Braz J Otorhinolaryngol. 2015;81(3):276–282

Brazilian Journal of
OTORHINOLARYNGOLOGY
www.bjorl.org

ORIGINAL ARTICLE

Clinical characteristics of patients with persistent postural-perceptual dizziness☆,☆☆

Roseli Saraiva Moreira Bittar[a], **Eliane Maria Dias von Söhsten Lins**[b,*]

[a] *Division of Otoneurology, Hospital das Clínicas, Medical School, Universidade de São Paulo (USP), Cerqueira César, São Paulo, SP, Brazil*
[b] *Department of Otorhinolaryngology, Medical School, Universidade de São Paulo (USP), Cerqueira César, São Paulo, SP, Brazil*

Received 17 January 2014; accepted 24 May 2014
Available online 6 September 2014

KEYWORDS
Anxiety;
Depression;
Dizziness;
Comorbidity

Abstract
Introduction: Persistent postural-perceptual dizziness is the dizziness that lasts for over three months with no clinical explanation for its persistence. The patient's motor response pattern presents changes and most patients manifest significant anxiety.
Objective: To evaluate the clinical characteristics of patients with persistent postural and perceptual dizziness.
Methods: statistical analysis of clinical aspects of patients with persistent postural-perceptual dizziness.
Results: 81 patients, average age: 50.06 ± 12.16 years; female/male ratio: 5.7/1; main reasons for dizziness: visual stimuli (74%), body movements (52%), and sleep deprivation (38%). The most prevalent comorbidities were hypercholesterolemia (31%), migraine headaches (26%), carbohydrate metabolism disorders (22%) and cervical syndrome (21%). DHI, State-Trait Anxiety Inventory – Trait, Beck Depression Inventory, and Hospital Anxiety and Depression Scale questionnaires were statistically different ($p < 0.05$) when compared to controls. 68% demonstrated clinical improvement after treatment with serotonin reuptake inhibitors.
Conclusion: Persistent postural-perceptual dizziness affects more women than men, with a high associated prevalence of metabolic disorders and migraine. Questionnaires help to identify the predisposition to persistent postural-perceptual dizziness. The prognosis is good with adequate treatment.

☆ Please cite this article as: Bittar RS, Sohsten Lins EM. Clinical characteristics of patients with persistent postural-perceptual dizziness. Braz J Otorhinolaryngol. 2015;81:276–82.
☆☆ Institution: Hospital das Clínicas, Universidade de São Paulo, Cerqueira César, São Paulo, SP, Brazil.
* Corresponding author.
E-mail: elianesohsten@gmail.com (E.M.D. von Söhsten Lins).

http://dx.doi.org/10.1016/j.bjorl.2014.08.012

PALAVRAS-CHAVE
Ansiedade;
Depressão;
Tontura;
Comorbidade

Caracterização clínica dos pacientes com tontura postural-perceptual persistente (TPPP)

Resumo
Introdução: A denominação tontura postural-perceptual persistente (TPPP) é atribuida à tontura que se mantém por mais de 3 meses em pacientes, sem que exista justificativa clínica para a sua persistência. A maioria dos pacientes possui perfil ansioso ou experimenta alto grau de ansiedade no início dos sintomas. O padrão de resposta motora apresenta-se alterado, com hipervigilância e hipersensibilidade a estímulos visuais e de movimento.
Objetivo: Avaliar as características clínicas de pacientes com diagnóstico de TPPP.
Método: Análise dos aspectos clínicos de pacientes do ambulatório de TPPPe quantificação do perfil ansioso ou depressivo.
Resultados: Foram avaliados 81 pacientes, com média de idade de 50,06 ± 12,16 anos; relação mulher/homem de 5,7/1; principais gatilhos para tontura: estímulos visuais (74%), movimentos corporais (52%) e privação de sono (38%). As comorbidades mais prevalentes foram hipercolesterolemia (31%), migrânea (26%), distúrbios do metabolismo do açúcar (22%) e síndrome cervical (21%). Os questionários DHI, STAI-Traço, Beck para depressão e HADS foram estatisticamente diferentes (P < 0,05) entre pacientes e controles. 68% de melhora clínica com o uso de inibidores da recaptação da serotonina.
Conclusão: TPPP acomete principalmente as mulheres, sendo alta a associação com distúrbios metabólicos e migrânea. Os questionários auxiliam na identificação da predisposição à TPPP. Há bom prognóstico com o tratamento adequado.

