Review Article

Diagnostic Criteria of Benign Paroxysmal Positional Vertigo

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Abstract

Benign paroxysmal positional vertigo (BPPV) is the mos common cause of peripheral vertigo. The pathophysiology of BPPV is canalolithiasis which includes free-floating otoconial debris within the endolymph of a semicircular canal, or cupulolithiasis comprising otoconial debris adherent to the cupula. The posterior and/or lateral semicircular canals are often affected in BPPV. It is characterized by a brief episode of rotatory vertigo associated with positional and/or positioning nystagmus, which is elicited by specific head positions or changes in head position relative to gravity. In the case of the posterior canal type of BPPV, torsional nystagmus is induced by the Dix-Hallpike test. In patients of lateral canal type of BPPV, horizontal geotropic or apogeotropic nystagmus is induced by the supine roll test. The observation of positional and/or positioning nystagmus is needed for the diagnosis of BPPV. The treatment of BPPV includes canalith repositioning procedure (CRP). By series of head position changes, the CRP moves the otoconial debris from the involved semicircular canal to the utricle. This review article presents operational diagnostic criteria for BPPV, formulated by the Committee for Classification of Vestibular Disorders of the Barany Society. This classification reflects the current knowledge of the clinical aspect and pathophysiology of BPPV. These diagnostic criteria will be helpful for the understanding of the disease and will lead to further development in the management of BPPV.

Keywords: Benign paroxysmal positional vertigo, diagnostic criteria, Dix-Hallpike maneuver, torsional nystagmus

Introduction

Benign paroxysmal positional vertigo (BPPV) is the most common cause of vertigo in routine clinical practice.[1] BPPV is an otoconial disorder that causes an episodic vestibular syndrome of short duration, generally under a minute.[2] This episodic vestibular syndrome is characterized by Nystagmus triggered by the abnormal stimulation caused by otoconia in the semi-circular canal affected.[3] In the majority of cases, BPPV is characterized by brief attacks of rotatory vertigo with torsional positioning nystagmus, which are elicited by changes in the head position relative to gravity.[3] The diagnosis of BPPV is supported if the changes in head position with respect to gravity-induced symptoms and elicit the patterns characteristic of BPPV. These symptoms are usually found when the patient goes to bed, turns in their bed, or bends their head down.[4] As many physicians are not familiar with the precise anatomical relationships of the semicircular canals in the skull, it will be a challenge to interpret the different types of positional nystagmus and perform the correct maneuvers.[4] Sometimes, BPPV involves multiple semicircular canals in one ear or is bilateral, making it difficult to find types of nystagmus and choose the best treatment. The diagnostic criteria of BPPV described here are part of the international classification of vestibular disorders (ICVD)-an endeavor for the classification of vestibular disorders steered by the Committee for Classification of Vestibular Disorders of the Barany Society.

Methods of Literature Search

Multiple systematic methods were used to find current research publications on diagnostic criteria of benign paroxysmal positional. We started by searching the Scopus, PubMed, Medline, and Google Scholar databases online. A search strategy using Preferred Reporting Items for Systematic Reviews and Meta-Analysis guidelines was developed. This search strategy recognized the abstracts of published articles,

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while other research articles were discovered manually from the citations. Randomized controlled studies, observational studies, comparative studies, case series, and case reports were evaluated for eligibility. There were a total number of 58 articles (20 case reports; 16 cases series; 22 original articles) [Figure 1]. This article focuses only on the details of diagnostic criteria of BPPV. This review article describes the epidemiology, history, pathophysiology, and diagnostic criteria of BPPV. This analysis provides a better understanding of BPPV and its diagnostic criteria. It will also serve as a catalyst for further study of the diagnosis of BPPV and the development of newer criteria for the better management of BPPV.

