BPPV A Review of Current Published Literature

Balasubramanian Thiagarajan
BPPV
A review of current published literature

February 17, 2012 · Otology

Authors
Balasubramanian Thiagarajan

Abstract
Benign paroxysmal positional vertigo is a commonly diagnosed condition. Studies reveal that this is the commonest cause of giddiness. This condition is commonly caused by dysfunction involving posterior semicircular canal. Lateral and superior canals have also been implicated rarely. It has been estimated that nearly 15-20% of all patients presenting with giddiness suffer from BPPV. This article attempts to review the current available literature in this topic.

BPPV (Benign Paroxysmal Positional Vertigo)

Introduction:
Benign paroxysmal positional vertigo is a commonly diagnosed condition. Studies reveal that this is the commonest cause of giddiness. This condition is commonly caused by dysfunction involving posterior semicircular canal. Lateral and superior canals have also been implicated rarely. It has been estimated that nearly 15-20% of all patients presenting with giddiness suffer from BPPV. With the advent of positional manoeuvres it should be stressed that BPPV is the most successfully treated condition of inner ear. This condition is characterised by brief spells of vertigo (mostly lasting for just a few seconds) and are experience by specific movements / positions of head.

History:
The first description of position induced vetigo is credited to Adler. It was later followed by Barany in 1921. Eventhough he failed to correlate the onset of nystagmus with specific head positions, he was the first to describe the components of this disorder (Nystagmus, fatiguability of nystagmus and vertigo). The earliest reference to BPPV was made by Shakespeare in his play "Romeo & Juliet". Charles Hallpike at Queen square Hospital came out with a thorough study of more than 100 patients who had vertigo when they assumed certain provoking positions. He also coined the term "Benign positional nystagmus" to describe this condition. Marie Jean Flourens by his classic experiments on pigeon ears demonstrated that semicircular canals were organs of balance and cochlea was the organ of hearing. This was an epoch making discovery those days. According to Politzer this was the most important turning point in the history of inner ear physiology.

Julius Ewalds Professor of Physiology at University of Strassburg conducted experiments by cannulating semicircular canals of pigeons. He applied positive and negative pressure to these cannula and observed the direction of nystagmus. This formed the basis of his famous Laws.

Ewalds law 1: The direction of nystagmus is in the plane of canal being stimulated.

Ewalds law 2: In lateral semicircular canal the ampullopetal flow of endolymph towards the vestibule causes the maximum nystagmus.
Ewald’s law 3: In posterior and superior semicircular canals ampullofugal flow of endolymph away from the vestibule causes the maximum nystagmus.

Invention of Electron microscope some 30 years later opened up new avenues for studying ultrastructural architecture of inner ear. In 1954 Wersall demonstrated that each vestibular sensory cell had one kinocilium and many stereocilia. In the lateral canal crista the kinocilium was found on the vestibule side of stereocilia; where as in the vertical canals the kinocilium was found on the canal side of stereocilia. This discovery went a long way in validating Ewald’s laws.

The exact relationship between the canal receptors and extraocular muscles became evident only in 1960’s after experiments with cat’s labyrinth. Each receptor is connected to one ipsilateral and one contralateral muscle.

Theories accounting for BPPV:

Schuknecht Theory:

Schuknecht proposed that BPPV is caused by loose otoconia from the utricle, which in certain positions displaces the cupula of posterior semicircular canal. He went on to modify his theory later suggesting that deposition of otoconia in the cupula of posterior canal could be the cause. He also coined the term cupulolithiasis. In a nutshell this theory proposes that calcium deposits become embedded on the cupula making the posterior semicircular canal sensitive to gravity.

Hall and Ruby theory:

This theory proposed jointly by Hall & Ruby suggests that deflection of cupula of posterior canal could be caused by debris in the posterior canal. This theory is also known as canal lithiasis theory. According to this theory the calcium deposits does not become adherent to the cupula of posterior canal but floats freely within the canal. Provoking head movements like looking up, down or rolling over to the affected side would cause giddiness and nystagmus.

Types of BPPV as described by Hall and Ruby:

Hall and Ruby while explaining their theory went on to describe two types of BPPV.

Type I BPPV: Nystagmus in this condition is fatigable and the calcium deposits are freely mobile within the cupula of posterior canal.

Type II BPPV: Nystagmus in this condition is nonfatiguing because the calcium deposits are fixed to the cupula of posterior canal.

Features of BPPV (As described by Hall & Ruby):

1. Canalithiasis mechanism – This explains the latency of the nystagmus as a result of the time needed for motion of the material within the posterior canal to be initiated by the gravity.

2. Duration of the nystagmus – is correlated with the length of time required for the dense material to reach the lowest part of the posterior canal.