Introduction

The field of Otoneurology evolved considerably in recent decades, from a simple vestibulometric evaluation of the vestibule-ocular reflex to a complex investigation of balance and posture. Nevertheless, there are patients who exhibit undiagnosed dizziness not explained by some otoneurologic disease, even with the help of the full range of diagnostic tests offered today. The tests present normal results; until a short time ago, these patients were labeled as psychogenic. Psychiatric syndromes also do not explain the symptoms found in these individuals. In 1986, Brandt[1] described the phobic postural vertigo (PPV), which still did not explain in detail the origin of symptoms nor suggested any kind of treatment. At the beginning of the 21st century, Staab and Ruckenstein related physical symptoms of PPV to behavioral factors, in may 2013, it was renamed persistent postural and perceptual dizziness (PPPD) for compatibility with DSM-5.[2,3]

PPPD is defined as a type of dizziness that persists for over three months with no identifiable etiology.[3] This is a somatoform disorder, representing an interface between otoneurology and psychiatry. Apparently, patients with PPPD present a profile that predisposes to the persistence of dizziness after an event of physical or emotional illness. When there is such a profile, the postural stability maintenance system becomes hyper-reactive to movement, especially in environments with high visual demands. From this sensitivity, there is an increase in the risk of behavioral disorders such as anxiety, phobias, and depression.[4] Thus, PPPD reflects the persistence of a vigilant pattern of posture control that was assumed during the acute phase of the disease.[4]

PPPD is a chronic condition that can last for months or years,[5–7] and is characterized by six basic aspects[4]: (1) Persistent sway or instability not detectable on physical examination; (2) Worsening of symptoms in the standing position; (3) Worsening of symptoms with head movements or with complex visual stimuli; (4) Presence of illness or emotional shock at symptom onset[4]; (5) Concurrent diseases, mainly that gave rise to the symptoms[7]; (6) Anxiety.

The association between dizziness and anxiety in otoneurological patients is attributed to neural interactions explained by neuroanatomy.[2] These interactions include connections between central-vestibular pathways and neural networks of anxiety and fear. The functional neurocircuitry of anxiety includes the amygdala, insula, anterior cingulate, prefrontal cortex, superior frontal gyrus, paracingulate, and inferior frontal gyrus. These structures are closely linked to emotion, and their dysfunction can result in impaired neural processing, with consequent anxiety.[8] The identification of the interdependence between otoneurologic and psychiatric diseases becomes crucial in the prognosis of dizziness.[9]

Based on the concepts presented by Staab and Ruckenstein,[9] PPPD can produce three different manifestations: (1) Psychogenic: anxiety is the sole cause of dizziness; (2) Otogenic: an otoneurologic disease acts as a trigger that activates the circuitry of anxiety; (3) Interactive: an otoneurologic disease triggers dizziness, which in turn exacerbates pre-existing anxiety symptoms. There are reports describing that the persistence of dizziness is directly related to the severity of the initial imbalance and to the anxiety generated by bodily sensations during the episode.[10] The anxiety impacts postural control, motor skills and eye tracking.[11,12]

The therapeutic approach to PPPD takes into account some basic principles. The first is the correct identification of the symptoms, followed by counseling for good dietary habits, medication, vestibular rehabilitation, and psychotherapy. Vestibular rehabilitation is based on the identification of troublesome movements and on the demonstration to the patient that the fear of their carrying out these movements is unfounded; thus, the classical protocols of adaptation and substitution do not apply. Cognitive-behavioral therapy, performed during a brief period, is the psychotherapy of choice. This treatment helps the patient to identify situations that cause distress, allowing the choice of the most suitable reactional behavior.[13] The use of psychometric testing aids in psychiatric screening and in the assessment of the degree of perception of dizziness-induced harm.