Epidemiology

BPPV is the most common vestibular disorder, with a reported prevalence between 10.7 and 64.0 cases per 100,000 population and a lifetime prevalence of 2.4%.[5] The prevalence of idiopathic BPPV is more among elderly persons and females, with peak onset between 50 and 60 years of age and female-to-male ratio of 2:1–3:1.[5] The cumulative incidence of BPPV in the general population during their lifetime is approximately 10%.[6] The recurrences of BPPV occur in approximately 50% of patients.[7] Although BPPV is usually self-limiting, it affects the personal and socioeconomic condition of the patients. BPPV often arises from the posterior semicircular canal, which is the most gravity-dependent canal; this variety of BPPV accounts for 60%–90% of all cases.[8] However, the proportion of BPPV patients that involves the lateral semicircular canal may have been underestimated, as involvement in this canal is more chance to remit spontaneously than involvement in the posterior semicircular canal.[9] Rarely, BPPV involves the anterior semicircular canal, due to its uppermost position in the labyrinth where otolith debris is unlikely to be trapped.[5,10]

History

Robert Barany first documented positional vertigo in 1921.[11] The term BPPV was coined by Dix and Hallpike in 1952.[12] Dix and Hallpike also reported that BPPV was caused due to lesion of the otolith organ.[12] In a postmortem examination of the temporal bones, Schuknecht then reasoned that otoconia coming from the otolithic membrane settled on the cupula (cupulolithiasis) and that the cupula responds to gravity.[13] Hall *et al*. later on documented that otoconial debris floats freely within the endolymph of the semicircular canal (canalolithiasis).[14] McClure first reported a lateral canal type of BPPV in 1985, in which a lateral semicircular canal was involved.[9] The Barany Society's ICVD established the diagnostic criteria for BPPV. These diagnostic criteria need to define the different clinical types of the disease as per characteristics of the nystagmus seen during positional tests. The classification determines two diagnostic types such as established positional syndromes (very common and documented in many studies) and controversial and emerging syndromes (less common and with fewer published studies).

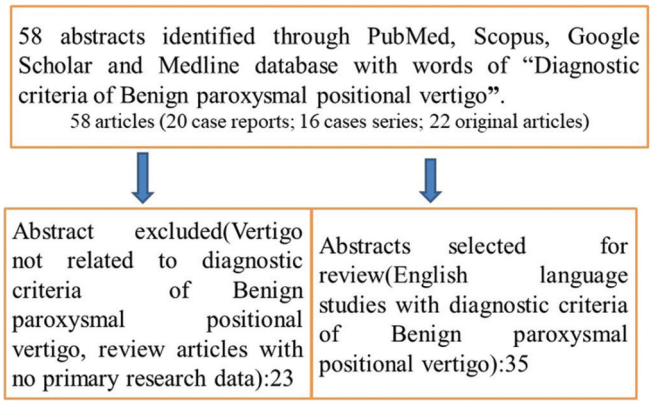

Figure 1: Flow chart showing methods of literature search

Pathophysiology

As per the accepted theory, BPPV is usually caused by the dislodgement of the otoconia from the otolith macula beds and is trapped in a semicircular canal.[15] The gravity result in their movement of them after changes of head position in the plane of the affected semicircular canal.[15] The inappropriate flow of the endolymph deflects the cupula and modulates the activity of the vestibular afferents of the affected semicircular canal, resulting in the attacks of positional vertigo and nystagmus (canalolithiasis).[16] Less popular, BPPV can occur due to otoconia that are attached to the cupula of a semicircular canal and render it sensitive to gravity (cupulolithiasis).[17] (paroxysmal positional) BPPV is not accompanied by cochlear manifestations (hearing loss and tinnitus) and headache including migraine.[18]

Examination for Diagnosis of Benign Paroxysmal Positional Vertigo

Positional and/or positioning nystagmus is usually observed with Frenzel's glasses or glasses equipped with an infrared charge-coupled device camera.[19] The characteristics of evoked nystagmus such as direction, amplitude, frequency, and torsional/vertical/horizontal components of the nystagmus are examined.[20] The latency that elapses before the appearance of the nystagmus and changes in the intensity of the nystagmus thereafter is also evaluated. In case, patients suffer from the cervical vertebral disease, the positional and/or positioning nystagmus test is avoided. This test must be canceled when the patient feels neck pain, sensory disturbance, and disturbance of consciousness. The positional test is done in a supine position and the head is rotated to the right ear down or left ear down position. The positional nystagmus test is called the supine roll test, which is helpful for the diagnosis of the lateral canal type of BPPV.[21] The Dix-Hallpike test is useful for the diagnosis of the posterior canal type of BPPV. In the case of the right posterior canal type of BPPV, right torsional nystagmus (the upper pole of the eye rotates to the right side) is induced by the right Dix-Hallpike test. The nystagmus

