Predictors of central vestibular disorders from videonystagmography tests

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Background
The diagnosis of central vestibular lesion is challenging and sometimes there is an overlap in symptoms and signs with a peripheral vestibular lesion. In some selected cases, dizziness is the only presenting symptom and in other patients, mild neurological symptoms as numbness are ignored. Videonystagmography (VNG) is considered a useful method for diagnosing vertigo of peripheral origin; however, not all the patients with central vertigo can be diagnosed easily. Benign paroxysmal positional vertigo and central positional vertigo share common criteria. The aim of the present study is to assess the usefulness of different VNG tests as predictors of central vestibular disorders, to determine the criteria that differentiate central positional nystagmus from the peripheral type, and to attempt to relate the abnormality in different VNG tests to certain central nervous system (CNS) levels.
Materials and methods
A retrospective study was carried out on 51 patients with possible central vestibular disorders from VNG tests battery and were referred for an MRI for further assessment. According to MRI results, the patients were divided into group A (31 patients), the group with manifest MRI findings, and group B, the group with free MRI (20 patients). Different VNG tests were compared between both groups.
Results
Three predictors of CNS lesion by VNG were determined: fixation index (FI), oculomotor tests, and central positional nystagmus; there was a statistically significant difference between both groups in FI, oculomotor tests, and central positional nystagmus, and apogeotropic criteria or nystagmus in multiple plains. There was no relation between any of those predictors and specific levels in the CNS.
Conclusion
VNG tests are a good diagnostic tool to differentiate between peripheral and central vestibular lesions. The inclusion of FI and positional tests to the oculomotor tests increases the sensitivity of the VNG. In some cases, it is difficult to distinguish between benign paroxysmal positional vertigo and central positional vertigo; apogeotropic nystagmus and nystagmus in multiple plains should raise the suspicion of CNS lesion. Anterior circulation ischemia may lead to chronic vertigo symptoms. Central vestibular vertigo could be caused by dysfunction or excitation of various structures in the CNS including the vestibular cortex.

Keywords:
central positional nystagmus, central vestibular lesion, vestibular cortex, videonystagmography

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Introduction

Dizziness is a common complaint among patients seen by general practitioners, neurologists, and otolaryngologists. Dizziness is a common term used to describe multiple sensations (vertigo, imbalance, presyncope), each with numerous etiologies. Peripheral vestibular disorders are the most common, but epidemiologic studies indicate that central causes are responsible for almost one-fourth of the dizziness experienced by patients [1]. Patients with missed central nervous system (CNS) disorders are at a higher risk for complications, with a higher mortality rate.

Ocular stability during most natural head movements results from a coordinated interaction of signals originating in vestibular, visual, and neck receptors, and failure to interact can cause vertigo of central origin. Visual–vestibular interaction is mediated through the subcortical accessory optic system and cortical pathway. Lesions of the nucleus of the pretectal nuclei (nucleus of the optic tract) and

inferior olive of the subcortical pathway, and the primary visual cortex, visual association cortex, dorsolateral pontine nuclei, and the flocculus result in an impairment in visual–vestibular interaction, followed by a vertiginous sensation [2].

Videonystagmography (VNG) is a computerized vestibular function test that uses infrared cameras to record eye movements directly. It is superior to electronystagmography (ENG) in terms of higher resolution and greater stability to observe, capture, and record torsional eye movement, although both assess the same functions [3]. Several studies underestimate the role of VNG/ENG in diagnosing central vestibular lesion, reporting that ENG is time-consuming and its usefulness in identifying central disturbance is controversial [4,5]. Others reported that nystagmography plays an important role in helping screen patients with suspected central vestibular disorders [6], but they emphasize that if the interpretation is made automatically by a computer, this can often affect the results and lead to an errant diagnosis.

In peripheral vestibular disorders, positional nystagmus is mostly observed in benign paroxysmal positional vertigo (BPPV). In BPPV, the nystagmus is mostly paroxysmal, but may rarely be persistent. Similarly, positional nystagmus can either be persistent or paroxysmal in central vestibular disorders [7].