3. The vertical (upbeating) and torsional (superior poles of the eye beating towards the lowermost ear). The nystagmus is more vertical when the patient looks away from the lowermost ear, and more torsional when looking towards the lowermost ear.

4. The reversal of nystagmus when the patient returns to the sitting position is due to retrograde movement of material in the lumen of the posterior canal back towards the ampula, resulting in
ampulo petal deflection of the cupula.

5. The fatiguability of the nystagmus evoked by repeated Dix Hallpike positional testing is explained by dispersion of material within the canal.

Role of History taking in the diagnosis of BPPV

Role of accurate history taking is very important in diagnosing BPPV. The following are the pointers towards the diagnosis:

Symptoms associated with certain head positions
Vertigo is episodic and rotatory in nature
History of previous episodes of neurolabyrinthitis i.e. Severe vertigo (rotatory).
History of head injury prior to history of vertigo
Symptoms are severe early in the day and becomes better as the day passes by
Absence of symptoms when provoking head position is assumed

Incidence:
BPPV is the most common cause of peripheral vertigo and nystagmus accounting for nearly 20 – 40% of all patients with vertigo. Females outnumber men by 2:1.
Patient may describe the sensation as if they are walking on pillows
Clinical examination reveals:
Vertical rotatory positional nystagmus seen when the patient assumes provacatory position
There is a definite latent period between the time the patient assumes the position and appearance of nystagmus
Nystagmus is of very short duration lasting for just 3 – 30 seconds
Nystagmus disappears on repetition of the provoking position

Probable causes of BPPV:
Infection
Trauma
Degeneration of peripheral end organ
Preceding neurolabyrinthitis (commonest)

According to Brandt BPPV is caused by positioning rather than positional. (It is not induced due to a particular position due to gravity but by rapid changes in head position). Hence the intensity of the nystagmus is dependent on the velocity of the maneuver, and the attacks can be avoided completely if the offending position is assumed slowly.

Dix Hallpike maneuver is virtually diagnostic test for BPPV.

Dix Hallpike Maneuver:
The patient is positioned on the examination table in such a way that when he/she is placed supine,
the head extends over the edge. The patient is lowered with the head supported and turned 45 degrees to one or the other side. The eyes are carefully observed; if no abnormal eye movements are seen, the patient is returned to the upright position.

This same maneuver is repeated with the head in the opposite direction and the patient's symptoms are noted.

The pattern of response consists of the following:

1. Nystagmus is a combination of vertical upbeating & rotatory (torsional) beating towards the downward eye. Pure vertical nystagmus is not seen in BPPV.

2. There is often a latency of onset of nystagmus

3. Duration is less than a minute

4. Vertiginous symptoms are invariably seen

5. Nystagmus disappears with repeated testing (fatiguability)

6. Symptoms often recur with the nystagmus in opposite direction on return of the head to upright position.

Note:

Canalithiasis involving the posterior canal is the commonest cause of BPPV. Posterior canal BPPV may rarely be bilateral, but while testing the head must be positioned in the plane of the posterior canal during testing of unaffected ear otherwise the debris in the affected side can rest against the cupula and stimulate an excitatory nystagmus from the unaffected ear.

Lateral canal BPPV:

Lateral canal has also been identified as the offender in 17 % of cases with BPPV. Lateral canal BPPV can be detected by a variation of Dix Hallpike maneuver. The patient's head is first brought to the supine position resting on the examination table (not hyperextended). The head is then turned rapidly to the right so that the patient's right ear rests on the table. The eye movements of the patient are monitored with Frenzel's glasses for 30 seconds. The patient's head is then turned to the supine position (eyes looking upward) and is then rapidly turned to the left so that the left ear rests on the table. Eye movements are monitored. The nystagmus with lateral canal BPPV is horizontal and may beat toward (geotropic) or away (ageotropic) from the downward ear. It begins with a short latency, increases in magnitude progressively, and is less susceptible to fatigue with repetitive testing than the vertical torsional nystagmus of posterior canal BPPV.

Cupulolithiasis, either alone or in combination with canalithiasis is more likely to be involved in the etiology of lateral canal BPPV than in the case of posterior canal BPPV. If the nystagmus is geotropic, the particles are likely to be in the long arm of the lateral canal relatively far from the ampulla, if the nystagmus is ageotropic, the particles could be in the long arm relatively close to the ampulla or on the opposite side of the cupula either floating within the endolymph or embedded in the cupula.

Superior canal BPPV is rather rare.