usually contains an additional vertical (upward) component. The nystagmus appears with short latency, lasts for less than a minute, and typically increases followed by a decrease in its intensity. In the majority of patients with the posterior canal type of BPPV, the transient torsional nystagmus with latency is induced by canalolithiasis inside the posterior semicircular canal.[22] Few patients of posterior canal type of BPPV show persistent torsional type nystagmus without any latency, the pathophysiology which is thought to be cupulolithiasis in the posterior canal.[23] The Dix-Hallpike test is the gold standard test where the patient sits on the bed and turns the head 45° toward the side that is to be evaluated.[1] Then, the patient is put into the supine position, extending his/her head about 15°–20° below the horizontal; this causes movement on the plane of the corresponding posterior canal which enables ampullofugal displacement of the canalith.[2] In the case of the right-sided Dix-Hallpike test, the right posterior semicircular canal is mobilized, and nystagmus is triggered with an up beating vertical and counterclockwise torsional component. The nystagmus is disconjugate (as it corresponds to the stimulation of the vertical canals), the same side eye shows the torsional component more markedly, and the contralateral eye the vertical component.

Diagnosis

The complete diagnosis of BPPV includes the specification of the affected semicircular canal(s) and the pathophysiology such as canalolithiasis or cupulolithiasis. The exact diagnosis of BPPV needs diagnostic positional maneuvers that lead to the observation of canal-specific positional nystagmus. The clinical features such as latency, direction, time course, and duration of the positional nystagmus are important parts of getting the diagnosis. Imaging the brain or ear is not essential in typical cases of BPPV.[24] The positional testing includes provocation of vertigo and nystagmus where different maneuvers test different semicircular canals. A canal-specific response is elicited when a rotation of the head in the plane of a semicircular canal induces positional nystagmus of the maximum intensity (in terms of slow-phase velocity). In contrast to central positional nystagmus, the positional nystagmus in BPPV always beats in the plane of the affected semicircular canal and the expected direction for canal excitation or inhabitation.[25]

Differential Diagnosis

The differential diagnosis of BPPV is central positional vertigo due to vestibular migraine and structural brainstem and cerebellar lesions, particularly in the vicinity of the fourth ventricle.[26] The diseases of central nervous diseases can be excluded by proper neurological evaluation, but a few cases may pose challenges to clinicians for diagnosis.[27] Magnetic resonance of imaging is usually advised when the symptoms or signs of concurrent cerebellar or brainstem dysfunction are present, or when positional vertigo and nystagmus are seen with atypical features or fail to resolve with repeated positional maneuvers.[24,28] The otological disorders which may mimic BPPV include Meniere's disease, acute unilateral vestibular syndrome (labyrinthitis, neuritis), superior semi-circular canal dehiscence, dilated vestibular aqueduct, and perilymphatic fistula.[29,30]

Diagnostic Criteria

Canalolithiasis of posterior canal benign paroxysmal positional vertigo

The diagnostic criteria of posterior canal-BPPV (pc-BPPV) include: (a) The recurrent attacks of positional vertigo or positional dizziness are provoked by lying down or turning over in the supine position. The intensity of vertigo decreases or disappears after repeated head positioning.[31] (b) The duration of vertigo is <1 min; (c) The vertigo is not associated with any cochlear symptoms such as hearing impairment, tinnitus, or aural fullness; (d) The nystagmus is elicited after a latency of one or few seconds by the Dix-Hallpike maneuver or side-lying maneuver (Semont's maneuver). The nystagmus is a combination of torsional nystagmus with the upper pole of the eyes beating toward the lower ear combined with vertical nystagmus beating upward (toward the forehead) classically lasting for <1 min.[32] (e) There is no attributable to other disorders.

