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Anxiety in Vestibular Disorders

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1. Introduction

Vertigo is one of the most common symptoms and complaints in the clinical practice. A frequent question in neurootological expertise is whether the vertigo of psychiatric patients suffering from anxiety disorder is caused by vestibular dysfunction or the vertigo is originated from psychiatric disease.

Several possibilities exist in the relationships between psychiatric disorders and otoneurological diseases. When organic cochleovestibular lesion occurred in a patient with normal mental state, the neurootologist must treat patients according to the cochleovestibular examination results. When the dizzy patients have normal vestibular function, the patient must be referred to the psychiatrist. The most challenging are the cases of a combined lesion: psychiatric disorder and organic cochleovestibular disease. A very interesting question is exactly why vertigo is the main symptom in such patients, whereas panic disease or anxiety disorder in other patients can cause other severe symptoms resembling heart attack, dyspnœa, and abdominal crisis. Vestibular neuritis, benign positional vertigo, Ménéâtre’s disease and migraine are the most common neurootological conditions, all of which can trigger the anxiety disorder and panic.

Several types of these combined problems can be observed: (1) organic vestibular dysfunction with consecutive anxiety and panic; (2) established psychiatric disease with a new cochleovestibular lesion; and (3) dizziness due to panic and an independent organic lesion of the vestibular system (e.g., dizziness due to agoraphobia with compensated, previously well-known unilateral loss of function). (Szirmai et al, 2005)

The differential diagnosis can only be attained by a careful interdisciplinary way of thinking and activity, given the fact that the vestibular, neurological and psychiatric disorders—considered as pathogenic factors—are being present simultaneously in triggering the symptoms, and there can be overlaps between the certain pathological processes. (Fazekas A, 2010). Dizziness accounts for an estimated 5 percent of primary care clinic visits. The patient history can generally classify dizziness into one of four categories: vertigo, disequilibrium, presyncope, or light-headedness. The main causes of vertigo are benign paroxysmal positional vertigo, Ménéâtre’s disease, vestibular neuritis, and labyrinthitis. Many medications can cause presyncope, and regimens should be assessed in patients with this type of dizziness. Parkinson disease and diabetic neuropathy should be considered with the diagnosis of disequilibrium. Psychiatric disorders, such as depression, anxiety, and hyperventilation syndrome, can cause vague light-headedness. (Post RE & Dickerson LM., 2010)
Psychiatric diagnoses are common among patients with dizziness referred for otologic evaluation that does not show evidence of a peripheral vestibular disorder. Specific psychiatric disorders should be part of the differential diagnosis of patients who present with dizziness. (Sullivan et al, 1993)

A high degree of psychiatric disorders has repeatedly been described among patients with organic vertigo syndromes and attributed to vestibular dysfunction. According to Eckhardt-Henn’s data patients with vestibular migraine and Ménière’s disease showed significantly higher prevalence of psychiatric co-morbidity (MD = 57%, VM = 65%) especially with anxiety and depressive disorders, than patients with VN (22%) and BPPV (15 %) compared to normal subjects (20 %). As a consequence, a structured psychological and psychometric testing and an interdisciplinary therapy should be proceeded in cases with complex and prolonged vertigo courses, especially in patients with vestibular migraine and Ménière’s disease. (Eckhardt-Henn et al, 2008)

An article by Yardley et al reviewed evidence for three mechanisms whereby psychological factors may aggravate dizziness and retard recovery from balance disorders. A common behavioural response to dizziness is to avoid activities and environments that provoke symptoms; yet, such avoidance deprives affected individuals of the exposure necessary to promote psychological and neurophysiologic adaptation. Also, anxiety arousal and hyperventilation may add to, amplify, and disinhibit the somatic symptoms induced by balance disorder. Further, attention and cognitive load may influence the central processing of information required for the perception and control of orientation (Yardley L&Redfern MS, 2001).

