

# Benign paroxysmal positional vertigo

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## ABSTRACT

Benign paroxysmal positional vertigo (BPPV) is a common clinical disorder characterized by brief recurrent spells of vertigo often brought about by certain head position changes as may occur with looking up, turning over in bed, or straightening up after bending over. It is important to understand BPPV not only because it may avert expensive and often unnecessary testing, but also because treatment is rapid, easy, and effective in >90% of cases. The diagnosis of BPPV can be made based on the history and examination. Patients usually report episodes of spinning evoked by certain movements, such as lying back or getting out of bed, turning in bed, looking up, or straightening after bending over. At present, the generally accepted recurrence rate of BPPV after successful treatment is 40%–50% at 5 years of average follow-up. There does appear to be a subset of individuals prone to multiple recurrences.

**Key words:** Benign paroxysmal positional vertigo, Benign paroxysmal positional vertigo, vertigo

## Introduction

Benign paroxysmal positional vertigo (BPPV) is a common disease in the ENT. It is important to understand BPPV not only because it may avert expensive and often unnecessary testing, but also because treatment is rapid, easy, and effective in >90% of cases.

BPPV is a common clinical disorder characterized by brief recurrent spells of vertigo often brought about by certain head position changes as may occur with looking up, turning over in bed, or straightening up after bending over. BPPV is an important cause of vertigo with a prevalence of 11–64 per 100,000 and a lifetime prevalence in general practice of 2.4%.<sup>[1]</sup>

## Anatomic Background and Mechanistic Basis

The vestibular part of the membranous labyrinth consists of 3 semicircular canals: the anterior, posterior, and the horizontal canals. These canals detect turning

movements of the head. The labyrinth also consists of 2 otolith structures, the utricle and saccule, that detect linear acceleration, including detection of gravity. The macula of the utricle is the presumed source of the calcium particles that cause BPPV. It consists of calcium carbonate crystals (otoconia) embedded in a gelatinous matrix, into which the stereocilia of hair cells project. BPPV is caused when otoliths originate from the utricular macula and move within the lumen of one of the semicircular canals. When the calcium carbonate crystals move within the semicircular canal, they cause endolymph movement that stimulates the ampulla of the affected canal, thereby causing vertigo. The direction of the nystagmus is determined by ampullary nerve excitation in the affected canal by direct connections to the extraocular muscles. Each canal affected by canalithiasis has its own characteristic nystagmus.

The reason for this shedding of calcium crystals from the macula is not well understood. The calcium debris may break off following trauma or viral infections, but in many instances it seems to occur without identifiable illness or trauma. It may have to do with age-related changes in the protein and gelatinous matrix of the otolithic membrane.<sup>[2]</sup>

BPPV may affect the posterior, horizontal, or anterior semi-circular canal, and in some cases it may even involve more than one canal at a time. Due to its gravity-dependent position, the most commonly affected

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semicircular canal is the posterior canal. The anterior canal and polycanicular forms are the least common.<sup>[3]</sup>

## Diagnosis

The diagnosis of BPPV can be made based on the history and examination. Patients usually report episodes of spinning evoked by certain movements, such as lying back or getting out of bed, turning in bed, looking up, or straightening after bending over. The episodes of vertigo last 10–30 s and are not accompanied by any additional symptoms other than nausea in some patients.

The diagnosis of BPPV of the posterior canal is confirmed by observing paroxysmal positional nystagmus with the Dix–Hallpike maneuver. The Dix–Hallpike maneuver is performed by rapidly moving the head from an upright to head hanging position with one ear 45 degrees to the side. The Dix–Hallpike maneuver results in torsional upbeating nystagmus corresponding in duration to the patient’s subjective vertigo, and occurring only after Dix–Hallpike positioning on the affected side.<sup>[4]</sup>

The most reliable way to diagnose horizontal BPPV is by a supine head turn maneuver. The patient’s head is turned to one side, then is turned back to the supine face-up position. Then the head is turned to the other side. The nystagmus of horizontal canal BPPV, unlike that of posterior canal BPPV, is distinctly horizontal and changes direction with changes in the head position. The paroxysmal direction changing nystagmus may be either geotropic or apogeotropic.<sup>[5]</sup>

The anterior canal form of BPPV is associated with paroxysmal downbeating nystagmus, sometimes with a minor torsional component following Dix–Hallpike positioning.<sup>[6]</sup> Polycanicular BPPV is uncommon, but indicates that 2 or more canals are simultaneously affected at the same time. The most common circumstance is posterior canal BPPV combined with horizontal canal BPPV.

## Treatment

The treatment of posterior canal and anterior canal BPPV is the canalith repositioning maneuver, sometimes referred to as the “Epley maneuver.” The most commonly used treatment for horizontal canal BPPV is the roll maneuver.

Evidence is lacking to recommend postmaneuver restrictions in patients treated with canalith repositioning therapies, although there is generally no associated harm with these instructions.<sup>[7]</sup>

Occasionally, freely mobile otoconia moving within the lumen of one semicircular canal can be moved during the course of treatment; not back to the vestibule as intended, but to one of the adjacent canals, as the canals all directly communicate with one another. The most common canal switch is from the posterior to the horizontal and posterior to the anterior canals.<sup>[8]</sup>

## Complications

The most common complications include nausea, vomiting, fainting, and conversion to lateral canal BPPV during the course of treatment due to “canal switch.”<sup>[9]</sup>

## Prognosis

At present, the generally accepted recurrence rate of BPPV after successful treatment is 40%–50% at 5 years of average follow-up. There does appear to be a subset of individuals prone to multiple recurrences.<sup>[10]</sup>

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