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Psychogenic Forms of Vertigo and Dizziness

**Introduction**

Somatoform disorders play a causal or contributory role in a large portion of patients presenting with complex forms of dizziness. In the course of their illness, even after several years, about 70% of these patients with complex somatoform dizziness still show symptoms and are more impaired in their professional and daily activities than those with organic forms of dizziness (Furman and Jacob 1997; Yardley and Redfern 2001; Eckhardt-Henn et al. 2003). The most frequent underlying psychiatric disorders are anxiety and depression as well as dissociative somatoform (ICD-10:F45) disorders.

Somatoform dizziness first occurs without psychopathological symptoms. Most often this causes the patients to go to otolaryngologists, neurologists or internists. The patients describe experiencing frequent postural imbalance or a diffuse feeling of dizziness (a feeling of numbness, light-headedness, unsteadiness when walking, a feeling of toppling over) or very rarely rotatory vertigo with accompanying vegetative symptoms and nausea. Depending on the underlying psychiatric illness (see above), the following additional symptoms can be present: disorders of motivation and concentration, decline in performance, restrictions in professional and daily activities that are subjective, vegetative symptoms that accompany the dizziness (accelerated heart rate, nausea, sweats, apnoea, fear of suffocating, loss of appetite, weight loss), emotional and mood disorders, sleep disturbances and symptoms of anxiety. Typically all of these symptoms are
experienced. The patients believe that the symptoms are triggered and induced by the dizziness. Patients seldom spontaneously report conflict and stress situations that can function as triggers of vertigo/dizziness; often they are initially totally unaware of them. This makes it difficult to establish the diagnosis.

The treatment depends on the clinical picture. Psychotherapy should be begun. Psychodynamic and also behavioural therapy are advisable; the choice depends on the clinical finding and the underlying conflict or stress situation. Outpatient therapy focused on the leading symptom can be quite successful in cases of short-term dizziness that is not very pronounced. Long-term techniques should be selected depending on the underlying conflict situation (e.g., psychoanalysis). For subjects with a strongly pronounced disorder and considerable suffering, we recommend combination therapy with a psychoactive drug; the drugs of choice are preparations belonging to the serotonin-reuptake inhibitors (e.g., paroxetine, citalopram or sertraline). In very few patients it is necessary to initially supplement these drugs with an anxiolytic drug (e.g., lorazepam) for a short time.

In the following section, phobic postural vertigo, an important form of somatoform dizziness and the second most common form of vertigo (see Table 1.1, p. 5), is discussed.

5.1 Phobic Postural Vertigo

5.1.1 Patient History

The cardinal symptoms and features of phobic postural vertigo include the following (Brandt and Dieterich 1986; Huppert et al. 1995; Brandt 1996):

- Patients complain about postural dizziness and subjective postural and gait unsteadiness without this being visible to an observer.
- Dizziness is described as a numbness with varying degrees of unsteadiness of posture and gait, attack-like fear of falling without any real falls, in part also unintentional body swaying of short duration.
- The attacks often occur in typical situations known to be external triggers of other phobic syndromes (e.g., large crowds of people in a store or restaurant, bridges, driving a car, empty rooms).
- During the course of the illness, the patient begins to generalise the complaints and increasingly to avoid the triggering stimuli. During or shortly after the attacks (frequently mentioned only when asked), patients report anxiety and vegetative disturbances; most also report attacks of vertigo without anxiety.
If asked, patients frequently report that the complaints improve after imbibing a little alcohol and during sports.

Frequently at the beginning, there is an organic vestibular illness, e.g., resolved vestibular neuritis, benign paroxysmal positioning vertigo (Huppert et al. 1995) or psychosocial stress situations (Kapfhammer et al. 1997).

Patients with phobic postural vertigo often exhibit obsessive–compulsive and perfectionistic personality traits and during the course of the disease reactive–depressive symptoms.

5.1.2 Clinical Aspects and Course of the Illness

The combination of postural vertigo with subjective instability of posture and gait in patients with normal neurological findings in vestibular and balance test results (otoneurological examination, electronystagmography including caloric irrigation, posturography) or disorders that cannot explain the complaints, and a compulsive personality structure, is characteristic. The monosymptomatic subjective disorder of balance is connected with standing or walking, manifests with attack-like worsening that occurs with or without recognisable triggers and with or without accompanying anxiety. The absence of recognisable triggers or vertigo without accompanying anxiety causes many patients and the doctor treating them to doubt the diagnosis of a somatoform disorder.

