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# Can Oculomotricity Be Altered in Patients with Tinnitus Only? A Preliminary Study

**Raquel Mezzalira,[1,2] Márcia Maria do Carmo Bilécki,[2] Breno Pardini Gontijo,[2] James E. Slusser,[2] George Eduardo Camare Bernarde,[1,2] and Oscar Antônio Queiroz Maudonnet[1,2]**

[1] *Department of Otorhinolaryngology–Head and Neck Surgery, State University of Campinas, and* [2] *Penido Burnier Institute, São Paulo, Brazil*

***Abstract:*** The study of oculomotricity is performed by evaluating three systems: saccadic ocular movements (SOMs), optokinetic nystagmus (OKN), and smooth pursuit eye movements (SPEMs). Our aim was to study oculomotricity in patients with a complaint of only tinnitus and to compare it with the value of our control group. We studied the SOMs, OKN, and SPEMs in 25 patients complaining only about tinnitus and in 35 normal adults and compared the results. The data analysis showed a significant difference in the value of the SOMs and SPEMs between the two groups. Sensorineural tinnitus can originate in the organ of Corti, in the cochlear nerve, or in the auditory pathways of the central nervous system. The auditory cortex connects with visual areas and with the superior colliculus. The latter structure is involved in the origin of SOMs and OKN. In our study, we found an increased delay in saccadic tests. In the SPEMs, we observed an increase in the degree of distortion, and a reduction in the gain. This outcome is in accordance with the literature. However, we detected a few alterations in the OKN, and this finding is in partial agreement with the studies analyzed. Alterations in oculomotricity can indicate involvement of the central nervous system in patients with a complaint of only tinnitus.

***Key Words:*** oculomotricity; optokinetic nystagmus; saccadic ocular movements; smooth pursuit eye movement; tinnitus

Tinnitus is a complaint in 80% of otolaryngologists' patients, especially in those who present with some kind of hearing problem, and can be so severe that it becomes incapacitating [1]. Tinnitus can be defined as a hearing illusion, a sonorous sensation without an external stimulating source. It can be the unique or the primary symptom in some diseases, potentially affecting patients' health and, indirectly, the health of their families. Thus, a correct diagnosis and effective treatment are required [2,3].

Sensorineural tinnitus may be peripheral when it originates from the organ of Corti or the cochlear nerve and central when it originates from the central nervous system (CNS) auditory structures. Tinnitus may be a symptom in many diseases. Therefore, a complete medical and audiological evaluation is an important initial step in the diagnostic approach. Commonly used tests are pure-tone and speech audiometry, imitanciometry, electrocochleography, otoacoustic emissions, and auditory brainstem response. However, the inner ear is a distinct complex organ, and the cochlear and vestibular parts work together, thereby achieving cochleovestibular unity. Thus, existing cochlear alterations can affect the posterior labyrinthine structures.

Many patients with tinnitus display abnormal vestibular test results even in the absence of vertigo or other balance disorders. This should alert specialists to the necessity for undertaking a complete evaluation of the vestibular and auditory systems in patients with tinnitus, owing to the anatomical and functional proximities of these systems, which supports the use of vestibular tests in patients with hearing disorders [4–7].

Reprint requests: Dr. Raquel Mezzalira, Vitoriano dos Anjos, 455, apto 12, Ponte Preta, Campinas–São Paulo, CEP:13041-317, Brazil. Phone: (19) 3739-1020; (19) 9798-7424; Fax: (19) 3232-4553; E-mail: raquelmezzalira@uol.com.br

Does this functional relation also exist between the auditory and the central vestibular pathways? Bergenius [8] observed oculomotricity alterations in perhaps one-half of the patients with deafness of retrocochlear origin. Wall et al. [9] described the triggering of tinnitus during oculomotricity tests in patients who underwent surgical removal of cerebellopontine-angle tumors with sectioning of the eighth cranial nerve.

Claussen et al. [10] studied the activity of cerebral electric mapping in tinnitus patients subjected to the evoked potential vestibular test. These authors discussed the possibility that tinnitus was being caused by some form of abnormal spontaneous CNS activity. Shulman and Goldstein [11] suggested a common final pathway for tinnitus: mainly the temporal and frontotemporal areas in severe cases.

The study of oculomotricity is performed by evaluating three systems: saccadic ocular movements (SOMs), optokinetic nystagmus (OKN), smooth pursuit eye movements (SPEMs). This overall action allows stabilization of one's field of vision during the various movements to which an individual is subjected.