The drugs of choice for this treatment are serotonin reuptake inhibitors (SSRIs); serotonin is the primary neurotransmitter for those previously described nuclei. These drugs change and regulate neural conduction through anxiety circuits and central vestibular neurons that respond to movement. With the proper use of SSRIs, a reduction of symptoms can be obtained in as many as 70% of individuals, in approximately three months of treatment.[14]

The knowledge of the clinical features of patients with PPPD allows for the establishment of a correct diagnosis and an appropriate treatment. This article aim to describe the main clinical features and therapeutic responses in outpatients with a diagnosis of PPPD.

Methods

This study assessed patients who attended the PPPD outpatient clinic during the year of 2013. This clinic is part of the PPPD research project, approved by the ethics committee of the institution under number 0411/10.

The sample consisted of 81 patients between 17 and 80 years (mean age 50.06 ± 12.16 years), 12 males and 55 females. All participants signed an informed consent form.

In order to be included in the PPPD outpatient clinic, the patient should have experienced persistent dizziness for over three months, with no identifiable causes for the symptomatology, i.e., all comorbidities (carbohydrate metabolic disorder, dyslipidemia, thyroid disorders, neck pain, heart disease, blood pressure disorder, dysautonomia, canalithiasis, cupulolithiasis, anemia, and other dizziness-inducing disorders) were compensated. All patients were previously attended to and treated by physicians at the general outpatient clinic. All participants were submitted to a full oculographic examination and serological tests; when clinically indicated, the participants were also submitted to a computerized rotational pendulum test, computerized dynamic posturography, evoked potentials test, and imaging studies. None of the others patients diseases has a cause effect relationship to the symptoms presented. Then, the patients followed a particular routine of investigation especially developed for patients with PPPD.

The control group consisted of patients whose dizziness ceased within three months without the use of psychotropic drugs. Sertraline at a dose of 25–100 mg/day was the first pharmacological option; it was replaced by paroxetine at a dose of 20–40 mg/day in a few patients due to lack of response after eight weeks of use or due to the emergence of side effects (increased anxiety, sexual dysfunction, gastrointestinal or sleep disorders). Patients were referred for psychotherapy and desensitizing, where desensitizing exercises i.e., dizziness-inducing stimuli for a gradual and progressive practice until tolerance, were prescribed.

Patients were classified as responders, when they presented complete resolution of symptoms; partial responders, when they presented symptomatic improvement, but with persistence of some symptoms; and non-responders, if there was no perception of improvement.

The control group consisted of only seven female volunteers, due to the difficulty of recruiting asymptomatic patients; this gender was chosen for being the most prevalent in the otoneurology outpatient clinic.

The study and control groups responded to several psychometric tests, all previously validated for Brazilian Portuguese.

Anamnesis

During the admission anamnesis, five key points were evaluated: time of onset of symptoms, symptom characteristics, circumstances in which the dizziness occurs, associated symptoms, underlying diseases, and predisposing factors to dizziness.[15,16]

Physical examination

The following tests to investigate the oculovestibular reflexes were performed: spontaneous and semi-spontaneous nystagmus, head shaking nystagmus, and head impulse test. The postural stability was then evaluated by Romberg, Fukuda, and walking tests.

Questionnaires

DHI (Brazilian Portuguese version):[17,18] comprises 25 questions, seven of which assess physical aspects; nine assess emotional aspects; and nine assess functional aspects. The maximum score, suggesting a high overall dizziness-induced damage, is 100 points.

