Nistagmo posicional *downbeat*: Uma revisão narrativa

Positional downbeat Nystagmus: A narrative review

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ABSTRACT
Objectives: To review the pathophysiology, differential diagnosis and treatment of central and peripheral positional downbeat nystagmus.
Study design: Narrative review.
Material and Methods: A search on "positional downbeat nystagmus" based on evidence presented in peer-reviewed journals was conducted on Pubmed® and ScienceDirect®databases. Reference lists of retrieved articles were manually searched to identify further relevant literature, including reference books.
Results and Conclusions: Positional downbeat nystagmus is a rare entity attributed either to central pathology, for example posterior cerebellar lesions close to the midline, especially if the flocculus and paraflocculus are involved or the result of APC or AC BPPV. Nystagmus characteristics may be not sufficient to distinguish between the two entities. The conversion into a typical posterior canal BPPV by liberatory manoeuvres is the only criteria to diagnose a definitely peripheral APC or AC BPPV.
Keywords: downbeat positional nystagmus; apogeotropic posterior canal; superior canal; paroxysmal positional vertigo; central dysfunction.

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MANUSCRIPT

1)Pathophysiology of spontaneous downbeat nystagmus:
Downbeat nystagmus is a rare entity and can be the result of both central and peripheral origin[1]. The ENT should be capable of identifying and distinguishing its possible aetiologies. Downbeat nystagmus can be spontaneous or positional.
Spontaneous downbeat nystagmus is usually attributed to central pathology, namely posterior cerebellar lesions close to the midline, especially if the flocculus and paraflocculus are involved[2]. Spontaneous downbeat nystagmus results from defective vertical gaze holding that allows for a pathologic upward drift of the eyes, which is then corrected with a downward saccade[3]. Spontaneus downbeat nystagmus in the primary gaze is usually persistent and not supressed by fixation. This type of acquired nystagmus has a tendency to increase both on lateral and downward gazes or with head extension. Oscillopsia and postural imbalance with a tendency to fall backward are usually present[3].
Although not totally understood, the pathophysiology of spontaneous downbeat nystagmus rely in tone imbalance caused by lesions of the pathways mediating 1) signals of vertical cerebellovestibular neural integrator; 2) central connections of the vestibular-ocular-reflex (VOR); including both the vertical semicircular canals and otolith response or
3) signals of the vertical smooth pursuit system. Structural or functional lesions in the craniocervical junction either on the floor of the IVth ventricle or vestibulocerebellar floccullus could also convey in a downbeat nystagmus[3].Rather than spontaneous, a paroxysmal positional induced downbeat nystagmus may also occur in central disorders and is typically accompanied by mild vertigo[4].
On the other hand, a positional paroxysmal downbeat nystagmus due to pure peripheral disease has already been well described. In benign paroxysmal positional vertigo (BPPV) when the vector of the displacement of the endolymph relative to the kinocilium within

the cristae ampullaris is excitatory on the plane of the semicircular anterior canal or inhibitory on the plane of the posterior semicircular canal a positional downbeat is also elicited, as we explain later on this review[5].

1.1) Central Positional downbeat nystagmus

The central positional downbeat nystagmus is usually the result of disinhibited otolith-canal interaction in nodulus lesions[6]. Sometimes a positional downbeat nystagmus could be the only sign in neurological patients. Patients with vestibular migraine can also present with positional downbeat nystagmus[7]. Diagnosis of central positional downbeat nystagmus can be challenging and some clinical features are usually used to distinguish it from infrequent variants of BPPV. Centrality should be suspected when a positional downbeat nystagmus presents in the absence of vertigo or it appears in a plane not related to a correspondent semicircular canal. Directional changing torsional component, when present, is also a sign of non-peripheral nystagmus. The latency of the positional downbeat nystagmus is usually short or absent, fatigue and habituation does not occur, although these characteristics could also present in atypical forms of BPPV[8].

Bertholon *et al*. reviewed 50 patients with positional downbeat nystagmus and only on 75% a central nervous system disorder was found. The remaining 25% were considered to have an idiopathic positional downbeat nystagmus[9]. Some causes of positional downbeat central nystagmus are listed on table 1.

Prognosis of central positional downbeat nystagmus depends on the underlying cause. Frequently, it is permanent when caused by structural lesions and reversible when caused by intoxication or metabolic deficiency. In cases of disturbing oscillopsy due to spontaneous downbeat nystagmus 4-aminopyridine (5–10 mg tid), 3,4- diaminopyridine (10–20 mg tid), baclofen (5 mg tid) and clonazepam (0.5 mg tid) have alredy been used with sucess[10]. 4-aminopyridine is also reported to be successful in cases of positional downbeat nystagmus[11].

