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# WHO IS AT RISK FOR ONGOING DIZZINESS AND PSYCHOLOGICAL STRAIN AFTER A VESTIBULAR DISORDER?

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**Abstract—Patients with vestibular vertigo syndromes often suffer from anxiety and depression, whereas patients with psychiatric disorders often experience subjective unsteadiness, dizziness, or vertigo. Thus, it has been hypothesized that the vestibular system may be interlinked with the emotion processing systems. The aim of the current study was to evaluate this hypothesis by correlating vestibular and psychiatric symptoms with the course of the disease over 1 year. This interdisciplinary, prospective, longitudinal study included a total of 68 patients with acute vestibular vertigo syndromes. Four subgroups of patients with benign paroxysmal positioning vertigo (BPPV, *n*=19), acute vestibular neuritis (VN, *n*=14), vestibular migraine (VM, *n*=27), or Menière's disease (MD, *n*=8) were compared. All patients underwent neurological and neuro-otological examinations and filled out standardized self-report inventories including the Vertigo Symptom Scale (VSS), the Vertigo Handicap Questionnaire (VHQ) and the Symptom Checklist 90R (GSI, SCL-90R) at five different times (T0–T4) in the course of 1 year. VM patients experienced significantly more "vertigo and related symptoms" (VSS-VER), "somatic anxiety and autonomic arousal" (VSS-AA), and "vertigo induced handicap" (VHQ) than all other patients (*P*<0.001–*P*=0.006). Patients with a positive history of psychiatric disorders had significantly more emotional distress (GSI, SCL-90R), regardless of the specific phenomenology of the four diagnostic subgroups. Finally, fluctuations of vestibular excitability correlated positively with the extent of subjectively perceived vertigo. VM patients are significantly more handicapped by vertigo and related symptoms. They show significantly elevated fluctuations of vestibular excitability, which correlate with the (subjective) severity of vertigo symptoms.** 

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*Abbreviations:* BPPV, benign paroxysmal positioning vertigo; GSI, global severity index; MD, Menière's disease; SCID, structural clinical interview for DSM-IV; SCL-90R, symptom checklist 90R; SD, standard deviation; SPV, slow phase velocity; SVV, subjective visual vertical; VHQ, vertigo handicap questionnaire; VM, vestibular migraine; VN, vestibular neuritis; VSS, vertigo symptom scale.

**Key words: vertigo, vestibular disorders, emotional distress, anxiety, depression.**

Vertigo and dizziness symptoms are two of the most common reasons why patients consult their general practitioner or are referred to a hospital. After headache and lower back pain, vertigo and dizziness symptoms are the most frequent complaints of patients seeking the advice of a neurologist (Brandt, 1996). The underlying causes of the complaints vary largely and include (a) peripheral and central vestibular disorders, (b) non-vestibular but organic disorders, and finally (c) non-organic, so-called psychiatric and somatoform disorders. A large percentage of patients with vestibular vertigo syndromes develop secondary psychiatric disorders in the course of their disease (Eagger et al., 1992; Clark et al., 1994; Eckhardt-Henn et al., 2008). The subjectively perceived symptoms of dizziness and vertigo persist longer in these patients than does the acute stage of the vestibular disease. Therefore, they are at risk of long-lasting dizziness, disease-specific handicap, and reduced psychosocial functioning (Mendel et al., 1999; Luxon, 2004). Due to their high prevalence, these risks pose a large societal problem.

Since vestibular deficits are a risk factor for the development of secondary psychiatric disorders (Jacob and Furman, 2001), a somatopsychic hypothesis has been postulated. A somatopsychic effect in this context is defined as a pathological influence of an organic disorder or symptom on the individual patient's mind which results in the persistence of vertigo symptoms after the underlying organic disorder is resolved. Conversely patients with psychiatric disorders often report dizziness as a concomitant phenomenon of their illness (Clark et al., 1992; Jacob and Furman, 2001; Yardley et al., 2001a). This interlinkage of vestibular and psychiatric symptoms in dizziness patients is the subject of an ongoing debate. Neuroanatomical connections between vestibular stimuli and the emotional response processing systems are thought to explain the extraordinary high rates of co-existence and co-morbidity observed clinically in patients with organic and psychiatric dizziness (Furman et al., 2005).

Contrary to studies reporting that vestibular deficits trigger psychiatric disorders (Jacob and Furman, 2001), cross-sectional studies of vestibular deficits found no impact on the extent of emotional distress in patients with dizziness (Perez et al., 2003; Staab and Ruckenstein, 2003; Best et al., 2006). Distress, however, was significantly elevated in patients with vestibular migraine (VM), Menière's disease (MD), and primary psychiatric dizziness, but it was independent of the amount of the vestib-

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**Table 1.** Demographic data

| | N | % | Age (y) | | | Sex | |
|---|---|---|---|---|---|---|---|
| | | | Mean | SD | Range | Female | Male |
| BPPV | 19 | 27.9 | 55.64 | 13.41 | 25–71 | 11 | 8 |
| Vestibular neuritis (VN) | 14 | 20.6 | 53.40 | 13.89 | 19–70 | 9 | 5 |
| Menière's disease (MD) | 8 | 11.8 | 59.24 | 10.81 | 42–73 | 2 | 6 |
| Vestibular migraine (VM) | 27 | 39.7 | 45.67 | 9.82 | 33–69 | 11 | 16 |
| Total | 68 | 100 | 51.46 | 12.72 | 19–73 | 33 | 35 |

ular deficit (Best et al., 2006; Eckhardt-Henn et al., 2008). Our working group recently found that patients with VM had the highest rates of psychiatric disorders within a 1 year follow up. Again, the amount of the vestibular deficit and dysfunction over the course of time had no impact on the development of secondary psychiatric disorders (Best et al., 2009).