The aim of the present study is to assess the usefulness of different VNG tests as predictors of central vestibular disorders, to determine the criteria that differentiate central positional nystagmus from the peripheral type, and to attempt to relate different VNG test abnormalities to certain CNS levels.

Materials and methods

A retrospective study was carried out on 51 patients with possible central vestibular disorders from VNG tests battery and were referred for MRI for further assessment between March 2012 and July 2015. The study was approved by the Assiut University Ethical Research Committee. Patients diagnosed with vestibular migraine according to the Neuhauser and Lempert [8] criteria or familial cerebellar ataxia were excluded from the study. All patients were subjected to an audiovestibular test battery, which included a detailed assessment of vertigo history, hearing evaluation, neuro-otological examination, and VNG.

A VNG tests battery was performed by two-channel monocular Micromedical Mobile Eyes spectrum7.2 B. Micromedical technologies, Illinois, USA. It included a search for spontaneous nystagmus both with and without fixation, with gaze evoked, and with a posthead-shaking test. Also, nystagmus was studied using both the Dix–Hallpike test (dynamic positional test) for the right and left side and static positional tests including the supine, head right and head left, and right lateral and left lateral positions. An assessment of saccades, smooth pursuit, and optokinetic nystagmus (OKN) was also performed. In addition, caloric function was studied using alternate bithermal caloric irrigation with water at 30 and 44°C. A difference of greater than 20% was considered unilateral caloric weakness and the magnitude of a nystagmus of a slow-component velocity of 6°/s was considered significant during positional and posthead-shake testing or nystagmus tests. Also, visual recording was performed to allow for playback and slow-motion evaluation of ocular movements if needed.

Central vestibular disorder was considered if one or more of the following criteria were fulfilled [6,9]:

(1) Pure torsional or pure vertical nystagmus, not abolished with fixation in any of the nystagmus tests, and direction-changing nystagmus in the gaze test. Gaze evoked nystagmus opposite to Alexander's law (more intense with gaze in the direction of the slow phases) 10. Dissociated nystagmus is also one of the central findings in which the rhythmic oscillations are different in the two eyes; internuclear ophthalmoplegia are included under this category [11]. However, binocular VNG is better to visualize this category of nystagmus. Vertical nystagmus induced by head-shaking tests is also considered a central vestibular finding [12].
(2) Central positional nystagmus, including apogeotropic nystagmus (after exclusion of lateral canal cupulolithiatic BPPV), direction-changing nystagmus in the same position, downbeating nystagmus, pure torsional or vertical nystagmus and not fatigable. More recent criteria included positional nystagmus induced in multiple planes or strange behavior of positional nystagmus [13,14].
(3) Abnormal oculomotor tests: Delayed saccades, saccadic slowing, and saccadic dysmetria. Symmetric or asymmetric defective tracking or saccadic pursuit. Symmetric or asymmetric low optokinetic gain.
(4) Impaired fixation suppression in caloric tests when the fixation index (FI) is greater than 60%.
(5) All patients were subjected to multiplaner MRI scanning of the brain performed by various

multiple pulse sequences. According to MRI results, patients were divided into group A (31 patients), with manifest MRI findings, and group B, with free MRI (20 patients).

Data were collected and analyzed using the computer program SPSS (ver. 21; IBM Corp. Chicago, Illinois, USA). Data are expressed as mean, SD, number, and percentage. The Mann–Whitney test was used to determine significance for numeric variables. The χ^2 was used to determine significance for categorical variables. The sensitivity and specificity of some diagnostic tests were also measured.

In all statistical procedures, *P* greater than 0.05 was considered not significant, *P* less than or equal to 0.05 as significant, *P* less than or equal to 0.01 as moderately significant, and *P* less than or equal to 0.001 as highly significant.

Results

The study included 51 patients with central vestibular disorders (according to VNG tests), 35 women and 16 men, mean age 52.58±15.29 years and age range from 18 to 83 years. Demographic characteristics, dizziness features, associated symptoms, and comorbidities in both groups are shown in Table 1. A significant difference was observed between both groups only in the duration of dizziness and associated neurologic symptoms, with shorter duration of dizziness and associated neurologic symptoms only in group A.