The term *saccadic* is defined as a quick eye movement of high velocity (on the order of hundreds of degrees per second), the purpose of which is to maintain the image over the fovea [12,13]. Controversies persist regarding neural mechanisms involved in the execution of SOMs. However, recent studies indicate that the generator of horizontal movement lies in the medulla-point reticular formation, around the abducent nucleus, whereas the generator of vertical movement originates in the medial rostral reticular formation to the oculomotor nucleus [14,15].

The movement of a visual scene through the field of vision evokes an involuntary and conjugated ocular movement denoted as OKN. The optokinetic function operates with visual signals from the fovea and retinas [12]. The mechanism of this function is also controversial, but it is believed that two distinct neural pathways are responsible for its control: the subcortical pathway, with initial processing in the optic tract nucleus but with input by the visual cortex, and another more recent pathway related to the SPEMs (voluntary).

The SPEMs comprise the oculomotor control mechanism that moves the eyes to stabilize the targeted image over the retina. The neural path involved in these movements passes by the occipital cortex, temporal cortex, parietal cortex, corpus callosum, pons, and cerebellum [16].

## ABNORMALITIES IN OCULOMOTRICITY

Oculomotor tests have been used in the study of certain diseases that compromise the CNS and that have tinnitus as one of their manifestations. Inhibition of OKN and alteration of SPEMs were described in one case of aqueduct stenosis with associated hydrocephaly [17]. In Arnold-Chiari malformation, there is reported damage to SPEMs and inhibition of OKN due to compression of the herniated cerebellum [18].

Dyssymmetry in SOMs and damage to the OKN and SPEMs were reported in Friedreich's ataxia and cerebellar and olivopontine atrophy [19–21]. Oculomotor alterations due to demyelination in multiple sclerosis are well-known. The SPEMs and OKN are compromised, and the latency of SOMs is increased owing to internuclear ophthalmoplegia [22,23].

Ohki et al. [24] described reduction in the velocity of the SOMs and alterations in OKN and SPEMs in patients with early-stage amyotrophic lateral sclerosis. Various severe oculomotricity disturbances were reported by Breuer et al. [25] in patients with progressive supranuclear paralysis.

In Parkinson's disease, well-known alterations occur in oculomotricity; however, a study by Degl'Innocenti et al. [26] reported the improvement of some oculography characteristics in phase-off, contrary to clinical evidence, suggesting that the oculomotor system is functionally separated from the system that controls arm movements. Oepen et al. [27] described reduction in the SOMs and OKN speed and alterations in SPEMs in patients with Huntington's chorea.

Advanced cases of essential tremor might display a compromise of cerebellar structures involved in ocular movement control. This fact was evidenced in studies by Helmchen et al. [28] on the difficulty of initiating SPEMs. In tension headache, reductions of velocity and amplitude of SOMs and in the gain of SPEMs have been reported [29].

Alterations in the three oculomotricity elements have been reported in schizophrenia, suggesting that the lesion site is over the brainstem [30,31]. Konrad et al. [32] reported in patients with cerebrovascular diseases a lower gain in SPEMs and a higher latency and reduction of the precision of SOMs.

Josefowicz-Korczynska et al. [33] used oculomotricity tests in evaluating patients with tinnitus due to vertebrobasilar insufficiency, finding alterations in SPEMs and SOMs. In presbyvertigo, oculomotricity tests are useful to suggest the compromised site of age-related changes concerning disturbances in oculovestibular integrations. One alteration is OKN asymmetry [34]. Individuals exposed to toluene can manifest increased SOM velocity due to neurotoxicity, as described by Hyden et al. [35].

Some infectious diseases (human immunodeficiency virus [HIV] and syphilis) might cause CNS dysfunction and provoke oculomotricity alterations [36,37]. However, none of theses studies points toward tinnitus as an isolated complaint or, at least, as a main symptom.

The purpose of this preliminary study was to evaluate

SOMs, OKN, and SPEMs by measurements obtained with oculography in patients with tinnitus but without any other cochleovestibular symptom. We sought to analyze whether differences are evident between the results of the studied group and those of the control group consisting of individuals without cochleovestibular symptoms.

## PATIENTS AND METHODS

The study group was composed of 25 patients (ages 15–72 years; 13 men, 12 women) having tinnitus complaints but no other cochleovestibular symptoms. We chose for the control group 35 adults (ages 22–70 years; 10 men, 25 women) without any medical history or physical examination data that might suggest abnormal ocular patterns.

