Anterior canal Benign Paroxysmal Positional Vertigo (BPPV)- Does it exist?  
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Anterior canal BPPV is a controversial subject and many wonder whether the canalithiasis variant exists at all. In the first of a series of discussions, an email was sent to BIG members to gauge their opinions. Here are responses, followed by some additional information to help you make up your mind. This information is not meant to be exhaustive but rather to spark thought and debate on the subject. What do you think?

Comments 1
- Do you believe it exists (some don’t!)
- I’m a believer!
- If so how does it present
  - Perhaps like a general BPPV picture but see below – it can be there ‘on and off’ in cases of tricky to treat BPPV where the nystagmus direction seems to vary depending on the day of test and recent treatments. I guess they are a very small minority of our general BPPV caseload, but we see them more often. One guy we saw had bilateral mixed canal post head injury – he would treat beautifully but never for more than a few weeks at a time – every time we saw him was it in a different place / places. He has given up coming in now, I think in disgust!
- How often do you see it in clinic
  - only occasionally, sometimes as mixed picture with bilateral / mixed canal / conversion issues (i.e. nystagmus all over the place)
- How do you treat (lots of different manoeuvres out there in addition to Epley and reverse Semont)
  - We would try a normal Epley on the non-suspect side first, (*ed see extra information 4) never tried reverse Semont!
- Have you ever seen common crus BPPV (which is meant to cause down beat nystagmus)
  - we have def seen occasional pure down-beat in context of fairly definite BPPV (e.g. a recurrence of previously treated posterior). Would be concerned if it was persistent following treatment though, I’ve also seen this with MS presenting very much like BPPV.
- Anything else you think is relevant and wish to share on the topic
  - I think I’ve also seen ‘canalith jam’ (*ed see extra information 1) once or twice where things go very wrong (dizzy ++++) on the way up from Epley - apparently the Rx is to reverse the Epley to shift the debris, which I have tried and it worked. Can’t remember nystagmus direction as I was panicking!

Comments 2
- Do you believe it exists (some don’t!)
- yes
- If so how does it present
  - Cupulolithiasis – relatively rarely, as sustained nystagmus > one minute (‘less rare’ for HC than PC). May need to exclude central as differential dx. Anterior – positive
Hallpike with torsional ageotrophic nystagmus e.g. (L) DHP, mixed torsion nystagmus away from under most ear = (R) AC

- How often do you see it in clinic
  - rarely e.g. 1% of positive Hallpikes

- How do you treat (lots of different manoeuvres out there in addition to Epley and reverse Semont)
  - Epley to start with - for the correct ear! Flow of otoconia direction?

- Have you ever seen common crus BPPV (which is meant to cause down beat nystagmus)
  - Seen vertical nystagmus but how know if common crus vs central?

Comments 3
- Do you believe it exists (some don’t!)
  - I definitely believe that anterior canal BPPV exits, I have had two cases of patients presenting in clinic and they responded positively to treatment. I used deep head hanging protocol on both with positive results

- If so how does it present
  - I’ve seen quite a lot of patients with what would normally be interpreted as anterior canal BPPV since we set up a specialist BPPV clinic and now see a lot more BPPV pts in Audiology (previously ENT treated them unless intractable, non-routine, or sent for vestibular assess for something else going on) - that is patients with a combined down-beating nystagmus and torsional nystagmus.

- How often do you see it in clinic
  - Quite a lot appear to have a false positive Dix-Hallpike on the non-affected ear i.e. torsion to other ear, as well as a positive DH on the affected ear i.e. torsion to test side ear. Often the stronger response is on the false positive side which can be confusing to patient and clinician - but this is expected.
  - A couple have only had positional symptoms in the vertical plane i.e.
    - looking up and down - which is in the literature - so patients with a BPPV like history but without the dizziness turning over in bed might give you a hint of AC BPPV.
  - Have also had patients with BPPV like symptoms and pure down-beat on occasion - would have to look back but think some of these have had torsion on other occasions, been treated effectively and not turned out to be central. Getting them to look in different directions sometimes elicits the torsion. But always have in back of mind that down beating nystagmus might need further investigation especially if treatment manoeuvres do not help.

