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Direction-fixed positional nystagmus following head-roll testing: how is it related with a vestibular pathology?

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ABSTRACT

Objective: The goal of this study is to analyze the clinical view of patients with direction-fixed positional nystagmus (DFPN) following head-roll maneuver.
Methods: Sixty patients with DFPN were reviewed retrospectively. Patients were categorized into 3 groups according to the direction of nystagmus based on rotation side. Associated problems were documented, and cumulative data were compared between groups. One-way analysis of variance (ANOVA test) was used for statistical analysis ($P < 0.05$).
Results: Thirty-three patients (55%) had stronger nystagmus beating towards the direction of head-roll (Group-A). Three patients developed geotropic LC-BPPV. Fourteen patients had inner ear disease. Sixteen patients (27%) had stronger nystagmus beating against the direction of head roll (Group-B). Nine patients had inner ear disease. None of the patients tested with head-shaking had change of direction of nystagmus. Eleven patients (18%) had DFPN with equal velocity during right or left head-roll maneuver (Group-C). Of those, nine patients had inner ear disease. None of the patients had change of direction of nystagmus. Comparison of the incidence of associated problems (migraine, vestibular neuronitis, Meniere's disease etc.) in each group was not statistically significant (P>0.05).
Conclusion: Patients with DFPN should be followed for a possibility of vestibular pathology since vestibular problem was documented for more than half of the patients in the follow-up. On the other hand, DFPN could be related with a temporary reason (thermal, physical or drug effect etc.) in some patients who do not exhibit any associated disease. Head-shaking testing is recommended to expose the lateral canal BPPV. But the incidence is low.

1. Introduction

Direction-fixed positional nystagmus (DFPN) in patients who have no spontaneous nystagmus at upright primary gaze position is a challenging condition, particularly when there is no medical story associated with the acute balance problem. Positional nystagmus in patients with benign paroxysmal positional vertigo (BPPV) is characteristically brief, transient and adapts to repeated tests. Presence of bi-directional, horizontal positional nystagmus (geotropic or apogeotropic) during head-roll maneuver indicates lateral canal benign paroxysmal positional vertigo (BPPV). Affected side is confirmed with respect to the velocity of nystagmus. Nystagmus velocity is greater on the affected side in patients with geotropic nystagmus and on the healthy side in patients with apogeotropic nystagmus (McClure, 1985). DFPN can be seen during treatment of patients with BPPV. Canalith re-positioning maneuver in some patients with lateral canal BPPV can result with acute increase of symptoms associated with direction-fixed nystagmus in which the condition was claimed to be due to canalith jam of the affected canal (Chang et al., 2014; Von Brevern et al., 2001; Ko et al., 2014). Debris may block the canal during the attempt of dragging the debris out of the canal and may cause continuous stimulation of

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the cupula leading to the intense nystagmus. On the other hand, DFPN has been proposed as a variant of lateral canal BPPV (Califano et al., 2013). Characteristic pictures of the patients are sudden development of a spontaneous nystagmus with vertigo regardless of head position, positive head impulse test with re-fixation saccades, distortion of VEMPs on some occasion and caloric weakness in the affected side mimicking acute vestibular deficit (Comacchio et al., 2018; Castellucci et al., 2019; Luis et al., 2013). Its pathogenesis is similar to seen in iatrogenic "canalith-jam" since diagnosis is confirmed with head-shaking maneuver which causes dispersal of debris and helps to understand the site of the affected canal.