TABLE 1

Causes of positional downbeat central nystagmus (adapted from [4])

Cerebellar degeneration	Multiple system atrophy Episodic ataxia type 2 Spinocerebellar atrophy type 6 Idiopathic
Drug toxicity	Amiadarona Phenytoin Carbamazepin Lithium
Developmental abnormalities	Arnold-Chiari malformation (commonest lesion associated)
Neoplastic	Primary or secondary occupying lesions around the IVth ventricle
Paraneoplastic	
Posterior circulation stroke	
Migraine	
Multiple Sclerosis	
Tyamine and Vitamin B12 deficiency	

2) Positional downbeat nystagmus due to anterior canal (AC) BPPV or apogeotropic posterior canal (APC) BPPV

a)Epidemiology of BPPV

BPPV is the most common cause of vestibular vertigo. It has a reported lifetime prevalence of 2.4%, an annual incidence of 0.6% and it increases with age[12]. When considering an otoneurological clinic setting, the rate of diagnosed BPPV may be as high as 28%[13]. Additionally, women are two to three times more affected than men[14]. A right ear predominance is usually reported.[15] Bilateral involvement is reported in up to 20%, mainly after head trauma[16].

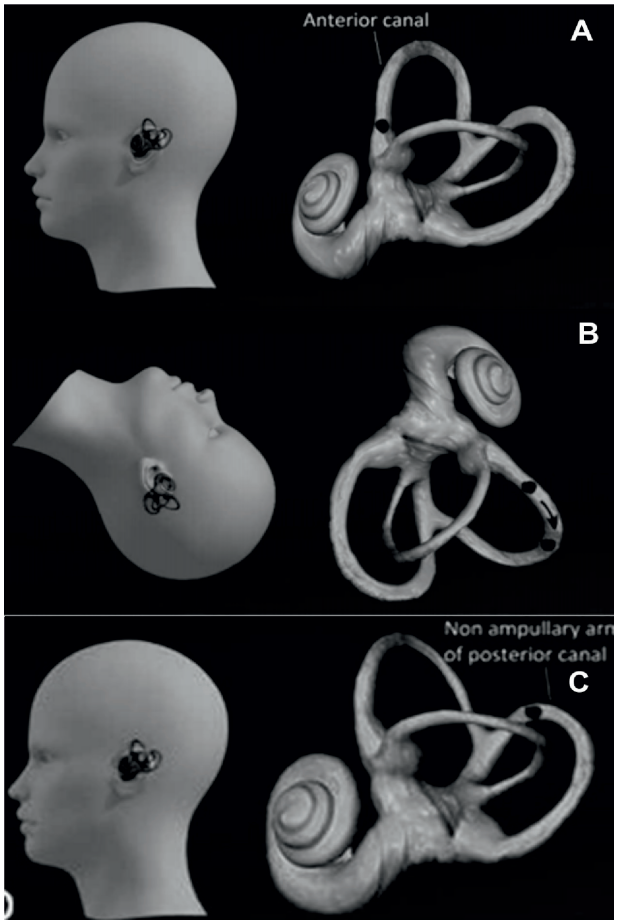
The posterior canal is, by far, the most affected canal (up to 88%), followed by the lateral/horizontal canal[17]. Rarer forms (5%) of BPPV include anterior canal BPPV, short arm canalolithiasis from the horizontal canal, and apogeotropic posterior canalolithiasis[18].

b)Positional downbeat nystagmus in BPPV – AC BPPV and APC BPPV

A downbeat nystagmus provocation by bilateral Dix-Hallpike and straight head-hanging may be explained by the vertical upwards orientation of the ampullary segment of the anterior canal in the normal upright head position; whereas in apogetropic posterior canal BPPV it emerges due to a ampullopetal flow (inhibitory) within the non-ampullary arm of the posterior canal (figures 1a, 1b,1c, adapted from Califano *et al*[18] with permission). Recently, Buki *et al* also proposed cupulolithiasis with debris attached to the inferior-most aspect of the cupula within the PC as a possible cause of a slow downbeat positional nystagmus during Dix-Hallpike manauver[19]. However, not much literature concerning this subtype of BPPV is yet available.

In both these rare forms of BPPV, a paroxysmal and positional downbeat nystagmus is evident, therefore differential diagnosis between AC and APC assumes a relevant importance. Besides the downbeat component, one must pay attention to the torsional component (usually more evident in APC than AC) and characteristics in sitting from supine position. Usually these atypical forms of BPPV present as follows on table 2[20]. The APC and AC forms BPPV are usually associated with a less intense symptomatology of vertigo compared to the typical forms of BPPV; patients usually complain of dizziness getting out of bed (or bending/extending the head) and marked neuro-vegetative symptomatology.

