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Brief communication (Original)

Retrospective review of oscillopsia: etiologies and clinical characteristics

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Background: Oscillopsia can result from dysfunction involving the vestibuloocular reflex (VOR). Few studies have detailed the full spectrum of clinical characteristics and laboratory findings of this disabling condition.
Objectives: To review causes and clinical characteristics of oscillopsia.
Methods: Retrospective review of clinical records of a series of patients with oscillopsia from the University Health Network Multidisciplinary Neurotology Clinic.
Results: Review of 109 patients with oscillopsia showed near equal sex distribution and a mean age of 54.9 years (standard deviation 15.7). The most common peripheral vestibular causes were ototoxicity (19.3%) and Meniere's disease (10.1%). The most common central cause involved cerebellar degeneration (7.3%). Only 43.1% complained of vertigo at any time. Head impulse testing demonstrated bilateral refixation saccades in 70% of patients, while over 95% had a loss of dynamic visual acuity ≥5 lines on LogMAR chart testing. Some 38.8% of patients demonstrated normal caloric responses, whereas only 26.5% exhibited evidence of a bilateral caloric loss. Over 50% of patients maintained otolithic function as demonstrated by cervical vestibular evoked myogenic potential testing. Most patients demonstrated low VOR gains bilaterally on magnetic scleral search coil testing.
Conclusions: A wide spectrum of clinical characteristics was identified in patients with oscillopsia. It is important for the physician to perform a thorough history, physical examination (including head impulse and dynamic visual acuity), and laboratory investigations.

Keywords: Bilateral vestibular loss, magnetic scleral search coil, oscillopsia, vestibuloocular reflex

Abbreviations
cVEMP = cervical vestibular-evoked myogenic potential
DVA = dynamic visual acuity
ENG = electronystagmography
MSSC = magnetic scleral search coil
vHIT = video head impulse test
VOR = vestibulo-ocular reflex

Oscillopsia is the perception that visual scenes are moving, usually during active head movement or locomotion. Vertical movement of one's surroundings during walking is often referred to as "bobbing oscillopsia" [1]. In a case series of 15 patients with bobbing oscillopsia from gentamicin ototoxicity, Ramsden and Ackrill reported that patients complained of difficulty focusing on objects when walking and "bouncing" with each foot-step [2]. Some authors have used the term oscillopsia somewhat differently to describe the illusion of visual movement in disorders associated with acute onset spontaneous nystagmus. However, more often it is a reflection that compensatory head generated eye movements do not adequately match, with opposite head movement because of an impairment involving the vestibuloocular reflex (VOR) [3]. Head movement-dependent oscillopsia is therefore described as illusory movement of the visual world that occurs during head movement [4]. In the present work, oscillopsia is defined as the symptom of moving visual scenes during head movement.

Visual acuity requires several mechanisms to optimize the compensatory eye to head movement that involves not only the VOR, but also visual tracking, motor preprogramming, prediction, and mental set. All of these mechanisms interact synergistically. However,

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there are limitations in their capacity; especially during high frequency, high velocity, or accelerative head movements [5]. The VOR stabilizes the visual image on the retina by generating conjugate eye movements in an equal, but opposite direction to head movement. This is vitally important for gaze stabilization during high frequency, high velocity, or accelerative head movement. Abnormalities in the VOR can subjectively cause visual blurring and dizziness while the head is moving because of so-called "retinal slip" [6, 7]. Some patients with a bilateral vestibular loss seem to be denied a diagnosis of oscillopsia, whereas others with normal VOR gains have been labeled as having oscillopsia [8].

There are many heterogeneous causes for oscillopsia arising from a bilateral vestibular loss, including aminoglycoside ototoxicity and autoimmune inner ear disease [9-13]. Oscillopsia may also arise from central nervous system pathology such as an Arnold–Chiari malformation, demyelinating polyneuropathy, or cerebellar lesions or degeneration [14-16].

Few studies appear to have specifically reviewed the clinical characteristics of patients with oscillopsia. In the present study we sought to review the causes, clinical characteristics, and investigative findings in a series of patients with oscillopsia.