State-Trait Anxiety Inventory (STAI):[19] two scales of self-assessment consisting of 20 items each, ranging from 1 to 4. These scales assess anxiety as a transitory emotional state (STAI-E [state]) or as a permanent characteristic of the individual (STAI-T [trace]).[20]

Beck Depression Inventory (BDI): The original scale consists of 21 items, ranging from 0 to 3 in terms of intensity.[20] For samples without a diagnosis of depression, the following intervals were adopted: <15: normal; 16–20: indicative of dysphoria; >20: indicative of depression.[21]

Hospital Anxiety and Depression Scale (HADS) (Brazilian Portuguese version):[22] a self-report scale with seven items for anxiety (HADS-A) and seven for depression (HADS-D). The score for each subscale ranges from 0 to 21. A score of eight or more on the subscales of anxiety or depression, or a total score (anxiety + depression) of 12 or more suggests clinically significant psychiatric symptoms.[22]

Table 1 Sample characterization in relation to gender and age.

Gender	*n*	Age (mean ± DP)	*p*
Male	12	49.41 ± 10.43	
Female	69	52.32 ± 12.5	
Total	81	50.06 ± 12.16	0.41

Table 2 Main triggers in the onset of symptoms of persistent postural and perceptual dizziness no lugar de chronic subjective dizziness.

Trigger	*n*	Percentage
Visual stimuli	60	74%
Body movements	42	52%
Sleep deprivation	31	38%
Crowding	21	26%
Stress	20	25%
Neck	11	14%

Study variables and statistical analysis

The clinical characteristics of patients with PPPD are described in Tables 1–5 with percentages of prevalence. For the questionnaires, comparative calculations were made by Student *t*-test for research participants, compared to the control group, which consisted of asymptomatic patients from the general outpatient clinic, with otoneurologic disease and a good response to treatment.

The level of significance was set at 5% ($p = 0.05$).

Results

The gender and ages of the subjects in the study sample are shown in Table 1. The mean age of subjects was 50.06 ± 12.16 years, with no statistically significant differences between genders ($p = 0.41$).

Table 3 Comorbidities associated to persistent perceptual and postural dizziness no lugar de chronic subjetive dizziness in the study group.

Disease	*n*	Percentage
Hypercholesterolemia	25	31%
Migraine	21	26%
CMD	18	22%
Cervical syndrome	17	21%
BPPV	12	15%
Dysautonomia	6	7%
Otitis media	6	7%
SAH	4	5%
Cardiac arrhythmia	4	5%
Diabetes	4	5%
Hypothyroidism	4	5%
Menière's syndrome	4	5%

CMD, carbohydrate metabolic disorders (hypoglycemia, intolerance, and hyperinsulinemia); BPPV, benign paroxysmal positional vertigo; SAH, systemic arterial hypertension.

When considering the type of PPPD according to the criteria defined by Staab and Ruckenstein,[9] 12 patients were classified as psychogenic PPPD (14.8%), 20 as otogenic PPPD (24.7%), and 49 as interactive PPPD (60.5%).

The main triggers that caused the abovementioned symptoms are described in Table 2. The three most prevalent stimuli were visual stimuli (74%), body movements (52%), and sleep deprivation (38%). Some subjects had two or more triggers for their symptoms, with a maximum of eight.

The comorbidities associated with PPPD can be observed in Table 3. Major diseases associated with PPPD were hypercholesterolemia (31%), migraine (26%), carbohydrate metabolic disorder (22%), cervical syndrome (21%), benign paroxysmal positional vertigo (15%) dysautonomia (7%), disorders of the middle ear (7%), hypertension (5%), cardiac arrhythmia (5%), diabetes (5%), hypothyroidism (5%), and Menière's syndrome (5%). In addition to the described comorbidities, the following were found, accounting for less than 2% of prevalence: elderly unbalance syndrome, peripheral facial palsy, vestibular neuritis, epilepsy, sudden deafness, hepatitis C. Some patients had more than one comorbidity associated with PPPD, with a maximum of six for this sample.

