

Anxiety affects vestibulospinal function of labyrinthine-defective patients during horizontal optokinetic stimulation

La comorbidità tra i disordini vestibolari e l'ansia sull'instabilità posturale indotta da stimolazione ottocinetica

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Key words

Vestibular disorders • Vestibular neuritis • Anxiety • Posturography • Optokinetic stimulation

Parole chiave

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Summary

Comorbidity of vestibular and anxiety disorders was suggested by epidemiological studies and, recently, new insights into potential neural circuits which subserve both balance control and emotions, appear to support this hypothesis. In particular, disorienting visual surroundings, such as those generated by full-field moving scenes, equally disrupt postural control of patients with vestibular or panic disorders. In the present study, behaviour of body sway was assessed in response to an optokinetic stimulation by means of static posturography in 20 patients with vestibular neuritis (10 patients with normal affect and 10 with generalized anxiety disorders, as diagnosed according to the American Psychiatric Association criteria), and 20 normal subjects who served as controls. Optokinetic responses and vestibulo-spinal function during a full-field, bi-directional horizontal optokinetic stimulation, were recorded simultaneously. Labyrinthine-defective patients with low and high level of anxiety showed a common pattern of asymmetric optokinetic reflexes. On the contrary, body sway was found to be increased more by eye closure and optokinetic stimulation towards the defective labyrinth in patients affected by high level of anxiety as compared to those with normal affect and controls. These data confirm the combined effect of anxiety and labyrinthine dysfunction on vestibulo-spinal function which is disclosed by both visual suppression and disorienting visual contexts.

Riassunto

La comorbidità tra disordini vestibolari e l'ansia è stata da tempo ipotizzata da studi sperimentali sia di tipo epidemiologico che neuroanatomico. In particolare sono stati recentemente identificati alcuni circuiti neurali del tronco ugualmente attivati sia dal sistema vestibolare afferente che dalle reazioni emotive. Sul piano comportamentale, è frequente il riscontro clinico di situazioni di conflitto sensoriale che si vengono a creare nel sistema nervoso centrale tra le informazioni provenienti dal sistema visivo, vestibolare e somatosensoriale come quelle generate da scene visive in movimento a tutto campo in grado di disturbare sia il controllo posturale dei pazienti vestibolopatici che di quelli affetti da agorafobia con attacchi di panico. In questo studio è stata eseguita l'analisi posturografica dell'oscillazione corporea in risposta ad una stimolazione ottocinetica a tutto campo in 20 pazienti affetti da neurite vestibolare in fase acuta, di cui 10 portatori di un disturbo d'ansia generalizzato e 10 privi di disturbi psicopatologici. Tale casistica è stata confrontata con 20 volontari sani. Le risposte ottocinetiche e l'attività vestibolospinale durante la stimolazione ottocinetica orizzontale e bidirezionale sono state simultaneamente registrate in due test stabilometrici. Si sono evidenziate asimmetrie del riflesso ottocinetico indipendenti dal livello di ansia e, all'opposto, il controllo posturale è risultato statisticamente differenziato in funzione delle diverse condizioni visive d'esame. Ad occhi aperti, con fissazione visiva di un target immobile, non si sono rilevate differenze tra i pazienti vestibolopatici con differenti livelli d'ansia ed i controlli, mentre ad occhi chiusi l'instabilità posturale è risultata aumentata nei vestibolopatici non patologicamente ansiosi rispetto ai normali e nei vestibolopatici con disturbo d'ansia generalizzato rispetto a quelli senza turbe dell'emotività. Inoltre la stimolazione ottocinetica verso il lato labirintico deficitario è risultata in grado di indurre una maggiore destabilizzazione rispetto a quella controlaterale e tale effetto è risultato più marcato nei pazienti affetti sia da neurite vestibolare che da disordine d'ansia generalizzato. Tali dati confermano non solo l'effetto destabilizzante dei pattern visivi in movimento nei soggetti affetti da vestibolopatia periferica, ma supportano ulteriormente l'esistenza di comorbidità tra disordini vestibolari e nuclei psicopatologici abnormi sulla funzione dell'equilibrio in determinate situazioni ambientali con potenziali ripercussioni negative sia sul compenso vestibolare che sul piano comportamentale del paziente labirintopatico.