According to Salhofer et al’s data the most prominent specific feature of migrainous vertigo was head motion intolerance. Patients with migraine showed anxiety more often and tended to have worse quality of sleep and higher depression scores. Migraine and vertigo is a risk factor for co-morbid anxiety. (Salhofer et al, 2010)

In previous studies, abnormal posturography scores under the eyes-open phase were related to high anticipatory anxiety, whereas those under the eyes-closed phase were related to phobic avoidance (Perna et al, 2001). Phobic postural vertigo (PPV) is a frequent diagnosis which can be challenging to treat. Most of these patients have psychiatric disturbances, which can cause compromised adaptive efficacy. Correlation was found between overall outcome on the Primary Care Evaluation of Mental Disorders Questionnaire and the Adaptive Operationalized Diagnostic Scale. Separate analysis revealed correlation between results of the AODS and anxiety disorders. Adaptive compromise was observed in individuals with PPV which was shown to be associated to psychiatric disorders. (Ferreira et al, 2010)

According to Brandt’s data, patients with psychiatric disorders may have pathological responses on vestibular testing. Patients with anxiety may have greater sensitivity and directional preponderance in vestibular testing, while organic vestibular symptoms may precipitate consecutive panic attacks with or without agoraphobia (Brandt 2000). Best et al (2006) didn’t find correlation between an acute or chronic vestibular dysfunction and pathology on psychometric testing. Their results do not support the hypothesis that latent vestibular dysfunction or imbalance triggers anxiety disorders, above all agoraphobia. Answering the question of whether this coexistence is only coincidental, their results suggested that special vestibular syndromes—for example, vestibular migraine and Ménière’s disease—may function as a trigger for a secondary somatoform disorder, but not vestibular disorders in general and especially not a subtle vestibular tone imbalance. (Best et al, 2006)
Patients with panic disorder, agoraphobia, or both are much more disabled by their dizziness than the patients with no psychiatric disorder. Anxiety and panic disorder was equally prevalent among patients with and without vestibular disease. In some cases panic disorder may provide an explanation for the dizziness, whereas in others it may be a co-morbid condition compounding the disability attributable to the vestibular disorder (Stein et al, 1994). In this study the main vestibular findings are analyzed in patients with and without anxiety disorders in few of main vestibular disorders were examined in our department. The main vestibular disorders are benign paroxysmal positional vertigo, true Ménière’s disease, the vertebrobasilar insufficiency, the vestibular neuronitis and vestibular migraine, sudden deafness with or without vestibular lesion, viral or bacterial labyrinthitis. The dizziness with headache, the Ménière’s disease, the vertebrobasilar insufficiency, the vestibular neuronitis, the BPPV, and their connection with anxiety are discussed in details.

Fig. 1. Romberg and sensitized Romberg test

2. Examination methods

The examination began with the detailed case-history, and followed by the routine oto-rhino-laryngological and neurological examinations. The cochleovestibular function of all the patients was examined by separate cochlear nerve and vestibular function tests. Cochlear function tests included the pure tone audiometry, acoustic reflex threshold and decay. The vestibular tests involved statokinetic tests (Romberg, sensitized Romberg and Babinski-Weil tests) (Figure 1.); spontaneous nystagmus with Frenzel’s glasses and with ENG registration as well, positional and positioning nystagmus examination using Frenzel’s glasses (Figure 2.). The patients were examined by the psychiatrist before or after the vestibular examination, so anxiety disorder was diagnosed by the psychiatrist.
The saccadic and smooth pursuit eye movement tests were performed by a computer-based ENG system (ICS Chartr ENG® system). Finally, bithermal caloric test was carried out by the computer-based ENG (Figure 3). The caloric test aims at establishing both the absolute levels of the two lateral semicircular canals responses to standardized thermal stimuli and the relative function between the two sides. It is usually done in a standardized fashion, using either temperature to begin, conducting testing of the two sides in a defined order, and then shifting to the other temperature. A minimum of 5 minutes should be spent between the end of the nystagmic reaction and the start of the next irrigation (Noaksson et al, 1998).

The ears were irrigated with the 50°C and 25°C air insufflations, the duration of stimulation being 40 seconds. The air caloric stimulation is the routine test battery of the otoneurological department even in the cases of normal external ear canal and normal ear drum, because the
Air caloric system is strictly connected to the computerized ENG system. Analyzing the results of the bithermal caloric test, the computer software gives the numeric results of the average slow phase velocities (ASPV) of the caloric nystagmus. The lateral preponderance between the left and right sides, according to Jongkees' formula, was calculated (Barber & Stockwell, 1980). The directional preponderance for left and right beating nystagmus was calculated accordingly. The figure 4 shows the Jongkees formula. Caloric weakness or canal paresis is pathological, when the asymmetry is more than 25%. Directional preponderance is pathological, when it is more than 40%. Normal range of average slow phase velocity of the caloric nystagmus with the above mentioned irrigation technique and parameters in between 20 and 35 deg/sec. The hyperreactivity (caloric nystagmus slow phase velocity is more than 40 deg/sec) often occurred in the central lesions and in anxiety disorders.