Abnormal VNG tests were compared between both groups (Table 2). In group A, five patients showed spontaneous and gaze nystagmus; two of them enhanced with fixation. Abnormal VNG findings were most frequently encountered with dynamic and static positional tests in both groups. Twenty-three (74.19%) patients fulfilled at least one criteria of central positional nystagmus in group A, compared with 14 (70%) in group B. Caloric test abnormalities were in the form of unilateral caloric weakness in nine patients, hyperexcitable response in two patients, and impaired FI in 10 patients of group A, with significantly increased FI compared with group B. The oculomotor tests also showed a significant difference between the two groups. Seventeen (54.83%) patients showed abnormality in at least one of the oculomotor tests in group A compared with two (10%) patients in group B.

The frequencies of different criteria of central positional nystagmus in both groups are shown in Fig. 1, with nystagmus in multiple plains being the most frequent, followed by downbeating nystagmus and apogeotropic nystagmus. Pure torsional nystagmus was observed in seven patients and direction-changing nystagmus in the same head position was observed in

Table 1 Demographic characteristics and clinical manifestations by number and percentage of group A versus group B

	Group A (manifest MRI findings)	Group B (free MRI)	*P*-value
Age in years (mean, SD)	53.38±16.1	50.1 ±12.87	0.561
Sex [*n* (%)]			
Male	11 (35.5)	5 (25)	0.308
Female	20 (46.5)	15 (75)	
Duration in weeks (mean, SD)	25.63±32.24	65±42	0.004**
Dizziness features [*n* (%)]			
Positional symptoms	24 (77.4)	16 (80)	0.935
Acute symptoms	7 (22.58)	4 (20)	0.928
Hearing loss [*n* (%)]	7 (22.58)	8 (40)	0.51
Tinnitus [*n* (%)]	18 (58.06)	12 (60)	0.605
Nausea or vomiting [*n* (%)]	18 (58.06)	10 (50)	0.471
Headache [*n* (%)]	21 (67.74)	8 (40)	0.169
Neurological symptoms [*n* (%)]	16 (51.61)	0 (0)	0.003**
Comorbidities [*n* (%)]			
Hypertension	7 (22.7)	6 (30)	0.89
Diabetes mellitus	3 (9.67)	2 (10)	
Vertebral illness	2 (6.45)	0 (0)	
Cardiac	2 (6.54)	2 (10)	

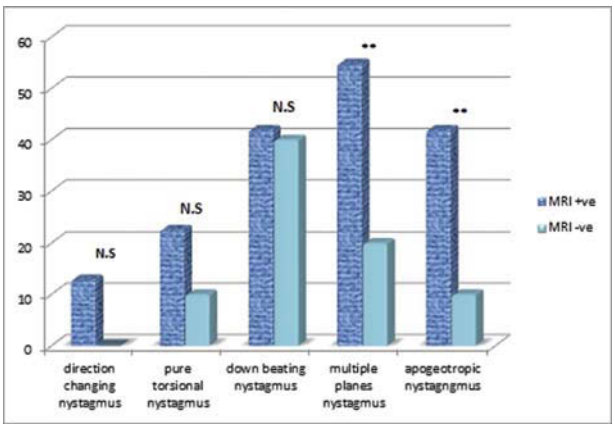
**Moderately significant.

Table 2 Videonystagmography findings in group A versus group B

VNG tests	Group A [*n* (%)]	Group B [*n* (%)]	*P*-value
Spontaneous nystagmus test	5 (16)	2 (10)	0.526
Gaze nystagmus tests	5 (16)	2 (10)	0.526
Posthead-shaking test	13 (42)	10 (50)	0.532
Dynamic positional tests	26 (83.8)	20 (100)	0.217
Static positional tests	27 (87.1)	18 (90)	0.633
Caloric test weakness	9 (29.03)	2 (10)	0.204
Impaired fixation index	10 (32.25)	1 (5)	0.05*
Saccade test	14 (45.1)	1 (5)	0.02*
Pursuit test	14 (45.1)	1 (5)	0.02*
Optokinetic nystagmus test	13 (42)	0 (0)	0.01**