Dumas et al. (2017) have demonstrated that nystagmus beating away from the affected side following skull vibration can be seen in patients with unilateral vestibular loss. Spontaneous nystagmus can be triggered by sudden impulsive forces following head movement in patients with unilateral vestibular dysfunction This condition can be regarded as intensification of spontaneous nystagmus by positional maneuvers which is sometimes not seen during primary gaze position. Hulshof and Baarsma (1981) have reviewed 151 patients with Meniere's disease. They have found that spontaneous nystagmus was present in 47% of cases. But 78% of cases have demonstrated DFPN. Acute unilateral peripheral vestibular dysfunction creates vestibular asymmetry which results from unilateral alteration of the resting neural potential. Ewald's first and second laws, "stimulation of the semicircular canal causes endolymphatic flow and eye movement in the plane of the stimulated canal" and "in the horizontal semicircular canals, an ampullopetal endolymph movement creates a greater stimulation than an ampullofugal one" are basic principles to understand the nystagmus. Head movement to each side exposes nonlinearity as indicated by Ewald's second law in case of unilateral hypofunction (Kim et al., 1978). It is associated with spontaneous nystagmus generally beating to the contralateral intact ear which is basically due to relatively prolonged stimulation of normal vestibulo-oculomotor system (McClure and Lycett, 1983). DFPN has been reported in patients with central vestibular disorders as well. Lemos et al. (2019) have reviewed fifteen patients with peripheral and fifteen patients with central vestibular syndrome. Ninety-three per cent of patients with peripheral pathology and 33% of patients with central vestibular syndrome demonstrated DFPN which was stronger when turning the head to the slow phase side.

The goal of this study is to document the clinical view of 60 patients with direction-fixed positional nystagmus (DFPN) following head-roll maneuver and to understand how it is or it is not related with lateral canal BPPV, peripheral vestibular disease or central vestibular disorder.

2. Material and methods

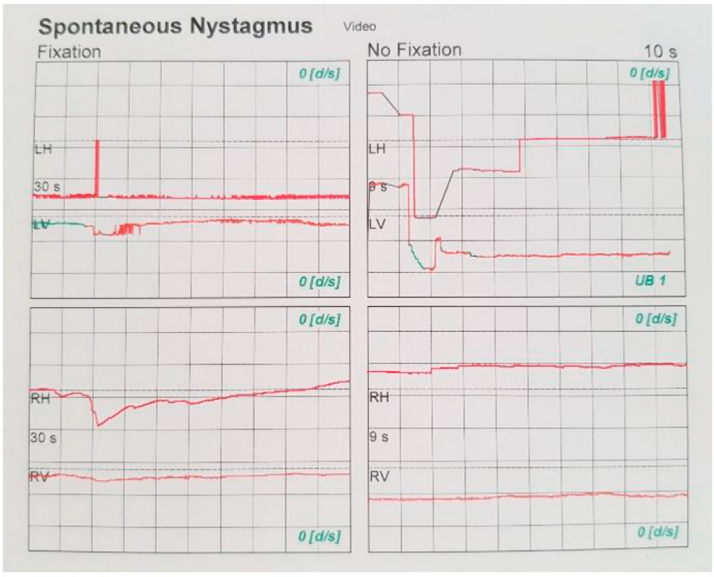
Sixty patients with DFPN following head-roll maneuver who have been examined at outpatient clinic between 2009 and 2019 were enrolled for the study. Patients charts were retrospectively reviewed from their first admission through the follow-up period (between 6 weeks and 2 years; 11.83 ± 7.09 months). Nystagmus was recorded by videonystagmography (VNG) (Micromed., Inc, USA) in all patients. All procedures were in accordance with the institutional review board (2019–4) and with the 1964 Helsinki declaration for ethical standards. The study was completed in a regional community hospital. Thirty-one males and 29 females aged between 21 and 83 were included (male; 27–83; 42.22 ± 11.81, female; 21–73; 45.10 ± 13.56). None of the patients had spontaneous nystagmus at primary gaze positions while sitting. All patients had normal otoscopic view and normal hearing threshold. Those with systemic problems, hearing loss, abnormal ear drum, muscular, ocular, and neurological symptoms and those having any medication one week prior to test which may have an impact on vestibular system were excluded. Positional tests (head-roll, head-hanging, head-bending in seated position) were done at the day of admission. Direction-fixed nystagmus following head-roll maneuver in supine position was present in both head rotation sides. The rolling sequence was always first to the right, then central, and then to the left. Video recordings were always checked with the graphics. Results were based on graphic analysis. However, printed eye movements were always matching with video recordings of eye movements. The duration of recordings was not less than 60 s at each position. Nystagmus did not disappear in any of the patients during recording despite decline in intensity towards the end of recording in some patients. Printed eye movement of a 41-year-old patient is presented in Fig.-1.

