Review

Revised criteria for suspicion of non-benign positional vertigo

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Summary

Benign paroxysmal positional vertigo (BPPV) is the most commonly diagnosed vestibular vertigo. BPPV can be diagnosed by clinical examination and its treatment is based on particle repositioning manoeuvres, and specialized equipment is not required. Therefore, most patients could be diagnosed and treated by their general practitioner. Unfortunately, not all positional vertigos are benign. Symptoms similar to those of BPPV can be caused by diseases that affect the central nervous system. It must be possible to define criteria that allow us to suspect, in a patient with symptoms of positional vertigo, the possibility of a cerebral origin (‘non-benign PV’). Requests for magnetic resonance imaging must be justified by the fulfilment of these criteria. That is especially relevant in primary care, because these criteria should make possible to distinguish between patients with positional vertigo that could be treated by general practitioner and patients that need to be directed to specialized units. We propose the following revised criteria for suspected non-benign PV: (i) association with signs or symptoms of neurological disorder, (ii) nystagmus without dizziness in positional diagnostic tests, (iii) atypical nystagmus direction, (iv) poor response to therapeutic manoeuvres and (v) recurrence (confirmed by positional tests) on at least three occasions.

Introduction

Since its original description by Bárány in 1921,¹ benign paroxysmal positional vertigo (BPPV) has been one of the most commonly diagnosed otoneurological entities, especially following its more precise characterization by Dix and Hallpike in 1952.² In our clinic, it accounts for ~13% of patients consulting for alterations of balance,³ and other authors have reported rather higher prevalences.⁴,⁵ Diagnosis is established from clinical features, based on anamnesis and physical examination, and its treatment comprises particle repositioning manoeuvres. Hence, this is a pathology that can be diagnosed and treated by general practitioner.

BPPV has generally been regarded as essentially due to the displacement of otoconia from the utricle...
to one or more of the semicircular canals, where they either adhere to the cupula (Schuknecht’s cupulolithiasis mechanism\(^6\)) or remain free to gravitate to whichever point of the canal is momentarily the lowest (Hall’s canalithiasis mechanism).\(^7\) Nevertheless, on the basis of histopathological findings it has recently been suggested that displaced otoconia are not the cause of BPPV, or not the only cause.\(^8\) The suggested alternative is neuronal degeneration leading to a reduction of the inhibition, by utricle and saccule, of afferent signals from the canals.

Conventional treatment of BPPV is based on the assumption of displaced otoconia. Otoconia in the posterior semicircular canal (the most frequent location) can be dislodged from the cupula by Semont’s manoeuvre\(^9\) and returned to the utricle by Epley’s manoeuvre\(^10\) or variants thereof. Analogous manoeuvres have been designed for otoconia located in the other semicircular canals.\(^11\)–\(^13\) The vast majority of BPPV patients can be treated effectively by means of these procedures, with or without supplementary exercises.\(^14\)–\(^15\)

Unfortunately, not all positional vertigoes are benign. Symptoms very similar to those of BPPV can be caused by diseases that affect the central nervous system (CNS), such as multiple sclerosis, brain tumours or brainstem stroke. Ideally, the differential diagnosis of BPPV would therefore include imaging studies—magnetic resonance imaging (MRI) is particularly efficient—to rule out the possibility of brain lesions. However, as the high incidence of BPPV makes routine MRI of all positional vertigo patients impracticable in the average public health service, requests for MRI must in practice be justified by the fulfilment of criteria for a tentative diagnosis of ‘suspected non-benign PV’. That is especially relevant in primary care, because these criteria should make possible to distinguish those patients with positional vertigo that could be treated by general practitioner and those patients that need to be directed to specialized units to rule out other causes distinct to BPPV.

Classical criteria for central positional vertigo

Traditionally, positional vertigo of central origin is suspected if patients test negative for BPPV according to the criteria proposed by Dix and Hallpike,\(^2\) according to which BPPV should be diagnosed if the nystagmus elicited by the Dix–Hallpike manoeuvre exhibits the following characteristics:

(i) **geotropic direction** (i.e. rotational or linear beating towards the side facing downwards) upon adoption of the test position, and reversal of this orientation (or absence of nystagmus) upon return to upright;
(ii) **latency**—a lag of several seconds between adoption of the test position and the appearance of nystagmus;
(iii) **limited duration**—the disappearance of nystagmus and vertigo if the supine position is maintained;
(iv) **refractoriness**—the absence of nystagmus, or the reduction of its duration and intensity, upon immediate repetition of the test.

Latency, limited duration and refractoriness are shared by, and are readily explicable in, patients with BPPV due to canalithiasis. Movement of the head only leads to movement of displaced otoconia, and hence to undesired endolympathic currents, after a lag of a few seconds. Movement of otoconia ceases when the otoconia reach the lowest point of the canal, and the ensuing decay of endolympathic currents limits the duration of nystagmus and dizziness. In addition, the test will not generate the symptoms again, or not as effectively, if, following the patient’s return to an upright position, it is repeated before the otoconia have time to regroup in their initial position.

