korea). 2013;9(4):203-13.

5. Kattah JC, Talkad AV, Wang DZ, Hsieh YH, Newman-Toker DE. HINTS to diagnose stroke in the acute vestibular syndrome: three-step bedside oculomotor examination more sensitive than early MRI diffusion-weighted imaging. Stroke. A journal of cerebral circulation. 2009;40(11):3504-10.

6. Oh SY, Kim DH, Yang TH, Shin BS, Jeong SK. classification and neuro-vestibular Clinical evaluation in chronic dizziness. Clinical neurophysiology: official journal of the International Federation of Clinical Neurophysiology. 2015;126(1):180-6.

7. Ross RG, Olincy A, Harris JG, Radant A, Adler LE, Compagnon N, et al. The effects of age on a smooth pursuit tracking task in adults with schizophrenia and normal subjects. Biological psychiatry. 1999;46(3):383-91.

8. Klein C, Fischer B, Hartnegg K, Heiss WH, Roth M. Optomotor and neuropsychological performance in old age. Experimental brain research. 2000;135(2):141-54.

9. Irving EL, Steinbach MJ, Lillakas L, Babu RJ, Hutchings N. Horizontal saccade dynamics across the human life span. Investigative ophthalmology & visual science. 2006;47(6):2478-84.

10. Roeser R, Valente M, Hosford-Dunn H. Audiology diagnosis, second edition, new York: thieme, . 2007, Page 551.

11. Katz J, chasin M, English K, Hood L, Tillery K. Handbook of clinical audiology, seventh edition, Philadelphia, Wolters Kluwer. 2015, page 414.

12. Herdman SJ, Tusa RJ, Blatt P, Suzuki A, Venuto PJ, Roberts D. Computerized dynamic visual acuity test in the assessment of vestibular deficits. The American journal of otology. 1998;19(6):790-6.

13. Niemann T, Lappe M, Buscher A, Hoffmann KP. Ocular responses to radial optic flow and single accelerated targets in humans. Vision research. 1999;39(7):1359-71.

14. Dowiasch S, Marx S, Einhauser W, Bremmer F. Effects of aging on eye movements in the real world. Frontiers in human neuroscience. 2015;9:46.
15. Buttner-Ennever JA, Buttner U. Neuroanatomy of the ocular motor pathways. Bailliere's clinical neurology. 1992;1(2):263-87.

16. Tilikete C, Pelisson D. Ocular motor syndromes of the brainstem and cerebellum. Current opinion in neurology. 2008;21(1):22-8.

17. Strupp M, Hufner K, Sandmann R, Zwergal A, Dieterich M, Jahn K, et al. Central oculomotor

Ocular Motor Function in Patients with Bilateral Vestibular Weakness

disturbances and nystagmus: a window into the brainstem and cerebellum. Deutsches Arzteblatt international. 2011;108(12):197-204.

18. Ajrezo L, Wiener-Vacher S, Bucci MP. Saccades improve postural control: a developmental study in normal children. PloS one. 2013; 8(11):e81066.

19. Whitney SL, Marchetti GF, Pritcher M, Furman JM. Gaze stabilization and gait performance in vestibular dysfunction. Gait & posture. 2009; 29(2): 194-8.

20. Strupp M, Brandt T. Diagnosis and treatment of vertigo and dizziness. Deutsches Arzteblatt international. 2008;105(10):173-80.

21. Guinand N, Boselie F, Guyot JP, Kingma H. Quality of life of patients with bilateral vestibulopathy. The Annals of otology, rhinology, and laryngology. 2012;121(7):471-7.