Introduction

Optokinetic stimulation (OKS), in man, produces a deterioration of postural control^{1,2} which is held to depend on a sensory conflict, within the central nervous system (CNS), between visual, vestibular and somatosensory information. Indeed, a full-field moving visual scene generates, in an observer, during quiet stance, a sensation of self-motion (vection) which is contradictory with the perception of stationarity provided by otolithic and somatosensory receptors³. Peripheral vision, rather than the central field, appears to be the more sensitive to moving visual patterns and responsible for the increased postural sway. Despite this ambiguity between the primary senses, the increase in body sway induced by OKS appears to be very limited in healthy individuals^{4,5} whose intact CNS is able to resolve the sensory mismatch and provide correct postural adjustments. Even if little information is available regarding the neural basis for this central processing of conflicting sensory signals⁶, it is thought that the CNS might reduce the weight (the "down-regulation" phenomenon) of misleading visual information and rely much more on appropriate vestibular and somatosensory signals for adaptive postural control. As indirect confirmation of this "reweighting" of sensory inputs, operated by the CNS in response to disorienting visual environments, recent studies have shown that patients with Parkinson's disease⁶, cerebellar damages⁵ and the elderly^{7,8}, i.e., subjects with disrupted or reduced integrity of the central neural pathways, find great difficulty in coping with moving visual patterns and present a pronounced body sway.

The increase in body sway, in response to optic flow, has been investigated also in patients with peripheral vestibular disorders⁹, since their postural control was shown to be challenged more by moving visual scenes, in the acute stage and to be less sensitive during compensation¹⁰, the intercritical period⁵ and after repeated exposure to optokinetic stimulations¹¹. Taken together, this experimental evidence suggests that both central and peripheral vestibular disorders could, potentially, produce an increased perceptual and postural instability, the so-called "visual vertigo syndrome"¹² in patients who are exposed to moving visual patterns such as those occurring in supermarkets, crowds and traffic. However, the question of whether the optokinetic-induced destabilization of posture, in patients with peripheral vestibular disorders, is specific to the direction of the visual motion and/or to the side of the lesion, has not been systematically investigated.

Another interesting point is that also patients with anxiety disorders¹³, agoraphobic and panic symptoms¹⁴ are destabilized by conflict between visual and vestibulo-proprioceptive inputs, thus supporting the hypothesis that abnormal psychological factors

could reduce the adaptability of the visuopostural system and lead to a balance disorder while subjects are viewing moving scenes.

Thus, it is not unreasonable to suspect that anxiety and vestibular disorders might have a synergic effect on balance function when discrepancy between proprioceptive, vestibular and visual sensory information is present.

The aim, therefore, of the present study was: a) to provide a detailed description both of body sway amount and optokinetic reflexes induced by bi-directional, horizontal, full-filled stimulation in unilateral labyrinthine defective-patients in the acute stage, and b) to establish whether anxiety and vestibular disorders are comorbid on vestibulospinal function.

Material

The study population comprised 20 patients with sudden unilateral peripheral vestibular loss due to vestibular neuritis, diagnosed according to Strupp's criteria¹⁵, recruited within 48 hours from the onset of vertigo. All were, therefore, clinically characterized by complaints of rotatory vertigo of acute onset, nausea and/or vomiting, persisting postural destabilization, horizontal spontaneous nystagmus away from the labyrinthine-defective side, a tendency to fall towards the affected side and absence of otologic symptoms.

This sample was selected from 1072 consecutive patients, referred to the Centre for Vestibular Testing of the University Hospital of Modena from the Emergency Unit, during the period 2001-2002, and included in the study only if they fulfilled the criteria of the American Psychiatric Association (Diagnostic and Statistical Manual, 4th Revision, DSM IV)¹⁶ for psychiatrically healthy individuals (Group A; 10 patients) or patients with general anxiety disorders (Group B: 10 patients). This study population included 9 males (45%) and 11 females (55%) of comparable age (males: 20-38 years, mean 32.1; females: 22-40 years, mean 37.4). Patients with a history of previous balance problems and/or vestibular dysfunction, impaired vision and neurological diseases were excluded. Patients with anxiety disorders were free of psychotropic medication for one week before examination. Limited use of anti-emetics, after hospitalisation, was not considered as exclusion criteria. Patients were compared with 20 healthy volunteers (N), for control purposes, matched according to sex (10 males, 50% and 10 females, 50%) and age (males: 24-38 years, mean 33.1; females: 20-40 years, mean 34.7). Healthy subjects were recruited among hospital staff personnel and students on the basis of a normal vestibular examination and no history of balance problems or psychological distress.