To perform the vestibular examination, we used computerized vestibulometry, an exam consisting of calibration and registration of SOMs, OKN, and SPEMs. The aspects studied were SOM right and left latency and precision, OKN right and left gain, and SPEMs right and left gain and distortion grade. The statistical data analysis was performed with the Student's *t* test to compare the average in both samples [38].

## RESULTS

The data analyses showed significant Student's *t* test results at level $\alpha < 0.05$ to the variables SOM right and left latency and precision and SPEM right and left gain and distortion grade. Table 1 lists the value of mean, standard deviation, and significance in both groups.

## DISCUSSION

The primary auditory cortex is located in the anterior transversal temporal gyrus, but the association areas connect it to the frontal and temporoparietal regions and to the vision and somatesthetic areas [39]. Connections between the auditory cortex and superior colliculus have also been described; this structure is involved in the origin of SOMs and OKN [16]. This anatomical configuration can aid in explaining alterations in the central vestibular system in patients with auditory symptoms.

CNS functional reorganization due to neuroplasticity may also be involved in tinnitus origin. Wall et al. [9] described the rise of tinnitus during oculomotricity tests in patients undergoing removal of a cerebellopontine-angle tumor with section of the eighth nerve. An abnormal interaction between the vestibular nucleus and the cochlear nucleus due to neural sprouting is a probable explanation. Coad et al. [40] and Biggs and Ramsden [41] also observed tinnitus worsening during oculomotor tests and attributed it to CNS neuroplasticity. This idea is consistent with that of Lockwood et al. [42], who described an association between tinnitus and plastic transformations in the central auditory system that occur by the appearance of aberrant auditory pathways.

Regarding SOMs, our study showed an increase in latency in patients with tinnitus only when compared to the control group. This increased SOM latency was described in ataxia, suggesting cerebellar involvement [21]; in multiple sclerosis, owing to medial longitudinal fasciculi injury; in the other brainstem structures functionally involved in the programming of saccades [22]; in Huntington's chorea [27]; and in cerebrovascular diseases [32]. However, Carlsson and Rosenhall [29] evaluated patients with tension headache and did not find alterations in SOM latency and precision. We observed compromise in the right and left SOM latency. We also observed increased precision of SOM and did not find any explanation for this in the literature we analyzed.

In our research, no significant differences were seen between the patient group and control group regarding OKN gain. Konrad et al. [32] studied patients with cerebrovascular diseases and observed that optokinetic responses were not affected. However, our results disagree

**Table 1.** Mean, Standard Deviation, and Significance in Both Groups

| | SOM | | | | OKN | | SPEMS | | |
|---|---|---|---|---|---|---|---|---|---|
| | **RL** | **LL** | **RP** | **LP** | **RG** | **LG** | **DG** | **RG** | **LG** |
| Mean values | | | | | | | | | |
| Tinnitus group | 329.00 | 321.18 | 106.35 | 106.35 | 92.35 | 90.08 | 27.03 | 57.53 | 58.50 |
| Control group | 194.49 | 180.86 | 90.60 | 94.20 | 82.52 | 80.11 | 5.77 | 85.06 | 81.54 |
| Standard deviation | | | | | | | | | |
| Tinnitus group | 177.42 | 189.84 | 29.61 | 20.72 | 34.33 | 40.49 | 11.16 | 18.97 | 18.11 |
| Control group | 41.05 | 32.97 | 12.71 | 12.19 | 12.22 | 12.72 | 6.00 | 17.15 | 18.50 |
| Significance value | 0.007* | 0.008* | 0.049* | 0.036* | 0.351 | 0.418 | 0.000* | 0.001* | 0.002* |

*Significant at $\alpha < 0.05$.
DG = distortion grade; LG = left gain; LL = left latency; LP = left precision; OKN = optokinetic nystagmus; RG = right gain; RL = right latency; RP = right precision; SOM = saccadic ocular movements; SPEMs = smooth pursuit eye movements.

with the majority of the publications, which describe OKN alterations (e.g., asymmetrical responses [43], damage in slow phases [19], and reduction in velocity of fast phase [27]). Some authors have reported unspecified OKN compromise [20,23–25,30,31]. Maybe in enlarging our study population, we will find abnormalities of OKN.

The SPEM research showed an evident increase in distortion grade and impairment gain values in patients with tinnitus only, and the literature described the same [19–24,27–30,32,36,37,44]. All alterations described suggested that CNS disruption and oculography are essential contributors to proving a topographical diagnosis of the pathology [25]. References to the importance of unilateral or bilateral tinnitus were not found in the literature. However, we did not consider unilateral or bilateral tinnitus important because of crossroads of the auditory pathways.