Regarding the formulated questionnaires, only STAI-E ($p = 0.36$) showed no significant difference between patients with a diagnosis of PPPD and those with no chronified dizziness after the vestibular event. All other questionnaires showed significant differences among the groups: DHI ($p = 0.008$), STAI-T ($p = 0.0008$), BDI ($p = 0.0005$), HADS-A ($p = 0.0001$), and HADS-D ($p = 0.0007$). The mean responses obtained from the questionnaires are described in Table 4.

Regarding the evaluation of responses to pharmacological treatment with SSRIs, the results can be seen in Table 5. Of the 45 treated patients, 46% responded satisfactorily to medication, 22% had partial response, and 31% exhibited no benefit.

Discussion

PPPD is a relatively new diagnosis that involves the vestibular-thalamic pathways, responsible for body movement awareness.[23,24] The predisposition to sensitivity of the structures involved in the connections between the vestibular-thalamic pathways and the circuitry responsible for anxiety and fear determine symptom onset. This disease is at the interface between otoneurology and psychiatry, and the patient has normal vestibular reflexes, while maintaining a vigilant pattern of postural control. This is a highly distressing condition for the patient, who will seek multiple physicians and will be submitted to several tests without finding answers to his/her symptoms.

The mean age of the study group did not differ from the recent literature.[25] In the present sample, the mean age did not differ between genders, but comprised a higher number of females, at a proportion of 5.7:1. Recent population survey in the city of São Paulo confirms the high prevalence of dizziness in females, in a ratio of 1.67:1, especially in the age group between 46 and 55 years.[26] The higher prevalence of females is attributed to hormonal changes. It is interesting to note that, at the age group between the fourth and fifth decades of life, the female climacteric occurs, with

Table 4 Responses to questionnaires formulated for patients with chronic subjective dizziness (study group) and patients who did not exhibited cronification of their dizziness after a vestibular event (control group).

Questionnaire	Study group (mean scoring)	Control group (mean scoring)	*p*
DHI	52.66 ± 21.74	13.66 ± 24.11	0.008[a]
STAI-T	52 ± 10.19	32.85 ± 9.44	0.0008[a]
STAI-E	38.66 ± 11.80	33.71 ± 11.49	0.36
BDI	14.57 ± 8.14	4.28 ± 3.14	0.0005[a]
HADS-A	12.57 ± 3.32	3.16 ± 3.12	0.0001[a]
HADS-D	7.71 ± 4.42	1.8 ± 2.22	0.0007[a]

STAI-T, State-Trait Anxiety Inventory – Trait; STAI-E, State-Trait Anxiety Inventory – State; BDI, Beck Depression Inventory; HADS-A, Hospital Anxiety and Depression Scale – Anxiety; HADS-D, Hospital Anxiety and Depression Scale – Depression.
[a] Statistically significant difference between groups.

peaks of migraine.[27,28] Staab and Ruckenstein also observed a female dominance, with a ratio of 2:1.[29] Due to the higher prevalence of PPPD in women, the outpatient clinic was initially only for women. The authors believe that the higher number of women in relation to literature data occurred because, for institutional reasons, this particular outpatient clinic admitted only women at the beginning of its operation.

According to the descriptions of Staab,[9] PPPD can be divided into three types: interactive, otogenic, or psychogenic. In the present sample, 60.5% of patients were classified as interactive, 24.7% as otogenic, and only 14.8% as psychogenic CSD. These data suggest that approximately 75% (60.5 + 14.8) of the present patients had a typically anxious profile before developing their symptoms. Staab and Ruckenstein also reported a high prevalence of anxiety disorders in 60% of their patients with PPPD.[29] Panic disorder prevailed in patients with psychogenic form, whereas generalized anxiety disorder was more common in interactive PPPD, and mild anxiety symptoms were more common in the otogenic form.[29] These observations highlight the need for psychiatric support for these patients. The partnership between psychiatry and otoneurology becomes critical for a good treatment of patients who present anomalous neural transmission of connections between the vestibular pathways and the circuitry of vigilance and fear.