**Unilateral Weakness:**
\[
\frac{(RW+RC)-(LW+LC)}{RW+RC+LW+LC} \times 100 = UW
\]

**Directional Preponderance:**
\[
\frac{(RW+LC)-(LW+RC)}{RW+RC+LW+LC} \times 100 = DP
\]

*RW = right warm, RC = right cool, LW = left warm, LC = left cool*

Fig. 4. Jongkees formula

Butterfly schema- Chartr® ENG system

Fig. 5. Bithermal caloric test. Symmetrical, normal responsiveness
The graphic representation is the Claussen butterfly schema (Claussen C.F. & Franz B, 2006), modified by ICS Medical Corp. The figure 5 shows the normal caloric test result. In this study the data of 700 patients with vertigo are analyzed retrospectively.

3. Patients

1249 patients were examined in the Otoneurological Department of the Semmelweis University ORL Clinic in 2010. 549 were referred because of hearing loss, tinnitus, and facial palsy, while 700 patients have vertigo or dizziness. Some patients have vertiginous attacks with or without fluctuating hearing loss with aural fullness and tinnitus and all of these patients were sent by neurologist or their family doctor with a suspicion of Ménière’s syndrome. Some patients have fluctuating vertigo with the symptoms of posterior fossae lesions, like loss of vision or drop-attacks. Some patients have sudden loss of balance, due to vestibular neuronitis. Some patients have BPPV. Some patients have migraine with vertigo. Some patients have constant imbalance or light-headedness. They are referred by neurologist or psychiatrist to confirm or exclude any type of unilateral or bilateral vestibular lesion.

The data of these 700 patients were analyzed retrospectively. The mean age of these patients was 46.2 years. (The youngest patient was 9, the oldest was 85 years old). 45.7% of them have anxiety disorder. Panic disorder was diagnosed in 15.7% of our dizzy patients, while 11.4% have depression. More than female than male were examined in every age-group and in every disease-group. 36.8% of the male patients, while 49.0% of the female patients have anxiety disorder. The sex distribution of our patients with and without anxiety is shown on Figure 6.

The patients’ diagnoses are shown on the figure 7.

Anxiety ratio is shown of figure 8.
Fig. 7. Diagnoses of patients (n=700)

Fig. 8. Anxiety ratio in several diseases

4. Anxiety symptoms during the vestibular examination

The patients’ examination starts with the detailed case history. Sometimes a patient’s interpretation is redundant and can be hysterical; thus, we must listen also for the tenor or “mood” of the interpretation. The next step is to ask in a circumspect fashion about the symptoms of disease, like in a questionnaire. The doctor needs information about the patients’ life style, living place and job. In our patients anxiety problems are more frequent in manual worker, and more frequent in town than in patients who are living in country. It is a time-consuming process, but very important in evaluating the connection between otoneurological disease and anxiety or sometimes depression. After this consultation but
before the examination the patients often ask about the outcome of the disease, and we often
must tell that we have to speak about after the examination. This fact shows the severe
anxiety because of the outcome of the examination and the uncertainty of the patients.

After the case history all of our patients have audiological examination, as a part of
differentialdiagnostic process of the vestibular examination. Several diseases can cause
hearing loss, like Ménière’s disease; in others the patients have normal hearing, like in
vestibular neuronitis and BPPV. Some patients with psychiatrical disorders can aggravate
the hearing loss or feels more severe than the audiometry shows. After the long
audiological process we start the patient’s vestibular examination. The examination of the
statokinetic tests seems to be easy, but sometimes it is very difficult. The dizzy patients
failed to stand up with closed eyes, or walk with closed eyes. The patients with anxiety often
told during the statokinetic test examination (for example Romberg and sensitized Romberg
tests, that “I cannot do it”, but most of them can carry out our commands. Most of them
have a problem with self-consciousness.

The spontaneous nystagmus examination can cause problems rarely, but some patients have
aversion with the Frenzel’s glasses (“It is too light, it is disturbing me!”). The presence of
spontaneous nystagmus might show, that the patient has vestibular lesion, which is not
compensated. Half of our patient with spontaneous nystagmus had anxiety disorder, half of
them weren’t anxious.

