Benign paroxysmal positional vertigo and vestibular myogenic evoked potential

https://doi.org/10.56238/sevened2024.001-043

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ABSTRACT

Benign Paroxysmal Positional Vertigo (BPPV) is considered a common disorder of the vestibular system. It corresponds to a hydro-mechanical disorder of the inner ear caused by abnormal stimulation of the dome of one or more of the three semicircular canals. BPPV represents approximately 25% of all vertigo of vestibular origin, it can manifest itself at any age, but it is more frequent after the age of 60. BPPV is known to be explained by the migration of otoconia from the utricle macula to the semicircular canals. The definitive diagnosis requires the performance of specific positional maneuvers, and the findings include: latency, direction, and duration of positional nystagmus. The vestibular myogenic evoked potential (VEMP) is a myogenic response evoked by brief pulses of sound and can be used as a complementary test for otoneurological evaluation, presenting several clinical applications in the diagnosis and follow-up of various vestibular diseases, including BPPV.

Keywords: Benign Paroxysmal Positional Vertigo, Vestibular System, Vestibular Diseases.

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INTRODUCTION

DEFINITION AND PATHOPHYSIOLOGY

Benign Paroxysmal Positional Vertigo (BPPV), is considered a common disorder of the vestibular system, also known as "labyrinth crystal disease", corresponding to a hydromechanical disorder of the inner ear caused by abnormal stimulation of the dome of one or more of the three semicircular canals, when there is a change in the position of the head1,2.

Its pathophysiology is characterized by the displacement of calcium carbonate crystals or otoconia within the semicircular canals. These otoconia, under normal conditions, are attached to the otolytic membrane, present in the macula of the vestibular organs: utricle and saccule. The movement of the otoconia within the semicircular canals abnormally stimulates the dome, generating nerve impulses3. In any channel that otoconia is deposited, it ends up hypo- or hyper-stimulating the semicircular canal, generating an imbalance, causing the sensation of vertigo. In this sense, BPPV can be classified into three categories4, 5, 6, 7.

- In the condition of canalithiasis, when the otoconia float freely in the endolymph, that is, in the fluid that circulates in the semicircular canals.
- In the condition of cupulolithiasis, when calcium carbonate crystals (otoliths or otoconia) adhere to the dome.
- Obstruction of the semicircular canal, a rarer form, in which the impacted particles obstruct the semicircular canal.

Canalithiasis and cupulolithiasis are not mutually exclusive and can coexist simultaneously in the same semicircular canal. BPPV may present unilateral or bilateral labyrinthine involvement, or simultaneous involvement of different channels8.

Posterior canal involvement is the most frequent form, in about 90% of cases, because in the upright position, the ampulla of the posterior canal is on a slope in the inner ear. The horizontal channel BPPV represents 9% of the BPPVs, while the anterior channel BPPVs are considered rare, as they represent only 1% of all BPPVs[9].

Barany10 described BPPV for the first time in 1921, at that time, vertigo and characteristic nystagmus associated with postural changes were linked to the otolithic organs. In 1952, Dix and Hallpike[11], during their provocative tests, described classical nystagmus and began to explain that the location of the pathology was the inner ear itself11.

EPIDEMIOLOGY

In the general population, the prevalence of dizziness and vertigo is approximately 20% to 30%[12,13,14]. BPPV represents approximately 25% of all vertigo of vestibular origin and is considered

the most frequent form of peripheral vertigo. It can manifest at any age, but it is less common in childhood and more frequent after the age of 60.

The scientific literature has shown that the prevalence of BPPV is higher in females, one of the hypotheses being hormonal variations such as estrogen and progesterone fluctuations, resulting from the senescence process, making women more susceptible to otoneurological alterations15,16. A cross-sectional study reported a lifetime prevalence of BPPV of 2.4%, being more prevalent in women (3.2%) than in men (1.6%)[17].