Materials and methods

After this research was approved by the University Health Network Research Ethics Board (REB 14-8093-BE, September 17, 2014), we examined the clinical records of patients with perception of moving visual scenes during head movement or locomotion who were patients referred to the University Health Network, University of Toronto, Multidisciplinary Neurotology Clinic, Canada. The clinical records of 129 patients in the database with oscillopsia were identified. We excluded 20 patients because of incomplete data. Ultimately data from complete or near complete clinical records from 109 patients were included and analyzed. Written informed consent for data collection and publication was obtained from all participants.

The symptoms, signs, and investigations were evaluated by multiple parameters that included a history of vertigo, hearing loss, and tinnitus. Physical examination included clinical otoscopy, a head impulse test, a head-shake test, and a Dix–Hallpike test. Horizontal dynamic visual acuity (DVA) testing of 88 patients was performed by LogMAR chart testing. The reference level at rest was compared to the smallest legible line read during horizontal head shaking. A loss of lines ≥5 was considered significant. Oculomotor testing evaluated ocular fixation, pursuit and saccade eye movements. All patients were assessed for spontaneous and gaze-evoked nystagmus along with the ability to suppress the VOR. Cerebellar testing evaluated both midline and hemispheric function. Romberg and tandem gait testing were evaluated in almost all patients.

Conventional audiometry was performed and the pure-tone air conduction average at 0.5, 1.0, 2.0, 3.0 kHz was calculated. Electronystagmography (ENG) or video nystagmography with bithermal caloric testing was performed in 94 patients and the caloric response was calculated using Jongkee's formula. Air caloric testing was performed in 4 patients. In addition, cervical vestibular-evoked myogenic potentials (cVEMP) and magnetic scleral search coil (MSSC) testing was performed in some patients. The MSSC was used primarily to detect vestibular dysfunction; especially during high frequency, high velocity, or accelerative head movement. The test involved placing a contact lens with an embedded copper coil in one eye of the tested individual who then sat in the center of an electromagnetic field (CNC Engineering, Seattle, WA, USA). A series of head thrust tests would be performed in the horizontal plane while the patient fixating on the target with an average of 20 rotations to either side. The eye movements were recorded with the coil, while the head movements were measured by a search coil mounted on a dental bite gripped by the teeth. A wide range of peak velocities between 50–500°/s were tested. VOR gain (defined as the corresponding mean of peak eye velocity/mean of peak head velocity) was analyzed digitally using a computer [17]. Data were collected and evaluated using IBM SPSS Statistics for Windows, version 20 (IBM Corp, Armonk, NY, USA). All variables were analyzed and reported as percentage.

Results

We identified 109 patients with oscillopsia. The sex distribution was near equal with 53 men and 56 women. The mean age of patients was 54.9 years (standard deviation 15.7). The etiology of the oscillopsia was identified in 73% of patients. A peripheral vestibular etiology was identified in 50%, a central etiology in 20%, and a mixed peripheral and

central (cerebellar ataxia with bilateral vestibulopathy) in 3%. Etiology was undiagnosed in 30 patients (28%) (**Table 1**). During the clinical course of their disease 11% of patients developed benign paroxysmal positional vertigo.

The most common peripheral vestibular causes were related to drug ototoxicity (gentamicin in 20 cases and chemotherapy in 1); followed by Ménière's disease and recurrent vestibulopathy. Other peripheral vestibular causes included vestibular neuritis, poststapes surgery, and trauma. The most common central etiology involved cerebellar degeneration or predominant vestibulocerebellar degeneration. Other central causes included multiple sclerosis, tumor, stroke, and meningitis or encephalitis. Miscellaneous causes included polyneuropathy, myelopathy, transverse myelitis, and multisensory deficits. One case involved a patient with a left lateral semicircular canal fistula and prior right labyrinthectomy (**Table 1**).

Most patients had a normal otoscopic examination; except for 6 who had previous mastoid surgery (n = 3) or a perforation of the tympanic membrane (n = 3). The head impulse test demonstrated bilateral overt saccade refixations in 70.1% (n = 75); was normal in 21.5% and unilaterally positive in 8.4%.

Almost 80% (n = 85) had no clinical evidence of postheadshake nystagmus. Nearly 95% of patients had a significant loss of DVA. Normal VOR suppression was found in 85%. Almost all patients with abnormal VOR suppression had evidence for central pathology (**Table 2**).

