Persistent Vertigo Following Particle Repositioning Maneuvers

An Analysis of Causes

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Objective: To analyze the causes of persistent vertigo following treatment with particle repositioning maneuvers (PRMs) in patients with benign paroxysmal positional vertigo.

Design: Prospective study of outcomes in patients with benign paroxysmal positional vertigo.

Study Setting: Outpatient clinic of a tertiary care referral center.

Patients: A sample of 90 consecutive patients with documented benign paroxysmal positional vertigo of the posterior semicircular canal who had persistent vertigo after at least 3 sessions of PRMs during a period of 2 weeks.

Intervention: Particle repositioning using a modified Epley maneuver.

Main Outcome Measure: Persistent vertigo following at least 3 sessions of PRMs over a period of 2 weeks.

Results: Seven patients showed partial or no improvement following treatment. The causes subsequently determined included coincident horizontal canal positional vertigo (2 cases), Menière’s disease (2 cases), persistent posterior canal benign paroxysmal positional vertigo in association with cervical spondylosis (2 cases), and a posterior fossa meningioma (1 case).

Conclusions: Patients with persistent or frequently recurring positional vertigo following treatment with PRMs should undergo detailed investigation to exclude coincidental pathology for which specific treatment is required. In patients in whom no coincident pathology requiring therapy is identified, treatment options other than the PRM already instituted should be considered.


Benign paroxysmal positional vertigo (BPPV) is a clinical condition characterized by transient episodes of vertigo when the affected ear is in the dependent position. Although first described by Barany in 1921,1 the specific characteristics of this disorder were defined in 1952 by Dix and Hallpike1 who devised a positioning maneuver to diagnose BPPV.

The treatment of BPPV has ranged from nonintervention (on the premise that it is a self-limiting disorder) to aggressive surgical procedures such as posterior amputary nerve section and posterior canal occlusion. Conservative procedures such as habituation exercises or particle repositioning maneuvers (PRMs) have been recommended by various authors.2-14 Habituation exercises, which were devised by Cawthorne in 19448 and later described by Brandt and Daroff5 and Norre and Beckers,7 aimed to increase the tolerance of the vestibular system to vertigo rather than eliminate its cause. These exercises have been performed by patients with BPPV either as initial treatment or following nonresponse to PRMs. In recent years various forms of PRMs have become popular,5-14 all based on either of 2 theories regarding the etiology of BPPV, the theory of cupulolithiasis and the theory of canalolithiasis.

Some maneuvers were devised on the basis of the Schuknecht theory of cupulolithiasis13 to disperse debris from their attachment to the cupula. These include the Semont liberatory maneuver5 and the Brandt-Daroff exercises.3 The latter consist of a series of repetitive exercises, which could also produce their effect by inducing habituation.

Other maneuvers based on the canaliolithiasis theory aim to displace particles from the semicircular canals into the utricle. The maneuver proposed by Epley,7 which is representative of this cat-
Me´nie`re's disease or vestibular ototoxicity was suspected and associated labyrinthine dysfunction due to conditions such as tinnitus, which suggested a diagnosis of Me´nie`re's disease.6 Those with unilateral tinnitus or asymmetrical hearing loss. Pure-tone audiometry was performed as a routine procedure to perform these exercises for 15 minutes every day for the ensuing 3 months.

• The patients' medical history was reviewed and a positional test was performed to exclude lateral semicircular canal BPPV. Patients who gave a positive response to the positional test were administered the lateral semicircular canal PRM as suggested by Lempert and Tiel-Wilck.12

• Nonpositional vertigo, if present, was treated with appropriate medications.

If not already done, contrast-enhanced computed tomography or magnetic resonance imaging was performed to exclude the possibility of an intracranial lesion.