Regarding the triggers for PPPD crisis, visual stimuli was observed as a trigger in 74% of individuals, comprising the 26% of subjects who reported symptoms when in crowded places. These patients appear to present visual dependency and a tendency to rely more on visual cues than in vestibular and somatosensory afferents for the maintenance of their posture. For reasons still unknown, anxious patients often develop visual dependency.[12] The second trigger, in order of frequency, are body movements, as described in the literature. The high level of anxiety experienced by these patients lead to an elevated muscle tension and stiffness in the neck, to avoid movements that cause the uncomfortable sensations of instability and fear.[30] From this perspective, it is easy to understand the high prevalence of cervical syndrome (21%) and neck pain as a trigger for their symptoms. An interesting fact is sleep deprivation, functioning as a trigger for the symptoms of PPPD in 38% of cases, together with stress in 25% of them. Stress acts as a trigger for sensitizing the neural pathways responsible for the reactions of vigilance. As previously mentioned, PPPD mainly affects women between the fourth and fifth decades of life, at the climacteric time, which is the peak incidence of migraine. The main triggers of PPPD are exactly the same as those for migraine attacks: visual conflict, head movements, stress, sleep deprivation.[27,28] The similarity of symptoms raises the question of whether there is an interdependence between PPPD, migraine, and metabolic and hormonal dysfunction. In fact, the three most frequent comorbidities in patients with PPPD are dyslipidemia (31%), migraine (26%), and carbohydrate metabolic disorders (22%). Staab and Ruckenstein observed a prevalence of 16.5% of migraine in patients with PPPD.[29]

Table 5 Response of patients with a diagnosis of chronic subjective dizziness pharmacologically treated with SSRIs.

Response to SSRIs	*n*	Percentage
Yes	21	46%
Partial	10	22%
No	14	31%
Total	45	100%

Regarding DHI, the mean score from patients in this study (52.66 ± 21.74) did not differ from the recent literature (59.8 ± 19.3). Patients with PPPD had a higher score when compared to the control group ($p = 0.008$). This figure allows for the conclusion that, compared to control subjects, the presented symptoms compromise the self-perception of body balance and interfere with the quality of life of these patients.[31] For questions relating to STAI questionnaire, no significant difference between groups in the STAI-E, which reproduces the state of anxiety at the time of consultation, was observed ($p = 0.36$). However, when evaluating STAI-T, it was noted that the PPPD group presented a significant difference when compared to controls ($p = 0.0008$). Gorestein and Andrade[20] classified STAI scores <33 as low; between 33 and 49, moderate; and >49, high. Eagger et al.[32] observed a STAI-E value of 43.5 ± 8.9 and a STAI-T of 40.4 ± 9.7, with no differences between symptomatic and asymptomatic subjects. In the present study, the STAI questionnaires scores were 38.66 ± 11.8 for STAI-E and 52 ± 10.19 for STAI-T, with a statistically significant difference when compared to controls only in STAI-T scale. Thus, the present sample had a mean STAI with high scores on both questionnaires, but mainly in STAI-T. It can be inferred that patients with PPPD do not arrive particularly

anxious during their clinical visit, but do exhibit anxiety as a characteristic trait of their personality. The anxiety pattern affects the assessment of postural challenges, and the patient assumes high-risk control strategies for movements of low demand, changing their gait and posture because of his/her hypervigilance. The higher the anxiety trait, the greater the fear of postural challenges and the more exaggerated the corrective strategies.[14]

The BDI reflects the degree of depression in the patient evaluated. A score of 15 points on the scale is considered normal, and patients who score between 16 and 20 points are considered as dysphoric (i.e., with a decreased level of humor, but not depression). In the present sample, a significant difference was observed between PPPD and control groups ($p = 0.0005$). The average for the PPPD group (14.57 ± 8.14) characterized most subjects as normal, but the high standard deviation of the sample shows that there is a greater tendency for a depressed mood in patients, when compared to controls.[20]