FIGURE 1
a) AC BPPV in sitting position; b) debris in AC BPPV in straight-head-hanging positioning; c) localization of debris in APC BPPV in sitting position (adapted from Califano *et al* [18] with permission)



Usually, the ocular movements have no latency, have longer duration and scarce fatigability[22].
Even considering the above mentioned characteristics, the involvement of the non-ampullary end of the posterior canal (APC) is, sometimes, hardly distinguishable from contralateral anterior canal canalolithiasis. In these situations, vHIT (where high-frequency VOR for vertical canals can be quickly assessed) may play a key role in the differential diagnosis of AC or APC BPPV[23]. Accordingly to Castellucci *et al*, otoconia debris can interfere with endolymphatic dynamics and cupular response mechanisms, resulting in high-frequency VOR deficit for the vertical involved canal due to cupula continuous activation to low-frequency stimuli (otoconial shifts) and inhibiting the ampullary receptor to respond to high-frequency inputs (head impulses)[23].
However, this hypothesis of VOR impairment is a matter of debate. Some degree of dysfunction occurs in the utricular macullae as this is the expected source of otoconia in idiopathic forms[24] but the majority of works published refer to a normality of VOR gain in the semicircular canals, even with BPPV with positional downbeat nystagmus[25]. Current clinical practice guidelines do not recommend vestibular testing in any type of BPPV[26].

c)Treatment and prognosis of AC and APC BPPV
There are many treatment manoeuvres proposed to these types of BPPV (table 3).
Accordingly to Anagnostou *et al* (2015), in a systemic review of the various treatments proposed for the AC canal BPPV, all manoeuvres demonstrated success rates of over 75%, and the overall sample-size-weighted

TABLE 2
Nystagmus characteristics of AC and APC BPPV in different head positions

	Anterior Canal BPPV*	Apogeotropic Posterior Canal BPPV
Dix-Hallpike	Sometimes present: downbeat, torsional clockwise if left side affected and counter clockwise if right side affected	Downbeat, torsional clockwise if right side affected and counter clockwise if left side affected
Head-hanging (Rose position)**	Downbeat, torsional to ipsilateral affected side	Sometimes present: downbeat, torsional to healthy side
Sitting from Supine	No reversal of nystagmus	Weak reversed nystagmus

*A pure downbeat nystagmus with lack of torsional component has been reported due to anatomical orientation of the AC: closer to the sagittal plane (about 41º) thant the posterior canal (56º) [9] or in bilateral AC BPPV, according to Zapala [21]
** The straight head hanging position may resolve AC BPPV or convert AC and APC BPPV into typical PC BPPV, a characteristic we should pay attention to in making the diagnosis,as further discussed.

TABLE 3
Treatment manoeuvres

AC BPPV	APC BPPV
Reverse Epley (by Honrubia *et al* in 1999) [27]	Quick Liberatory Rotation Manauver (by Califano *et al* in 2003) [28] *
Rahko manauver (by Rahko *et al* in 2002) [29]	Demi Semont (by Vanucchi *et al* in 2015) [22]
Crevits manauver(by Crevits *et al* in 2004) [30]	45° forced prolonged position (by Vanucchi *et al* in 2015) [22]
Kim manauver (by Kim *et al* in 2005) [31]	
Yacovino (by Yacovino *et al* in 2009) [32]	

*first described to treat typical posterior canal BPPV, but later used in APC by the same authors [18]

mean was 85.6%[33]. Still, a controlled randomized study of a rational AC maneuver is needed.

The authors use preferably the Yacovino manoeuvre due to its simplicity, comfort for the patient and no need to determine laterality. In the Yacovino manoeuvre, also known as the deep hanging manoeuver, the patient lies supine with 30ºdegrees of neck extension. The patient then moves his chin to his chest before sitting up. The patient remains about 30 seconds in each of the 3 positions (figure 2)[32]. The outcome of Yacovino's manoeuvre seems to be affected by the duration of the positional downbeat nystagmus: patients demonstrating positional nystagmus lasting for less than 1 min have significantly better outcomes[34].

Quick liberatory manoeuvre, prolongued 45º degrees position and demi Semont manoeuvres act as liberataroy manoeuvres, conducting the otoconia from the non ampullary end of the posterior canal to the utricle. To our knowledge, no systematic review or meta-analysis comparing manoeuvres in the APC BPPV form exist.