The type, slow phase velocity and the direction of nystagmus were documented. Patients were classified into 3 groups according to the direction of nystagmus based on the rotation side. Group-A included the patients with stronger nystagmus beating towards the direction of head roll. Group-B included the patients with stronger nystagmus beating against the direction of head-roll. Group-C included the patients having nystagmus with equal intensity during right or left head-roll maneuvers beating towards the right or left. Head-roll test was repeated in 43 patients following head-shaking maneuver by shaking the head to either side forcefully for 30 s to see any change in direction of nystagmus. Magnetic resonance imaging (MRI) of the temporal bone and/or Doppler ultrasound analysis of the carotid and vertebral arteries was performed in 25 patients at first admission or in the follow-up period. Audiometry was ordered and VNG was repeated in the follow-up period if the patient returns to the clinic with otologic symptoms. One-way analysis of variance (ANOVA test) was used to analyze the groups. Statistical significance was set at $P < 0.05$.

3. Results

Thirty-three patients (55%) had stronger nystagmus beating towards the direction of head-roll (Group-A) either to the left (23 patients) or to the right (10 patients). Comparison of average slow phase velocity of stronger nystagmus towards the direction of head-roll with the weaker one was statistically significant (stronger; 5.22 ± 2.91°/sec, weaker; 3.32 ± 1.92°/sec, p<0.05). Three patients in this group who had right sided DFPN (stronger nystagmus beating in the direction of right head-roll) were later developed right-sided geotropic type lateral canal BPPV following head-shaking and cured after barbeque maneuver. Five patients had migraine and eight patients had vestibular neuritis during follow-up. One patient later developed Meniere's disease with recurrent episodes of vertigo and low frequency hearing loss. Sixteen patients in this group did not exhibit any associated disease. Sixteen patients (27%) had stronger nystagmus beating against the direction of head-roll (Group-B). Nystagmus was beating to the left but stronger in the right head roll in 10 patients and nystagmus was beating to the right but stronger in the left head-roll in 6 patients. Comparison of average slow phase velocity of stronger nystagmus against the direction of head-roll with the weaker one was statistically significant. (stronger; 4.88 ± 2.02°/sec, weaker; 2.94 ± 1.47°/sec, p<0.05). Three patients later developed hearing loss and recurrent episodes of vertigo associated with Meniere's disease in the follow-up period. Two patients had migraine and four patients had vestibular neuronitis in the follow-up. Seven patients in this group had no associated inner ear disease. None of the patients in this group had diagnosis of lateral canal BPPV after head-shaking maneuver at first admission and in the follow-up period.