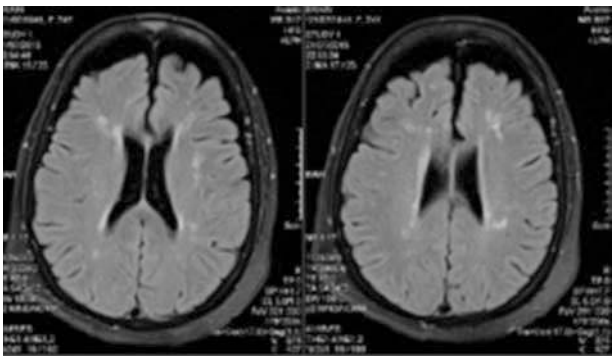
VNG, videonystagmography.
*Significant.
**Moderately significant.

Figure 1



Frequency of different criteria of central positional nystagmus in both groups. NS, nonsignificant. **moderately significant.

Figure 2



MRI scanning of the brain showing multiple bilateral small focal lesions, scattered along the periventricular white matter of both frontoparietal regions, suggesting multiple lacunar infarctions.

Table 3 Sensitivity and specificity of different videonystagmography parameters

VNG parameter	True positive patients (*N*)	Sensitivity (%)	True negative patients (*N*)	Specificity (%)
Fixation index	10	32.25	19	95
Oculomotor tests	17	54.83	18	90
Dynamic positional tests [*n* (%)]	26 (83.8)	83.8	0	0
Static positional tests	27	87.1	2	10
Direction-changing nystagmus	4	12.9	20	100
Pure torsional nystagmus	7	22.58	18	90
Downbeating nystagmus	13	41.93	14	60
Multiple plains nystagmus	17	54.83	16	80
Apogeotropic nystagmus	13	41.93	18	90

VNG, videonystagmography.

four patients, and only one patient showed pure vertical nystagmus. A significant difference between both groups was detected in multiple plains and apogeotropic nystagmus.

In the present study, three main predictors of central vestibular lesions were determined through VNG: impaired FI, impaired oculomotor tests, and central positional nystagmus.

The sensitivity and specificity of these predictors were studied and are shown in Table 3. The oculomotor tests, multiple plains nystagmus, and apogeotropic nystagmus showed moderate sensitivity with high specificity. FI showed low sensitivity and high specificity. Direction-changing nystagmus and pure torsional nystagmus also showed low sensitivity and high specificity, whereas downbeating nystagmus showed moderate sensitivity and specificity.

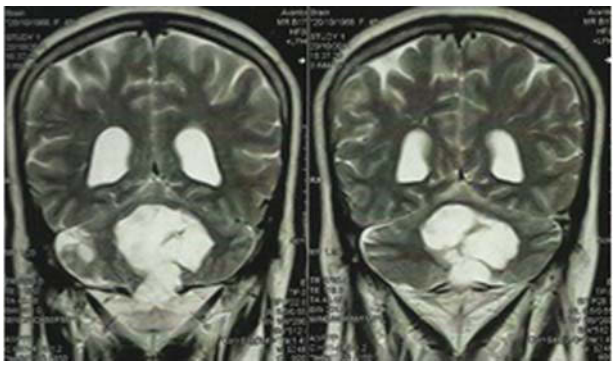
According to the MRI findings in group A, 23 patients showed vascular disorders, five patients had a space-occupying lesion (SOL), and three patients had multiple sclerosis (MS).

Among patients with vascular disorders (23 patients), 21 showed either multiple lacunar ischemic infarctions or small blood vessel disease (Fig. 2), one case with left occipital encephalomalacia and one case with cavernous sinus thrombosis. The most frequent site of lesion in this group was the periventricular area in16 patients and specifically the parietal periventricular area in nine of them, followed by the basal ganglia in three patients, the occipital lobe in three patients, and the frontal lobe in three patients and thalamic infarction in one patient.