During the examination of positional vertigo most of the patients have aversion of motion.
Most of the patients with BPPV have a resistance to the head motion, sometimes with a stiff
neck. When the patient has a long lasting recurrent BPPV, the X-ray of the neck shows a
compulsion port of vertebrae. When we rotate the patient’s neck, we can feel the resistance
and the stiffness of the neck. After verbal persuasion we can examine the patients, meaning
that the stiffness has a psychic but no neurological or rheumatologic reason. Not only in the
typical BPPV, but in the central positional vertigo the patients might have aversion of
motion. In our vertiginous patients with anxiety disorder positional vertigo was observed in
34.4%, while 61% of the patients with BPPV have anxiety disorder. In patients without
anxiety the ratio of positional nystagmus is 23.7%. It means that all of the positional types
of vertigo can generate anxiety, not only the typical BPPV. We can explain it with the
disturbing effect of head movement, which can generate short oscillopsia.

The examination of the optokinetic and smooth pursuit eye movements could be tiring for
the patients, but there aren’t aversions to the tests.

During the caloric stimulation most of the patients with anxiety have a severe fear from the
provoked vertigo. The caloric test can provoke vertigo, even in lying position which could
be very severe with vegetative symptoms. If the patient have a hyporeactivity of the
vestibular end organ, the vertigo is not severe, and bearable in the cases of normal vestibular
responsiveness. In the cases of hyperaesthesia, hyperresponsiveness the feeling of vertigo is
very unpleasant. In the patient with anxiety three type of mood can be observed during the
caloric test. When the hyperresponsiveness of the vestibular end organ can be measured, the
patients have severe vegetative symptoms. In these cases the uncomfortable feeling is
almost normal, and based on the severe vegetative status (uncomfortable level). In patients
with vegetative dystonia the vegetative symptoms are more severe, than we wait, based
onto the vestibular responsiveness. In patients may have hyperreactivity with vegetative
symptoms, but in patients with anxiety disorder we often see a normal or decreased
responsiveness without vegetative symptoms, but more uncomfortable level told by the
patients. These reactions without any vegetative symptoms can occurred in patients with

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anxiety; we mention it as a psychogenic reaction. Sometimes the combinations of these findings occurred. Sometimes patients are shouting and crying during the three minutes of provoked vertigo, but vegetative symptoms are missing (Figure 9). Twenty patients rejected the electronystagmographical examination after the information, that the complete ENG test might provoke vertigo four times during the caloric test.

**Fig. 9. Caloric test analysis**

Analysis of the electronystagmographic results of the caloric weakness, and the directional preponderance are very important, which data are characteristic for the peripheral and central vestibular lesion. The ENG results are shown on figure 10.

**Fig. 10.**

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Canal paresis or caloric weakness shows, that the patient has peripheral lesion, the pathological ratio is almost the same in the anxiety and the non-anxiety groups. The directional preponderance is more frequent in the anxiety group, signalling the role of the central pathways in the pathomechanism not only in the central vestibular disorders but also in the anxiety disorders. The hypersensitivity or hyperresponsiveness of the vestibular end organ also shows the pathological function of the central vestibular pathways. Interesting and unsettled question is which the primary dysfunction was. Whether the hypersensitive vestibular system cause the motion sickness of the patients, and this motion sickness generates anxiety disorder or the anxiety disorder modulates the central vestibular pathways to give hyperactive response for the caloric stimulus. The question needs further investigations.

5. Anxiety in vestibular disorders

5.1 Headache, motion sickness and anxiety in vestibular migraine
Patients with vestibular dysfunction, migraine and/or anxiety may experience visual vertigo, whereby symptoms are provoked by disorienting visual environments (e.g. supermarkets). Patients with visual vertigo over rely on vision for balance (i.e. visually dependent). According to Pavlou et al’s opinion visual vertigo significantly improves when vestibular rehabilitation incorporates exposure to optokinetic stimulation. (Pavlou et al, 2011). But when the patients have anxiety disorder, they often reject the visual stimulation and vestibular training as a therapeutical possibility. Most of the patients with migraine have motion sickness from the early young age. Patients with migraine frequently had abnormal caloric test responses, especially with a directional preponderance (figure 11.) In the migraine attack the patients are presumed to have hypersensitivity of the labyrinth with nausea and vomiting, (Szirmai 1997). What is the connection between the migraine, the motion sickness and the hypersensitivity of the labyrinth in the caloric test?