Approximately 50% to 70% of BPPV cases occur without a known cause, and are classified as primary or idiopathic BPPV18. The remaining cases are called secondary BPPV and are often associated with an underlying pathology, such as: traumatic brain injury, vestibular neuronitis, labyrinthitis, Ménière's disease, migraine, among others19.

Studies point to the following BPPV frequencies: 41 to 65% posterior channel, 21 to 45% horizontal channel, 20% multiple channels, and 17% anterior channel. The posterior semicircular canal is the most frequently involved structure due to its anatomical position[20]. The variants of the horizontal and anterior canal are less prevalent because they are not in a gravity-dependent position21.

SIGNS AND SYMPTOMS

The pathogenesis of BPPV is still not well understood. However, BPPV is known to be explained by the migration of otoconia from the uterine macula to the semicircular canals. Two theories are raised here: in the first theory, called Cupulolithiasis, the fragments would be adhered to the dome of the posterior semicircular canal. The second theory, called Canalithiasis or Ductolithiasis, suggests that the degenerated fragments float in the endolymph of the semicircular canal.

The triggering of its clinical manifestations occurs through gravity-related head movements, i.e., a sudden movement from the upright to the supine position. They are often provoked by everyday activities and usually occur when rolling over in bed or tilting the head to look up or lean forward, for example. The result is a transient spinning sensation, i.e. brief, violent episodes of vertigo, which usually disappear within 20 to 30 seconds, and which may or may not be accompanied by an oculomotor phenomenon (nystagmus). The vertiginous sensation caused by BPPV may persist, causing from mild symptoms to disabling manifestations that last for years22.

The severity of each episode is variable. Patients with BPPV do not experience dizziness all the time, however, in extreme cases, even the slightest movement of the head can result in nausea and vomiting. Between episodes, patients have few or no symptoms at rest. Other symptoms of

BPPV include imbalance, increased risk of falling and fear of falling, decreased activity levels, anxiety, impaired vision, and headache.

Vestibular diseases have had an increasing impact on patients' quality of life. Many of these patients restrict their activities, such as social and occupational interactions, in an attempt to reduce the probable onset of symptoms, often limiting their routine movements to avoid causing vertigo episodes23.

DIAGNOSIS

A detailed medical history and careful physical examination are essential to assess vertigo, since differentiating peripheral and central vestibular processes is of paramount importance. The most important biomarkers of BPPV are clinical history and positional tests, i.e., the detection of positional nystagmus24. Detecting the semicircular canal involved in BPPV is important for the treatment to be performed, i.e., for the precise choice of the repositioning maneuver.

Definitive diagnosis requires specific positional maneuvers, and the essential clinical findings for diagnosis are: latency, direction, and duration of positional nystagmus. BPPV may present unilateral or bilateral labyrinthine involvement, or simultaneous involvement of different channels8.

The most commonly used diagnostic procedures are: the Dix-Hallpike test for the diagnosis of anterior and posterior canal BPPV and the Pagnini-McClure test for the diagnosis of horizontal canal BPPV. These procedures induce a nystagmic response and vertiginous sensation and, in addition to confirming the diagnosis of BPPV, enable the identification of the semicircular canal, the affected side, and the probable pathophysiology (canalithiasis and cupulolithiasis)[25].

The Dix-Hallpike maneuver, considered the gold standard test, consists of moving the patient's head in such a way as to promote a displacement of the endolymph in the posterior semicircular canal. In this maneuver, the patient is quickly moved from the sitting posture to the supine position, with lateral rotation of the head by 45 degrees to the tested side, in this position, the patient remains staring at the examiner and after 20 to 30 seconds, the patient is taken back to the sitting position. If there is no nystagmus, the same procedure is repeated on the opposite side. Lopez-Escamez et al.[26] indicated sensitivity of 82% and specificity of 71% in the Dix-Hallpike maneuver in patients with posterior canal BPPV.

The pathognomonic sign of BPPV is rotational nystagmus with latency and short duration, classified as objective when nystagmus is observed during the test, or subjective when vertigo is present, without the presence of nystagmus27.