## CONCLUSION

This preliminary study permits us to conclude that the value of some oculomotricity elements differed between patients with and patients without tinnitus, suggesting that the abnormality in these test results in patients who complain of tinnitus can indicate functional compromise of the CNS. By extension, then, these tests may be useful in evaluating patients who complain of tinnitus only.

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## REFERENCES

1. Pulec JL, Hodell SF, Anthony PF. Tinnitus: Diagnosis and treatment. *Ann Otol Rhinol Laryngol* 87(6–1):821–833, 1978.
2. Tyler RS, Babin RW. Tinnitus. In CW Cummings, JM Fredrickson, LA Harker, et al. (eds), *Otolaryngology–Head and Neck Surgery*. Baltimore: Mosby–Year Book, 1993:3031.
3. Bento RF, Minitti A, Marone SAM. Doenças do Ouvido Interno. In *Tratado de Otologia*. São Paulo: EDUSP, 1998:322–384.
4. Norrè ME. The early detection of the acoustic neurinomas: Otoneurological indications of the radiological investigation. *Acta Otorhinolaryngol Belg* 32(2):136–151, 1978.
5. Seabra JC, Diamantino H, Faria E, Almeida J. Neurootological evaluation of tinnitus. *Int Tinnitus J* 1(2):93–97, 1995.
6. Seabra JC. The medical audiological evaluation of tinnitus patients. *Int Tinnitus J* 5(1):53–56, 1999.
7. Mezzalira R, Maudonnet O, Pereira RG, Ninno JE. Contribution of otoneurological evaluation to tinnitus diagnosis. *Int Tinnitus J* 10(1):65–72, 2004.
8. Bergenius J. Vestibular findings in sensorineural hearing disorders: Results of caloric, oculomotor and hearing tests in 205 patients with unilateral hearing dysfunction. *Acta Otolaryngol* 99:83–94, 1985.
9. Wall M, Rosemberg M, Richardson D. Gaze-evoked tinnitus. *Neurology* 37(6):1034–1036, 1987.
10. Claussen C-F, Schneider D, Koltchev C. On the functional state of central vestibular structures in monaural symptomatic tinnitus patients (BEAM-VbEP Study). *Int Tinnitus J* 1(1):5–12, 1995.
11. Shulman A, Goldstein B. Quantitative electroencephalography: Preliminary report—tinnitus. *Int Tinnitus J* 8(2): 77–86, 2002.
12. Moreira MMF, Maudonnet OAQ. Conceitos e funções dos movimentos oculares de rastreio lento, sacádico e nistagmo optocinético. *Acta AWHO* 17(3):135–138, 1998.
13. Dufour A, Mira E, Pignataro O. *Otoneurologia Clínica*. Milão: CRS, 1993:245.
14. Morrow MJ, Sharpe J. Smooth Pursuit Eye Movement. In JA Sharpe, HO Barber (eds), *The Vestibulo-Ocular Reflex and Vertigo*. New York: Raven, 1993:141–162.
15. Moreira MMF, Maudonnet OAQ. Neurofisiologia dos movimentos oculares. *Acta AWHO* 17(4):213–217, 1998.
16. Maudonnet OAQ. Anátomo-Fisiologia da Oculomotricidade. In *Avaliação Otoneurológica*. São Paulo: Editora BYK, 1999:29–34.
17. Suzuki M, Harada Y, Ishida M, et al. Aqueductal stenosis—results of vestibular functions tests. *J Laryngol Otol* 99(2):151–161, 1985.
18. Spooner JW, Baloh RW. Arnold-Chiari malformation: Improvement in eye movements after surgical treatment. *Brain* 104(1):51–60, 1981.
19. Furman JM, Perlman S, Baloh RW. Eye movements in Friedreich's ataxia. *Arch Neurol* 40(6):343–346, 1983.
20. Wessel K, Moschner C, Wadinger KP, et al. Oculomotor resting in the differential diagnosis of degenerative ataxic disorders. *Arch Neurol* 55(7):949–956, 1998.
21. Buttner N, Geschwind D, Jen JC, et al. Oculomotor phenotypes in autosomal dominant ataxias. *Arch Neurol* 55(10):1353–1357, 1998.
22. Reulen JP, Sanders EA, Hogenhus LA. Eye movement disorders in multiple sclerosis and optic neuritis. *Brain* 106(1):121–140, 1983.
23. Huygen PL, Verhagen WI, Hommes OR, Nicolasen MG. Short vestibule-ocular reflexes time constants associated with oculomotor pathology in multiple sclerosis. *Acta Otolaryngol* 109(1–2):25–33, 1990.
24. Ohki M, Kanayama R, Nakamura T, et al. Ocular abnormalities in amyotrophic lateral sclerosis. *Acta Otolaryngol Suppl* 511:38–42, 1994.
25. Breuer TJ, Wuisman PG, Korten JJ. Electrooculographic findings in progressive supranuclear palsy. *Clin Neurol Neurosurg* 89(2):87–95, 1987.