Abnormal ocular movements including saccadic pursuit were demonstrated in 21.3% of patients, dysmetric saccades in 14.8%, and gaze-evoked nystagmus in 15.7%, and were usually found in patients with central pathology. Normal finger-to-nose testing was noted in 90.7%. There was no evidence for dysdiadochokinesia in 96.3% (3 patients had positive left dysdiadochokinesia, 1 patient had positive bilateral dysdiadochokinesia). Up to 47.2% of patients demonstrated an ataxic tandem gait, but only 10.2% had an abnormal Romberg test (**Table 3**).

Table1. Causes of oscillopsia

Cause of oscillopsia	**n**	**%**
Drug ototoxicity	21	19.3
Ménière's disease	11	10.1
Cerebellar/primary vestibulocerebellar degenerations	11	10.1
Recurrent vestibulopathy	9	8.3
Vestibular neuronitis	4	3.7
Multiple sclerosis	4	3.7
Post stapes surgery	3	2.8
Post trauma	3	2.8
Stroke	3	2.8
Tumor	3	2.8
Meningitis/encephalitis	2	1.8
Miscellaneous	5	4.6
Unknown	30	27.5
Total	109	100

Table 2. Neurotological examination findings

Neurotological examination	**n (N = 107)**	**%**
Abnormal otoscopy	6	5.5
Bilateral positive head thrust	75	70.1
Unilateral positive head thrust	9	8.4
Normal head thrust	23	21.5
Post head-shake nystagmus	22	20.6
Abnormal VOR suppression	16	14.9

Table 3. Ocular movement and cerebellar examination findings

Ocular motor /cerebellar examination	n (N = 108)	%
Saccadic pursuit	23	21.3
Dysmetric saccade	16	14.8
Gaze-evoked nystagmus	17	15.7
Dysmetria finger to nose	10	9.3
Dysdiadochokinesia	4	3.7
Positive Romberg	11	10.2
Ataxic tandem gait	51	47.2

Pursuit eye movements on ENG was similarly identified on physical examination in 28% and evidence of gaze-evoked nystagmus on ENG in 20%. Abnormal saccade eye movements on ENG testing were found in only 5%. Normal caloric responses were identified in 38 patients (38.8%, n = 98). A unilateral caloric reduction, which was defined as excitability difference more than 15% from Jongkees' formula, was identified in 34 patients (34.7%). A bilateral vestibular loss was found in 26 (26.5% absent response or sum of nystagmus generated from bithermal testing < 20°/s) (**Figure 1**).

Of the 66 patients with cVEMP testing, bilateral absent responses were identified in 9 (13.6%), whereas 23 (34.8%) had an absent unilateral response and 33 (50.1%) had responses present bilaterally. MSSC testing was performed in 35 patients. Nine patients (25.7%) demonstrated normal VOR gains, which was defined as VOR gain >0.8, 21 patients (60%) demonstrated bilaterally low gains, and 5 patients (14.3%) had a unilateral low VOR gain (**Figure 2**). Ninety-three patients underwent conventional audiometry. The median of pure-tone average was 16.25 decibels. Eleven patients had a unilateral profound hearing loss and 2 were deaf in both ears.

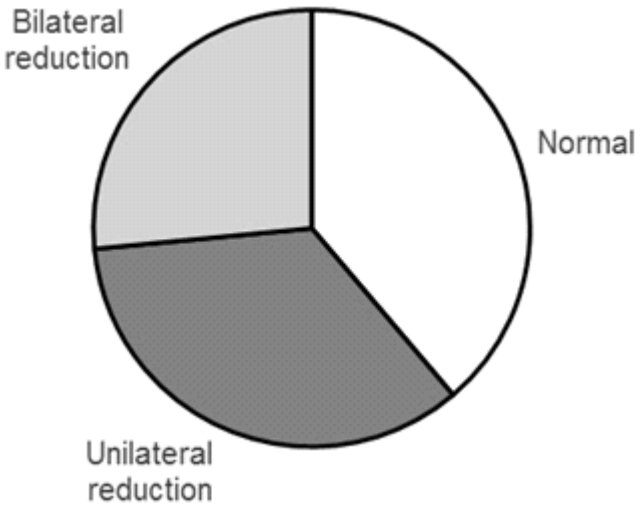

Figure 1. Caloric test results showed bilateral reduction of response in 26 patients (26.5%), unilateral reduction in 34 (34.7%), and normal caloric response in 38 (38.8%; N = 98)