Regarding HADS, the present results are in agreement with those in the literature. Staab et al. found an average of 10.9 ± 3 for HADS-A and of 8.1 ± 4.4 for HADS-D.[25] The present sample had an average score of 12.57 ± 3.32 (normal <8) for HADS-A and 7.71 ± 4.42 (normal <8) for HADS-D. Both responses differ significantly from the control group ($p = 0.0001$ and $p = 0.0007$, respectively), demonstrating that PPPD patients have changes within the anxiety and depression area, compared to the asymptomatic population. These values, however, do not characterize individuals with PPPD as depressive, since the scoring for depression is borderline. However, the HADS-A was higher, just a little above the limit accepted for normality, again characterizing these patients as anxious.

According to literature, the response to SSRIs stays around 70% to 75%.[14,33] The present data do not differ from published reports, and the treatment produces satisfactory results in 46% of patients, with partial results in 22% of them. The sum of the positive responses reached 68% – a spectrum expected for the population of patients with PPPD.

Finally, the present experience with patients of PPPD show features very close to the international literature reports, and brings to light many aspects still little known to otorhinolaryngologists. Since this is a group of somatoform patients, the association between somatic and psychic evaluation is crucial for a better resolution of the symptoms.

Conclusion

PPPD is a dysfunction generated by psychic and somatic interactions, which affects more women in the menopausal age group, with high association with metabolic disorders and migraine. The main triggers of PPPD are visual conflict, head movements, stress, and sleep deprivation. The questionnaires help in the identification of predisposition to this disease. An adequate therapy confers to PPPD a good prognosis, with improvements observed in almost 70% of patients.

Funding

This study was conducted by FAPESP (Fundação de Amparo à Pesquisa do Estado de São Paulo).

Conflicts of interest

The authors declare no conflicts of interest.