Fig. 11. Directional preponderance to the right side on electronystagmogram
25.7% of our vertiginous patients have headache, half of them have anxiety disorders. Only 5.7% of them have vestibular migraine, the others have cervical headache or tension headache. Motion sickness was observed in 14.3% of our patients, but 90% of them has anxiety disorders. In the vestibular migraine patients group 25% of the patients have severe anxiety, but in 75% of them have motion sickness.

These data suggests that there isn’t tight connection between migraine and anxiety and motion sickness. In the everyday medical experience most of the patients accept the fact, that they have motion sickness from early childhood. When the anxiety disorder develops in these patients, the motion sickness is worsened, and become more disturbing. When the patients have migraine, they used to bear the nausea during the headache, but they cannot accept the same kind of nausea as a concomitant symptom of vertigo. It suggests that the vertigo to the higher degree than migraine can provoke anxiety disorders.

5.2 Ménière’s disease
The true Ménière’s disease is caused by the hydrops of the endolymph. According to the Committee of Hearing and Equilibrium, (1995, cit. in Szirmai 2004) the classic triad is the recurrent, episodic vertigo, hearing loss, which is not always fluctuating, and the aural fullness or/and tinnitus. The criteria of the Ménière’s disease at least 2 attacks, which are more than 20 minutes long, and at least once documented hearing loss. The Ménière’s disease considerably deteriorates the quality of life. During the attacks the patients may have a fear from death, especially at the beginning of the disease. Later the patients know that the symptoms will disappear after few hours. As the attacks become more frequent, the patients have constant anticipated anxiety, thinking: „When will the next attack come?”

The vegetative symptoms, especially the vomitus is increasing the patients’ anxiety. Because of the vegetative symptoms, the patients have a feeling of uncomfortableness during the attacks. The attacks with vomiting can come unexpectedly, sometimes in an overcrowded place, or in the patients working place during a conference. Some patients have an attack at home with vomiting. Although the family is supporting them, the patients have anxiety of the situation. One of my patients told me: „When I have an attack of Ménière’s disease, and I’m laying in the bed and vomiting, my dear wife hold the pot. Although I use the toothbrush after the attack, but how can I kiss my dear wife with the same mouth? I fear of that she will disgust me!”

Later, when the patients have hearing loss, the problem is the communication with the other people. The sensorineural hearing loss and the tinnitus are very disturbing for the patients. The sensorial hearing loss with the recruitment can decrease the speech discrimination. Most of the patients cannot recognise the direction of the sounds. The patients fear from becoming ridiculous because the hearing loss and of the misunderstanding speech. Ménière patients fear loss of friends because of the communication problems. Because of the recruitment the patients have an increased sensitivity of noise. This fact can results, that patients can disturbed by several frequencies of noise and speech. Some interpersonal conflict situation can occurred because of this fact. One of my Ménière patients has a severe sensitivity of the sounds of 2000 Hz frequency. While this is one of the speech frequencies, these facts can results severe deterioration of her interpersonal connections. Because of the recurrent attacks of vertigo and the communication problem caused by the hearing loss, the patient could have a depression or a fear from the loss of their job. They could have problem with keeping of their living standards after becoming disabled.
Tinnitus connected to the hearing loss is very disturbing for the Ménière patients. The tinnitus is fluctuating. Before the vertiginous attack the noise and fullness in the ear is increasing, forecasting the severe attack. When the patient is anxious, or tired, the tinnitus can increasing without attack of vertigo, but this increasing of the tinnitus can provoke an anticipatory anxiety, whether the attack is coming or not. In the later phase of the Ménière’s disease, the tinnitus is constant, and the patients are disturbed in falling asleep, or in listening to music, or in reading. For these patients the silence is not golden, but very disturbing. These patients could be nervous and impatient with their family, and this fact can cause conflict situation in the family and in job.

In some cases, the Ménière patients have other diseases also. The co-morbidity causes further deterioration of the quality of life, and causes difficulties in the treatment and rehabilitation (Figure 12).

