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PSYCHOLOGICAL-PSYCHIATRIC FACTORS IN CHRONIC DIZZINESS

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Summary

Investigations demonstrated a correlation between oto-neurological illnesses manifested subjectively by instability and dizziness and anxiety and other psychiatric disorders.

The concept of chronic vertigo offered a systematic approach to patients with a lasting dizziness not caused by an evident patophysiological vestibular damage. According to newer neurobiological investigations, there are three subtypes of chronic dizziness: otogenic, psychogenic and interactive.

Nowadays there is a greater diagnostic accuracy and insight into the basic pathophysiological processes of the vestibular migraine, post-concussional syndrom and dysautonomias that can cause chronic dizziness.

Selective serotonin reuptake inhibitors, rehabilitation therapy for restoring the balance, and cognitive-behavioural therapy can be effective in treatment, but this effectiveness is limited.

Key words: anxiety, phobia, chronic dizziness, postconcussional syndrome, dysautonomia, migraine

INTRODUCTION

Psychosomatic medicine points out the uniqueness of the body and soul, as well as their interaction. Today there is a growing consensus that psychological factors are important in the development of all diseases as well as in chronic dizziness. They play a role in the predisposition to the illness, its emergence, progression, course, severity

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and, finally, in the reaction to the somatic illness. Psychosomatic medicine involves a great part of behavioural medicine. The American National Academy in 1978 defined the behavioural medicine as an interdisciplinary branch dealing with the development and integration of behavioural and biomedical scientific cognitions and techniques important for the health and disease, and the application of this knowledge and techniques in the prevention, diagnosis, treatment and rehabilitation [1].

Through decades the chronic dizziness without identifiable vestibular deficits was called *psychogenic dizziness*. It was, however, a vague idea which could not include the complex correlation between the psychiatric and oto-neurological factors of chronic dizziness. In 1980 Brandt contributed to a better clarification of the psychogenic chronic dizziness concept. He introduced the concept of *phobic postural vertigo (PPV)*, a syndrome of subjective unsteadiness and illusory perturbations of posture, frequently accompanied by anxiety and autonomic arousal. Patients with PPV tend to have obsessive-compulsive personalities and may develop a PPV after vestibular insults, other medical illnesses, or periods of stress. Brand emphasized the importance of the positive identification of the core features of PPV rather than considering it to be a diagnosis of exclusion [2]. Recent studies confirmed that PPV could be reliably differentiated from other neuro-otological illnesses [3]. Treatment studies gave different results. The physical symptoms of PPV generally improved, but rarely resolved and often recurred. The majority of patients with PPV developed anxiety or depressive disorders that required psychiatric interventions [3]. Thus, PPV offers a more specific neuro-otological concept than psychogenic dizziness, but does not account for the range of psychiatric symptoms in patients with chronic dizziness and has not produced fully effective treatments [1].

Further, it was found that patients with vestibular disorders had high rates of panic disorders, and that patients with a panic disorder had high rates of dizziness and non-specific abnormalities on balance function tests (e.g. changes non diagnostic of a specific vestibular illness). It is considered that anxious disorders can cause psychosomatic dizziness and that vestibular dysfunction can cause somatopsychic anxiety [4].

Recent retrospective and prospective studies examined the longitudinal relationship between physical oto-neurological illnesses and anxiety disorders in chronic dizziness [5]. Also, studies of pharmacological, psychotherapeutic and rehabilitative procedures emerged [6-9]. These investigations paralleled research into the causes and treatments of other somatoform disorders such as noncardiac chest pain [10].

Taking this information into account the concept of *chronic subjective dizziness (CSD)* was introduced, which reformulates PPV and updates the psychosomatic dyad to provide clearer insights into the events that trigger and sustain chronic dizziness, a better understanding of therapeutic shortcomings, and the potential for preventive interventions [6,8].



Separate investigations examined three conditions that are easily confused with CSD, specifically vestibular migraine, traumatic brain injury and dysautonomia. All three may cause chronic or recurrent dizziness in the absence of identifiable vestibular deficits and are frequently accompanied by anxiety or depressive symptoms that mask the physical illnesses. Key clinical features distinguish them from CSD and other chronic neuro-otological conditions, allowing for appropriate diagnoses.