Eleven patients (18%) had nystagmus with equal velocity during



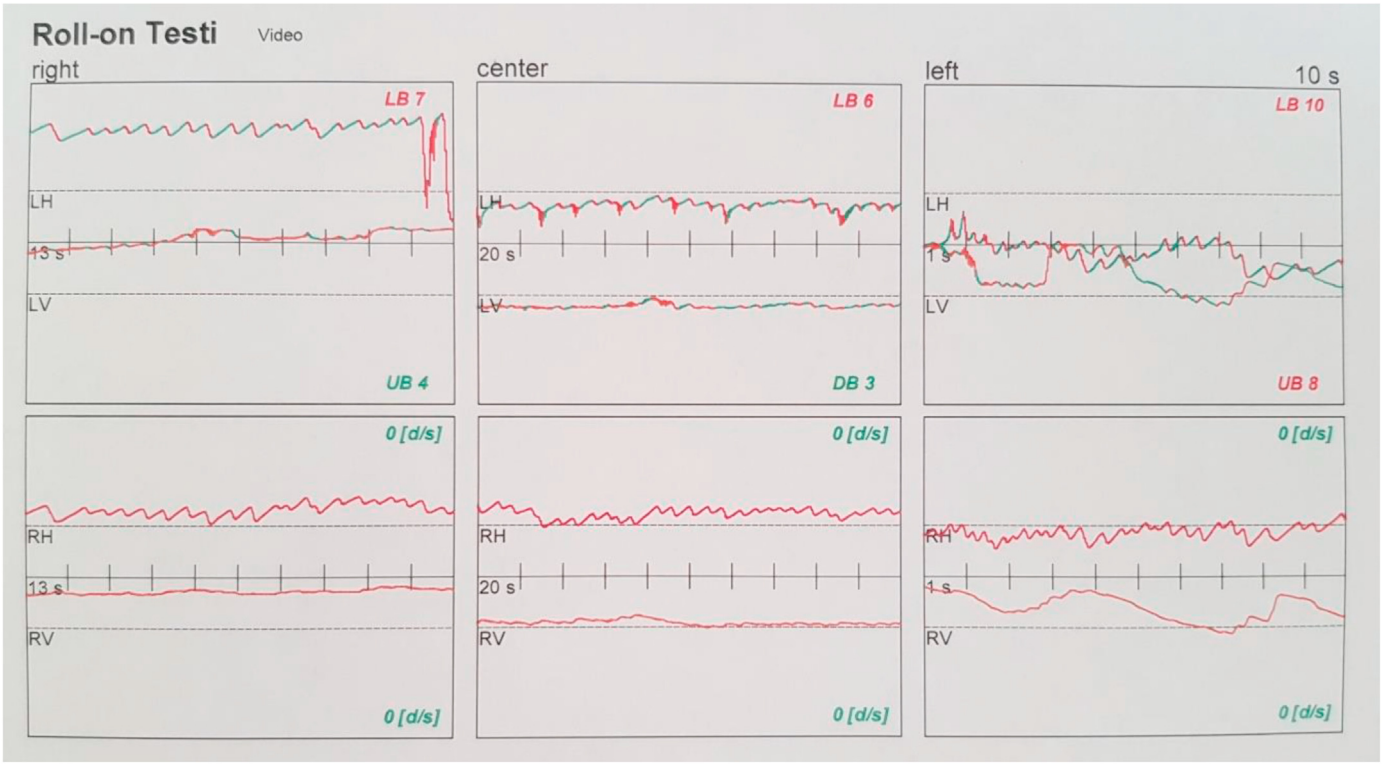


Fig. 1. This is a 41 year-old-man with acute onset balance problem. Upper recording (A) shows that the patient has no spontaneous nystagmus during upright and primary gaze position with and without optical target. B. lower recording shows that the patient has a long-lasting left beating DFPN with stronger SPV in the plane of head-roll movement to the left (7°/sec in the right, 10°/sec in the left head-roll).

right or left head-roll maneuver beating towards the right (3 patients) or left (8 patients) (Group-C). No statistically significant difference was found when comparing SPV of direction-fixed nystagmus beating to the left (Average SPV; 2.87 ± 0.83°/sec) and beating to the right (Average SPV; 2.50 ± 0.70°/sec) during head-roll maneuver to the right or to the left (p = 0.581). Three patients in this group later developed hearing loss and recurrent episodes of vertigo associated with Meniere's disease in the follow-up period. Three patients had migraine and three patients had vestibular neuronitis. Two patients in this group had no associated inner ear disease. None of the patients in group-C had diagnosis of lateral canal BPPV after head-shaking maneuver and in the follow-up period. Fig. 2 shows the distribution of the associated inner ear problems among three groups. Comparison of the means of two independent samples separately was not statistically significant (P>0.05). However, the incidence of Meniere's disease was slightly different between groups. When three groups were reviewed, twenty-five patients (42%) had no associated disease in the follow-