![Co-morbidity of Ménière’s disease](image12)

Fig. 12. Co-morbidity of Ménière’s disease

![Psychiatric disturbances in Ménière’s disease](image13)

Fig. 13. Psychiatric disturbances in Ménière’s disease

Patients need more pills because of the co-morbidity and sometimes the drug-interactions can cause side-effects, for example stomach-ache. In these cases the patient will stop taking medications. The psychiatric co-morbidities are one of the most important problems in the
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patient’s rehabilitation. The Patients with Ménière’s disease could have self-confidence disturbances, panic disease, depression, or anxiety disorders. The distribution of these co-existing psychiatric problems is shown in the figure 13.

5.3 Vertebrobasilar insufficiency
Vertigo, tinnitus and hearing loss occurred very frequently in the vascular posterior fossa lesions. In a slow blood flow of the temporal lobe vessels, brainstem vessels and labyrinthine artery wide range of cochleovestibular dysfunction could be observed. The evaluation of the vascular risk factors is very important in the diagnostical procedure. In the patients with vertigo, hearing loss, and tinnitus the regional slow blood flow of the brain were diagnosed by MRI, or single photon emission computer tomography. Although the blood vessels of the inner ear are invisible, but using MR and/or SPECT for the differential diagnosis in incapacitating vertigo similar to Ménière’s syndrome, we can deduce for the vascular origin. Despite of the peripheral lesion of true Ménière’s disease, in most of the cases of vertebrobasilar insufficiency we can diagnose central or combined vestibular lesion with ENG. (Figure 14) (Szirmai 2004).

Fig. 14. Combined lesion in vertebrobasilar insufficiency
The typical symptoms of vertebrobasilar insufficiency are the rotatory vertigo provoked by head movements, with mild pain in the nuchal region, bilateral tinnitus and/or hearing loss, and drop-attacks. The vertigo can occur after head turning backwards (“upper shelves disease”). Hypertension, atherosclerosis and cervical spondylosis could be a risk factor for the disease. Most of these patients (52%) have multiple co-morbidity and risk factors (Figure 15). In 19% of the vertebrobasilar insufficiency cases the patients have psychiatric disorders previously. In our cases 21.1% of the VBI patients have consecutive anxiety disorder, and 5.3% have depression. We can evaluate fear from becoming completely deaf, and fear from the motion and public transport. Most of the patients have anxiety when they know the diagnosis, which defined as vascular disorder of the brain. They have a fear from having a stroke, becoming paralysed, or going stupid because of the slow blood flow of the brain. Most of the patients have problem with the treatment, the antiaggregation therapy with aspirin could provoke stomach-ache. When they read about dizziness in the side effect list of...
aspirin, some patients reject taking it. The patients have aversion of much pills, they want to stop the medical treatment. The doctors have to persuade them, that the treatment of all the risk-factors, co-morbid diseases and consecutive psychiatric disorders are necessary.

Fig. 15. Co-morbidity in vertebrobasilar insufficiency

5.4 Vestibular neuronitis
Vestibular neuronitis is a considerably frightening disorder. The sudden loss of vestibular function can cause severe, few days long rotatory vertigo with extreme severe vegetative symptoms like nausea and vomitus. The patient cannot stand up and walk. In the acute phase the patients have fear from a heart attack, or death. Dizziness or vertigo presents itself as a profound illness, giving the sensation of “imminent death”. The differential diagnostic procedure is not easy; we have to exclude heart attacks, and gastroenteral disorders. The presence of spontaneous nystagmus could be helpful. When the doctor recognises, that the problem exists in the vestibular system, we can calm the patients, that their disease is not a life-threatening disease. In the acute phase of the vestibular neuronitis it seems to be enough for the patients, but later patients have to face to fact, that the recovery is a long process. Nobody dies from vertigo, but it is an illness that invalidates everybody who suffers from it preventing from carrying out a normal life, with the social consequences that it produces.

At the patients examination in the beginning of the disease we can see the harmonic vestibular syndrome, which is characteristic for the acute peripheral lesion. The patient has a severe nystagmus beating to the healthy side. If the patient can stand up, we can see deviation and tilting to the affected side. When the patient is unable to stand up, the tilting is visible in the sitting position. In the ENG we can registrate the unilateral loss of function. (Figure 16) In the first few days of disease the patient has severe vertigo and vegetative symptoms. The severity of the vegetative symptoms depends on the sensitivity of the vestibular responsiveness. If the patients have a hyperresponsiveness, the sudden loss of the hyperreactive vestibular end-organ can cause more severe vegetative symptoms than the loss of hyporeactive one (Figure 17 and 18). The treatment could be sedative drug administration and rest in bed. Later, when the vegetative symptoms are decreased, the patients have to stop sedative drugs, and try to move. In the early phase of the recovery the
patients need help. Vestibular training is necessary. Eye-movements and neck movements during the walking is advisable for the patients. In this phase the family is supporting the patient, but later, when the patients can walk, but have fear from the walk, the supporting tendency of the family could be diminished.