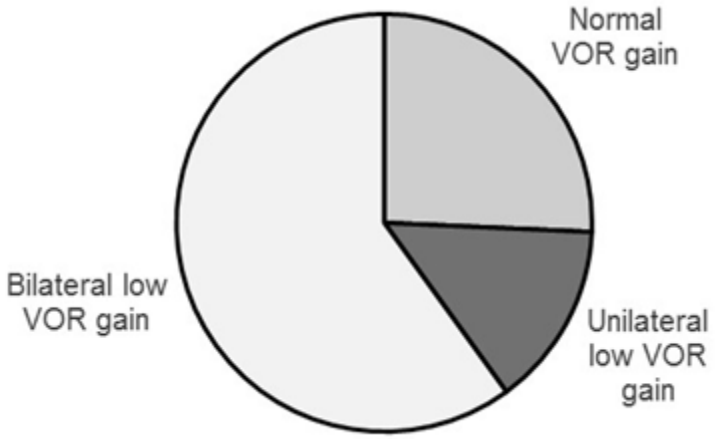

Figure 2. Magnetic scleral search coil test results from 35 patients demonstrated bilateral low vestibuloocular reflex (VOR) gain in 21 (60%), unilateral low VOR gain in 5 (14.3%), and normal VOR gain in 9 (25.7%).

Discussion

Oscillopsia is an uncommon complaint and to our knowledge only a few publications have extensively reviewed the etiologies and its clinical characteristics. This study reviewed the clinical records of patients who complained of oscillopsia and were assessed in a multidisciplinary neurotology clinic in Toronto, Canada. There were various causes for their oscillopsia: peripheral vestibular (49.5%), central (20.2%), or mixed peripheral, and central (2.7%) pathologies. Up to 27.5% had unknown etiology. The most common causes for oscillopsia were aminoglycoside ototoxicity, which was also the most common cause of bilateral vestibular loss. Oscillopsia is experienced by 44%–50% of patients with a bilateral vestibular loss [10, 17-19]. Moving visual scene during head movement results from insufficient VOR response. The pathology could be in peripheral or central pathways and our study supported that peripheral vestibular and central lesions can cause patients to complain of oscillopsia.

Patients with Ménière's disease and recurrent vestibulopathy typically experience attacks of vertigo. Some patients with a central etiology also experienced vertigo although not frequently. None of the patients with an ototoxic drug exposure complained of vertigo. These findings were similar to those reported by Ahmed et al. [18] who reviewed 103 patients with gentamicin ototoxicity. Patients with vestibular ototoxic typically present with imbalance, oscillopsia or both. Black et al. [20] reviewed 33 patients with gentamicin ototoxicity. All patients had complaints of disequilibrium and ataxia and 32 of 33 patients complained of oscillopsia. Other common complaints from patients in this series were tinnitus, cognitive dysfunction, nausea, and visual sensitivity. By contrast with the study by Ahmed et al. [18], a third of patients also had complaints of vertigo. It is possible that if the patients had prior vestibular asymmetry, they might experience vertigo because of a change in vestibular function.

In the present study, abnormal otoscopic findings were identified in 6 patients. Two patients with a unilateral tympanic membrane perforation had a history of topical gentamicin use on that side. However, there was no other explanation from their history for the oscillopsia. One patient who underwent mastoidectomy had a left lateral semicircular canal fistula and had a previous right labyrinthectomy resulting in a bilateral vestibular loss. The other 3 patients with abnormal otoscopic findings were also diagnosed to have evidence of cerebellar degeneration, Ménière's disease, and vestibular neuritis.

The head impulse test is a useful bedside screening examination of vestibular function [19, 20]. Saccade refixation occurs after a quick horizontal head rotation ipsilateral to semicircular canal dysfunction. These findings are also referred to as overt or "catch-up" saccades and can be detected by visual observation [21]. Bilateral refixation saccades were an important characteristic of oscillopsia and were identified in 70.1% of patients in the current study. It is important to recognize that a normal head impulse test does not definitely imply that the VOR is normal. Covert saccades can occur during active head rotation and may be undetectable by observation causing

misinterpretation [20, 22, 23]. Video head impulse testing (vHIT) has provided a utilitarian means for objective measuring VOR gain and whether overt or covert saccades, or both, are present. vHIT overall appears generally comparable to MSSC at head velocities up to 180°/s [21]. Six of our 23 patients with normal clinical head impulse testing underwent MSSC. Of these patients, only one demonstrated normal findings, while the others all demonstrated low VOR gains and decreased caloric responses. Of the 23 of 75 patients with a bilateral positive head impulse test who underwent MSSC testing, 15 demonstrated bilateral low VOR gains, 3 patients demonstrated a unilateral low VOR gain, and an unusual 5 patients demonstrated normal VOR gain. MSSC testing is generally considered the best for high frequency/velocity/acceleration VOR evaluation. An abnormal head impulse test with normal MSSC testing may be consistent with a false positive head thrust result [17].