Standard electrooculography or 2 dimensional video nystagmography devices donot record the typical eye movements associated with BPPV. Thus clinical examination of the patient is of paramount importance.
Differences between central and peripheral nystagmus

<table>
<thead>
<tr>
<th>Symptom /sign</th>
<th>Peripheral</th>
<th>Central</th>
</tr>
</thead>
<tbody>
<tr>
<td>Latency</td>
<td>1-40 secs</td>
<td>No latency</td>
</tr>
<tr>
<td>Duration</td>
<td>Persists for just less than a minute</td>
<td>Symptoms may persist for a long time</td>
</tr>
<tr>
<td>Habituation</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Nystagmus direction</td>
<td>Fixed, torsional, upwards</td>
<td>Direction changing</td>
</tr>
<tr>
<td>Intensity</td>
<td>Vertigo intense, nystagmus marked, vomiting ++</td>
<td>Mild vertigo, less intense nystagmus, nausea is rare</td>
</tr>
<tr>
<td>Reproducibility</td>
<td>Inconsistent</td>
<td>Consistent</td>
</tr>
</tbody>
</table>

Management:

Repositioning maneuvers have been found to be very useful in managing this problem.

Currently BPPV is managed by repositioning maneuvers that, in cases of canalithiasis use gravity to move canalith debris out of the affected semicircular canal and into the vestibule. For posterior canal BPPV the maneuver developed by Epley is effective.

Epley Maneuver:

This is performed by placing the head of the patient in Dix Hallpike position that evoke vertigo.

The posterior canal on the affected side is in the earth vertical plane when the head is in this position. After the cessation of initial nystagmus, the head is rolled through 180 degrees, (this is done in two 90 degree increments, stopping in each position until the nystagmus resolves) to the position in which the offending ear is up. The patient is then brought to the upright sitting position. This procedure is likely to be successful when nystagmus of the same direction continues to be elicited in each of the new position (as the debris continues to move away from the cupula). This maneuver is repeated until no nystagmus is elicited. This is successful in 90% of cases. Posterior canal BPPV can be converted to lateral canal BPPV during Epley maneuver. The lateral canal BPPV resolves in several days. Drugs are usually not prescribed, but low dose meclizine or calmpose can be given 1 hour before the procedure if the patient is anxious or prone to vomiting.

Sermont maneuvers – is also effective in posterior canal BPPV, but is most difficult to perform and it has no significant advantages over the Epley maneuver. This is being described here for the sake of completion. In this maneuver the patient is moved quickly in to the position that provokes the vertigo and remains in that position for 4 minutes. The patient is then turned rapidly to the opposite side ear down, and remain in the second position for 4 minutes before slowly getting up.

In both these maneuvers gravity is the stimulus that move the particles within the canal, so there is no need to turn the head on the body, enbloc movement of the head and body as much as possible is the plan.

Exercise therapy:

This therapy can be selfadministered. It is also fairly successful.

The patient is first asked to be seated properly in a couch before begining the exercise. The patient begins in the seated position and then leans rapidly to the side placing the head on the bed or table. The patient remains there until the vertigo subsides and then returns to the seated upright position.
remaining there until all symptoms subside. The maneuver is repeated toward the opposite side completing one full repetition. Ten to twenty repetitions should be performed three times a day. Patients should be cautioned not to do this procedure forcibly as this could cause neck injuries.

If the patient has intense symptoms of giddiness / vomiting following exercise therapy then they need to be admitted and administered labyrinthine sedative and intravenous fluids.

Semont’s Liberatory maneuver:

This maneuver has been found to be helpful in managing BPPV.

This maneuver is begun with the patient seated on a couch. The patient is swung down quickly with the offending ear down and head slightly inclined. If nystagmus appears, the patient is kept in that position until nystagmus disappears. Wait for 3 minutes.

If nystagmus doesn't appear the head is turned so that face is up 45 degrees from the horizontal. Nystagmus should appear. The patient is kept in this position for 2-3 minutes after nystagmus stops.

The head and neck of the patient is held with two hands and is swung quickly to the opposite side. The speed of the head should be zero the moment it touches the table.

In patients with classic BPPV, a rotatory nystagmus will develop with the fast phase of nystagmus beating towards the offending ear (the topmost ear). If the fast component of nystagmus beats towards the bottom ear then in all probability the diagnosis of BPPV is wrong.

If no nystagmus develops still, the head is moved slowly to 90 degrees facing up. The head is then turned to the opposite direction to 135 degrees so that it is 45 degrees facing down below the horizontal (with sick ear up). Nystagmus should occur.

If nystagmus is produced the patient is held in this position for full 5 minutes and then the patient is brought back to the sitting position rather gradually and very slowly.

After this exercise patient is instructed to keep their head absolutely vertical in space during the next 48 hours. If this maneuver is not successful then it can be repeated a week later.