Fig. 16. Vestibular neuronitis. Caloric weakness of the left side on electronystagmogram

Later, when the patients know the diagnosis, they have anxiety from the long process of central compensation. When their vestibular status is compensated, the patients may have few minutes long oscillopsia at looking to the affected side. This oscillopsia can provoke severe anxiety and fear of motion in 25% of our patients.

Normal responsiveness on right, hyporesponsiveness on left side

Butterfly schema- Chartr® ENG system

Fig. 17. Bithermal caloric test
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We can avoid the anxiety of the patients, if we explain the pathomechanism of the vestibular dysfunction to the patients and teach them to keep under control their mind and give them a vestibular training program during their recovery.

5.5 Benign paroxysmal positional vertigo

Positional vertigo is caused by several central and peripheral vestibular lesions. The head or body position changing can provoke different type of positional nystagmus in central and peripheral lesions. In the central lesions the positional vertigo is direction fixed, non-fatigable. The peripheral positional nystagmus in most of the cases in direction fixed, but fatigable. The peripheral type of the positional nystagmus is often caused by canalolithiasis or cupulolithiasis. Vertiginous patients with anxiety positional vertigo were observed in 34.4%, while in patients without anxiety the ratio of positional nystagmus is 23.7%.

The disease called BPPV is a separate entity among the position-dependent types of vertigo. It might be one of the most frequent vestibular disorders, but often remained undiagnosed. The typical symptom in BPPV is the nystagmus in Dix-Hallpike manoeuvre (Figure 19.)

If we miss the Dix-Hallpike manoeuvre during the patient’s examination, we will misdiagnose BPPV. Sometimes the few seconds’ long vertigo results a differentialdiagnostic problems, the disease is misdiagnosed as vertebrobasilar insufficiency. This fact could result several targets for anxiety disorders: the patients have fear from vertebral artery occlusion, fear from stoke, fear from death, or being disabled. Most of patients are anxious from expectation being crazy or having anxiety disorder.

The long lasting or recurrent BPPV can provoke severe anxiety disorder, fear of motion. 61% of our patients have severe anxiety because of the recurrence of benign paroxysmal positional nystagmus.

Most of the patients with BPPV have a fear of head - motion, especially to the side of lesion. They try to avoid the head and neck movement. The result will be a stiff neck and a panic reaction after a head movement. These patients can reject the Epley manoeuvre and the
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vestibular training. Cognitive-behavioural therapy is an effective treatment for anxiety disorders, and vestibular rehabilitation exercises are effective for vestibular disorders. (Holmberg et al 2006). These patients have to be convinced again and again about the usefulness of vestibular training manoeuvres and they often have to use anxiolytic drugs.

![Dix-Hallpike manoeuvre](image)

Patient’s position on the table: head tilt with 45° in hanging position. Nystagmus is observed by +18 Diopter glasses.

Some patients have anxiety disorder during the self-treatment. Few of my patients had severe anxiety because of BPPV. I have convinced them about the necessity of the training, but they have written one or more electronic letters daily to me or called me daily, whether the training technique, what they were making, was good or not.

The treatment of BPPV combined with anxiety disorder is a long lasting process and requires more empathy and patience of the doctor.

6. Anxiety disorders with dizziness without vestibular disorders

Patients with anxiety disorders could have dizziness without vestibular dysfunction. Most of our patients have non rotatory vertigo, but constant imbalance. Patients have normal hearing and no vegetative symptoms. It seems to be very important diagnostic fact, when the patients’ rotatory vertigo is organic; most of them have vegetative symptoms like nausea. During the examination the symptoms of sever anxiety are visible. Most of the young ladies with vertigo wear not normal, but elegant shoes with spike heel, while patients with vestibular dysfunction wear normal shoes. The everyday activities (shopping, working in a crowded place) are impaired because of their feeling of dizziness, while other activities like using bicycle, sports like skiing are unimpeded. Patients with organic vestibular symptoms cannot work on the latter, cannot use bike, and cannot ski, only after their complete recovery.