- How do you treat (lots of different manoeuvres out there in addition to Epley and reverse Semont)
  - Have appeared to have had success with Reverse Semonts for what looked like AC cupulolithiasis i.e. nystagmus >30 secs, but then I think the non-believers only question whether AC canalithiasis exists and acknowledge AC cupulolithiasis can exist.
  - Would initially try Epley for what looks like AC canalithiasis on affected side. When I first started out it used to be advised to do the mEpley from the non-affected side but Neil Shepard on the Southampton Balance course convinced me that starting
from affected side should move the crystals in the correct direction. Think there has been apparent success but would have to look back.

- **Have you ever seen common crus BPPV (which is meant to cause down beat nystagmus)**
- But have had at least 1 patient which could support the common crus (not AC BPPV canalolithiasis) believers in that the torsional component may not have been pointing to the affected ear i.e. patient with down-beat and torsion to right ear but did not get better with initial appropriate treatment for right AC BPPV. Tried mEpley on left ear as previous PC BPPV this side and everything else pointing to this ear operations, other ear symptoms. Think they then got better. ??

- I've tried deep head hanging in one patient when I wasn't sure which ear was affected but don't think it was effective in this case, maybe because they couldn't get their head back far enough - needs to be very deep apparently to have any chance of working. Can't remember the outcome in the end for this patient. Wouldn't put me off having another go, but do think there will only be very limited numbers of patients with the neck flexibility to manage it.

**Comments 4**
- **Do you believe it exists (some don’t!)
- Yes, although rare. This is because I have seen nystagmus conforming to what one should see in anterior canal BPPV with a history suggestive of it. Treatment for it has worked, and previously symptomatic patients with nystagmus become symptom-free

- **If so how does it present**
  Torsional nystagmus to the side of interest with a down beat component. Sometimes the torsional component is minimal. This can sometimes be bought out by a rose (straight head hanging) test or by deviation of gaze to enhance the torsional component

- **How often do you see it in clinic**
  Rarely, but I do see it

- **How do you treat (lots of different manoeuvres out there in addition to Epley and reverse Semont)**
  Epley on suspect side, reverse Semont with head turned to towards suspect side or deep head hanging when side unclear

- **Have you ever seen common crus BPPV (which is meant to cause down beat nystagmus)**
  I have seen down beat nystagmus in isolation and it has responded to deep head hanging – whether this is common crus BPPV or anterior canal im unclear about

**Siena international conference on balance in 2013.** Doctors Soumit Dasgupta, Marco Mandala, Timothy Hain, David Zee and Stefano Ramat devoted special time to common crus BPPV.
Dr Dasgupta says: ‘The consensus which is yet to be published is that theoretically ASCC canalolithiasis may exist but in practical situation it will be very rare almost non existent
due to the effects of gravity. The theory of a common crus BPPV was put forward to explain a downbeat torsional nystagmus on Dix Hallpike which has a latent period and does not last for more than 30 seconds'.

Hain (www.dizziness-and-balance.com) says more about this about this:

‘Considering causes within the ear, a mixed-DBN/contratorsional nystagmus might also be caused by debris close to the common crus of the PC. In other words, following along the same logic as geotrophic-ageotrophic LC BPPV, especially after an Epley or Dix Hallpike maneuver, debris in the PC might move towards the ampulla/cupula rather than away. Here, the nystagmus should be strongest with the bad ear down, and the torsion should be oppositely directed. This nystagmus pattern is very uncommon (i.e. contra torsion). Although to our knowledge, this idea was first posted on this web page, this mechanism was also discussed at a 2013 conference in Siena Italy by two different research groups. Califano et al (2014) discussed this mechanism in detail in a recent paper and used the name “apogeotropic posterior canal BPPV”. Califano used the term "apogeotropic" rather than "contra" for the torsional component, but they are the same entity. The term apogeotropic posterior canal was first used by Vannuchi and colleagues (2011, 2012)’.