Canalolithiasis of the horizontal canal benign paroxysmal positional vertigo

There are two acknowledged types of horizontal canal-BPPV (hc-BPPV), canalithiasis, and cupulolithiasis, although the former is a more common variety.[33] There are again two subvariants of canalithiasis, based on whether the particles are in the posterior or anterior arm of the canal.[34] The diagnostic criteria of hc-BPPV include: (a) Attacks of rotatory vertigo or dizziness are occurred by changes in the head position relative to gravity. Recurrent attacks of positional vertigo or positional dizziness induced by lying down or turning over in the supine position; (b) Vertigo appears with short latency and The duration of vertigo attacks is <1 min followed by a decrease in its intensity; (c) The intensity of vertigo decreases after repeated head positioning; (d) Positional nystagmus elicited after a brief latency or no latency by the supine roll test and beats horizontally toward the undermost ear with head turned to either side (geotropic direction-changing nystagmus) and lasting for <1 min; (e) The vertigo is not associated with any cochlear symptoms such as hearing loss, tinnitus, or ear fullness; (f) Not attributable to another disorder.[35] One of the problems with the horizontal semicircular canal is that the diagnostic tests act on both sides of horizontal semicircular canals at the same time, making it difficult to determine the involved side, which is needed for planning the treatment.[36] Pagnini-McClure or roll test is a provocation test for the horizontal semicircular canal and it is performed with the patient in a supine position. It is ideally done with their head bent 30° so that the plane of the two horizontal semicircular canals is vertical.

Canalolithiasis of the anterior canal benign paroxysmal positional vertigo

The anterior canal is the least commonly affected (3% BPPV).[37] Canalolithiasis of the anterior semicircular canal is uncommon compared to posterior and horizontal semicircular canal variants. This may be due to the anatomical orientation of the anterior canal, which allows the particle to leave the canal simply after lying down and sitting up again. The diagnostic criteria of the anterior canal-BPPV include: (a) Recurrent episodes of positional vertigo or positional dizziness induced by lying down or turning over in the supine position; (b) Duration of attacks is <1 min; (c) Positional nystagmus is elicited immediately or after a latency of one or few seconds by the Dix-Hallpike test (in one or both sides) or in a supine straight head-hanging position, beating predominantly vertically downward and lasting for <1 min.[38]

Cupulolithiasis of the posterior canal benign paroxysmal positional vertigo-cupulolithiasis

The diagnostic criteria of the pc-BPPV-cupulolithiasis include: (a) Recurrent episodes of positional vertigo or positional dizziness provoked by lying down or turning over in the supine position; (b) Positional nystagmus elicited after a brief or no latency by a half Dix-Hallpike test, beating torsionally with the upper pole of the eye to the lower ear and vertically upward (to the forehead) and lasting for more than 1 min; (c) No attributable to another disorder.[39]

Cupulolithiasis of the horizontal canal-benign paroxysmal positional vertigo-cupulolithiasis

The diagnostic criteria of hc-BPPV-cupulolithiasis include: (a) Attacks of rotatory vertigo or dizziness are induced by specific head positions. Recurrent episodes of positional vertigo or positional dizziness provoked by lying down or turning over in the supine position; (b) Positional nystagmus is detected after a brief latency or no latency by supine roll test, beating horizontally toward the uppermost ear with the head turned to either side (apogeotropic direction changing nystagmus) and lasting for more than 1 min; (c) The vertigo is not associated with any cochlear symptoms such as hearing loss, tinnitus, or ear fullness; (d) No attributable to another disorder.[39]

Lithiasis of multiple semicircular canals-benign paroxysmal positional vertigo

This is included in the Barany Society as an emerging diagnosis.[19] It might be underdiagnosed and more common in cases of posttraumatic BPPV. The most frequently described combination is of the posterior and horizontal canals of a labyrinth, although any combination would be possible.[19] The diagnostic criteria of multiple semicircular canals-BPPV include (a) Recurrent episodes of positional vertigo or positional dizziness provoked by lying down or turning over in the supine position; (b) The duration of the attack is <1 min; (c) The positional nystagmus is compatible with canalolithiasis of more than one semicircular canal during the Dix-Hallpike test and the supine roll test; (d) Not attributable to another disorder.[32,40]

Conclusion

BPPV is the commonest episodic inner or vestibular ear disorder. It is due to the displacement of otoconia that dislodge from the utricular otolithic membrane and shift toward the semicircular canal. The intensity, frequency, and duration of the symptoms in the case of BPPV depend on the affected semicircular canals and sites of the otolithic debris. Clinical findings such as latency, reversibility, crescendo, transience, and fatigability are usually present in BPPV which support the diagnosis. A diagnosis of BPPV is often made through clinical history along with diagnostic maneuvers. BPPV is often amenable to in-office repositioning techniques.