Definition and classification

The term psychosomatic is eliminated from the revised 4th edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV). The psychological factors influencing the somatic state are described (one or more psychological or behavioural factors that significantly affect the course and development of the general physical condition or significantly increase the risk of an adverse outcome).

According to the DSM-IV-TR diagnostic criteria psychological factors can unfavourably affect the general medical condition by one of the following ways:

1. Influencing the course of the general health condition, which can be estimated by a narrow time linkage between the psychological factors and the deterioration or delay of recovery of the general health condition.
2. Impeding the treatment of the general health condition.
3. Containing additional health risks for the patient.
4. Psychological reactions to stress precipitate or impair the general health condition.

The term psychological factors (I consider it better to use the term psychological-psychiatric factors, because not only the psychological symptoms, stress reactions, personal characteristics and behavioural disorders are included, but psychiatric diseases as well) is based on the nature and way they affect the general health condition causing chronic dizziness or some other psychosomatic illness.

Mental disorders, i.e. great depressive disorders, slow the recovery of chronic dizziness caused by vestibular neuronitis, psoriasis, myocardial infarction, and cerebral insult.

Psychological symptoms, i.e. anxious and depressive symptoms, cause the delay of the recovery of chronic dizziness, asthma, and urticaria.

Psychological reaction to stress causes the deterioration of the stress related chronic dizziness, arterial hypertension, tension headache, neurodermitis, urticaria, and arythmia ecc.

Personal characteristics, i.e. continuous anxiety, insecurity, lack of self-confidence, depressiveness, phobia of problem solving, have been linked with chronic dizziness.



Unadapted behavioural patterns (overeating, reduction of physical activity, unsettled sexual life).

Other or nonspecific behavioural patterns, i.e. interpersonal, cultural and spiritual factors.

The criteria of the International Classification of Illnesses and Health Problems, tenth revision (MKB-10) are more general, and are related to the psychological and behavioural factors linked with disorders or illnesses not classified in the chapter of psychiatry but in other chapters of somatic medicine. The psychiatric diagnostic category F54 is to be used when the influence of the psychological and behavioural factors on somatic disorders or illnesses is considered significant for the appearance and course of the somatic disorder [12]. It is the question of long lasting, mild psychological factors as worry, emotional conflict, anxiety, depressiveness, isolation, which cannot be classified by themselves, either in the group of mental or behavioural disorders in MKB10. Chronic dizziness related to the psychological and somatic factors which are in a changeable interrelation together responsible for the appearance, deterioration, gravity and duration of the disorder can be classified in the psychiatric diagnostic category F54 when the influence of the psychological and behavioural factors is considered significant [12].

From the above mentioned psychologic-psychiatric factors influencing the occurrence of the illness excluded are the following mental disorders with somatic symptoms:

1. classic mental disorders with somatic symptoms in the clinical picture, i.e. conver- sive disorders in which the somatic symptoms are caused by a psychological con- flict;
2. Somatic disorders in which there is no organic basis for the somatic symptoms;
3. Hypochondria, when the patient expresses an exaggerated worry for his/her health;
4. Mental disorders with somatic symptoms, i.e. dystimic disorders associated with the somatic symptoms like muscle weakness, fatigue, exhaustion;
5. Drug abuse and dependency with somatic symptoms (i.e. cough in smokers, fat infiltration in the liver because of the alcohol abuse).

A review of somatoform disorders is indispensable for the evaluation of the concept of chronic subjective dizziness. The fourth edition of DSM-IV-TR and the tenth edition of MKB-10 don not have well defined categories for the medically inexplicable somatic symptoms emerging from one organic system, like chest pain without heart damage, chronic dizziness without neurological or vestibular damage and others. They are called undifferentiated somatoforms. These disorders are defined as somatic symptoms lasting for 6 months continuously during most of the time without the evidence of organic damage. The dominant disorder is a persistent, vigorous, anxious and distressing dizzi-

ness that cannot be completely explained by physiological and somatic disorders and is considered to be caused by the stress as a response to an emotional conflict or psychosocial problems. Persistent somatoform dizziness (chronic subjective dizziness) is caused by psychological factors, which have a major role in its appearance, deterioration, maintenance and intensity. It is preoccupation with dizziness without the influence of a somatic illness. The somatic disorder, if it exists, has no significant influence on dizziness [12].

