

Meta-Analytic Study on the Necessity of Post-Maneuver Postural Restriction in Treating Benign Paroxysmal Positional Vertigo

For Partial Fulfillment of Master Degree in Otolaryngology

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Summary

Comprehension of the physiopathological mechanism of BPPV of the PSC has permitted the development of maneuvers capable of immediate symptomatic relief. After a liberatory maneuver, many authors advise patients to adhere to postural restrictions with the aim of avoiding a resettlement of the otolithic mass inside the affected canal.

These restrictions include avoidance of quick head movements, keeping the head erect, sleeping at a 45° angle, refraining from lying on the pathologic side, and even wearing a cervical collar to prevent certain head movements. In various clinics, patients are instructed to abide by these restrictions 24 to 48 hours or even up to a week following treatment.

This study was conducted to verify the role of postural restrictions after repositioning maneuvers in treating patients with benign paroxysmal positional vertigo and if these restrictions interfere in the clinical progression of patients with BPPV concerning symptomatology.

We analyzed 8 randomized controlled trials selected from 37 articles and abstracts according to the previously selected inclusion criteria and its analysis using Meta analytic software (Revman 5.1).

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List of Abbreviations

BPPV	: Benign Paroxysmal Positional Vertigo.
PC-BPPV	: Posterior canal Benign Paroxysmal Positional Vertigo.
HC-BPPV	: Horizontal canal Benign Paroxysmal Positional Vertigo.
AC-BPPV	: Anterior canal Benign Paroxysmal Positional Vertigo.
P-SCC	: Posterior semicircular canal.
H-SCC	: horizontal semicircular canal.
A-SCC	: Anterior semicircular canal.
CRM	: Canalith repositioning maneuver.
PRM	: Particle repositioning maneuver.
SLM	: Semont liberatory maneuver.
ENG	: Electronystagmography.
CT	: Computed Tomography.
MRI	: Magnetic Resonance Imaging.
Fig.	: Figure.
vs	: Versus.

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بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

قَالُوا سُبْحَانَكَ لَا عِلْمَ لَنَا إِلَّا مَا
عَلَّمْتَنَا
إِنَّكَ أَنْتَ الْعَلِيمُ الْحَكِيمُ
صدق الله العظيم

البقرة الآية ٣٢

Introduction

Benign Paroxysmal Positional Vertigo (BPPV) is defined as a vestibular syndrome of peripheral origin characterized by short and intensive episodes of vertigo, triggered by quick change of head position such as getting up from bed or rotating around the body and associated with nystagmus **(Simoceli et al., 2005)**.

BPPV is the most common cause of vertigo, resulting from migration of otoconia into the semicircular canals. The symptoms are caused by an abnormal interaction of the semicircular canal cupula and displaced otoconia from the utricle. The presence of the displaced otoconia causes the involved canal to become sensitive to changes in head position in the plane of the involved canal **(Roberts et al., 2005)**.

The diagnosis of BPPV is based on characteristic history of positionally provoked vertigo, physical examination, videonystagmography and Dix-Hallpike test. Posterior semicircular canal BPPV is the most common, but involvement of other canals is seen **(Macias et al., 2000)**.

Several treatment methods involving repositioning maneuvers that return the otoconia to the utricle have been described. Following treatment, most patients are provided with a variety of activity restrictions **(Roberts et al., 2005)**.

The use of different postmaneuver restrictions has been investigated to improve the success of repositioning maneuvers. Several studies have examined the use of restrictions in comparison with unrestricted controls .The results of these studies have varied and there is still a question of whether specific restriction hold a significant improvement over another, or over no restriction at all (**Devaiah and Andreoli, 2010**).

Pathogenesis of BPPV

1) Aetiology of BPPV:

Positional vertigo is defined as a spinning sensation produced by changes in head position relative to gravity. Benign paroxysmal positional vertigo is defined as a disorder of the inner ear characterized by repeated episodes of positional vertigo. Traditionally, the terms benign and paroxysmal have been used to characterize this particular form of positional vertigo. In this context, the descriptor benign historically implies that BPPV was a form of positional vertigo not due to any serious CNS disorder and that the overall prognosis for recovery was favorable. The term paroxysmal in this context describes the rapid and sudden onset of the vertigo associated with an episode of BPPV. BPPV has also been termed benign positional vertigo, paroxysmal positional vertigo, positional vertigo, benign paroxysmal nystagmus, and paroxysmal positional nystagmus (**Bhattacharyya et al., 2008**).