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Conflicts of interest

There are no conflicts of interest.

References

1. Swain SK. Benign paroxysmal positional vertigo in patients with Meniere's disease. Saudi J Otorhinolaryngol Head Neck Surg 2022;24:51-5.
2. Swain SK, Sahu MC, Samal R, Padhy RN. Incidence of hearing loss, tinnitus and vertigo among diabetes patients. Siriraj Med J 2014;66:179-84.
3. Kim HJ, Park J, Kim JS. Update on benign paroxysmal positional vertigo. J Neurol 2021;268:1995-2000.
4. Instrum RS, Parnes LS. Benign paroxysmal positional vertigo. Adv Otorhinolaryngol 2019;82:67-76.
5. Bhattacharyya N, Baugh RF, Orvidas L, Barrs D, Bronston LJ, Cass S, *et al.* Clinical practice guideline: Benign paroxysmal positional vertigo. Otolaryngol Head Neck Surg 2008;139:S47-81.
6. von Brevern M, Radtke A, Lezius F, Feldmann M, Ziese T, Lempert T, *et al.* Epidemiology of benign paroxysmal positional vertigo: A population based study. J Neurol Neurosurg Psychiatry 2007;78:710-5.
7. Nunez RA, Cass SP, Furman JM. Short- and long-term outcomes of canalith repositioning for benign paroxysmal positional vertigo. Otolaryngol Head Neck Surg 2000;122:647-52.
8. Furman JM, Cass SP. Benign paroxysmal positional vertigo. N Engl J Med 1999;341:1590-6.
9. McClure JA. Horizontal canal BPV. J Otolaryngol 1985;14:30-5.
10. Swain SK, Shajahan N, Acharya S. A scoping review of vestibular paroxysmia: An acute disabling clinical entity. J Acute Dis 2020;9:248-52.
11. Yamane H, Konishi K, Anniko M. Visualization of horizontal canal benign paroxysmal positional vertigo using 3DCT imaging and its assessment. Acta Otolaryngol 2021;141:482-9.
12. Dix MR, Hallpike CS. The pathology symptomatology and diagnosis of certain common disorders of the vestibular system. Proc R Soc Med 1952;45:341-54.
13. Schuknecht HF. Cupulolithiasis. Arch Otolaryngol 1969;90:765-78.
14. Hall S, Ruby P, McClure J. The mechanics of benign paroxysmal positional vertigo. J Otolaryngol 1979;8:151-8.
15. Swain SK. Benign paroxysmal positional vertigo in pediatric age group: A review. Int J Contemp Pediatr 2022;9:863-8.
16. Brandt T, Steddin S. Current view of the mechanism of benign paroxysmal positioning vertigo: Cupulolithiasis or canalolithiasis? J Vestib Res 1993;3:373-82.
17. Lechner C, Taylor RL, Todd C, Macdougall H, Yavor R, Halmagyi GM, *et al.* Causes and characteristics of horizontal positional nystagmus. J Neurol 2014;261:1009-17.
18. von Brevern M, Bertholon P, Brandt T, Fife T, Imai T, Nuti D, *et al.* Benign paroxysmal positional vertigo: Diagnostic criteria. J Vestib Res