Clinical picture and diagnosis

Table 1 shows the usual characteristics of undifferentiated somatoform disorders using the non-cardiac chest pain as an example. Chronic dizziness has similar characteristics. The two steps approach to somatoform syndromes is considered better than DSM-10 approach [10]. The first step is to identify and precisely describe the somatic symptoms. The second step is to identify the psychiatric comorbidity.

The concept of CSD follows this approach, solves the border diagnostic problems, clears up the definition of somatoform dizziness and enables the treatment of anxious

Table 1. Common characteristics of monosymptomatic somatoform syndromes

Characteristics	Examples (Chest pain)
<ul style="list-style-type: none">• Somatic symptoms involve one organ• Patient seeks help from a specialist of somatic medicine, not a psychiatrist• There are no somatic disorders or they have not been diagnosed• There is a low correlation between symptoms and the somatic state• Patients have a phobic-anxious or neurotic, not hysteric personality• High correlation with a variety of anxious disorders• Illness is determined by psychological not somatic factors• Diagnoses in medical literature are descriptive syndromic• Organ directed treatment has limited effect• Treatment with SSRIs is more effective• Disorder shows tendency of recurrence and chronicity	<ul style="list-style-type: none">• chest pain, heart palpitations, fluttering• internist, cardiologist, primary care doctor• short attacks of supraventricular tachycardia on holter• there is no correlation between chest pain, EKG and biochemical findings• specific phobia, panic disorder, generalized anxiety• catastrophic fear of grave illnesses, anticipatory anxiety, phobic avoidance• chest pain is not correlated with heart disease• β-blockers can control somatic symptoms linked with anxiety• In some cases placebo can also be effective• 30% of patients with chest pain not orrelated to organic heart disorder continuously use unnecessary heart medications

and depressive disorders of these patients [3]. Chronic subjective dizziness can appear together with the cardiovascular, gastrointestinal, neurological, vestibular and other somatic symptoms, which can lead to diagnostic and therapeutic difficulties. The advance in the diagnosis and treatment in one field always has a positive influence on the progress in other fields.

Table 2 contains the diagnostic criteria for CSD [6]. Dominant sensations are persistent unsteadiness, rocking, swaying or fullness in the head, but patients may experience fleeting spins or tilts. They feel unstable while walking, but they do not have ataxia and rarely fall.

Table 2. Criteria and definition of chronic subjective dizziness

Somatic symptoms

Subjective dizziness and unsteadiness: Persistent (≥ 3 months) sensation of dizziness without dizziness, mild headache, grave headache, sense of imbalance lasting for the great part of the day.

Motion hypersensitivity: chronic oversensitivity (≥ 3 months) to one's own movement which is not direction specific, and to the movement of objects in the environment.

Visual vertigo: aggravation of symptoms following a complex visual stimulation such as in groceries, or when performing precision visual task (e.g. using a computer).

Neuro-otological evaluation

History and exam: Absence of active physical neuro-otological illnesses, definite medical conditions, or medications that may cause dizziness. History may include episodes of true vertigo or ataxia until the withdrawal of the state causing the vertigo.

Neuroimaging: Normal radiographic imaging of the brain.

Balance function tests: Normal or nondiagnostic findings. Included in these criteria are patients who clinically recovered from past neuro-otological illnesses and demonstrate fully compensated vestibular deficits on balance function tests and those with isolated test abnormalities that cannot explain the presenting symptoms.

They are very sensitive to vestibular, visual and proprioceptive motion stimuli and can develop a visual and spatial dependency with a tendency to favour the visual and proprioceptive over vestibular factors in motion and keeping balance. Patients are also intolerant to complex visual environments and demanding visual tasks. However, they do not report diplopia or oscilloscopia. These visual disturbances are defined as visual vertigo, which is proved not to be caused by ocular illnesses [13]. A neuro-otological examination of patients with a possible CSD can discover the signs of an overcome vestibular insult without any evident active oto-neurological dysfunction or existing organic cause of dizziness.