The demi Semont manoeuvre consists of a contralateral "half Semont" with the head turned 45° to the healthy side. In detail, the patient's head is turned by 45° to the healthy side, and then the patient is brought onto that

FIGURE 2
Yacovino Manauver (from [18] with permission)

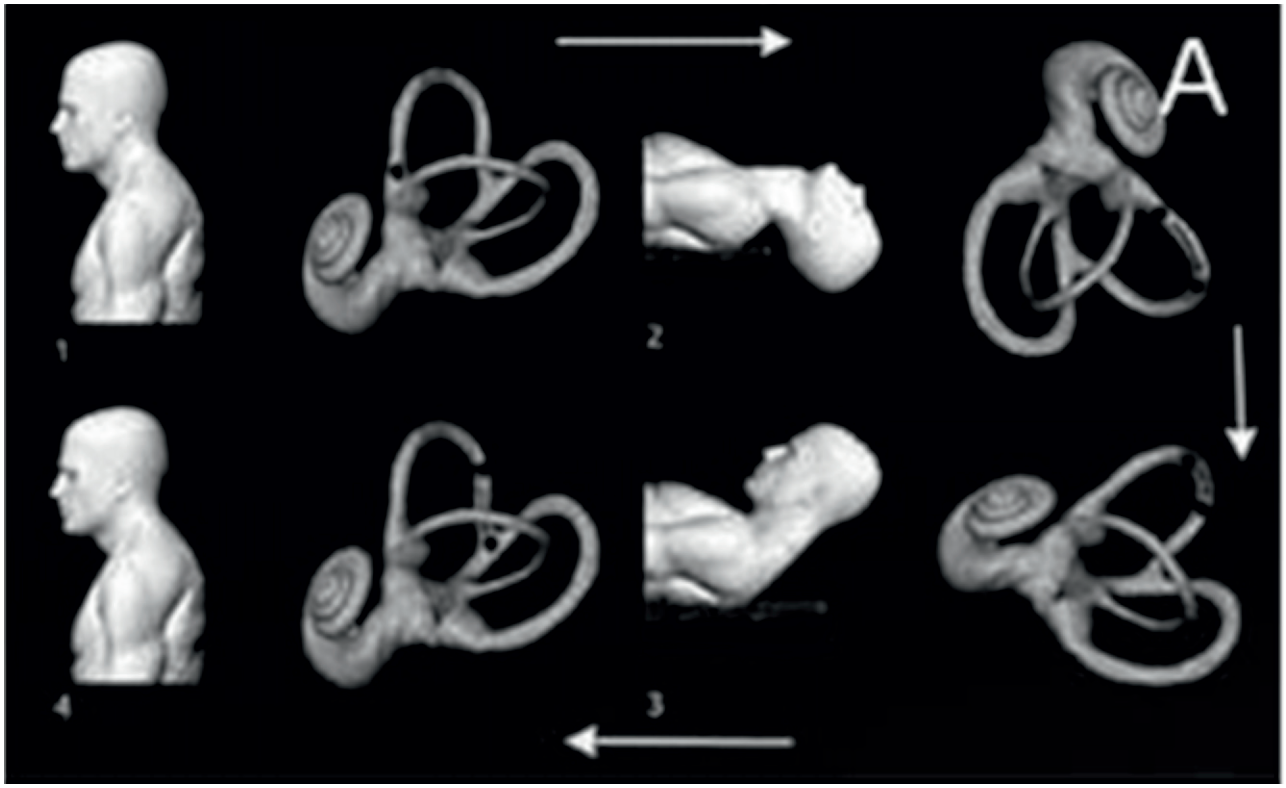


FIGURE 3
Dynamics of QLR. From left to right: A. Starting position (–45°); B. "Dynamic" middle position (about +45°); C. Final lying position (about +135°). Velocity of head is about 180°/sec. (from [28], with permission)