Analyzing the symptoms or our patients with normal vestibular system, in 67% of them generalised anxiety disorder was diagnosed. 18% of them have depression, diagnosed by psychiatrist, and 18% of them have panic disease.
Most of these patients are convinced themselves, that they have organic lesion. Most of them ask doctors again and again to send them to imaging methods (CT and MRI scan) to exclude organic lesion. (“I must have been some severe disease, because I’m suffering from dizziness.”) It is very difficult to persuade them about the psychogenic reason. Few of them reject all of medical treatments and the help of psychiatrist (“I’m not crazy, I’m dizzy!”). Few of them use homeopathic drugs and several alternative ways of therapy.

7. Conclusions
The sensation of vertigo, a subjective complaint is sometimes defined as a movement illusion. Psychogenic vertigo or psychogenic superposition is relatively common in patients. To complicate the matters, vestibular dysfunctions frequently cause psychiatric illness, especially anxiety because of its incapacitating nature. The importance of the vestibular system for the internal representation of our body image accounts for the secondary psychiatric symptomatology in patients with primary vestibular disorders (Shilder, 1933, cited by Brandt, 2000).

In our experience, vertigo proved to have a bi-directional connection with psychiatric disorders. The panic disorder can be superimposed on chronic diseases with vertigo, and psychiatric patients with a cochleovestibular lesion have a diminished chance for complete recovery, and the recovery is longer. This relationship can be explained as a somatopsychic mechanism by which the vestibular symptoms provoke anxiety in the patient, who feels that the disease is a life-threatening catastrophe. Patients have severe anxiety at the beginning of the vertigo because of the uncertainty of the diagnosis. Some patients with vertigo have misdiagnosed disorder, mostly the BPPV remained undiagnosed. Undiagnosed peripheral vestibular lesion can cause psychiatric disorders, and increases somatisation tendencies in patients. In vestibular dysfunctions the mild movement of environment, like oscillopsia is often complained by patients. This feeling is very disturbing. If the doctor explains the pathophysiology of these feelings to the patients, the consecutive anxiety disorders could be avoided. If the patients don’t know, what the reason of their complaints is, the vestibular symptoms can provoke anxiety. Few years after the beginning of anxiety, we can suggest based on the case history that the organic vertigo was the trigger factor of the generalised anxiety disorder. If we examine the patient in that phase, we can find normal vestibular system.

Psychogenic superposition is suggested when there is a clear dissociation between objective and subjective disequilibrium, the patients complains severe rotatory vertigo without concurrent spontaneous nystagmus. In most of these psychogenic cases the vegetative symptoms associated to acute rotatory vertigo, like nausea and vomitus are missing. There are several controversies in the patients’ handicapping in everyday life. Anxiety can cause severe problems for example the patient cannot shopping, cannot use metro because the moving steps cause motion sickness and the metro is overcrowded. On the other hand, patient can use the latter at home for working, and can make excursions with bike. Alternatively, the psychosomatic mechanism might operate in such a way that the anxiety and panic increase vestibular responses to positional tests and caloric and rotational provocations. It can cause difficulties in the patient’s examination; the patients often reject the provocative tests of the vestibular examination.

The co-existing anxiety and vertigo needs parallel treatment. Treatment possibilities include antivertiginous drugs and SSRIs, vestibular training, and psychotherapy. The vertigo
treatment could be a longer process, than in non-anxious patients, and needs more empathy from the doctor. Treatment of vertigo in patients suffering from anxiety disorders requires cooperation between neurootologist and psychiatrist.

8. References


Anxiety disorders are one of the most common psychiatric disorders worldwide and many aspects of anxiety can be observed. Anxious patients often consult primary care physicians for their treatment, but in most cases they do not accept the diagnosis of anxiety disorder. Anxiety is a symptom that could be seen in many organic disorders and can accompany almost any psychiatric disorder. Anxiety disorders are frequent and are associated with significant distress and dysfunction. Stigmatization is an important factor in insufficient diagnosis. The problems of anxiety cover all fields of life. This book intends to describe the epidemiological aspects and the main co-morbidities and consecutive diseases of the anxiety disorders.

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