The experimental protocol was consistent with the

Declaration of Helsinki for Human Experimentation and informed consent was obtained from all individuals before entering the study.

Methods

POSTUROGRAPHY

Static posturography was performed in all subjects by means of a computerized stable force-plate sensitive to vertical force (S.Ve.P Amplifon)¹⁷. The force-plate was mounted on three strain-gauge force transducers which are positioned at the vertices of an equilateral triangle, providing description of body sway in terms of displacement of the centre of pressure of the patients (i.e., approximately the projection of the centre of mass to the ground). Posturographic recordings were performed in standardized conditions: both healthy controls and patients were requested to maintain a relaxed, motionless upright stance, stand bare foot at a forward open angle of 30°, with a natural head-neck posture, both arms hanging beside the trunk, in 4 consecutive trials: 1. gazing at a steady, vertical light bar in front of them, at a distance of 150 cm (EO), 2. with eyes closed (EC) in total darkness, 3-4. facing the wall in front, on which a special projector provided a full-field horizontal OKS which was represented by vertical alternating dark and light stripes, 30 cm in diameter, moving at a 30°/s velocity (Fig. 1). Patients were instructed to look carefully at the moving stripes and to mentally count them; they were also informed that they would be asked the result after each test. This mental arithmetic task was added in order to avoid a decrease of attention which could deteriorate the optokinetic reflex¹⁸. The direction of the OKS was, firstly, delivered towards the intact labyrinth and then to the affected one, in two separate trials. This order of presentation was selected to avoid any hypothetical improvement of postural control due to the 'learning effect'¹⁹. The direction of the OKS was, firstly, delivered to the right side in the first 10 normal controls and to the left, in the others. The duration of each test was 30 seconds. Stabilometric data were sampled at 10 Hz in each test. Sway path was computed by continuously detecting the body's centre of pressure and calculating an elliptic area corresponding to 90% of the positions of the centre of pressure over time. This procedure was designed to eliminate 10% of the more extreme positions which could be due to involuntary perturbations of quiet stance. The mean magnitude of sway path (expressed in square millimetres) was computed, in each trial, and a stabilometric Romberg's quotient was calculated using the following formula:

(sway path with eye closed/ sway path with eye open)

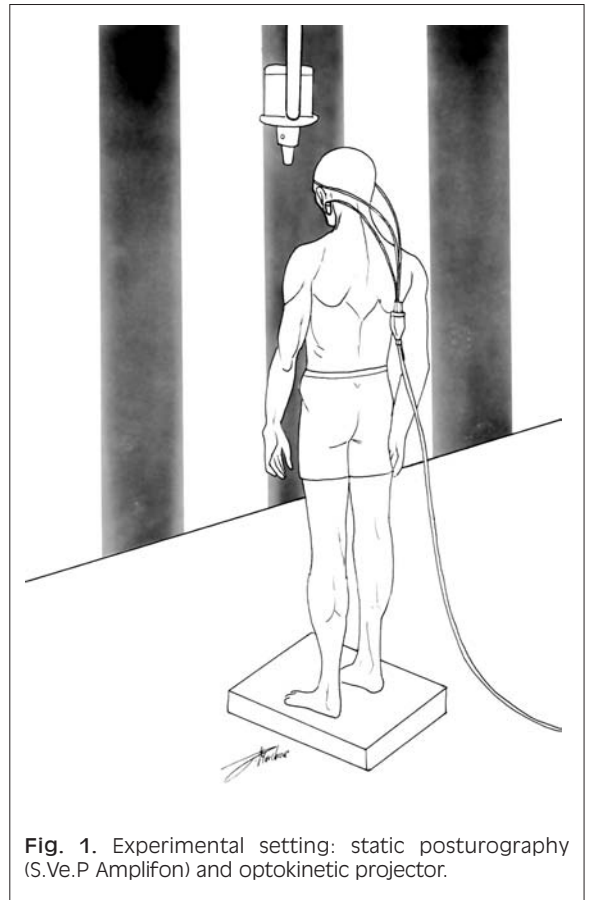


Fig. 1. Experimental setting: static posturography (S.Ve.P Amplifon) and optokinetic projector.

which indicates the weight of static visual cues on postural sway control.