SOL in the form of left parietal meningioma, right hemispheric cerebellar astrocytoma, and two cases with posterior fossa epidermoid cyst (Fig. 3). The fifth patient had multiple metastasis in both cerebral hemispheres compressing on the lateral ventricles.

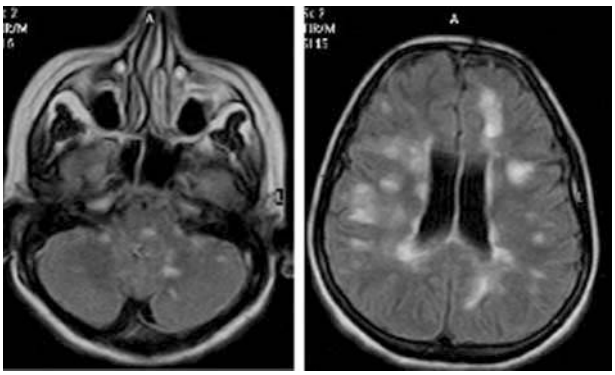
MRI of patients with MS showed MS plaques either in the medulla in one patient or the parietal lobe in the other patient. In the third patient, there were MS plaques in both cerebral hemisphere, including frontal, temporal, parietal, and occipital regions in relation to the ventricular system; also, there was an

Figure 3



MRI scanning of the brain showing a posterior fossa cystic lesion, involving the fourth ventricle (FV). The lesion measured 5×7 cm, insinuating through the foramina of FV into both cerebellopontine angles and below the vermis.

Figure 4



MRI scanning of the brain showing MS plaques in cerebellar hemispheres, brainstem, and cerebral hemisphere, related to the ventricular system.

involvement of both cerebellar hemispheres, thalami, and brainstem (Fig. 4).

Discussion

Narrative reviews have highlighted the importance of accurately assessing the risk of dangerous central vestibular disorders and have emphasized the utility of a focused history and physical examination in these patients [15,16]. Some reviews have generally suggested that the presence of neurologic symptoms or signs indicates a central cause of vestibular syndrome and some authors have gone so far as to suggest that their absence indicates a peripheral cause [17,18]. Our results are similar to those studies that included patients with general neurologic symptoms or signs (e.g. diplopia and numbness), and reported that the presence of such symptoms or signs was strongly associated with central causes, but their absence was a relatively poor predictor of a peripheral cause [19,20].

Expert opinion grounded in well-established neuroanatomy suggests that auditory symptoms usually point to a peripheral cause in patients with dizziness. Unfortunately, this anatomic fact may lead to the mistaken diagnosis of a more benign cause [21]. In the present study, 22.58% of patients with manifest MRI lesion had hearing loss compared with 40% of those with free MRI.

In many balance disorders, the ENT and neurological evaluations are inconclusive; in continuation, performing a contrast-enhanced MRI scan for a patient who have BPPV would also be an overkill of sorts. VNG can detect subtle ocular motility abnormalities and reflect the effect of balance disorder on the ocular system. It has thus been said that the eyes serve as a window to the vestibular system [6].

The sensitivity and specificity of different VNG predictors were studied [22] (Table 3); the results showed that the oculomotor tests and central positional nystagmus in the form of multiple plains nystagmus and apogeotropic nystagmus have moderate sensitivity, which means that they have moderate ability to correctly identify those patients with central vestibular lesion. However, the FI and the central positional nystagmus in the form of direction-changing nystagmus in the same position and pure torsional nystagmus have low ability to correctly identify those patients with central lesion. All of the above tests (VNG predictors of central lesion) have high specificity, which means that, when they are positive, it is very likely that the patient has a central vestibular lesion.