References

1. Brandt T. Phobic postural vertigo. Neurology. 1996;46:1515–9.
2. Staab JP. Chronic dizziness: the interface between psychiatry and neuro-otology. Curr Opin Neurol. 2006;19:41–8.
3. Staab JP. Assessment and management of psychological problems in the dizzy patient. Continuum (Minneap Minn). 2006;12:189–213.
4. Staab JP. Chronic subjective dizziness; review article. Continuum (Minneap Minn). 2012;18:1118–41.
5. Huppert D, Strupp M, Rettinger N, Hecht J, Brandt T. Phobic postural vertigo – a long term follow up (5 to 15 years) of 106 patients. J Neurol. 2005;252:564–9.
6. Kapfhammer HP, Mayer C, Hock U, Huppert D, Dieterich M, Brandt T. Course of illness in phobic postural vertigo. Acta Neurol Scand. 1997;95:23–8.
7. Staab J, Eggers S, Neff B. Validation of a clinical syndrome of persistent dizziness and unsteadiness. Abstracts from the XXVI Bárány Society Meeting, Reykjavik, Iceland. 2010; 18–21. J Vestib Res. 2010;20:172–3.
8. Paulus MP. The role of neuroimaging for the diagnosis and treatment of anxiety disorders. Depress Anxiety. 2008;25:348–56.
9. Staab JP, Ruckenstein MJ. Which comes first? Psychogenic dizziness versus otogenic anxiety. Laryngoscope. 2003:113.
10. Heinrichs N, Edler C, Eskens S, Mielczarek MM, Moschner C. Predicting continued dizziness after an acute peripheral vestibular disorder. Psychosom Med. 2007;69:700–7.
11. Staab JP, Balaban CD, Furman JM. Threat assessment and locomotion: clinical applications of an integrated model of anxiety and postural control. Semin Neurol. 2013;33:297–306.
12. Staab JP. The influence of anxiety on ocular motor control and gaze, Review. Curr Opin Neurol. 2014;27:118–24.
13. Mahoney AEJ, Edelman S, Cremer PD. Cognitive behavior therapy for chronic subjective dizziness: longer-term gains and predictors of disability. Am J Otolaryngol. 2013:115–20.
14. Staab JP, Ruckenstein MJ, Amsterdam JD. A prospective trial of sertraline for chronic subjective dizziness. Laryngoscope. 2004;114:1637–41.
15. Balow RW, Honrubia V. The history of the dizzy patient in Clinical Neurophysiology of the Vestibular System. 2nd ed. Oxford Press; 2001. p. 111–31.
16. Tusa RJ. Dizziness. Med Clin North Am. 2009;93:263–71.
17. Garcia FV, Luzio CS, Benzinno TA, Veiga UG. Validação e adaptação do Dizzness Handicap Inventory para a língua e população portuguesa de Portugal. Acta ORL Técnicas em Otorrinolaringologia Edição. 2008;2.
18. Castro ASO, Gazzola JM, Natour J, Ganança FF. Versão Brasileira *do* Dizziness Handicap Inventory Pró-fono. Revista de Atualização Científica. 2007;n. 1:97–104.
19. Spielberger CD, Gorsuch RL, Lushene RE. Manual for the Strait-Trait Anxiety Inventory. Palo Alto, CA: Consulting Psychologists Press; 1970.
20. Gorenstein C, Andrade L. Validation of a Portuguese version of the Beck Depression Inventory and the State Trait Anxiety Inventory in Brazilian subjects. Braz J Med Biol Res. 1996;29:453–7.
21. Kendall PC, Hollon SD, Beck AT, Hammen CL, Ingram RE. Issues and recommendations regarding use of the Beck Depression Inventory. Cognit Ther Res. 1987;11:289–99.
22. Zigmond AS, Snaith RP. The hospital anxiety and depression scale. Acta Psychiatr Scand. 1983;67:361–70.

23. Baloh RW, Halmagyi GM. Disorders of the vestibular system. Oxford University Press; 1996. p. 113–25 [chapter 10].
24. Lopez C, Blanke O. The thalamocortical vestibular system in animals and humans. Brain Res Rev. 2011;67:119–46.
25. Staab JP, Robe DE, Eggers SDZ, Shepard NT. Anxious, introverted personality traits in patients with chronic subjective dizziness. J Psychosom Res. 2014;76:80–3.
26. Bittar RSM, Oiticica J, Bottino MA, Ganança FF, Dimitrov R. Population epidemiological study on the prevalence of dizziness in the city of São Paulo. Braz J Otorhinolaryngol. 2013;79:8–11.
27. Merikangas KR. Contributions of epidemiology to our understanding of migraine. Headache. 2013;53:230–46.
28. Loder E, Rizzoli P, Golub J. Hormonal management of migraine associated with menses and the menopause: a clinical review. Headache. 2007;47:329–40.
29. Staab JP, Ruckestein MJ. Expanding the differential diagnosis of chronic dizziness. Arch Otolaryngol Head Neck Surg. 2007;133:170–6.
30. Herdman SJ. Reabilitação Vestibular. 2nd ed. Editora Manole; 2002. p. 312–24 [chapter 14].
31. Jacobson GP, Newman CW. The Development of the Dizziness Handicap Inventory. Arch Otolaryngol Head Neck Surg. 1990;116:424–7.
32. Eagger S, Luxon LM, Davies RA, Coelho A, Ron MA. Psychiatric morbidity in patients with peripheral vestibular disorder: a clinical and neuro-otological study. J Neurol Neurosurg Psychiatry. 1992;55:383–7.
33. Staab JP, Ruckenstain MJ, Solomon D. Serotonin reuptake inhibitors for dizziness with psychiatric symptoms. Arch Otolaryngol Head Neck Surg. 2002;128:554–60.