QUESTIONNAIRES

Before taking part in the study, all patients and controls were submitted to a structured diagnostic interview for DSM-IV Axis I Anxiety disorders (SCID-I), Italian version²⁰, by a well-trained psychologist, under the supervision of a hospital psychiatrist. All subjects were invited to complete the Spielberger State-Trait Anxiety Inventory²¹, Italian version²² that is a validated psychometrically self-report measure of anxiety which provides separate indications on how much subjects *habitually* experience anxious feelings (trait) and their emotional reaction to a specific situation, *at the present time* (state). At this point, subjects were instructed to complete the trait portion of the questionnaire before examination and to state one to assess the level of their discomfort immediately after exposure to OKS. Each portion is a 20-item list and items are rated on a scale from 1 to 4 (absent, weak, moderate, strong). After reversing scores on items where a high score indicates low anx-

ity, the summed score for each scale ranges from 20 (minimum anxiety) to 80 (maximum anxiety).

ELECTROCULOGRAPHY

During the third and fourth posturographic trials (i.e., under bi-directional OKS), horizontal eye movements were recorded by an analogue-digital converter which provided averaged values for the slow phase velocity, expressed in angular degree/second² (SPV), and for the total number of fast phases (FP) of the optokinetic responses over a period of 15 seconds.

STATISTICS

Optokinetic and posturographic differences between psychologically disturbed patients, patients with normal affect and normal controls were analysed with the ANOVA (analysis of variance) procedure and post-hoc analysis between groups was obtained by Bonferroni's test. All data were expressed as mean \pm standard deviation. The State-Trait Anxiety scores were compared by the Kruskal-Wallis test for non-parametric variables $p < 0.05$ was considered significant in all procedures.

Results

As expected, the mean score for trait anxiety was increased in group B (53.2 ± 14.1) as compared to group A (41.6 ± 5.3) and controls (39.6 ± 6.9) (Kruskal-Wallis' $H = 0.000$), with no relevant difference between the latter (Table I). Also state anxiety was markedly increased in psychologically disturbed patients (60.1 ± 11.2), as compared to patients with normal affect (39.7 ± 6.7) and controls (38.2 ± 6.9) (Kruskal-Wallis' $H = 0.016$). Since state anxiety is defined as a perturbed emotional condition characterized by a transient feeling of tension and apprehension which could be triggered by particular situations perceived as threatening, this result clearly indicates that a full-field optokinetic stimulation promotes a more intense emotional reaction in patients affected by anxiety disorders than those with a normal affect.

The quantitative analysis of optokinetic parameters showed a significant increase of both SPV ($F = 22.8$, $p < 0.000$) and FP ($F = 3.8$, $p < 0.05$) in response to the visual stimulation towards the affected labyrinth in the two pathological groups, as compared to controls (*post-hoc analysis*: $p < 0.000$ and $p < 0.05$, respectively) (Table II), but no difference was observed between the two pathological samples (*post-hoc analysis*: $p = 1.0$ and $p = 0.87$, respectively) thus confirming that different levels of anxiety were not associated with a significant variation in the optokinetic responses between labyrinthine-defective patients with normal and perturbed affect. On the contrary, no differences in the optokinetic parameters were noted between the groups, in response to the optic flow directed to the intact side ($p > 0.05$). This behaviour was attributed by previous experiments to spontaneous nystagmus which, in the acute stage, enhances both slow and fast phases of optokinetic reflex when the direction of the optical flow is delivered to the defective labyrinth^{23 24}.

Furthermore, no difference in the sway path was observed between patients and controls in the EO condition ($F = 2.4$, $p > 0.05$). On the contrary, body sway varied significantly between groups in the EC condition ($F = 33.4$, $p < 0.000$). A more pronounced sway path was demonstrated for group B, in the EC condition, as compared both to group A and controls (*post-hoc analysis*: $p < 0.000$). Furthermore, a greater amount of sway path was observed in group A as compared to normal subjects (*post-hoc analysis*: $p = 0.012$). This result was corroborated by the behaviour of Romberg's quotient, the mean values of which varied between groups ($F = 10.2$, $p < 0.000$); this resulted increased in group B with respect both to group A (*post-hoc analysis*: $p = 0.020$) and controls (*post-hoc analysis*: $p < 0.000$); moreover, also group A showed a greater postural instability than controls (*post-hoc analysis*: $p = 0.038$) (Table III). These results clearly indicate that neither anxiety nor labyrinthine hypofunction affect body sway when static visual cues are available and, furthermore, that eye closure (i.e., no visual information available)

Table I. Mean scores for trait and state anxiety (Spielberger State-Trait Anxiety Inventory) during optokinetic stimulation in patients with and without generalized anxiety disorders (groups B and A) and controls.