Murai *et al.* [23]. examined 10 cerebellar and 23 peripheral disequilibrium patients using caloric testing and showed an impairment in FI in cerebellar disorders. In another study carried out by Shiomi *et al.* [4], vertiginous patients with cerebellar disorder also showed impaired visual suppression (VS). In addition, VS was deteriorated in patients with disorders of the brainstem, cerebellopontine angle, or vertebral and basilar artery lesions. Furthermore, VS was also impaired in patients who had a cerebral contusion or had a hemorrhagic scar at the orbital gyri. Diffuse cortical damage has been reported to result in impairment in the vestibuloocular reflex and VS of the vestibuloocular reflex [24]. In the present study, impaired FI was detected in four patients with multiple lacunar ischemic infarctions, in bilateral basal ganglia and hippocampal region in one patient, the

parietal and frontal periventricular region in two patients, and the parietal periventricular region in one patient. Three patients had SOL: two in the posterior fossa and one with a cerebral metastatic lesion compressing on the lateral ventricle. Also, impaired FI was reported in two patients with MS: one in the parietal region with periventricular orientation and the other in the brainstem. These results indicate that impaired FI can be a useful parameter to differentiate not only between peripheral and cerebellar lesions but also between peripheral and cerebral equilibrium disorders.

The second predictor of central lesion was oculomotor tests, including saccade, pursuit, and OKN testing. It is known that the presence of ocular motor test abnormalities is associated strongly with central vestibular disorders [6,25,26]. However, in the present study, about 45.17% of patients with manifest central vestibular lesions showed normal findings in oculomotor tests, which means that their absence does not exclude central cause.

Oculomotor tests could be affected in old age, inattention, poor vision, sedation, sleep deprivation, and medications, and these factors should be considered during testing. The generation of saccades involves the paramedian pontine reticular formation as well as multiple sites within the CNS and its impairment often localizes to lesions of the brainstem or the cerebellum [25] or in basal ganglia disorders [6]. The smooth pursuit system relies on multiple sites within the CNS, including the pons and cerebellum. OKN shares pathways with the pursuit system and other eye movements, but the nucleus of the optic tract plays an important role [21]. Brainstem, cerebellar, and cerebral lesions produce impairments in pursuit and OKN tests [26].

In the present study, most of the patients with oculomotor tests abnormalities showed an impairment in the three tests, except in a few patients. The patients either had a vascular lesion, SOL, or MS; collectively, they had lesions in the brainstem, cerebellum, subcortical, parietal lobe, occipital lobe, periventricular area, and multiple cortical lesions.

Positional nystagmus can be either persistent or paroxysmal in central vestibular disorders [7]. The paroxysmal type was diagnosed when the nystagmus induced by position changes decayed within 1 min. In contrast, the persistent type was diagnosed when the nystagmus lasted for more than 1 min without a discernible change in the slow phase velocity in a certain position [13]. In this study, 77.4% of patients with manifest MRI lesion had positional symptoms and about 71% of patients fulfilled at least one criterion suggesting central positional nystagmus.

Significant differences between group A and group B were observed only in multiple plains nystagmus and apogeotropic nystagmus. Nystagmus in multiple plains is a mixture of horizontal, torsional, and vertical components with a variable combination of each component depending on the positioning maneuver. It was once suggested that the direction of CPPN (central paroxysmal positional nystagmus) is typically pure vertical and torsional, and does not correspond to the stimulated canal plane [7]. In a recent study carried out by Choi *et al.* [13], it was reported that the downbeat nystagmus was most prominent during straight head hanging. The horizontal and torsional components were also associated with the downbeat nystagmus during straight head hanging and became more apparent during Dix–Hallpike maneuvers. Upon supine head turning, prominent horizontal nystagmus was induced in association with a torsional component. In the present study, we did not study the straight head-hanging position, but the picture was the same in the Dix–Hallpike maneuvers. Vertical and horizontal components were recorded graphically and by video, but the torsional component was recorded only by video.