2015;25:105-17.
19. Swain SK, Debta P, Lenka S, Samal S, Dani A, Mahapatra S. Vestibular migraine-A challenging clinical entity. Indian J Forensic Med Toxicol 2020;14:8835-41.
20. Swain SK. Vertigo following cochlear implantation: A review. Int J Res Med Sci 2022;10:572-7.
21. Swain S, Behera IC, Sahu MC. Prevalence of benign paroxysmal positional vertigo: Our experiences at a tertiary care hospital of India. Egypt J Ear Nose Throat Allied Sci 2018;19:87-92.
22. You P, Instrum R, Parnes L. Benign paroxysmal positional vertigo. Laryngoscope Investig Otolaryngol 2019;4:116-23.
23. Imai T, Takeda N, Ito M, Sekine K, Sato G, Midoh Y, *et al.* 3D analysis of benign positional nystagmus due to cupulolithiasis in posterior semicircular canal. Acta Otolaryngol 2009;129:1044-9.
24. Bhattacharyya N, Gubbels SP, Schwartz SR, Edlow JA, El-Kashlan H, Fife T, *et al.* Clinical practice guideline: Benign paroxysmal positional vertigo (Update) executive summary. Otolaryngol Head Neck Surg 2017;156:403-16.
25. Büttner U, Helmchen C, Brandt T. Diagnostic criteria for central versus peripheral positioning nystagmus and vertigo: A review. Acta Otolaryngol 1999;119:1-5.
26. Brandt T. Positional and positioning vertigo and nystagmus. J Neurol Sci 1990;95:3-28.
27. Kim HA, Yi HA, Lee H. Apogeotropic central positional nystagmus as a sole sign of nodular infarction. Neurol Sci 2012;33:1189-91.
28. Swain SK. Vertigo during pregnancy: A review. Int J Otorhinolaryngol Head Neck Surg 2022;8:695-701.
29. White J, Krakovitz P. Nystagmus in enlarged vestibular aqueduct: A case series. Audiol Res 2015;5:120.
30. Swain SK, Anand N, Mishra S. Vertigo among elderly people: Current opinion. J Med Soc 2019;33:1-5.
31. Baloh RW, Yue Q, Jacobson KM, Honrubia V. Persistent direction-changing positional nystagmus: Another variant of benign positional nystagmus? Neurology 1995;45:1297-301.
32. Beyea JA, Agrawal SK, Parnes LS. Transmastoid semicircular canal occlusion: A safe and highly effective treatment for benign paroxysmal positional vertigo and superior canal dehiscence. Laryngoscope 2012;122:1862-6.
33. Fu W, Han J, Chang N, Wei D, Bai Y, Wang Y, *et al.* Immediate efficacy of Gufoni maneuver for horizontal canal benign paroxysmal positional vertigo (HC-BPPV): A meta-analysis. Auris Nasus Larynx 2020;47:48-54.
34. Aron M, Bance M. Insights into horizontal canal benign paroxysmal positional vertigo from a human case report. Laryngoscope 2013;123:3197-200.
35. Bisdorff A, Von Brevern M, Lempert T, Newman-Toker DE. Classification of vestibular symptoms: Towards an international classification of vestibular disorders. J Vestib Res 2009;19:1-13.
36. Bhandari A, Bhandari R, Kingma H, Zuma E Maia F, Strupp M. Three-dimensional simulations of six treatment maneuvers for horizontal canal benign paroxysmal positional vertigo canalithiasis. Eur J Neurol 2021;28:4178-83.
37. Soto-Varela A, Santos-Perez S, Rossi-Izquierdo M, Sanchez-Sellero I. Are the three canals equally susceptible to benign paroxysmal positional vertigo? Audiol Neurootol 2013;18:327-34.
38. Bertholon P, Bronstein AM, Davies RA, Rudge P, Thilo KV. Positional down beating nystagmus in 50 patients: Cerebellar disorders and possible anterior semicircular canalithiasis. J Neurol Neurosurg Psychiatry 2002;72:366-72.
39. Bertholon P, Chelikh L, Tringali S, Timoshenko A, Martin C. Combined horizontal and posterior canal benign paroxysmal positional vertigo in three patients with head trauma. Ann Otol Rhinol Laryngol 2005;114:105-10.
40. Baloh RW, Yue Q, Jacobson KM, Honrubia V. Persistent direction-changing positional nystagmus: Another variant of benign paroxysmal positional vertigo? Neurology 1995;45:1297-301.