Dr Dasgupta continues: ‘An ASCC cupulolithiasis, on the other hand is a practical possibility as the debris by virtue of their adherence to the cupula are not moved by gravity. Same nystagmus is noted but it might not have a latent period as may last for more than 60 seconds with or without autonomic symptoms. The problem with Dix Hallpike as Timothy says does not tell you the side of the ASCC BPPV as there are other influencing factors.

Treatment
For ASCC cupulolithiasis where side is unclear: Dr Dasgupta and Dr Hain suggest a brisk deep head hanging exercise. Essentially, this addresses both sides and is not side dependent, one of the advantages. The head is extended beyond the couch to about 40 degrees asking the patient to lie down rapidly and then holding it for 30 secs or until nystagmus or dizziness disappear; then briskly flexing it so that the chin almost touches the chest and holding there as usual for 30 seconds or until dizziness disappears and finally briskly sitting up the patient with the head still flexed in the same position so that he looks down.

A common crus BPPV can be treated by a brisk or modified Epley as Epley at one point does entail sending the particles to the common crus’.

Further thoughts from Tim Hain’s thoughts on anterior canal BPPV

1. Canalith jam


‘Epley himself first described canalith jam (Epley, 1995). The situation here is that the episodic/positional triggered dizziness of ordinary BPPV converts into a constant spontaneous nystagmus. This is attributed to debris "jamming" the canal (Epley 1995; Von Brevern, 2001). So far, there has been no proof that this mechanism is correct. Still, there are few reasonable alternatives to this picture.
In our practice (where we treat several BPPV patients on a daily basis, for 20 years), we have only seen this a few times -- perhaps 3 in 20 years.

On the other hand, we may be just missing it, as it is fairly common to attribute downbeating nystagmus after the Epley to mysterious and unknown events. Perhaps this is canal jam of the posterior canal.

This condition is very rare and little is known about treatment'.

Richard Gans also talks about canalith jam

(http://www.hearingreview.com/2000/09/overview-of bppv-treatment-methodologies/)

Canalith Jam: ‘A rare, but often frightening, occurrence is a canalith jam. This can best be described as an inability of the otolith debris to clear the common crus as the debris falls downward from the posterior canal into the utricle, the final stage of the Canalith Repositioning Maneuver. In the instance of a jam, the patient will become symptomatic and the symptoms will not abate. The sensation of falling or rapidly tumbling can be extremely severe, and the only way to clear this is to reverse the Canalith Repositioning protocol in the order in which it was administered –

In performing well over 500 Repositioning and Liberatory Maneuvers, the author has only had two patients experience a canalith jam. However, it is imperative for the clinician to quickly identify what is occurring and to be able to reverse the process so that the patient can recover’

A case history is given by Chang et al (see references at the end)

2. How anterior canal BPPV could cause a bilaterally positive Dix Hallpike (DH)
unilaterally positive DH, or theoretically –ve DH.. and even self clear

Contra lateral Dix Hallpike

- Angular acceleration and gravity both in plane of anterior canal.
- Particles likely to be displaced.
- Particles move along centreline of canal – causes a pressure difference across the cupula → deflection of cupula → big response.
Ipsilateral DH

- Angular acceleration is perpendicular to plane of anterior canal → no force on cupula.
- Ampullary segment of anterior canal points down at 37° from vertical, so particles can be displaced but only under force of gravity so less violently.
- Also – angular acceleration shifts particles off the centreline of anterior canal – movement off-centre → smaller drag effect → smaller deflection of cupula → smaller response.