Anxiety is responsible for most of the morbidity in somatoform dizziness, but it is not included in the core definition of CSD for diagnostic and therapeutic purposes. The best clinical approach to a patient with chronic dizziness is the evaluation of the neuro-otological state, and only when that is done, the patient can be examined for anxiety and the diagnosis of CSD can be made. Clinicians concentrating on anxiety first may fail to diagnose the existence of an active neuro-otological illness and could prematurely conclude that the only cause of dizziness is a psychiatric disorder [3,4]. Recent advances in the psychology, genetics and neurophysiology of anxious disorders can help in differentiating the somatoform dizziness and various anxious disorders.

Stress and chronic dizziness

It is considered that a particular psychosomatic illness, besides to the stress or intensity, is related to a specific personality and to intrapsychic conflict. Specific psychic stress can be defined as specific for a given personality, or as an unconscious conflict that disrupts the homeostatic equilibrium thus contributing to the development of a psychosomatic illness. Thus, there is a special type of a coronary personality with aggressive impulses, competitive and prone to coronary illnesses, and the opposite personality type, which is calm and relaxed, and not prone to cardiac illnesses [14].

For the majority of experts the non-specific theory of stress is more acceptable than the specific one. Chronic stress, usually associated with changeable anxiety, has physiological correlates that, combined with genetic organic oversensitivity or weakness, make predisposition to psychosomatic illnesses. The sensitive organ can be in any part of the body. The susceptibility or weakness is probably genetic in origin, but the susceptibility can also be acquired (like pulmonary insufficiency in smokers). Various organs and systems can be susceptible, so patients can be: "gastric reactors", "cardiovascular reactors", "skin reactors", "vestibular reactors", etc. [15-17].

Many investigations confirmed the connection between the dizziness, temperament and anxious disorders. Temperaments are innate, highly heritable and stable behavioural patterns. Research on the temperament as a predisposing factor for psychiatric illnesses began in 1950s, but expanded rapidly in the recent years with the ability to link behavioral and physiological processes to genetic polymorphism. Two temperaments - phobic/anxious and neurotic, are characteristic for anxiety disorders. Epidemiological studies showed that the phobic/anxious temperament predisposes to panic and phobic disorders, while neuroticism predisposes to general anxiety disorders and major depression [18].

Neurotic persons are chronically worried, often of a bad mood, and react to stress by psychosomatic symptoms rather than an acute autonomic arousal. This construct of neuroticism is narrower than older formulations, which included an obsessive personality that is a part of PVV [18].

There is a mild association between neuroticism and the short allele of the serotonin transporter gene promoter region (5HTTLPR) [19]. Functional neuroimaging studies found that human homozygotes for the short allele had reduced amygdala and cingulate gyrus gray matter volumes as compared with those for the long allele [20], reduced hippocampal activity [21] and overactive, but uncoupled frontolimbic feed-back loops that extinguish negative affects. Short allele homozygotes also had a reduction in the serotonin 1A receptors throughout the brain [22,23].

Persons with phobic/anxious temperaments are timid, withdrawn, and prone to exaggeration. They avoid new situations and slowly adapt to them [18]. They have a high cerebrospinal fluid level of corticotrophin releasing hormone and serum cortisol (24) and disproportionate amygdalar responses [25]. Persons with a single nucleotide polymorphism affecting the gene for catechol-O-methyltransferase had twice more often the prevalence of the panic disorder and phobic behaviour than unaffected persons, but no differences in the levels of neuroticism or rates of generalized anxiety [26].

Characteristics of chronic subjective dizziness

A longitudinal investigation of 122 patients with CSD and anxiety demonstrated three possible patterns of correlation between oto-neurological illnesses and anxious disorders [5].

Otogenic CSD is caused by transitory neuro-otogenic illnesses, such as vestibular neuronitis, and sustains psychological-psychiatric symptoms and illnesses, such as pathologic phobic avoidance, anticipated anxiety and panic attacks, in patients without a pre-morbid anxiety. Pathologic avoidance is defined as avoidance of situations and activities provoking dizziness because of the anticipated anxiety and fear from the consequences. Panic attacks occur in situations incited by dizziness, as well as in those independent of dizziness. Their physical symptoms are considered to be caused by the same pathophysiological mechanisms as anxiety; they are a counterpart of their anxiety. It is considered, nevertheless, that they are less sensitive to psychological manifestations of anxiety. Patients with CSD, with or without anxiety, also demonstrate visual vertigo [27] and somatoform PPV [3].