As already mentioned, BPPV is largely a clinical diagnosis, and often the battery of laboratory and imaging tests ordered only helps to rule out other conditions.

REPOSITIONING MANEUVERS

Repositioning maneuvers have been increasingly useful in the treatment of BPPV in order to move the otoconia out of the canal and back into the vestibule. Medications usually given to suppress vestibular influence are not an effective treatment option. Nausea and vomiting, neurovegetative symptoms in general, are common complaints of BPPV and can be treated with antiemetics as needed. Studies have shown a trend towards improvement in symptoms, especially for vertigo, dizziness and nystagmus, after otolithic repositioning maneuvers.

Although repositioning maneuvers are effective, it is worth remembering their contraindications: unstable cardiovascular disease, suspected vertebrobasilar disease, severe cervical disease, high-grade carotid stenosis, among others[22].

The scientific literature describes the most commonly used maneuvers for the treatment of BPPV:

1. Epley's maneuver - used for the treatment of posterior canal BPPV;
2. Gufoni maneuver - used for the treatment of horizontal canal BPPV;
3. Lempert's maneuver or BBQ Roll - used for the treatment of horizontal canal BPPV and
4. Yacovino maneuver - used for the treatment of anterior BPPV.

Epley30 reports 400 patients treated using the repositioning maneuver for the posterior semicircular canal, created by him, with a cure rate in 95% of the patients. This maneuver has been widely used by professionals because it is well tolerated by patients.

It is worth mentioning that, despite adequate and successful treatment, there are recurrences, studies point to a recurrence rate of 5 to 25%, so additional treatment may be necessary. The risk of recurrence is higher in women, older patients, and those with psychiatric comorbidities.

It should be noted that the most important part of treatment is accurate diagnosis, considering the patient's clinical history and the result of positional tests. An incorrectly performed maneuver, even during evaluation, can lead to worsening of symptoms.

VESTIBULAR MYOGENIC EVOKED POTENTIAL IN BPPV

Vestibular myogenic evoked potential (VEMP) It is a myogenic response evoked by brief pulses of sound and can be used as a complementary examination of the otoneurological evaluation31,32. Currently, it has been widely used in clinical practice to evaluate patients with vertigo and body balance disorders.

The VEMP is considered an objective, non-invasive test for the patient, fast, easy to perform and aims to evaluate the functional integrity of the sacculo-colic reflex (saccule, inferior vestibular

nerve, vestibular nucleus, spinal nerve nucleus and sternocleidomastoid muscle), which is called cervical VEMP (cVEMP)[31,32]. Therefore, the otolytic function can be assessed with VEMPs.

Studies that have used VEMP in patients affected by BPPV describe the alterations that can be found in patients with BPPV and demonstrate significantly greater differences in VEMP responses in patients with BPPV when compared to recurrent and non-recurrent groups.

A study33 examined the characteristics of the cervical vestibular myogenic evoked potential (cVEMP) in patients with BPPV and found a greater number of changes on the pathological side compared to healthy controls. Another study by Hong et al.[34] in patients with BPPV describe abnormalities, such as increased cVEMP latency, on the affected side.

VEMP has several clinical applications in the diagnosis and follow-up of various vestibular diseases, including BPPV. However, it is important to emphasize the need for further studies on this pathology.

CONCLUSION

BPPV is one of the most prevalent vestibular diseases, characterized by the displacement of calcium carbonate crystals within the semicircular canals or the dome of these canals, with a higher prevalence of posterior canal involvement and age group over 60 years. Classified as the most frequent form of peripheral vertigo, most cases have unknown etiology, its clinical manifestations are often triggered during daily activities, resulting in a transient spinning sensation. The vertiginous sensation can be persistent and extend over long periods.

The diagnosis consists of positional maneuvers that induce a nystagmic response and vertiginous sensation. BPPV may present with unilateral or bilateral labyrinthine involvement, or simultaneous involvement of different channels. Repositioning maneuvers are used in rehabilitation in order to redirect the otoconia to the vestibule.