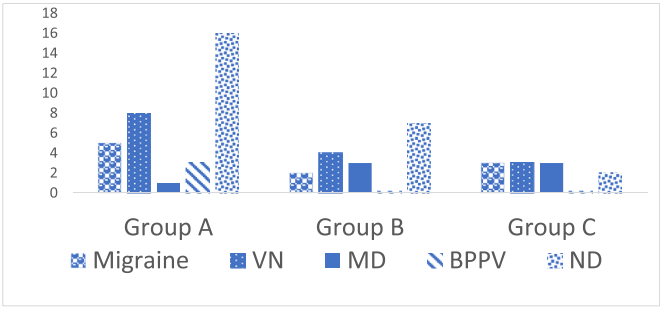


Fig. 2. Distribution of patients with migraine, vestibular neuritis (VN), Meniere's disease (MD), benign paroxysmal positional vertigo (BPPV) and those with no identified pathology (ND; no disease) in 3 groups which are categorized according to the severity of positional nystagmus as compared to the direction of head-roll test. Note that in group A (nystagmus is stronger in the plane of head movement either to the right or left, No.33; 55%), 5 patients (15%) had migraine, 8 patients (24%) had VN, 1 patient (%3) had MD, 3 patients (9%) had BPPV and 16 patients (49%) had no disease. In group B (nystagmus is stronger against the direction of head movement, No.16; 27%), 2 patients (13%) had migraine, 4 patients (25%) had VN, 3 patients (%19) had MD and 7 patients (43%) had no disease. In group B (severity of nystagmus is equal in the right and left head movement, No.11; 18%), 3 patients (27%) had migraine, 3 patients (27%) had VN, 3 patients (27%) had MD and 2 patients (19%) had no disease.

up, 15 patients (25%) had vestibular neuritis, 10 patients (17%) had migraine and 7 patients (11%) had Meniere's disease and 3 patients (5%) had LC-BPPV. Two patients in group A and two patients in group B had mild ischemic gliosis on brain MRI. Five patients in group A and two patients in group C had mild bulging of cervical disc. None of the patients in groups had any MRI abnormality, space occupying or demyelinating lesion. Patients who have later diagnosed as Meniere's disease, migraine and vestibular neuritis received proper medical therapy.

4. Discussion

Unilateral vestibular dysfunction may present with DFPN. Young et al. (1992) have studied on an animal model of experimental perilymphatic fistula to investigate the vestibular pathophysiology. Spontaneous nystagmus directed toward the normal side was noted in 57.4% of animals during the acute stage. One week postoperatively, DFPN beating towards the lesioned ear was present in 22.7% of the animals. Choi et al. (2018) have reviewed 33 patients with idiopathic sudden hearing loss without dizziness and have observed nystagmus in 22 patients (67%). Of which 14 patients exhibited direction-fixed nystagmus (9 paretic, 5 irritative type). Direction-fixed positional nystagmus either beating to the healthy or to the pathologic side is associated with vestibular disorder in which the direction of nystagmus is simply related with the active or compensated phase of the inner ear. Kim et al. (2018a,b) have studied the incidence of positional nystagmus in 28 patients with Ramsey-Hunt syndrome having balance problem. He subjected his patients to the head-roll test. Direction-fixed nystagmus beating away from the affected side was the most common type (61%). Direction-fixed nystagmus beating toward the affected side was documented in 14% of patients. Fifty-three per cent of patients with DFPN in the presented study had peripheral vestibular dysfunction.

DFPN is correlated with head-shaking nystagmus and canal paresis in patients with peripheral vestibular pathology (Tseng and Chao, 1997; Takahashi et al., 1990). Provocation of nystagmus with head motion is related with vestibular imbalance. Head motion does not expose nystagmus in normal subjects and in patients with bilateral vestibular loss (Hain et al., 1987; Katsarkas et al., 2000). Appearance of nystagmus associated with head movement indicates vestibular asymmetry in neural firing. However, direction of provoked positional nystagmus could not always be correlated with the side of peripheral vestibular dysfunction. Head movement during positioning of the patient could provoke a neural firing from the labyrinth which would be expected to be direction-fixed with the fast phase of nystagmus directed towards (paretic) or against (recovery) the intact ear (Uemura et al., 1975). Nystagmus is usually directed to the intact ear in the acute phase. Recovery type nystagmus is mostly due to adaptation of vestibular function at the compensatory stage and indicates damaged canal function (Kim et al., 2018a,b). We assume that DFPN in the direction of head movement in patients from group-A indicates the intact ear. These findings in acute stage were confirmed in 8 patients with vestibular neuronitis and one patient with Meniere's disease in the follow-up period. On the other hand, nystagmus beating the opposite direction of the head movement is caused by adaptation of the vestibular system. Kim et al. have (2018a,b) reported that paretic direction-fixed nystagmus (towards the intact ear) is more common in acute vestibular neuritis than in follow-up period of patients with vestibular neuritis and Meniere's disease. They have found that recovery type direction-fixed nystagmus (beating against the intact ear) was more common in Meniere's disease than in acute and follow-up vestibular neuritis. Patients with vestibular neuronitis presented paretic type and patients with Meniere's disease presented recovery type positional nystagmus. It is interesting to note that chronic peripheral vestibular pathologies could also be associated with direction-fixed positional nystagmus (Park et al., 2009). We have found more patients with Meniere's disease presenting recovery type nystagmus.