CSD symptoms can be found in some DSM-IV diagnoses [11], such as panic attack, agoraphobia without panic, specific phobia, anxious disorders, undifferentiated and in other parts classified somatophorm disorders. Pathophysiological processes at the basis of CSD manifest themselves in anxiety, personal characteristics and the response to treatment. The sensation of dizziness in persons with a predisposed character is incited by innate anxious answers (e.g. fight/flight), the increase of awareness of the stimulus causing CSD and anxiety. In the prospective study of the systems threat/stress in organic



CSD 17 from 20 patients had a high-level anxiety during an acute vestibular crisis [7]. They developed oversensitivity to motion and obsession with dizziness in spite the fact that they had physically recovered.

Patients with psychogenic CSD have panic attacks and there are no neuro-otological illnesses in their medical history [5]. It is considered that a panic attack occurs when innate anxious answers are caused by a harmless physical stimulus i.e. transitory unsteadiness, which produces conditioned and exaggerated worrying and expectation of a hypothetic catastrophic outcome with anticipatory anxiety, phobic avoidance and recurrent panic attacks. Therefore, the treatment of the hypothetic and conditioned exaggerated worrying can also be applied to otogenic and psychogenic CSD. In both cases of stimulus conditioning motions are of cardinal importance. The use of visual and proprioceptive, at a higher rate than vestibular, stimuli can cause visual dependency. For the diagnosis and treatment of otogenic and psychogenic subtypes of CSD necessary are only the simple and well-defined pathophysiological mechanisms, and not the two way process in terms of somatophysic and psychosomatic dizziness [4].

In the interactive CSD transitory neuro-otological illnesses, occurring in the preexisting symptoms of anxiety, produce CSD and stimulate anxiety by the prevalence of general anxiety disorders and not panic and phobic disorders [5]. The mechanism stress/conditioning is operable in general anxiety. Interactive CSD is consistent with anxious disorders and is in a closer relationship to the neurotic than to phobic/anxious character. Three observations support this concept: in psychogenic CSD the original panic disorder is significantly linked to the phobic/panic predisposition [6], in otogenic CSD the correlation to the phobic-anxious temperament is not so pronounced, and in various interactive CSDs there is a neurotic and generalized anxious disorder at the same time.

Treatment

Selective serotonin reuptake inhibitors (SSRIs) are effective in the treatment of CSD [6]. Significantly more patients with otogenic and psychogenic CSD reach full remission after being treated with SSRI, as compared to those with interactive CSD. There are no controlled treatment trials for CSD, but the use of SSRI, vestibular and balance rehabilitation therapy (VBRT) and cognitive behavioural therapy (CBT) is recommended.

SSRIs were effective in more than 63% of patients with CSD and psychiatric symptoms. Patients with the panic disorder and at least one long allele 5HTTLPR responded better than the short allele homozygotes [8]. A vestibular and balance rehabilitation therapy can be psychologically useful since they lessen the hypersensitivity to motion stimuli, improve confidence in balance and diminish avoidance behaviour [9]. A cognitive behavioural therapy is accepted for anxious and depressive disorders and is therefore ideal for CSD [27].



Chronic dizziness and other medical conditions

Chronic dizziness can be triggered by various causes besides CSD. Symptoms similar to CSD can be caused by vestibular migraena, traumatic brain injuries and dysautonomia. These patients can also have symptoms of anxiety and depression. According to DSM-IV-TR diagnostic criteria psychological factors adversely influencing general health condition can significantly contribute to the development of CSV: psychiatric illnesses like depression, psychological symptoms like anxiety and depressiveness, psychological reactions to stress, personal characteristics (continuous worrying, insecurity, lack of self-confidence, anxiety, depressiveness, phobia of problem solving), non-healthy behavioural patterns (overeating, decrease in physical activity, unsettled sexual life) and other non-specific psychological factors (interpersonal, cultural or spiritual factors) [11,12].