Table 1. Clinical Data and Management of 7 Patients Who Did Not Respond to PRMs

<table>
<thead>
<tr>
<th>Age, y/Sex</th>
<th>Associated Medical Condition</th>
<th>Cause for PRM Failure</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>60/M</td>
<td>Hypertension, diabetes mellitus</td>
<td>BPPV from left HSC</td>
<td>PRM</td>
</tr>
<tr>
<td>49/F</td>
<td>Hypothyroidism</td>
<td>Ménie`re's disease</td>
<td>Betahistine hydrochloride</td>
</tr>
<tr>
<td>60/M</td>
<td>Hypertension</td>
<td>Ménie`re's disease</td>
<td>Betahistine hydrochloride</td>
</tr>
<tr>
<td>65/M</td>
<td>Cervical spondylosis</td>
<td>Persistent BPPV from PSC</td>
<td>Vestibular habituation therapy</td>
</tr>
<tr>
<td>70/M</td>
<td>Cervical spondylosis</td>
<td>Persistent BPPV from PSC</td>
<td>Refused further treatment</td>
</tr>
<tr>
<td>60/M</td>
<td>Posterior fossa meningioma</td>
<td>Persistent BPPV from PSC</td>
<td>Neurosurgery referral</td>
</tr>
</tbody>
</table>

Abbreviations: BPPV, benign paroxysmal positional vertigo; HSC, horizontal semicircular canal; PRM, particle repositioning maneuver; PSC, posterior semicircular canal.

Of 90 patients who underwent the Epley maneuver, 7 (8%) did not achieve complete relief of symptoms after 2 weeks (Table 1). These 7 patients were administered a PRM with the vibrator. Two patients were discovered to have horizontal semicircular canal BPPV and underwent a specific PRM to which they responded. Of the 5 remaining patients, 2 developed persistent nonpositional episodic vertigo with associated aural fullness and tinnitus, which suggested a diagnosis of Ménie`re's disease. Pure-tone audiometry showed a mild, bilateral, low-frequency sensorineural hearing loss. In both these patients BPPV preceded the first attack of Ménie`re's disease, which appeared about 2 weeks after PRMs were initiated. After detailed investigation to exclude other causes, the patient was given 16 mg of betahistine hydrochloride 3 times daily and experienced relief. Two other patients had persistent BPPV of the posterior semicircular canal with no associated condition except for cervical spondylosis. Of these patients, 1 responded to 3 months of home-based vestibular habituation therapy as detailed above while the other refused further treatment. The remaining patient, who presented 3 times to the out-
Table 2. Causes and Management of PRM Failure in Previous Reports

<table>
<thead>
<tr>
<th>Source</th>
<th>Patients, No.</th>
<th>Failures, No. (%)</th>
<th>Diagnosis</th>
<th>Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>Barnes and Price-Jones, 1993</td>
<td>38</td>
<td>4 (10.5)</td>
<td>BPPV from PSC</td>
<td>PSC occlusion</td>
</tr>
<tr>
<td>Herdman et al, 1993</td>
<td>30</td>
<td>3 (10)</td>
<td>BPPV from PSC</td>
<td>Retreatment</td>
</tr>
<tr>
<td>Harvey et al, 1994</td>
<td>25</td>
<td>8 (32)</td>
<td>BPPV from PSC</td>
<td>Brandt-Daroff exercises</td>
</tr>
<tr>
<td>Welling and Barnes, 1994</td>
<td>27</td>
<td>4 (22.2)</td>
<td>PLF following stapedectomy</td>
<td>Fistula closure, neurosurgery referral, vestibular habitation exercises and posterior canal occlusion</td>
</tr>
<tr>
<td>Weider et al, 1994</td>
<td>44</td>
<td>2 (4.5)</td>
<td>BPPV possibly from PSC</td>
<td>Further treatment refused</td>
</tr>
<tr>
<td>Steenerson and Cronin, 1996</td>
<td>20</td>
<td>3 (15)</td>
<td>BPPV possibly from PSC</td>
<td>Complete relief with vestibular habituation therapy</td>
</tr>
</tbody>
</table>

Abbreviations: BPPV, benign paroxysmal positional vertigo; HSC, horizontal semicircular canal; PLF, perilymph fistula; PRM, particle repositioning maneuver; PSC, posterior semicircular canal.