DVA was assessed during horizontal or vertical head movements [5]. A visual acuity loss of >5 lines during active head movement relative to static stage testing was observed consistently in those with a bilateral vestibular loss [24]. In the present study, 95% of patient loss with at least a 5 line DVA and bilateral low VOR gains, confirmed these findings. While the bedside DVA test seems to be highly specific, it is not highly sensitive [25]. Some patients with a unilateral vestibular loss or with high myopia might be expected to have problems reading during head movement [26.] Limitations of bedside DVA testing include such variables as the velocity and frequency of head movement and memorization of content [27].

Abnormal VOR suppression, dysmetric finger-to-nose and dysdiadochokinesia were present only in patients with central pathology. Almost half of patients, regardless of whether a peripheral vestibular or central etiology was present, demonstrated an ataxic tandem or unsteady gait. Similar to previous findings, patients with unsteadiness in their gait were typically found to have a bilateral vestibular loss or cerebellar disease [10, 28].

Bithermal caloric testing is frequently used as a reference test for vestibular function. There are many limitations: such as being only able to test one ear. Unpredictable thermal energy transfer may cause both false positive and false negative results, and correlates primarily with low frequency vestibular function [17, 29]. Normal caloric responses were demonstrated in 38 of 98 patients (38.8%). Our findings confirmed these limitations. Relevant to the discussion is the recognition that a normal ENG does not always indicate normal vestibular function; normal caloric responses can exist with vestibular deficits identified on MSSC testing [17, 23].

cVEMPs are biphasic, short latency, electrical changes measured at the sternocleidomastoid muscle resulting from sound stimulation. Many studies have demonstrated their efficacy in the assessment of saccular function [30]. As the saccule predominantly detects movement along the vertical plane, a saccular defect could theoretically be the cause of walking-induced vertical oscillopsia. It is not known whether saccular or vertical semicircular canal dysfunction is the predominant cause for vertical oscillopsia [31]. From this study only 9 of 66 patients had bilateral absent cVEMP responses with 3 of them also having bilateral low VOR gains on MSSC testing. The relative sparing of saccular function suggests greater pathological involvement of semicircular canal function in an involved individual. It suggests that the saccule generated VOR alone is probably insufficient to maintain vision. Vertical oscillopsia might also be caused by inadequate vertical semicircular canal VOR compensation in the event of a loss involving these receptors [31]. In patients with a bilateral vestibular loss, saccular function was less affected than function in the horizontal semicircular canals by analogy [32, 33].

MSSC is considered a powerful tool for evaluating high frequency/velocity/acceleration vestibular function [17]. Seven of 35 (20%) patients had normal MSSC testing although one patient had ocular flutter, which may have affected test results. Most of them had unknown etiology or central lesions, which confirms that oscillopsia can result from peripheral or central pathology.

Conclusions

Oscillopsia (moving visual scene during head movement) can arise from peripheral vestibular, central, or mixed pathologies. In 30 patients (27.5%) a formal diagnosis for oscillopsia was not forthcoming in this series. However, rarely does oscillopsia occur without some evidence for pathology involving the vestibular system. A wide spectrum of clinical characteristics was identified in patients with oscillopsia. For diagnosis, it is important for the physician to perform a thorough history, physical

examination (especially head impulse and dynamic visual acuity), and other investigations (particularly vHIT or MSSC). The relative maintenance of otolithic function may largely explain the ability to maintain relatively normal balance in our patient cohort.

Conflict of interest statement

The authors declare that there is no conflict of interest in this research.

Authors' contributions

PC acquired, analyzed, and interpreted data, and drafted the manuscript. PS analyzed and interpreted data, and helped to draft and revise the manuscript. DP contributed to the conception and design, analyzed and interpreted data, and critically revised the manuscript. JR contributed to conception and design, and revised important intellectual content. All authors read, approved, and take responsibility for the final manuscript.

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