26. Degl'Innocenti F, Ginanneschi A, Cipparrone L. Oculomotor findings in the stages "on" and "off" in Parkinson disease patients. *Rev Neurol* 58(5):210–215, 1988.
27. Oepen G, Clarenbach P, Thoden U. Disturbance of eye movements in Huntington's chorea. *Arch Psychiatr Nervenkr* 229(3):205–213, 1981.
28. Helmchen C, Hagenow A, Miesner J, et al. Eye movement abnormalities in essential tremor may indicate cerebellar dysfunction. *Brain* 126(6):1319–1332, 2003.
29. Carlsson J, Rosenhall U. Oculomotor disturbances in patients with tension headache. *Acta Otolaryngol* 106(5–6): 354–360, 1988.
30. Lipton RB, Levy DL, Holzman PS, Levin S. Eye movement dysfunction in psychiatric patients: A review. *Schizophr Bull* 9(1):13–32, 1983.
31. Pawlak-Osinka K, Kazmierczak H, Osinski P, Michorzewski A. Electronystagmographic study in chronic schizophrenia. *Int Tinnitus J* 6(2):172–174, 2000.
32. Konrad HR, Rybak LP, Ramsey DE, Anderson DR. Eye tracking abnormalities in patients with cerebrovascular disease. *Laryngoscope* 93(9):1171–1176, 1983.
33. Josefowicz-Korczynska M, Lukomski M, Pajor A. Electronystagmographic evaluation of vestibular system in tinnitus patients with degenerative cervical spine lesions. *Otolaryngol Pol* 58(2):349–353, 2004.
34. Kazmierczak H, Pawlak-Osinska K, Osinski P. Visuoocular reflexes in presbyvertigo. *Int Tinnitus J* 7(2):112–114, 2001.
35. Hyden D, Larsby B, Andersson H, et al. Impairment of visuo-vestibular interaction in humans exposed to toluene. *ORL J Otorhinolaryngol Relat Spec* 45(5):262–269, 1983.
36. Rosenhall U, Lowhagen GB, Roupe G. Oculomotor dysfunction in patients with syphilis. *Genitourin Med* 63(2): 83–86, 1987.
37. Rosenhall U, Hakansson C, Lowhagen GB, et al. Otoneurological abnormalities in asymptomatic HIV-seropositive patients. *Acta Neurol Scand* 79(2):140–145, 1989.
38. Beiguelman B. O Teste *t* para Comparação de Médias. In R Preto (ed), *Curso Prático de Bioestatística*. Brazil: Revista Brasileira de Genética, 1996:152–159.
39. Santi PA, Mancini P. Cochlear Anatomy and Central Auditory Pathways. In CW Cummings, JM Fredrickson, LA Harker, et al. (eds), *Otolaryngology–Head and Neck Surgery*. Baltimore: Mosby–Year Book, 1993:2497–2524.
40. Coad ML, Lockwood A, Salvi R, Burkard R. Characteristics of patients with gaze-evoked tinnitus. *Otol Neurotol* 22(5):650–654, 2001.
41. Biggs ND, Ramsden RT. Gaze-evoked tinnitus following acoustic neuroma resection: A de-afferentation plasticity phenomenon. *Clin Otolaryngol Allied Sci* 27(5):338–343, 2002.
42. Lockwood AH, Salvi RJ, Burkard RF, et al. Neuroanatomy of tinnitus. *Scand Audiol* 51(suppl):47–52, 1999.
43. Morinaka S, Hidaka A, Nagata H. Abrupt onset of sensorineural hearing loss and tinnitus in a patient with capillary telangiectasia of the pons. *Ann Otol Rhinol Laryngol* 111(9):855–859, 2002.
44. Josefowicz-Korczynska M, Pajor A. Evaluation of oculomotor tests in patients with tinnitus. *Int Tinnitus J* 8(2): 100–103, 2002.