Benign paroxysmal positional vertigo (BPPV) is the most common cause of vertigo (Bath et al., 2000). BPPV is characterized by intense, positionally provoked vertigo. The posterior semicircular canal is the most commonly involved canal, although 3–8% of BPPV is due to horizontal canal involvement (Herdman & Tusa, 1996; Korres et al., 2002). Horizontal canal benign paroxysmal positional vertigo (HC-BPPV) is elicited by positioning the patient laterally on an exam table with the dependent ear oriented, toward the ground. HC-BPPV causes the patient to exhibit a purely horizontal nystagmus that is most commonly geotropic (Appiani et al., 2001). That is, when the right ear is on the underside, the fast phase of the nystagmus beats toward the right. When the left ear is undermost, the fast phase beats toward the left. Movement of displaced otoconial debris within the horizontal canal (canalithiasis) causes geotropic nystagmus. Less commonly, ageotropic nystagmus has been reported with HC-BPPV (Baloh et al., 1995; Casani et al., 2002). In this case, the displaced otoconial debris is thought to adhere to the cupula of the horizontal canal (cupulolithiasis).

Patients diagnosed with migraine vertigo can also present with episodic positional vertigo (von Bremen et al., 2004). This has been observed in our clinic and such patients in this category have been diagnosed as having migraineous positional vertigo (MPV). Differentiation of MPV from typical cases of HC-BPPV is accomplished easily based on the type and duration of nystagmus. This is true unless the patient is actually experiencing the less common cupulolithiasis variant of HC-BPPV, which produces a persistent ageotropic nystagmus (Baloh et al., 1995; Casani et al., 2002).

Neuhauser et al. (2001) suggest that migrainous vertigo may be identified using several criteria shown in Tables 1 and 2. A patient meeting the criteria for migrainous vertigo, but with an episodic positional component, would be given a diagnosis of MPV. In this article, a case report, we suggest an additional diagnostic criterion that may assist in differentiating MPV from cupulolithiasis of the horizontal canal.

**Method**

**Case description**

A 66-year-old female presented with positional vertigo. The patient indicated that lying on her left side provoked her vertiginous symptoms, although she also reported less intense symptoms when lying on her right side. Initially, the patient experienced week-long periods of time during which this positional vertigo could not be provoked. Over a period of...
Differentiation of migrainous positional vertigo (MPV) from horizontal canal benign paroxysmal positional vertigo (HC-BPPV)

### Table 1 Criteria for probable migrainous vertigo (Neuhauser et al, 2001)

<table>
<thead>
<tr>
<th>Probable migrainous vertigo</th>
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<tr>
<td>- Episodes of symptoms including rotational vertigo, positional vertigo, other perception of motion, and/or head motion intolerance of at least moderate severity</td>
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<tr>
<td>- At least one of the following:</td>
</tr>
<tr>
<td>- migraine according to International Headache Society criteria</td>
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<tr>
<td>- migrainous symptoms during vertigo</td>
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<td>- and/or migraine-specific precipitants of vertigo (i.e., food triggers, sleep irregularities, hormonal changes, response to anti-migraine drugs)</td>
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<td>- Other causes ruled out by appropriate investigation</td>
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</table>

eleven months, the episodes transitioned into a more constant state at which time positional vertigo could be induced consistently by lying on her side. At the time of her appointment at our facility, the patient experienced symptoms whenever she was in a provocative position. Prior otolaryngologic evaluation yielded a diagnosis of Meniere's disease even though the patient had no auditory changes during her episodes of vertigo. Based on suspicion of BPPV, the patient was referred to our clinic for further vestibular evaluation and possible management. However, our case history revealed a life-long history of migraine headache with a decrease in the frequency and intensity of her headaches postmenopause.

### Examination and evaluation

Otologic history was negative with the exception of a high-frequency sensorineural hearing loss readily attributable to presbycusis. Vestibular evaluation was completed using binocular infrared videonystagulography. Standard static positional testing revealed a persistent, ageotropic nystagmus of 43 degrees per second in left lateral, and 8 degrees per second in right lateral positions. The patient reported experiencing strong, subjective vertigo with the left ear positioned downward and much less intense vertigo with the right ear downward. Oculomotor function, caloric responses, and results of Dix-Hallpike positioning were unremarkable.