Postconcussional cerebral syndrome. Postconcussional syndrome can be defined by symptoms such as headache, short-term memory loss, sleep disturbances, as well as persistent dizziness and depressiveness [11]. Many patients complaining of dizziness following a traumatic brain insult or whiplash have subjective dizziness and unsteadiness, hypersensitivity to motion cues and visual vertigo, which are similar to CSD [28]. True vertigo and ataxia occur rarely in those suffering a direct vestibular trauma or developing a posttraumatic benign paroxysmal vertigo. Patients with symptoms of CSD have no significant neuro-otological deficiencies. CSD following a traumatic brain injury is, however, associated with poorer psychosocial functioning and a smaller possibility of returning to work [29]. Patients who develop benign positional vertigo or vestibular migraena after a cerebral trauma return to work much faster than those with CSD-like symptoms [28].

In patients with a milder traumatic brain injury or a whiplash dizziness and unsteadiness could not be confirmed objectively. Besides, many patients have financial claims and the possibility of malingering cannot be excluded [30].

Vestibular migraena. This term does not exist in the International Headache Society (IHS) nomenclature for migraine. Nevertheless it is very probable that migraena is the most common cause of recurrent vertigo attacks and subjective dizziness [31]. Migraena is present in 60-80% of patients with a recurrent subjective dizziness without auditory symptoms, but there is no consistent temporal relationship between dizziness and headache. Migraena and anxiety disorders often appear in comorbidity. More than 35-50% of those suffering from migraena suffer also from anxiety, so that it was proposed to name this disorder descriptively as a migraine-anxiety related dizziness [32].

Dysautonomia. A neurovegetative dysfunction leading to a neurocardiogenic syncope and orthostatic intolerance can cause persistent dizziness. Symptoms are very similar

to those of CSD, but their symptoms usually appear or increase during position changing (rising), standing for a long period of time, or after physical exhaustion [33]. Six out of 345 CSD patients treated in the Balance Rehabilitation Center had a dysautonomia [34]. These persons are usually the first to visit cardiologists, otolaryngologists and neurologists.

Conversive disorder, factitious disorder and malingering are rare in patients with somatoform dizziness [27]. Conversive patients have unusual gait and posture abnormalities, while those with a factitious disorder and malingering simulate their symptoms, which are therefore often unconvincing and inconstant.

Conclusion

A close correlation was found between the disorders presenting with “dizziness without dizziness”, subjective sensation of unsteadiness and other psychiatric symptoms of oto-neurological disorders and anxiety. The term CSD was introduced and the need of a systematic approach to patients with persistent dizziness without any evident vestibular damage was emphasized. According to newer neurobiological investigations there are three subtypes of CSD: otogenic, psychogenic and interactive.

Better diagnostic possibilities and better insight into the basic pathophysiological processes enabled the definition of the key features of some disorders causing chronic dizziness.

Treatment studies need further evaluation. SSRIs have, however, been undoubtedly effective in the majority of patients, as well as balance rehabilitation and cognitive behavioural therapy.

CSD caused by a vestibular migraena, traumatic brain injury and dysautonomia can cause diagnostic difficulties and can be misdiagnosed as a psychogenic disorder.

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Sažetak

Psihološko-psihijatrijski čimbenici kronične vrtoglavice

Istraživanja su pokazala da postoji povezanost između nekoliko neuro-otoloških kliničkih stanja koja se javljaju subjektivnim osjećajem nestabilnosti i vrtoglavice uz anksioznost i druge psihijatrijske simptome.

Pojam kronične subjektivne vrtoglavice ponudio je sustavni pristup bolesnicima sa ustrajnom vrtoglavicom koja nije uzrokovana očitim patofiziološkim vestibularnim oštećenjem. Sukladno sa novim neurobiološkim istraživanjima postoje tri podtipa kronične subjektivne vrtoglavice (otogeni, psihogeni, interaktivni) koje čine fizički i psihološki simptomi. Danas postoji veća dijagnostička točnost i uvid u temeljne patofiziološke procese vestibularne migrene, postkontuzijskog sindroma mozga i distonije autonomnog živčanog sustava, koji mogu prouzročiti simptome nalik kroničnoj subjektivnoj vrtoglavici.

Selektivni inhibitori ponovne pohrane serotonina-SIPPSa, rehabilitacijska terapija ravnoteže i kognitivno-bihevioralna terapija pokazuju ograničenu, ali korisnu učinkovitost u liječenju kronične subjektivne vrtoglavice.

Ključne riječi: kronična subjektivna vrtoglavica, neuro-otološka stanja, anksioznost, fobičnost