Most cases of BPPV are idiopathic and respond well to PRMs. Some resolve without specific treatment, proving the self-limiting nature of the disease. A lack of response to at least 3 consecutive sessions of PRM within 2 weeks, or frequent recurrence of symptoms despite partial or total response to PRM, should prompt the physician to investigate the possibility of other conditions—for instance, an intracranial space-occupying lesion. Thus, of the 2 patients who did not respond to PRMs in Welling and Barnes’s series of 27 patients, 1 was found to have a glioma. Similarly, in the present series, 1 patient had 3 episodes of severe positional vertigo within 3 months, each episode resolving partially with PRMs. On computed tomographic scanning, this patient was found to have a moderate-sized meningioma of the posterior fossa. Rarely, patients with tumors of the fourth ventricle or with vestibular schwannomas present with persistent positional vertigo. The exact causal relationship between episodic BPPV and an intracranial space-occupying lesion is debated. One of the mechanisms suggested is that there is vascular compromise of the affected labyrinth as the tumor causes ischemia of the anterior vestibular artery, which leads to posterior canal BPPV. Other treatable conditions that should be excluded include local aural conditions like chronic otitis media, Ménière’s disease, or perilymph fistula. The 2 patients with Ménière’s disease in the present series had their first attack 2 weeks after starting PRM. The vertigo that appeared was found to be historically different from the characteristic positional vertigo that occurred initially. The appearance of concomitant tinnitus and aural fullness was diagnostic. Perez et al describe patients with Ménière’s disease in whom attacks of BPPV may precede, accompany, or follow the attacks of Ménière’s disease. Careful monitoring of patients who have had PRMs is therefore essential.

Once it is clear that BPPV is not improved by a particular PRM and no other treatable cause for posterior canal BPPV has been identified, an attempt should be made to try an alternative PRM or habituation procedure. It may be that cupulolithiasis, and not canalithiasis, is causing BPPV; hence, the appropriate PRM is required. Some authors describe differentiating between cupulolithiasis and canalithiasis by means of the Dix-Hallpike maneuver. In cupulolithiasis, when the head is placed in the Dix-Hallpike position, there is no latency in the onset of vertigo and nystagmus. Further, nystagmus and vertigo last as long as the head is maintained in the provoking position. In canalithiasis, however, there is usually a latency period of about 1 to 40 seconds before the onset of vertigo and nystagmus when the head is placed in the Dix-Hallpike position, and vertigo and nystagmus disappear within 60 seconds if the head’s position is maintained. When the patients of Harvey and colleagues responded neither to the PRM nor to a modified liberatory maneuver, the authors prescribed the Brandt-Daroff exercises. Occasionally, vestibular habituation therapy is more effective than PRMs. Steenerson and Cronin found that the 3 patients who had persistent BPPV following PRMs had complete relief of symptoms following institution of vestibular habituation exercises.

Some patients with BPPV have either horizontal canal canalithiasis or a combination of horizontal canal and posterior canal canalithiasis. Such patients do not attain complete relief with PRMs such as the Epley or Semont maneuver alone. Indeed, Herdman and Tusa suggest that a few patients who have been treated for posterior semicircular canal BPPV may be found to have horizontal canal BPPV after treatment. The conversion of posterior semicircular canal BPPV to lateral semicircular canal BPPV is believed to be due to the shift of otocional debris from the posterior canal to the horizontal canal via the utricle. This appeared to be the case in 2 patients in the present
series. Testing for horizontal canal involvement followed by administration of the specific PRM\textsuperscript{12} is required in these cases.

Finally, a certain proportion of patients who have persistent BPPV may benefit from surgical procedures like posterior ampullary nerve section or posterior semicircular canal occlusion. Included in this group are those with vestibular atelectasis. This entity was first described by Merchant and Shucknecht\textsuperscript{21} in a postmortem diagnosis made in individuals whose temporal bones had collapsed walls of the ampullae of the semicircular canal and utricle, causing restriction of movement of the cupula and otolithic membrane. These patients had experienced positional vertigo and persistent unsteadiness. In such patients PRMs are unlikely to be effective. The surgical procedures for BPPV are associated with a small but significant risk of hearing loss, which may be acceptable to patients with severe symptoms.

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### REFERENCES