The aim of the current study was to analyze the relationship between vestibular disorders, disease-specific handicap, and emotional distress in a 1 year follow up study. Our objective was to identify risk factors for ongoing dizziness and elevated psychological strain. Therefore, the following questions were of special interest: (1) Did the various organic vertigo syndromes differ in the occurrence of psychological strain? (2) Did the psychiatric history of the patients correlate with the subjective impairment after the onset of the vestibular disorder? (3) Did the severity of the vestibular deficit correlate with vertigo-induced handicap, subjectively perceived dizziness, or emotional distress?

## EXPERIMENTAL PROCEDURES

A total of 68 patients with vestibular vertigo syndromes (benign paroxysmal positioning vertigo (BPPV, $n=19$); vestibular neuritis (VN, $n=14$); vestibular migraine (VM, $n=27$); Menière's disease (MD, $n=8$)) were recruited for this interdisciplinary longitudinal study (Table 1). Only two patients who fit all inclusion criteria refused to participate in the study because of the remoteness of our clinic. None of the 68 included patients dropped out.

The aim was to include patients as early as possible after the onset of their disease in order to reduce effects of recurrence and chronicity. VN patients were recruited within the first days of disease onset. VM, MD, and BPPV patients were included in the study, if their vestibular vertigo syndrome had lasted less than 6 months. All patients underwent detailed diagnostic procedures in the Departments of Neurology and Psychosomatic Medicine and Psychotherapy after having given their informed written consent. The study was in accordance with the Helsinki Declaration and was approved by the local ethics committee.

### Inclusion and diagnostic criteria

*BPPV.* BPPV is the most common cause of peripheral vestibular vertigo. It is a benign disorder, in which mainly degenerative, dislocated otolith particles (otoconia) are trapped inside one of the semicircular canals. The posterior semicircular canal is affected most often, but also the horizontal and rarely the anterior semicircular canals can be affected. When the otoconia are inside one of the semicircular canals, body and/or head movements parallel to the plane of the affected canal induce an ampullofugal vection or ampullopetal deflection of the endolymph, which activates the macular hair cells. These patients develop nystagmus. If they have the typical complaints of a positioning vertigo, BPPV is diagnosed. In addition, the Dix-Hallpike head positioning test[1] must be positive; if it was negative, patients were not included in the study.

*VN.* VN accounts for the second most common cause of peripheral vestibular vertigo. Evidence supports the view that VN is induced by reactivation of herpes simplex virus type 1. After an acute or sub-acute onset patients suffer from a permanent rotatory vertigo with a spontaneous horizontal-rotatory nystagmus. The main symptoms are rotational vertigo, oscillopsia, unsteadiness of stance and gait, nausea with or without vomiting, and an intense vegetative activation (increased heart rate, sweating, raised blood pressure). VN was diagnosed in our patients, if (a) the patients had typical complaints, (b) the clinical neurological examination revealed spontaneous nystagmus and ipsilateral body tilts, and (c) neuro-otological evaluation yielded a pathological head-impulse test, ipsilateral tilts of the subjective visual vertical, ocular torsion, and additional caloric hyporesponsiveness.

*VM.* VM is an important subtype of migraine syndromes. In clinical practice, it is the most common cause of central vestibular vertigo. The individual course of the disorder varies greatly. Attacks can last from seconds, to minutes, and up to hours; they can re-occur with a very low frequency of a few attacks yearly or with a very high frequency of weekly attacks. As no internationally IHS criteria for VM are currently available, a diagnosis of VM was established in patients with episodic vestibular symptoms according to Neuhauser and Lempert (Neuhauser et al., 2001) if they had (a) a positive history or positive family history of common migraine according to the criteria of the International Headache Society, (b) migrainous symptoms occurred in at least two attacks of vertigo (e.g., hemicranial headache, photo- or phonophobia), and (c) typical migraine-precipitants (food triggers, sleep irregularities, or hormonal changes) were present and the patients responded positively to migraine medication in more than 50% of the attacks (reduction of attack duration, intensity, or attack frequency).

*MD.* MD is caused by an imbalance of secretion and absorption of endolymph which results in recurrent episodes of an endolymphatic labyrinthine hydrops, with periodical rupturing of the membrane separating the endolymph space from the perilymph space. The overflow of the potassium-rich endolymph into the perilymphatic space leads to a potassium-induced depolarization of the vestibulocochlear nerve. Attacks are characteristically abrupt with vestibular and/or cochlear symptoms; they fluctuate with slowly progressive hearing reduction and tinnitus, and persist for a minimum of 20 min or up to hours. MD was diagnosed

[1] Dix-Hallpike head positioning test: Testing for BPPV due to canalolithiasis of a posterior semicircular canal. While the patient is sitting, his head is turned by 45° to one side, then the patient is rapidly put in a supine position with the head hanging over the end of the examination couch. In case of a positive Dix-Hallpike test, a typical crescendo-decrescendo nystagmus with rotatory movement towards the tested canal can be induced.