Patients with phobic postural vertigo generally have a compulsive primary personality (in the sense of “pronounced personality traits”) and a tendency to intensified introspection and the need “to keep everything under control”. They are more likely to be ambitious and place high demands on themselves, and are often easily irritated and fearful.

Such patients rarely go to a psychiatrist first; they tend to see the “specialist” for their symptom, especially as they feel themselves to be organically sick. However, as phobic postural vertigo is not yet part of the diagnostic repertoire of most neurologists and otolaryngologists, the illness often lasts quite a long time before a diagnosis is established (a mean of 3 years for 154 patients with phobic postural vertigo; Huppert et al. 1995). Diagnosis is established only after a number of visits to different specialists, superfluous laboratory examinations, and erroneous classifications such as “cervicogenic vertigo” or “recurrent vertebrobasilar ischaemia”, with correspondingly unsuccessful treatment attempts. A psychiatric longitudinal study confirmed that phobic postural vertigo is a unique medical entity, which can be clearly differentiated from panic disorder with or without agoraphobia (Kapfhammer et al. 1997).

Phobic postural vertigo can manifest in adults of every age, most often in the second and fifth decades; it is the most common
form of vertigo in this age group (Strupp et al. 2003). There is no sexual predominance. If phobic postural vertigo remains untreated, the complaints are exacerbated, generalisation develops, and avoidance behaviour increases until the patient is unable to leave his own apartment without help.

5.1.3 Pathophysiology and Therapeutic Principles

We have tried to explain the illusory perception of postural vertigo and postural instability by hypothesising that there is a disturbance of space constancy, which results from a decoupling of the efference-copy signal for active head and body movements. Under normal conditions, we do not perceive such slight, self-generated body sway or involuntary head movements during upright stance as accelerations. The environment also appears to be stationary during active movements, although there are shifts of retinal images caused by these relative movements. Space constancy seems to be maintained by the simultaneous occurrence of a voluntary impulse to initiate a movement and the delivery of adequate information in parallel to identify self-motion (Figure 5.1). According to von Holst and Mittelstaedt (1950), this efference copy may provide a sensory pattern of expectation based on earlier experience, which by means of the movement-triggered actual sensory information is then so interpreted that self-motion can be differentiated from the motion of the environment. If this efference copy is missing, e.g., if we move the eyeball by a finger on the eyelid, illusory movements of the environment occur, so-called oscillopsia. The sensation of vertigo described by phobic patients (involving involuntary body sway and the occasional perception of individual head movements as disturbing external perturbations) can be explained by a transient decoupling of efference and efference copy, leading to a mismatch between anticipated and actual motion. Healthy persons can experience similar mild sensations of vertigo without simultaneous anxiety during a state of total exhaustion, when the difference between voluntary head movements and involuntary sway becomes blurred. In phobic patients, this partial decoupling may be caused by their constant preoccupation with anxious monitoring and checking of balance. This leads to the perception of sensori-motor adjustments that would otherwise occur unconsciously by means of learned (and reflex-like) muscle activation programmes called up to maintain upright posture.

Precise posturographic analyses show that these patients increase their postural sway during normal stance by co-contracting the flexor and extensor muscles of the foot. This is evidently an expression of an unnecessary fearful strategy to control stance. Healthy subjects use this strategy only when in
real danger of falling. During difficult balancing tasks, such as tandem stance with closed eyes, the posturographic data of the patients do not differ from those of healthy subjects, i.e., the more difficult the demands of balance, the more “healthy” the balance performance of the patients with phobic postural vertigo (Querner et al. 2000). Patients with phobic postural vertigo often report a particularly increased unsteadiness when looking at moving visual scenes. However, when exposed to large-field visual motion stimulation in the roll plane, body sway does not exhibit any increased risk of falling (Querner et al. 2002). Vibratory stimulation showed that the patients were more sensitive to proprioceptive disturbances than healthy subjects, and less apt to use visual information to control upright stance (Holmberg et al. 2003).