Figures from Korres et al 2008 with annotations in read

Depending on the side you test first

- Unilateral +ve DH (either ipsilateral or contralateral with direction of torsion denoting affected side
- Bilaterally +ve DH but with direction of torsion denoting a unilateral anterior canal BPPV ie the same in both right and left Dix Hallpike
- Bilaterally –ve despite presence of anterior canal BPPV. Theory dictates that If you test ipsilateral side (with smaller response may not get nystagmus
- Habituation then occurs when testing second side so that even though contralateral side give larger response this may have habituated ie negative bilaterally even thought anterior canal BPPV

Korres argues that in some cases the testing for anterior canal bppv can also treat at the same time (ie the BPPV self clears). This is because as the patient sits up the otoconia can move upwards from the cupula and into the common crus

Figure from Korres et al 2008 with annotations in read
3. **Anterior canal BPPV presenting with down beat nystagmus only**

Because of the sagittal orientation of the anterior canals, often a torsional component is difficult to identify and nystagmus can be vertical (down beat) in nature only.

A torsional component can sometimes be identified by use of Ewalds first law as follows:

- Torsional component of nystagmus is enhanced in anterior canal BPPV when the direction of gaze is perpendicular to the canal - ie gaze away from affected side.
- Vertical component enhanced by deviating gaze parallel to affected canal ie towards affected side.
- Affected side = side to which torsional nystagmus beats.

A number of studies have looked at the causes of positional down beat nystagmus. Bertholon looked at 50 cases, 12 of whom who had positional down beat nystagmus in the absence of central signs (3 had an additional torsional component) and were subsequently diagnosed with anterior canal canalithiasis.

Lopez et al also presents a series of patients with positional down beat nystagmus for whom the diagnosis with anterior canal BPPV.

Whilst these cases presented in the absence of other central signs and with features consistent with BPPV such as habituation on repeat testing, some central positional vertigo can present very much like a peripherally generated vertigo. Johkura et al in 2007 presented 8 such cases which turned out to be secondary to cerebellar haemorrhage. Six of these cases presented with bilateral apogeotropic nystagmus (similar to horizontal canal cupulolithiasis, one with horizontal geotropic nystagmus bilaterally (similar to horizontal canal canalithiais), and one with nystagmus similar to that seen in posterior canal canalithiasis.

It is important that if a patient is presenting with what appears to be straightforward BPPV but this does not respond to treatment, that this is kept in mind and appropriate onward referral made.

4. **Treatment of anterior canal BPPV**

Again there is lots of controversy..

A good overview is given in Tim Haines website [www.dizziness- and-balance.com](http://www.dizziness- and-balance.com)

Unfortunately as far as the author is aware, there are no Randomized control trials on any of these manoeuvres.

1. Many feel that the anatomy dictates that an **Ipsilateral Epley** is the treatment of choice- otherwise otoconia do not move all the way round to the common crus (personal communication Shepard)
2. **The Reverse epley** has been suggested by Honrubia et al 1999 but results are from 4 patients only

   **For right anterior canal BPPV**
   - start seated head front (last position in Epley)
   - turn head right and lie onto right side
   - roll onto back with head right
   - head turn to left, then sit up

3. **A reverse semont** with head turned to the side of BPPV is also used by many
There are a set of manoeuvres in which more significant neck extension than the (15-20 degrees in the Epley) is required

4. **Crevits et al forced 2004** prolonged positioning manoeuvre: a deep head hanging (at least 60 degree of extension), then sit upright and immobilise head for 24 hours

5. **Kim et al 2005** : head right or left, then lying supine with 30 degrees of neck extension, then patient lies supine (with head turn), then finally sits up with chin tilted 30 degrees down

6. **Yacovino et al 2009 and 2014** Deep head hanging manoeuvre. In this manoeuvre the patient lies supine with 30 degrees of neck extension (meaning the anterior canal is 50 degrees below the horizontal). The patient then puts his chin to his chest before sitting up. Essentially this should treat anterior BPPV of either side, and so is useful when no torsional component to the nystagmus is seen and the side of BPPV is unclear

**References**


Yacovino DA, Hain TC, Olivier MA, HermanLaflue A, Gualteri FJ. Barany Society (abstract) May, 2014