Physiologic positional nystagmus associated with head rotation has been reported in normal subjects. Its intensity is quite low, and it is seen when the subject's head is moved rapidly to a constant-velocity rotation (Martens et al., 2016). It always occurs with the fast phases in the direction of rotation. On the other hand, pathologic process should be suspected when the positional nystagmus seen during head rotation on both sides is direction-fixed even if the intensity is very low. This kind of nystagmus is not expected in healthy subjects. It should be kept in mind that direction-fixed positional nystagmus can be a manifestation of central pathology (13). However, thermal or drug affect should be considered in some patients when radiological investigation is normal. MRI was normal in 25 patients with DFPN. Patients with DFPN who are resistive to medical therapy should be closely followed and care should be taken if there is gaze-evoked spontaneous nystagmus, skew deviation, impaired vertical smooth pursuit and normal vestibulo-ocular reflex with head impulse test which all together are sensitive than the presence of normal MRI at the early period (Kattah et al., 2009).

Califano et al. (2013) have reviewed 272 patients with lateral canal BPPV and reported 5 patients with DFPN as another form of lateral canal BPPV since they were able to transform all cases into geotropic type lateral canal BPPV through head pitch maneuver. The proposed pathophysiological mechanism for the apogeotropic nystagmus of the pathological side was ampullofugal flow of the otoliths located close to the cupula during lying down of the patients on the affected side. However, explanation of the geotropic nystagmus on the healthy side was more complex. The authors state that nystagmus on the healthy side was somehow linked to the endolymphatic flow of the pathologic side. The incidence of transformation of DFPN to lateral canal BPPV following head-shaking test in the presented series was low. We have documented only 3 patients who had initially persistent direction-fixed positional nystagmus. Geotropic type lateral canal BPPV was diagnosed following head-shaking and they were treated with barbeque maneuver.

One limitation for such retrospective studies could be the lack of

target-specific questioning of balance problem before subjecting them to the VNG analysis during first admission of patients. Forty per cent of patients in the presented series do not seem to develop any central or peripheral vestibular pathology and the diagnosis is unclear. Not all patients with DFPN necessarily develop further pathology. DFPN could just be an isolated finding for some reason. Another limitation is technical and can be experienced in the retrospective analysis of video recording of eye movements. Sometimes, resolution of infra-red cameras is not enough, and justification based on video data is misleading. Therefore, match-up between video of eye movement and graphics is important.

In conclusion, in some conditions, it is not possible to distinguish "positioning" or "positional" nystagmus that is the one caused by the head movement itself and the one caused by the final head position with respect to gravity (Von Brevern et al., 2015). Therefore, "positioning" nystagmus of lateral canal BPPV and "positional" nystagmus of vestibular pathology may be confused during head-roll maneuver. Nystagmus of lateral canal BPPV is provoked by the act of moving the head from one position to the next and eventually decays unless the debris is stuck in the canal (canalith jam). Head-shaking is recommended to expose the geotropic type lateral canal BPPV. But the chance was low in the presented series. DFPN can be indicative of vestibular asymmetry. Those patients should be followed for a possibility of vestibular pathology. However, there was no peripheral or central pathology in some patients during follow-up period. One may speculate that this could be due to unrelated temporary conditions affecting the vestibule or central vestibular pathways like drugs, infection, thermal or physical impacts.

Declaration of competing interest

This study was conducted at Anadolu Medical Center (Affl. Johns Hopkins Medicine), Dept of ORL & HNS, a regional community health center, Kocaeli - Turkey. We hereby declare that we don't have any conflicts of interest in the manuscript, including financial, consultant, institutional and other relationships that might lead to bias or a conflict of interest. This study has no grant or funding. All procedures performed in the study involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration. An informed consent was obtained from all individual participants included in the study.

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