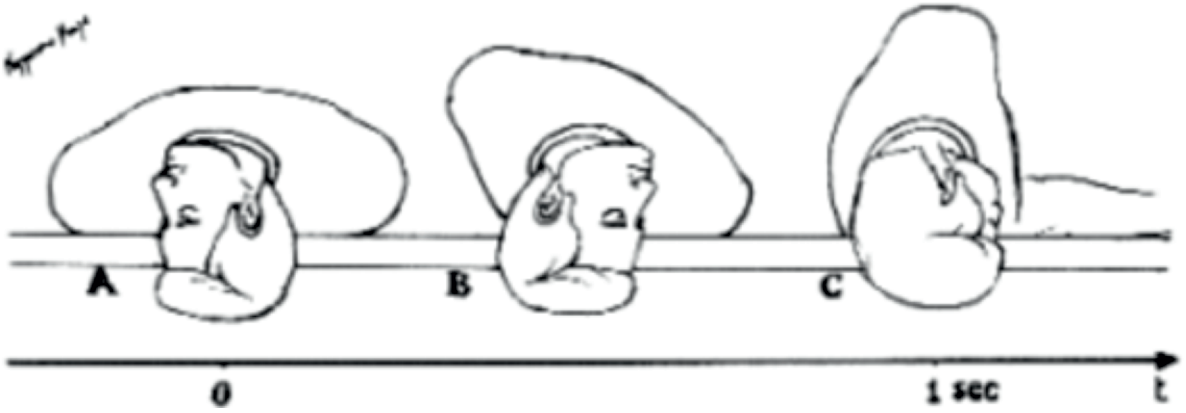


TABLE 4
Diagnosing AC BPPV and APC BPPV with liberatory manauvers, according to Califano *et al*.

	Anterior Canal BPPV (AC)	Apogeotropic Posterior Canal BPPV (APC)
Certain	•Presence of a positional vertical downbeating paroxysmal nystagmus evoque through the straight head-hanging positioning and sometimes through the Dix-Hallpike test; •In such positions, possible presence of a clockwise torsional component for the left AC or a counter-clockwise torsional component for the right AC; •Canal conversion in typical posterior BPPB during or immediately after (no more than two days) the therapeutic manoeuvre	•Presence of a downbeating nystagmus, torsional clockwise for the right canal and counter-clockwise for the left canal, evoked through the Dix-Hallpike test and sometimes through the straight head-hanging positioning •Possible presence of a vertical downbeat component in the same positioning tests. •Canalar conversion in typical posterior canal BPPV during or immediately after (no more than two days) the therapeutic manoeuvre.
Probable	•As reported for "certain" AC BPPV, but with a direct resolution without canalar conversion in typical posterior canal BPPV	•As reported for "certain" APC BPPV, but with a direct resolution without canalar conversion in typical posterior canal BPPV
Possible	•Persistence of symptoms after five cycles of therapeutic manoeuvres •MRI does not show any neurological disease as a presumed cause of the nystagmus	•Persistence of symptoms after five cycles of therapeutic manoeuvres •MRI does not show any neurological disease as a presumed cause of the nystagmus

side. A liberatory nystagmus with opposite direction may be seen in this position. Finally, after 30 seconds, the patient is brought back quickly to the sitting position. The complete sequence of movements is repeated up to five times[22].

The 45° forced prolonged position can free the canal from debris through gravity. It is preferably to use in patients with low mobility, obese or older. The patient should lie down on the healthy side with head turned 45° downwards for, at least, 8 hours[22].

In the Quick Liberatory Rotation (QRL) manoeuvre, first described by Califano et al for the treatment of typical posterior BPPV, the patient is tested by Dix-Hallpike manoeuvre and two minutes after paroxysmal nystagmus disappears, the patient is brought from the affected, to the contralateral, side by a quick rotation (less than one second) of the head of about 180° in the horizontal plane; the head is about 45° downward, staying in this position for two minutes after secondary nystagmus disappears or four minutes, if secondary nystagmus is not detected; finally the patient is brought to the sitting position (figure 3)[28].

In the literature, is consensual that cerebral MRI is required in cases of positional downbeat nystagmus if there are associated neurological signs or symptoms or in the case of failure of therapeutic manoeuvres[35].

Finally, Califano et al proposed a useful grading AC and APC BPPV from "certain" to "possible" (table 4)[18].

CONCLUSION

Positional downbeat nystagmus is a rare entity and can be the result of both central and peripheral origin. This type of nystagmus has been reported in various structural, metabolic, or degenerative disorders affecting the cerebellum as well as in of both apogeotropic posterior canalithiase or anterior canalithiase forms of BPPV. It is essential to correctly distinguish between the central and peripheral entities in order to instigate correct and prompt investigations and subsequent management. Distinction based on assessment of latency, direction, habituation, fatigability and reversal of nystagmus may be not sufficient to distinguish both entities.

This narrative review draws special attention to the rare peripheral AC and APC forms of BPPV that can present to an ENT with a downbeat positional nystagmus and highlights its characteristics and treatment. As so, in the case of a downbeat positional nystagmus, without central signs, one should first perform liberatory manoeuvres; the conversion into a typical posterior canal BPPV is the only criteria to diagnose a definitely peripheral APC or AC BPPV. Probable and possible AC or APC BPPV needs a close follow up; one should ask for MRI in any case of doubt.

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Disclosures

The authors have nothing to disclosure.

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