REFERENCES

1. Kim, J. S., & Zee, D. S. (2014). Benign paroxysmal positional vertigo. *New England Journal of Medicine*, *370*, 1138-1147.

2. Casani, A. P., Nacci, A., Dallan, I., et al. (2011). Horizontal semicircular canal benign paroxysmal positional vertigo: effectiveness of two different methods of treatment. *Audiology and Neurotology*, *16*, 175-184.

3. Suzuki, M., Kadir, A., Hayashi, et al. (1996). Functional model of benign paroxysmal positional vertigo using an isolated frog semicircular canal. *Journal of Vestibular Research*, *6*(2), 121-125.

4. Epley, J. M. (2001). Human experience with canalith repositioning maneuvers. *Annals of the New York Academy of Sciences*, *942*, 179-191.

5. Schuknecht, H. (1969). Cupulolithiasis. *Archives of Otolaryngology*, *90*, 765-778. http://dx.doi.org/10.1001/archotol.1969.00770030767020.

6. Hall, S. F., Ruby, R. R., & McClure, J. A. (1979). The mechanics of benign paroxysmal vertigo. *The Journal of Otolaryngology*, *8*(2), 151–158.

7. Epley, J. M. (1980). New dimensions of benign paroxysmal positional vertigo. *Otolaryngology–Head and Neck Surgery*, *88*(5), 599–605. http://dx.doi.org/10.1177/019459988008800514.

8. Lopez-Escamez, J. A., Molina, M. I., Gamiz, M. J., et al. (2005). Multiple positional nystagmus suggests multiple canal involvement in benign paroxysmal positional vertigo. *Acta Oto-Laryngologica*, *125*(9), 954-956.

9. Sauvage, J. P., & Grenier, H. (2017). Reabilitação vestibular - guia prático. 1ª ed. Rio de Janeiro: Revinter.

10. Andaz, C., Whittet, H. B., & Ludman, H. (1993). Uma causa incomum de vertigem posicional paroxística benigna. *Journal of Laryngology and Otology*, *107*(12), 1153-1154.

11. Hornibrook, J. (2011). Benign paroxysmal positional vertigo (BPPV): History, pathophysiology, office treatment and future directions. *International Journal of Otolaryngology*, *2011*, 835671.

12. Karatas, M. (2008). Central Vertigo and Dizziness: Epidemiology, Differential Diagnosis And Common Causes. *The Neurologist*, *14*, 355-364.

13. Chang, J., Hwang, S. Y., Park, S. K., Kim, J. H., Kim, H. J., Chae, S. W., et al. (2018). Prevalence of dizziness and associated factors in South Korea: a cross-sectional survey from 2010 to 2012. *Journal of Epidemiology*, *28*, 176-184.

14. Murdin, L., & Schilder, A. G. (2015). Epidemiology of balance symptoms and disorders in the community: a systematic review. *Otology & Neurotology*, *36*(3), 387-392.

15. Moreira, M. D., Costa, V. S. P., Melo, J. J., & Marchiori, L. L. M. (2014). Prevalência e associações da vertigem posicional paroxística benigna em idosos. *Revista CEFAC*, *16*(5), 1533-1540. doi: 10.1590/1982021620149513.

16. Guzmán, P. V., Zeigelboim, B. S., Hassan, S. E. F., Diniz Junior, J., & Caovilla, H. H. (2000). A manobra de Brandt & Daroff modificada na reabilitação da vertigem postural. *Acta AWHO*, *19*(4), 189-192.

17. Von Brevern, M., Radtke, A., Lezius, F., Feldmann, M., Ziese, T., Lempert, T., & Neuhauser, H. (2007). Epidemiologia da vertigem posicional paroxística benigna: um estudo de base populacional. *Journal of Neurology, Neurosurgery & Psychiatry*, *78*(7), 710-715.