The direction of paroxysmal nystagmus during the Dix–Hallpike maneuver may be explained by the vector sum of the rotational axes of the ipsiversive anterior canal and contraversive horizontal canal, which are normally inhibited during this positioning. This finding explains why the downbeat nystagmus during straight head hanging and Dix–Hallpike maneuvers shows a pattern similar to that observed in the anterior canal BPPV [13]. Approximately three-quarters (76.4%) of the patients in the Choi and colleagues study had apogeotropic paroxysmal nystagmus, which was similar to the nystagmus observed in cupulolithiatic horizontal canal BPPV. However, in the cupulolithiatic horizontal canal BPPV, the apogeotropic nystagmus was not associated with other types of nystagmus. Therefore, the cooccurrence of various types of nystagmus, such as downbeat, upbeat, and apogeotropic, should increase the suspicion of central positional vertigo rather than multiple canal BPPV (e.g. anterior canal BPPV and horizontal canal cupulolithiatic BPPV). Also, in BPPV, the intensity vertigo is correlated with the degree of nystagmus and the BPPV could be treated in the majority of patients with different canalith repositioning maneuvers. Other studies used the

temporal pattern of the nystagmus to differentiate between central positional nystagmus and BPPV; they reported that in CPPN, the intensity of evoked nystagmus is at its peak initially and then decreases exponentially over time. In contrast, the intensity of nystagmus gradually builds up over 10–20 s and then decreases slowly in both anterior canal BPPV and cupulolithiatic horizontal canal BPPV [13,27,28].

Multiple studies have implicated cerebellar lesion as a cause for central positional nystagmus. Lesions involving the nodulus and uvula could explain the frequent coincidence of central paroxysmal positional nystagmus and persistent apogeotropic central positional nystagmus [29,30]. Lesions dorsolateral to the fourth ventricle or in the dorsal vermis are often found in central paroxysmal positional vertigo [7]. The flocculus was only occasionally involved in another study [13]. In the present study, not only was the cerebellum involved in patients with central positional nystagmus but also lesions in subcortical and cortical areas including more frequently periventricular and parietal areas, suggesting the involvement of multiple sites within the CNS.

It was found in this study that the parietal lobe and the periventricular parietal area were the most frequently affected sites in MRI of our patient group with various CNS lesions, especially those with cerebral ischemia.

Dieterich and Brandt [31], in their recent study, carried out to determine the vestibular areas that in exceptional cases manifest with transient vertigo or dizziness in acute strokes of the middle cerebral artery, found that overlap areas were located either in the posterior retroinsular cortex, that is, the parieto-insular vestibular cortex, or the separate parietal vestibular cortex. They concluded that rare vestibular cortical vertigo is mostly elicited by acute lesions of the core region of the retroinsular vestibular network. They suggested that a bilateral lesion is needed to induce dizziness symptoms. They propose a concept to explain how, in a unilateral lesion, the unaffected opposite hemisphere can suppress vertigo. This is based on visual–vestibular interaction for motion perception and orientation. It is the hemisphere in which vestibular and visual inputs are in agreement, which is more reliable, and determines the overall perception of body orientation and motion.

The present study included patients with middle cerebral artery ischemia (white matter ischemia of the parietal lobe); 66.6% showed a bilateral lesion. However, the majority of them had subacute or chronic symptoms. This finding raises the suspicion of chronic vertigo induced by anterior circulation ischemia, added to the long-known posterior circulation ischemia.

Conclusion

VNG tests are a good diagnostic tool to differentiate between peripheral and central vestibular lesions, provided that they are performed and analyzed by clinicians and not technicians. Patients with CNS lesions could have normal oculomotor tests, but adding FI and searching for central positional nystagmus increases the sensitivity and the specificity of the VNG. In some cases, it is difficult to distinguish between BPPV and central positional vertigo; strange behavior nystagmus and nystagmus in multiple plains should raise the suspicion of a CNS lesion. Anterior circulation ischemia may induce chronic vertigo symptoms. Central vestibular vertigo could be caused by dysfunction or excitation of various structures in the CNS including the vestibular cortex.

Recommendations

(1) Study of the criteria and direction of the positional nystagmus by VNG or video goggles during the attacks is recommended to differentiate central from peripheral types.
(2) The role of anterior cerebral circulation ischemia in the development of chronic vestibular symptoms should be further investigated.
(3) The vestibular areas in the cortex that could cause transient vertigo or dizziness in a central vestibular lesion should be determined.
(4) The usefulness of otolith function tests such as skew deviation and subjective visual vertical in differentiating peripheral from central vestibular lesions should be assessed.

Conflicts of interest

There are no conflicts of interest.

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