Variable	Group						p
	Controls		A		B		
	Mean	SD	Mean	SD	Mean	SD	
Trait anxiety	39.6	6.9	41.6	5.3	53.2	14.1	0.000
State anxiety	38.2	6.9	39.7	6.7	60.1	11.2	0.016

Table II. Optokinetic parameters (mean slow phase velocity and total number of fast phases) during bi-directional visual stimulations in groups A and B and controls.

OKN stimulation	OKN responses		Controls	A	B	p
Away from lesion (or to left)	SPV	Mean	23.2	21.5	24.1	0.603
		SD	7.5	3.9	4.5	
Towards lesion (or to right)	SPV	Mean	23.5	36.0	34.7	0.000
		SD	7.0	3.1	3.9	
Away from lesion (or to left)	FP	Mean	58.9	57.1	63.7	0.163
		SD	8.5	7.8	5.5	
Towards lesion (or to right)	FP	Mean	59.0	64.2	68.3	0.029
		SD	9.2	8.3	7.4	

promotes an increase in postural imbalance which could be related to a dual interference of sensory loss and abnormal emotional condition on vestibulo-spinal function.

Posturographic data also demonstrated a significant variation in patients' sway path in the trial with OKS towards the affected side ($F=32.5$, $p<0.000$) and no difference with OKS to the intact one ($F=1.8$, $p>0.05$). Conflicting sensory cues actually caused a destabilization of posture which was higher in group A patients than in controls (*post-hoc analysis*: $p=0.07$); a further increase in body sway was observed between B and A patients (*post-hoc analysis*: $p<0.000$) (Table III). These results are not only a confirmation of previous experimental data documenting deterioration of postural control due to optic flow in patients with peripheral vestibular disorders⁹, but al-

so indicate an additional contribution of anxiety to the impairment of vestibulo-spinal function.

Discussion

Results of this study confirm that patients with peripheral vestibular deficiency in the acute stage show asymmetry of the optokinetic reflexes, in response to a full-filled visual stimulation, which is due to an increase in slow phase velocity and frequency of the fast phases when the optic flow is delivered towards the affected labyrinth. The influence of the asymmetry of vestibulo-ocular reflex on optokinetic reflex is a further demonstration of the functional synergy between these two oculomotor subsystems²⁵. It also revealed a simultaneous increase in postural imbalance

Table III. Posturographic parameters (sway path) in trials under different visual conditions (eye open, eye closed, Romberg's quotient, bi-directional optokinetic stimulation); (A = psychologically intact patients, B = patients with generalized anxiety disorders).

Posturography		Control	A	B	p	
Eye open	Sway path	Mean	184.9	206.9	233.6	0.166
		SD	66.7	63.2	55.7	
Eye closed	Sway path	Mean	229.8	354.5	582.6	0.000
		SD	66.8	85.5	183.8	
Romberg's quotient		Mean	1.3	1.8	2.5	0.010
		SD	0.3	0.4	0.6	
OKN towards lesion (or to right)	Sway path	Mean	195.6	357.0	623.1	0.000
		SD	53.7	99.2	249.0	
OKN away from lesion (or to left)	Sway path	Mean	193.4	217.6	238.9	0.172
		SD	54.0	62.2	72.9	

as revealed by static posturography when the patient's eyes are directed by visual stimulation towards the affected side. Since it has been demonstrated that an interaction of visual object motion and vestibular self-motion exists in man²⁶, it is tempting to suggest that the vestibulo-spinal impairment, due to a sudden loss of peripheral vestibular function, could be further challenged by visually induced self-motion perception (vection). The neural correlates for this interaction, in man, has been explored by recent functional magnetic resonance imaging studies which indicate that the parieto-insular vestibular cortex responds to both vection²⁷ and vestibular stimulation²⁸, thus suggesting the existence of cortical areas where vestibular and visual motion information converge for orientation in space and appropriate motor response.