### Intervention

Following vestibular evaluation, it was clear that the patient did not have posterior or anterior canal BPPV (negative Dix-Hallpike to either side). Further, the persistent ageotropic nystagmus was inconsistent with a typical transient geotropic nystagmus that is observed with most cases of HC-BPPV, due to canalithiasis, as noted above. Although it was felt that MPV was the most likely diagnosis, a cupulolithiasis variant of HC-BPPV could not be ruled out conclusively.

Horizontal canal BPPV is easily treated using the methods of Appiani et al (2001) or Casani et al (2002). The Appiani maneuver is preferred for patients with geotropic nystagmus while the Casani maneuver has been used for patients with ageotropic nystagmus. For the Appiani maneuver, the patient is initially seated on the exam table. The patient is then moved into a lateral side-lying position with the unaffected ear down and kept in this position for two minutes. This duration allows the relatively heavy otolith debris to travel through the endolymphatic fluid in the canal. The head of the patient is then rotated 45° downward. After two minutes, the patient is returned to an upright position. The Casani maneuver is quite similar to the Appiani with one key exception; the patient is moved from the initial seated position to a lateral side-lying position with the affected ear down. The head is then rotated 45° downward. After two minutes, the patient is returned to an upright position. The authors developed this maneuver to attempt to free any otoconial debris that may be adherent to the cupula of the horizontal semicircular canal.

Therefore, to provide further diagnostic information in the presently reported case, the patient was treated with HC-BPPV liberatory maneuvers. The signs and symptoms exhibited by this patient were most consistent with a cupulolithiasis form of HC-BPPV on the left side. This form of HC-BPPV is thought to be most amenable to treatment using the method described by Casani et al (2002). The patient here was treated via the Casani maneuver. Although our results were consistent with left ear involvement, we proceeded to perform a Casani maneuver on the right ear as well. Given that treatment of all BPPV, including horizontal canal involvement, is highly efficacious, it was felt that failure to eliminate the nystagmus and vertigo would provide additional support that this patient was experiencing MPV. Since medical management with pharmacological intervention is indicated for MPV an appropriate diagnosis was essential for successful management of this case.

### Outcomes

Treatment did not eliminate or ameliorate either the nystagmus or vertigo, leading to the conclusion that the patient was experiencing MPV instead of the cupulolithiasis variant of HC-BPPV. Subsequent referral and medical evaluation by a neurologist confirmed these findings. At this time, coincidentally, the patient reported experiencing a migrainous headache with a week-long bout of vertigo. The patient was placed on Topamax 25 mg b.i.d. and has not experienced an episode of vertigo in the past 22 months. She is currently managed by a neurologist.

### Discussion

The quandary in appropriate management of this patient was to differentiate MPV from HC-BPPV. The patient presented with intense episodic positional vertigo and a longstanding history of migraine, although the patient reported her migraine headaches were typically less frequent and less intense postmenopause. In addition, the patient reported experiencing headache during a

### Table 2 Criteria for definite migrainous vertigo (Neuhauser et al, 2001)

<table>
<thead>
<tr>
<th>Definite migrainous vertigo</th>
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<tbody>
<tr>
<td>- Episodes of symptoms including rotational vertigo, positional vertigo, other perception of motion, and/or head motion intolerance of at least moderate severity</td>
</tr>
<tr>
<td>- Migraine according to International Headache Society criteria</td>
</tr>
<tr>
<td>- At least one of the following symptoms:</td>
</tr>
<tr>
<td>- migraine headache</td>
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<tr>
<td>- photophobia</td>
</tr>
<tr>
<td>- phonophobia</td>
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<tr>
<td>- abnormal visual perception</td>
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<tr>
<td>- Other causes ruled out by appropriate investigation</td>
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Roberts/Gans/Kastner 225
week-long episode of intense vertigo. The patient thus readily meets the criteria for probable migrainous vertigo (see Table 1), and arguably meets the criteria for definite migrainous vertigo (see Table 2), as suggested by Neuhauser et al. (2001). Nevertheless, the cupulolithiasis variant of HC-BPPV could possibly provide similar vertiginous symptoms. Misdiagnosis would lead to inappropriate intervention and continued discomfort with poor quality of life for this patient.