Horizontal canal BPPV:

This is commonly identified when the patient is undergoing Dix Hallpike test. Sometimes it can become apparent after a successful canal repositioning maneuver for posterior canal BPPV. Vertigo happens to be more intense than that of posterior canal BPPV.

Head roll test:

This test is virtually diagnostic of Lateral canal BPPV. While performing this test the patient is placed in midline and in horizontal plane. The head is gently turned towards one side and then to the other. There is clear brisk nystagmus beating towards the undermost ear (maximum geotropic). A weaker nystagmus occurs when the opposite ear is down (apo geotropic). When the offending ear is identified then a different repositioning technique known as “Barbecue Procedure” is performed. When performing this Barbecue positioning procedure the examiner is seated at the head end of the couch, and the patient is asked to rotate 360 degrees in four stages, a minute apart. During the third stage of this maneuver the patient would be resting on the elbows, with the neck flexed. In this position the horizontal canal is vertical, and the particle will exit the canal during this stage. The head roll test should be repeated, and if negative the treatment is considered successful. If unsuccessful rotation is performed in the opposite direction.
Superior canal BPPV:

While performing Dix Hallpike test, if the nystagmus is downbeat then superior canal BPPV should be considered. This should be differentiated from nystagmus due to central causes. If nystagmus is due to central causes then it is immediate in onset, (no latency) and the patient does not experience vertigo. In superior canal BPPV the nystagmus produced will have a definite latency, and would be associated with vertigo. The particle may be present in either ear, but commonly it is seen in upper most ear.

Li maneuver^{17} is performed to treat this condition. In this maneuver the patient is moved rapidly from a supine (midline) head hanging position to a face down position at the opposite end of the couch.

After these repositioning maneuvers patients are instructed to avoid bending over and are told to sleep with the head elevated atleast 45 degrees for the next couple of days.

Vibrator therapy:

Some physicians use a small hand held vibrator over the mastoid to agitate the particles and make it move. This mastoid vibrator is to be avoided in patients with retinal detachment or in patients who may be susceptible to retinal detachment due to high myopia.

Brandt Doroff exercises – can be performed by the patient in the home environment. These exercises are performed in 3 sets / day for 2 weeks.

It is started like this:

Position 1 – The patient must be seated upright on the bed. Then he moves to side lying position (position 2) the head is kept angled upwards about half way. The patient should stay in this position atleast for 30 seconds or till the giddiness subsides. If the giddiness does not subside thee patient must revert back to position 1. After 30 seconds the procedure is repeated on the opposite side. Most of the patients get relief within a period of 10 seconds.

Surgical management:

Singular neurectomy – is a very demanding procedure. The posterior canal is supplied by singular branch of vestibular nerve. This nerve when preferentially sectioned alleviates the patient’s symptom due to posterior canal BPPV.

Posterior canal plugging procedure – is a easier procedure. Through a mastoidectomy incision the labyrinth is exposed. The posterior canal is drilled exposing the membranous portion of the canal. The canal is sealed and packed off thereby preventing the debris from floating. After the procedure the patient may feel slightly giddy. The patient needs to be kept in the hospital till giddiness subsides.

Recent trends:

Epley’s canalith theory explains majority of the clinical features of BPPV. It also substantiates the logic and efficacy of repositioning maneuvers as a treatment modality.

Majority of BPPV can get better even without management. This leads to the inference that otoconia can make a spontaneous exit also. Experiments involving frogs^{18} have demonstrated that otoconia in them rapidly dissolve in endolymph when the calcium levels are within physiologic limits, It dissolves rather slowly if the calcium levels are raised.

Studies have also demonstrated that 75% of women aged between 50 – 85 with BPPV show evidence
of osteopenia / osteoporosis. 

Role of VEMP’s in diagnosis of BPPV:

Measurement of vestibular evoked myogenic potential is good for measuring saccule and otolith function. In patients with vestibular neuronitis absense of cervical vemps is a good predictive indicator for non development of BPPV. It occurred only if cervical VEMP is present. The recently identified ocular vemp also measures utricle function and its presence could be seen as a recovery indicator in patients with BPPV.

---

Anatomy of inner ear

---

Epley's repositioning maneuver

---

Barbecue maneuver

---

Li maneuver

---
Appendix 1

[gallery link="file" columns="4"]

References

3. 3. http://www.drtbalu.co.in/bppv.html
7. 7. J. R. Ewald, Physiologische Untersuchungen Ueber das Endorgan de Nervus Octavus, Bergmann JF Publishers, Wiesbaden, Germany, 1892.

entscholar.wordpress.com/article/bppv/