No significant difference was shown concerning the asymmetry of the optokinetic reflex in relation to the degree of anxiety. However, it is worthwhile pointing out that this experimental setting was not specifically designed to address this issue.

The role that anxiety plays on vestibulo-spinal dysfunction, depending on the three different visual conditions, appears, to a large extent, to be consistent with previous observations concerning the interface between psychological distress with balance function²⁹. When static visual cues are available, no postural differences, between patients with low and high level of anxiety and controls, were noted thus suggesting that the contribution of the underlying foveal mechanism (retinal slip of images due both to self-motion and object motion) to balance function is not substantially disrupted either by a deficient sensory functioning³⁰, or by an abnormal emotional condition. On the contrary, labyrinthine-defective patients were more destabilized by eye closure than controls and this behaviour indicates a reduced postural control in response to a combination of disrupted vestibular information and absence of retinal signal as recently indicated by Guerraz et al.³¹. Taken together, these results clearly confirm that postural control involves a dynamic central processing of peripheral sensory inputs which promotes a reweighing of the retinal signal whereas vestibular information is misleading³². In a similar 'visual dependence' condition, a remarkable destabilization of posture, as a consequence of eye closure, is likely. Furthermore, anxious patients showed a greater sway path when tested in EC conditions than those with no disturbed affect. This further deterioration of balance control could be attributed to the phobic stimulus, generated, in anxious patients, by eye closure, i.e., whenever they could no longer operate the conscious and 'overdimensioned' visual vigilance of the surroundings³³. Finally, the most intriguing result, emerging from this study, is the different postural reaction across

groups in response to moving visual cues. It was confirmed that patients with vestibular disorders⁹ showed an increased postural imbalance with respect to controls in a perceptual conflicting situation. Since patients were also invited to perform an arithmetic mental task, while standing on the force-plate, it is possible that attentional demands for postural control and mental task performance compete the same cortical processing resources^{19 34} for orientation when vestibular information is impaired. Moreover, since a greater destabilizing effect was observed in labyrinthine-defective patients, with a higher level of anxiety, it could be argued that also emotional arousal, due to disorienting visual surroundings, affects central elaboration of balance information on account of the well-known interference between cognitive and emotional processes³⁵. However, the interference between anxiety and vestibulo-spinal function, as shown by our data, could be observed only when the optokinetic visual stimulation was delivered to the defective labyrinth and this result provides a new clinical insight into co-morbidity between psychological factors and vestibular disorders on balance function^{36 37} which has recently been supported by the identification of a common neurological circuitry in the brainstem which involves direct connections between vestibular nuclei, para-brachial nucleus and the amygdala³⁸ which is considered a crucial structure for pathological anxiety. It is tempting to conclude that since opto-kinetic reflexes were not significantly different between patients with a high and low level of anxiety. This study indicates that co-morbidity between vestibular and anxiety disorders is suggested only as far as concerns the vestibulospinal function.

Conclusions

The occurrence of somatic symptoms, such as dizziness and imbalance, often reported by patients with vestibular dysfunction, when exposed to repetitive, moving visual scenes, corresponds not only to a perceived, but also to an actual postural instability. The possibility of a consequent increase in untoward symptoms could lead these patients to avoid complex physical activities and provocative sensorial situations, such as shopping in supermarkets, driving in traffic, walking in a crowd or taking lifts, generally regarded as necessary for adaptation and/or extinction of symptoms caused by a vestibular lesion. These avoidance behaviours, at the same time, could be even more evident in patients with vestibular disorders and high level of anxiety and phobic reaction, the postural stability of which is demonstrated to be much more challenged by disorienting visual situations. Therefore, results of this study appear to sug-

gest that the comorbidity of vestibular/anxiety disorders, on balance function, could be correctly approached only if both components are adequately investigated and treated. On the other hand, since also psychiatrically anxious patients are equally destabilized by sensory conflicting situations, a possible underlying vestibular dysfunction should be investigated which might provoke reduced adaptability of the

visuo-postural system thus providing the neural substrate for the perpetuation of their dizziness (visual and phobic vertigo). Finally, since static posturography with a full-filled optokinetic stimulation provides a valid analysis of an otherwise not measurable visually-induced balance disturbance, it should be performed to test patient's postural adaptability to dynamic visual cues.

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