Figure 5.1. Schematic diagram of how dizziness develops as a result of an impairment of the space constancy mechanism during active movements. Intentional head movements cause sensory stimulation of the vestibular, visual and somatosensory organs. Their signals are compared with a multisensory pattern of expectation calibrated by earlier experience of movements. The pattern of expectation is prepared by the efference-copy signal, which is emitted parallel to and simultaneously with the voluntary movement impulse. If concurrent sensory stimulation and the pattern of expectation are in agreement, self-motion is perceived while “space constancy” is maintained. If there is a partial “decoupling” of the efference-copy signal and thus a sensorimotor mismatch between the input and the expected pattern, vertigo and imbalance develop. The patient no longer experiences an intentional self-generated head motion in a stationary environment, but rather an exogenic head perturbation and simultaneously an illusory movement of the environment.
A doctor–patient consultation that provides a detailed explanation of the mechanism of the disease and of the necessity of self-controlled desensitisation, i.e., the patient consciously confronts those situations that induce dizziness, is essential for the therapy to succeed.

5.1.4 Pragmatic Therapy

The treatment is based on three or four measures:

- thorough diagnosis and diagnosis of exclusion
- “psychoeducational” explanation
- desensitisation by self-exposure to triggers and regular exercise
- if complaints persist, behavioural therapy with or without accompanying pharmacotherapy (Brandt 1996).

In our experience, the most important therapeutic measure is to relieve the patient of his fear of having an organic illness by carefully examining him and explaining the psychogenic mechanism (“increased self-observation” in the context of the corresponding primary personality structure). Desensitisation by exposure to the causative situations should follow, i.e., the patient should not avoid such situations but, on the contrary, seek them out. At the same time, regular exercise has proven to be helpful to give the patient confidence in his own sense of balance. If the explanation and self-densensitisation do not result in sufficient improvement after weeks to months, behavioural therapy with or without drug therapy should be started, i.e., with a selective serotonin-reuptake inhibitor (e.g., paroxetine, 10–40 mg/day) or a tri-/tetracyclic antidepressive for 3–6 months. In rare cases of situation-dependent attacks, tranquillisers can also be administered to certain patients, although there is a danger of addiction.

In a follow-up study (0.5–5.5 years after the initial diagnosis) involving 78 patients, we showed that 72% of the patients were free of symptoms or exhibited a clear improvement after receiving therapy (Brandt et al. 1994). In a more recent long-term follow-up study (5–15 years) of 106 patients, the improvement rate was 75%; 27% reported a complete remission (Huppert et al. 2005). There was a negative correlation between the duration of the condition before assessment of the diagnosis and the improvement/regression rate. There was no indication in both follow-up studies that we had misdiagnosed anyone.

The readiness of most of the patients, who experience much stress as a result of their suffering, to understand the psychogenic mechanism and to overcome it by desensitisation is a positive experience for both the physician and the patient.
5.1.5 Differential Diagnosis and Clinical Problems

The differential diagnosis of phobic postural vertigo includes psychiatric–psychogenic syndromes as well as vestibular and non-vestibular organic syndromes.

The most important psychiatric syndromes include:

- panic disorder with or without agoraphobia
- space phobia (Marks 1981)
- visual vertigo (Bronstein 1995, 2004)
- mal de debarquement syndrome (Murphy 1993)
- depression.

The most important organic syndromes include:

- primary orthostatic tremor with a pathognomonic frequency peak of 14–16 Hz in electromyography and posturography (Yarrow et al. 2001)
- bilateral vestibulopathy (Section 2.5)
- vestibular paroxysmia (Section 2.4)
- perilymph fistula or superior canal dehiscence syndrome (Section 2.6)
- basilar/vestibular migraine (Section 3.2)
- episodic ataxia types 1 and 2
- neurodegenerative disorders (spinocerebellar ataxias, multisystem atrophy)
- central vestibular syndromes (Section 3.1)
- orthostatic dysregulation.

In contrast to the long list of possible differential diagnoses, the combination of traits connected with the complaints, normal physical findings and primary personality type is so characteristic that there is seldom any doubt as to the diagnosis after the first examination.

References

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