Duration of symptoms
The classic positive response observed with BPPV includes a latent onset, characteristic nystagmus indicative of the involved semicircular canal, and subjective vertigo of a transient duration (Korres et al., 2002). The response is caused by the mechanical interaction of otoconial debris from the utricle and the cupula of the involved semicircular canal. Most authorities are in agreement that the duration of the response must be limited since the effect on the cupula will only occur until the debris settles into another portion of the involved canal (Korres et al., 2002; Bisdorff & Debatissé, 2001). This temporal characteristic should aid in differentiating MPV with a persistent nystagmus and typical BPPV with a transient nystagmus.

It has been reported that for cases of BPPV in which the otoconial debris actually becomes attached to the cupula (cupulolithiasis), the patient may exhibit a response with a persistent duration (Baloh et al., 1995). Patients with MPV may also exhibit a persistent response similar to the cupulolithiasis variant of BPPV, making differential diagnosis based on this factor alone difficult (Neuhauser et al., 2001; von Brevern et al., 2004).

Type of nystagmus
Another factor that may assist with differentiation of BPPV and MPV is the type of provoked nystagmus. In 90% of BPPV cases, the posterior canal is involved (Korres et al., 2002). Posterior canal BPPV is easily identified by a rotary-torsional, upbeat nystagmus toward the involved ear. This type of nystagmus is observed for posterior canal BPPV given the connection between this canal and the superior oblique and inferior rectus extraocular muscles (Honrubia & Huffman, 1997). The current patient exhibited a horizontal ageotropic nystagmus which is consistent with either BPPV of the horizontal canal due to cupulolithiasis, or with migrainous vertigo (Neuhauser et al., 2001). In the current case, Casani liberatory maneuvers were used to clear any possible otoconial debris from the canals and cupulae of both horizontal canals even though the left ear would be the most likely involved. Incidentally, it is worth mentioning that when a Casani liberatory maneuver is performed for a left ear HC-BPPV involvement, the actual maneuver is identical to performing an Appiani liberatory maneuver for a right ear involvement. So, by treating both ears with a Casani, the Appiani is also performed. Both liberatory methods are reported to be effective at clearing debris from the horizontal canal (Appiani et al., 2001; Casani et al., 2002). Appiani et al. (2001) reported successful treatment of 100% of their patients with HC-BPPV who presented with geotropic nystagmus. Casani et al. (2002) reported successful treatment of 90% of their patients with geotropic nystagmus and 75% of their patients with ageotropic nystagmus.

Possible mechanisms of migrainous positional vertigo
Although vertigo has been reported to be approximately three times more common in migraineurs than in controls (Kuritzky et al., 1981; Kayan & Hood, 1984), the underlying mechanism for this symptom remains speculative. von Brevern et al. (2004) indicate that the multiple anatomical and functional interconnections between the vestibular system and mechanisms known to be associated with migraine make the task of identifying the source of migrainous vertigo with any degree of clarity quite difficult. Such is the case with MPV. von Brevern et al. (2004) go on to state that migraine events such as vasoconstriction and spreading depression can involve cortical and brainstem structures that process vestibular signals. Von Brevern and colleagues suggest that positionally-induced migrainous vertigo (termed MPV in the current report) results from dysfunction of inhibitory fibers from the vestibulocerebellar nodulus and uvula to the vestibular nuclei.

Conclusions
The fact that both horizontal canal treatments are highly efficacious but did not resolve the symptoms of this patient provides further evidence that this was a case of MPV, not BPPV. Successful differentiation led to appropriate referral and management of the patient. We suggest that in cases when MPV must be differentiated from typical BPPV, symptom duration and type of nystagmus should be considered. When HC-BPPV must be differentiated from MPV, incorporation of the treatment proposed by Appiani et al. (2001) for cases with geotropic nystagmus, or by Casani et al. (2002) for ageotropic nystagmus may facilitate diagnosis, thereby leading to appropriate patient management.

References