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COMMENTARY

Role of vestibular testing in diagnosis of benign paroxysmal positional vertigo

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ABSTRACT

Diagnosis and treatment of benign paroxysmal positional vertigo (BPPV) is a mixture of empiricism of particle repositioning with the rationally-based knowledge obtained from clinical observations, histopathology, and neurophysiological experiments. The recently published clinical practice guideline on BPPV makes recommendations on the management of BPPV. One of the statements discourages the use of radiographic or vestibular testing, unless the diagnosis was uncertain or there were additional signs or symptoms unrelated to BPPV. The role of video-oculography in diagnosis and treatment of BPPV is argued, since vestibular testing has provided key relevant information to understand positional nystagmus in patients with BPPV.

The clinical practice guideline (CPG) on benign paroxysmal positional vertigo (BPPV) published by Bhattacharyya et al[1] makes recommendations on managing BPPV, and these will be used as a source of evidence for clinical practice in any setting. This CPG provides statements for: 1) diagnosis of posterior and lateral canal BPPV; 2) differential diagnosis of BPPV from other causes of imbalance, dizziness, and vertigo; 3) radiographic and vestibular testing; 4) repositioning maneuvers, vestibular rehabilitation, or observation as initial therapy; 5) medical therapy; 6) evaluation of treatment failure; and 7) education.

The most relevant aspect in the CPG on BPPV is its educational purpose for the general clinician. Despite BPPV being the most common vestibular disorder, it is not well known in clinical practice outside specialty neuro-otologic clinics, and this lack of information among clinicians leads to a delay in diagnosis and treatment of BPPV.[2] The natural history of BPPV is poorly understood and the disease is recurrent in around 40 percent of patients at two years, with an increase of recurrences associated with longer follow-up periods.[3]

However, the panel has made recommendations against radiographic or vestibular testing, unless the diagnosis was uncertain or there were additional signs or symptoms unrelated to BPPV. This statement will not help clinicians or patients in the management of BPPV. It is important to perform a detailed physical examination to search for clinical neurological signs, since most patients will show some abnormalities. The CPG should comment on the diagnostic value of a basic neuro-otological examination in the management of a patient with vertigo, including oculomotor, saccades, spontaneous, gaze-evoked, and head-shaking nystagmus; head-impulse test; cranial nerve examination; and Romberg, Barany, and Fukuda tests. BPPV is currently under diagnosis, but the disease is more complex than posterior and sometimes horizontal canal variants.

Vestibular testing can provide useful information in a patient with a positive Dix-Hallpike test. First, video-oculography (VOG) is the best way to analyze positional nystagmus at different positional tests (supine, Dix-Hallpike, head-hanging in midline during supine) to determine which canals are involved in the generation of the nystagmus. Second, if we do not investigate the status of the vestibular system, we will miss essential information to know whether the patient has a noncompensated vestibular lesion.

Vestibular testing should be used if neurological signs are found in the clinical examination. These could be oculomotor abnormalities of saccades, smooth pursuit, or an impairment of the vestibulo-ocular reflex (positive head-thrust test, spontaneous, gaze-evoked, or head-shaking nystagmus). Moreover, the observation of atypical positional nystagmus (nonrotatory, lack of latency, adaptation, or fatigue) or nystagmus observed at different positional tests warrants the use of VOG to distinguish between central and peripheral positional nystagmus. The adaptation of the response, decrease of velocity of the positional nystagmus with an increase of the interval between nystagmuses, can be recorded by VOG and is a feature of peripheral positional nystagmus. The involvement of other canals such as the horizontal or anterior, or the simultaneous contribution of two or three canals, has been demonstrated by vectorial analysis of the rotational axis of positional nystagmus in BPPV.[4,5] There is ongoing research on this topic by different groups to diagnose the different clinical variants, and two-dimensional and three-dimensional VOG is the instru-