In contrast, the Dix–Hallpike test nystagmus of patients with BPPV due to cupulolithiasis may exhibit neither latency, nor limited duration, nor refractoriness. The adherence of otoconia increases both the instability of the cupula in its neutral position and the inertia it opposes to restoring forces once displaced. It therefore reacts to the Dix–Hallpike manoeuvre immediately, without any latency; fails to recover its neutral position until the patient regains the upright; and once it has recovered its neutral position can react again immediately, without any refractory period. Refractoriness is likewise often absent in cases of BPPV due to canalithiasis of the horizontal semicircular canal.\(^16\)

The Dix–Hallpike criteria are also ambiguous or unreliable as regards the direction of nystagmus. Although BPPV of posterior canal origin and anterior canal origin both give rise to geotropic rotatory beating, in both cases the observed nystagmus also has a vertical component, which is downward in the latter case but upward in the former. And if it is the horizontal canal that is affected, the associated purely horizontal nystagmus is geotropic or ageotropic depending on whether BPPV is due to canalithiasis or cupulolithiasis. These directions have been recently characterized in a three-dimensional spatio-temporal study.\(^17\)

The above shortcomings of the Dix–Hallpike criteria all court false negative results of the test for BPPV, and hence unnecessary requests for imaging studies. More serious is the fact that the Dix–Hallpike criteria for BPPV can be satisfied by patients with CNS lesions.\(^18\)–\(^22\) It has been reported,
for example, that patients with positional vertigo due to lesions of the vermis cerebelli exhibit refractory nystagmus in the Dix–Hallpike test.23

New criteria for suspected non-benign positional vertigo

In view of the above shortcomings of the Dix–Hallpike criteria, and on the basis of our experience in this field, we propose the following revised criteria for suspected non-benign positional vertigo.

1. Presence of other signs or symptoms of neurological disorder: This criterion naturally applies to all patients with balance problems, not only positional vertigo patients, but should not be overlooked during anamnesis and examination. Even when the patient complains chiefly of typical BPPV symptoms, he or she should be asked about accompanying symptoms (headache, seeing double, etc.), and cranial nerve and cerebellar function should be checked. Nevertheless, the presence of neurological symptoms not excludes the diagnosis of BPPV; in fact, BPPV is not unusual in multiple sclerosis. It has been reported a good response to repositioning manoeuvres in these patients.24,25

2. Appearance of nystagmus without dizziness in positional tests: The opposite, the appearance of dizziness without nystagmus, should not necessarily arouse suspicion of a central lesion, because it is often seen in BPPV patients in late stages of spontaneous resolution (probably because the endolymph currents caused by vestigial otoconia are strong enough to cause dizziness but too weak to trigger nystagmus).

3. Atypical nystagmus direction,26 especially in the following two cases.

a. Downward-beating vertical positional nystagmus: Although the presence of downbeating nystagmus in healthy people with migraine has been reported,27 the downbeating ‘positional’ nystagmus should be an absolute indication for an imaging study, usually MRI of the posterior fossa. Although vertical downbeating, accompanied by rotation, is also seen in patients with BPPV originated by the anterior semicircular canal, the rarity of this type of BPPV suggests that an imaging study is a prudent measure even when rotation is observed. Figure 1 shows the videonystagmographic record from a patient with a downbeating vertical

Figure 1. Videonystagmographic register of a positional central nystagmus (Arnold–Chiari malformation) in the Dix–Hallpike test.
nystagmus in all decubitus positions of the Dix–Hallpike test, which did not cease. This patient suffered an Arnold–Chiari malformation.

b. Nystagmus that takes different directions in positional tests carried out at different times: If repositioning manoeuvres have not been developed (in this case, probably debris has moved from one canal to another). This should be an absolute indication for MRI of the posterior fossa, and if the MRI shows no lesions, evaluation by a neurologist should be requested. Note that the nystagmus should occur in different directions when on different occasions the ‘same’ position is adopted; if different directions are followed when the patient is placed in different positions, he or she probably has BPPV originated by more than one canal.\(^\text{28,29}\)

4. Poor response to therapeutic manoeuvres: If repeated performance of manoeuvres designed to dislodge and relocate otoconia fails to resolve the symptoms, MRI of the posterior fossa should be requested. MRI of the posterior fossa should of course always be carried out before referral for surgical therapy. In patients with poor response to ‘manoeuvres’, MRI is also useful for detecting anatomical abnormalities in semicircular canals.\(^\text{30}\)

5. Frequent recurrence of symptoms: The recurrence of BPPV following successful treatment with therapeutic manoeuvres is not uncommon. The expected rate of recurrences in a follow-up period of 10 years in our patients diagnosed with posterior semicircular canal BPPV (in a series of 419 patients) was 27%.\(^\text{31}\) This data were similar to those reported in a study made in a geographical area close, and published recently.\(^\text{32}\) However, other authors have reported recurrence rates of up to 50% at 5-year\(^\text{13}\) or 10-year\(^\text{34}\) follow-up. Multiple recurrence should nevertheless not be automatically interpreted as recalcitrant BPPV (perhaps, due to some property of the patient’s otoconia making them particularly prone to displacement), but should instead suggest that the symptoms may have a central origin. As only 3% of the above-mentioned 419 patients suffered more than two recurrences, requesting MRI studies in these cases would not overload MRI services.

### Conclusion

Experience of the results of using classical criteria for suspicion of non-benign PV, together with advances in our understanding of BPPV, suggest that these criteria are currently less than satisfactory. We propose the following revised criteria for suspected non-benign positional vertigo.

1. Association with signs or symptoms of neurological disorder.
2. Nystagmus without dizziness in positional diagnostic tests.
3. Atypical nystagmus direction, especially
4. Downward-beating vertical nystagmus, and
5. Nystagmus that takes different directions in positional tests carried out at different times.

4. Poor response to therapeutic manoeuvres.
5. Recurrence (confirmed by positional tests) on at least three occasions.

### References