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18. Parnes, L. S., Agrawal, S. K., & Atlas, J. (2003). Diagnóstico e tratamento da vertigem posicional paroxística benigna (VPPB). *CMAJ*, *169*(7), 681-693.

19. Katsarkas, A. (1999). Vertigem posicional paroxística benigna (VPPB): idiopática versus pós-traumática. *Acta Oto-Laryngologica*, *119*(7), 745-749.

20. Epley, J. (1992). The canalith repositioning procedure: for treatment of benign paroxysmal positional vertigo. *Otolaryngology–Head and Neck Surgery*, *107*, 399-404.

21. Cakir, B. O., Ercan, I., Cakir, Z. A., Civelek, S., Sayin, I., & Turgut, S. (2006). What is the true incidence of horizontal semicircular canal benign paroxysmal positional vertigo?. *Otolaryngology–Head and Neck Surgery*, *134*, 451-454.

22. Palmeri, R., & Kumar, A. (2022, December 26). Benign Paroxysmal Positional Vertigo. *National Library of Medicine*.

23. Taguchi, C. Z., & Bohlsen, Y. A. (2015). Reabilitação vestibular. In: Boéchat EM, Menezes PL, Couto, CM, et al. *Tratado de audiologia* (2nd ed., pp. 551-559). Rio de Janeiro: Guanabara Koogan.

24. Bhattacharyya, N., Baugh, R. F., Orvidas, L., Barrs, D., Bronston, L. J., Cass, S., et al. (2008). Clinical practice guideline: benign paroxysmal positional vertigo. *Archives of Otolaryngology*, *139*(5 Suppl 4), 47-81.

25. Libonati, G. A. (2014). Vertigem posicional paroxística benigna. In: Maia FCZ, Albernaz PLM, Carmona S. *Otoneurologia atual* (1st ed., pp. 275-317). Rio de Janeiro: Revinter.

26. Lopes-Escamez, J. Á., Gamiz, M. J., Fernandez-Perez, A., & Gomes-Finana, M. (2005). Long-term outcome and health-related quality of life in benign paroxysmal positional vertigo. *European Archives of Oto-Rhino-Laryngology*, *262*, 507-511.

27. Haynes, D. S., Resser, J. R., Labadie, R. F., Girasole, C. R., Kovach, B. T., & Scheker, L. E., et al. (2002). Treatment of benign positional vertigo using the Semont maneuver: efficacy in patients presenting without nystagmus. *The Laryngoscope*, *112*(5), 796-801.

28. Maia, R. A., Flávia, L., Diniz, F. L., & Carlesse, A. (2001). Treatment of benign paroxysmal positional vertigo with repositioning maneuvers. *Revista Brasileira de Otorrinolaringologia*, *67*(5). https://doi.org/10.1590/S0034-72992001000500003

29. Cal, R., Maia, F. C. Z., Araújo, M. S., & Brusco, T. R. (2014). In: Maia FCZ, Albernaz PLM, Carmona S. *Otoneurologia atual* (1st ed., pp. 275-317). Rio de Janeiro: Revinter.

30. Colebatch, J. G., Halmagyi, G. M., & Skuse, N. F. (1994). Myogenic potentials generated by a click-evoked vestibulocolic reflex. *Journal of Neurology, Neurosurgery & Psychiatry*, *57*, 190-197.

31. Rosa, M. S., Campagnoli, M., Masnaghetti, D., et al. (2023). Clinical and prognostic implications of cervical and ocular vestibular evoked myogenic potentials (cVEMP and oVEMP) in benign paroxysmal positional vertigo (BPPV): A prospective study. *Audiology Research*, *13*(5), 700-709. https://doi.org/10.3390/audiolres13050061.

32. Hong, S. M., Parque, D. C., Si, S. G., & Cha, I. C. (2008). Potenciais evocados miogênicos vestibulares em pacientes com vertigem posicional paroxística benigna envolvendo cada canal semicircular. *Journal of Otolaryngology*, *29*, 184–187.