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ment that will help quantify the parameters of positional nystagmus. These techniques have demonstrated that the prevalence of atypical positional nystagmus is higher in patients with BPPV than was previously reported.[6-8] Atypical positional nystagmus includes horizontal and anterior canal positional nystagmus and multiple positional nystagmus (observed at different positional tests), which suggests the presence of lithiasis at different canals at once.[7]

In our neuro-otology clinic, we have been using VOG during positional testing for diagnosis of BPPV for the last eight years. The Research and Ethics Hospital Review Board has approved all previous studies on BPPV performed in our institution. Between September 1999 and December 2001, 94 patients with BPPV were diagnosed by direct observation of positional nystagmus during the Dix-Hallpike or the roll test. In this period, we found 95 percent posterior canal and 5 percent horizontal canal BPPV. Between March 2002 and December 2006, diagnosis was performed in the office followed by two-dimensional VOG recording in 132 individuals with BPPV. Then, we found 45 percent unilateral posterior canal, 7 percent with a bilateral posterior canal affectation, 17 percent horizontal, 15 percent with positional down-beating nystagmus (anterior canal, central positional nystagmus), and 15 percent with multiple canal affectation. These patients with multiple positional nystagmus in our series showed a direction-changing positional nystagmus, with horizontal and vertical components, which cannot be explained by a single canal affectation. Some of these patients showed changes in the nystagmus during the follow-up, confirming the complex scenario of some cases of BPPV.[7] Nothing has changed in our setting, a general hospital, except the systematic use of VOG in diagnosis of BPPV since 2002, and our series has changed dramatically. Treatment is based on the analysis of positional nystagmus recorded. When the VOG analysis indicates the involvement of two canals, the severity of perceived vertigo and the velocities of positional nystagmuses registered help to decide which canal should be treated first.

BPPV is not a diagnosis that can exclude other causes of vestibular disorder. It can be observed in patients with vestibular hypofunction, migraine, or Ménière's disease, and these comorbidities may change the functional status of the vestibular system and the vestibulo-ocular responses. In a prospective series, horizontal canal paresis (CP) has been shown in 25 percent (16/64) of patients with BPPV at diagnosis.[9] One year after, seven individuals with CP showed a normal caloric response, another seven demonstrated persistent CP, and one case developed a bilateral CP. Three of the patients in this series with normal caloric response at diagnosis developed a mild CP (31%-32%) one year after.

Vestibular evoked myogenic potentials is an emerging vestibular test able to evaluate the integrity of the saccule and the inferior vestibular nerve by testing the vestibulo-collic reflex. Recently, it has been reported that BPPV patients showed prolonged p13 and n23 latencies compared with those of the control group, suggesting that some patients with BPPV could have neural degeneration in the afferent fibers from saccule or in the inferior vestibular nerve itself.[10]

A patient with vertical spontaneous nystagmus may develop a positional nystagmus, since a latent nystagmus may be triggered by the force of gravity, and I would recommend vestibular testing and MRI for this individual. Some patients report imbalance when they walk and turn their head, after successful therapy for BPPV, and it is necessary to demonstrate that they do not have an uncompensated vestibular dysfunction.

I will not recommend vestibular testing for a patient with a first episode of unilateral posterior canal BPPV without any neurological sign in clinical examination. However, I will perform it in patients with recurrent BPPV, atypical nystagmus, or neurological signs.

The CPG on BPPV finishes with a list of research needs, including studies to identify which subsets of patients, according to specific history or physical examination findings, should be submitted for additional vestibular testing or radiographic imaging.

Diagnosis and treatment of BPPV is a mixture of empiricism of particle repositioning with the rationally based knowledge obtained from clinical observations, histopathology, and neurophysiological experiments. Vestibular testing has provided relevant information to understand positional nystagmus in patients with BPPV, and VOG will incorporate new knowledge to the diagnosis and treatment of the disease.

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AUTHOR CONTRIBUTION

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