As the name implies, BPPV is most often a benign condition, however, in certain situations it may become dangerous. For example, a painter looking up from the top of a ladder may suddenly become vertiginous and lose his or her balance, risking a bad fall. The same would hold true for underwater divers who might get very disoriented from acute vertigo. Heavy machinery operators should use great caution especially if their job involves significant head movement (**Parnes et al., 2003**).

The etiology of BPPV is considered idiopathic although there may be increased susceptibility among persons with head and neck trauma, infections, inner ear disorders, and advanced age. In particular, BPPV is the most common cause of dizziness among older adults. This disorder can be seen in any age group, but incidence and prevalence increases with increasing age (**McGinnis et al., 2009**).

In most cases, BPPV is found in isolation and termed "primary" or "idiopathic" BPPV. This type accounts for about 50%–70% of cases. The most common cause of "secondary" BPPV is head trauma, representing 7%–17% of all BPPV cases. A blow to the head may cause the release of numerous otoconia into the endolymph, which probably explains why many of these patients suffer from bilateral BPPV. Viral neurolabyrinthitis or so-called "vestibular neuronitis" has been implicated in up to 15% of BPPV cases. Ménière's disease has also been shown to be strongly associated with BPPV. There is large variation in the literature regarding what proportion of patients with BPPV also have the diagnosis of Ménière's disease. Recently, migraines have been found to be closely associated with BPPV.

Ishiyama et al (2000) found an increased incidence of migraine in patients with BPPV and higher recurrence rates of BPPV after successful positioning in patients with migraine. It has been suggested that spasm of the inner ear arteries may be a possible causative mechanism, because vasospasm is well documented in migraines (**Parnes et al., 2003**).

1-2) Pathophysiology of BPPV:

The currently accepted mechanisms of BPPV are cupulolithiasis and canalolithiasis but the exact pathophysiology of BPPV is still not completely understood (Angeli et al., 2003).

1-2-1) Mechanisms underling BPPV:

1-2-1-a) Cupulolithiasis and canalolithiasis theories:

The first attempt to determine the pathogenesis of BPPV was made by Schuknecht and Ruby. They postulated the theory of cupulolithiasis based on their finding of basophilic deposits over the cupula of the posterior semicircular canal figure (1). (Simhadri et al., 2003).

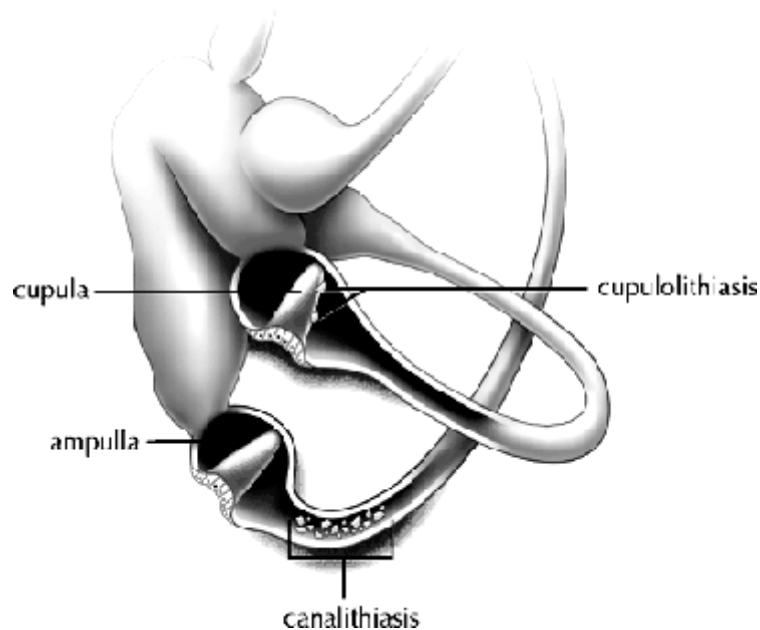


Fig. (1): Canalolithiasis of the posterior canal and cupulolithiasis of the lateral canal in the left inner ear (Parnes et al., 2003).

Benign paroxysmal positional vertigo is caused by abnormal mechanical stimulation of 1 or more of the 3 semicircular canals within the inner ear. The fluid-filled canals normally act to detect rotation of the head through the deflection of sensory hair cells embedded within a gelatinous membrane, the cupula. The weighted sensory membrane of the maculae normally acts to detect gravitational forces on the head. In BPPV, calcite particles (otoconia), which normally weight this membrane, become dislodged and sediment in the canals, changing the dynamics of the canals. There are 2