J Am Acad Audiol 17:69–80 (2006)

Differentiation of Ménière's Disease and Migraine-Associated Dizziness: A Review

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Abstract

A significant body of literature exists demonstrating a relationship between migraine disorders and dizziness. In the characterizations of the migraine-associated dizziness, the signs and symptoms show overlap with those characterizing Ménière's disease. There has been literature, beginning with Ménière himself, suggesting a relationship between Ménière's disease and migraine-associated dizziness. This implicates a possible underlying link in pathogenesis. This review article presents a discussion of the overlap in signs and symptoms between the two disorders. Suggestions for differentiating between the disorders based on recent literature protocols and use of test results are also discussed. Vestibular and balance rehabilitation programs have a role in both of the disorders but differ in the overall management aspects of the disorders. The distinction in the use of vestibular rehabilitation is discussed.

Key Words: Dizziness, Ménière's disease, migraine, vertigo

Abbreviations: BPPV = benign paroxysmal positional vertigo; CEOAE = click-evoked otoacoustic emissions; DPOAE = distortion-product otoacoustic emissions; ENG = electronystagmography; HIS = International Headache Society; MAD = migraine-associated dizziness; MD = Ménière's disease; VEMP = vestibular evoked myogenic potential; VBRT = vestibular and balance rehabilitation training

Sumario

Existe una significativa cantidad de material en la literatura demostrando una relación entre los trastornos migrañosos y el mareo. En la caracterización del mareo asociado a la migraña, los signos y síntomas muestran un traslape con aquellos que caracterizan la enfermedad de Ménière. Han existido reportes, comenzando con el propio Ménière, sugiriendo una relación entre la enfermedad de Ménière y el mareo asociado a la migraña. Esto sugiere un posible vínculo subyacente en la patogénesis. Este artículo de revisión presenta una discusión sobre el traslape de síntomas y signos entre estos dos trastornos. Se discuten también sugerencias para diferenciar entre estos trastornos con base en protocolos recientes de la literatura y el uso de resultados de pruebas. Los programas de rehabilitación vestibular y del equilibrio juegan un papel en ambos tipos de trastornos, pero difieren en los aspectos generales de manejo. Se discuten la caracterización en el uso de la rehabilitación vestibular.

Palabras Clave: Mareo, enfermedad de Ménière, migraña, vértigo

Abreviaturas: BPPV = vértigo de posición paroxístico benigno; CEOAE = emisiones otoacústicas evocadas por clic; DPOAE = emisiones otoacústicas por productos de distorsión; ENG = electronistagmografía; HIS = Sociedad Internacional de Cefaleas; MAD = mareo asociado a migraña; MD = enfermedad de Ménière; VEMP = potencial miogénico evocado vestibular; VBRT = entrenamiento en rehabilitación vestibular y del equilibrio

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The symptoms of "dizziness," including spontaneous vertigo and motion-provoked vertigo, imbalance, disorientation, or disequilibrium in association with headache, more specifically migraine, have been reported for an extensive interval of time beginning in 100 AD as a description by Aretaeus of Cappadocia (Sacks, 1970) and followed in the neurology and otolaryngology literature by numerous authors (Living, 1873; Atkinson, 1943; Toglia et al, 1981; Kayan and Hood, 1984; Neuhauser et al, 2001; Furman et al, 2003). Epidemiology studies demonstrate a significantly greater prevalence of vestibular symptoms in populations of migraineurs (Kayan and Hood, 1984; Bayazit et al, 2001), and a significantly greater incidence of migraine in patients with dizziness complaints than is found in the general unscreened population (Neuhauser et al, 2001). More germane to this discussion is the reported high incidence of migraine in patients diagnosed with Ménière's disease, suggesting a possible common link between Ménière's and migraine (Ménière, 1861; Atkinson, 1944; Radtke et al, 2002). There is higher prevalence of Ménière's disease in a population screened for migraine compared to the incidence in the general population (Hinchcliffe, 1967; Kayan and Hood, 1984).

Descriptions of the symptoms in migraine-associated dizziness (MAD) include tinnitus, perceived fluctuant hearing (unilateral and bilateral), spontaneous spells of vertigo that can last from seconds to days, motion intolerance and motion-provoked vertigo with lightheadedness (Cutrer and Baloh, 1992; Cass et al, 1997; Radtke et al, 2002; Reploeg and Goebel, 2002). The studies vary as to the percentage of patients presenting a repeated temporal relationship between headache and the symptoms of vertigo or disequilibrium. However, it is clear from the studies that a regular temporal pattern is the exception not the rule. It is reported that from 30 to greater than 50% of the patients have their symptoms of dizziness inconsistently associated with a headache or completely independent of head pain (Kayan and Hood, 1984; Cutrer and Baloh, 1992; Cass et al, 1997; Johnson, 1998; Reploeg and Goebel, 2002). The frequency of abnormal test findings on electronystagmography (ENG), rotational chair, and posturography is summarized by Furman and colleagues (2003). Their review covering a number of studies indicates an average of 9.9% of 534 patients with spontaneous nystagmus. Fifty-four percent of the 534 patients had abnormal asymmetry on rotary chair. In another review of multiple individual studies, Battista (2004) reports the finding of sensorineural loss of auditory sensitivity in patients diagnosed with MAD. In general, the losses of sensitivity were typically in the mild-to-moderate range and of a variety of configurations.

Estimates of the overall prevalence of migraine in the general population range from 18 to 25% (Stewart et al, 1992; Radtke et al, 2002). The disorder is indicated to be significantly more frequent in females and most common between the ages of 30 and 50 years. However, estimates of lifetime prevalence of migraine disorders in a general dizziness population is 38% (Neuhauser et al, 2001). Given a prevalence of vestibular symptoms in migraine patients as high as 54.5% (Kayan and Hood, 1984), it is highly likely that practitioners who manage patients with complaints of dizziness and balance disorders will encounter a patient with migraine-associated dizziness. A prospectively designed study determined the lifetime prevalence of migraine in well-defined Ménière's disease patients to be 56% compared to a 25% prevalence in an age-/gender-matched control group (Radtke et al, 2002). Therefore, it would be likely if a clinic handles a large percentage of Ménière's disease patients to have migraine disorder mixed in with the group.

The potential problem that presents with the issue of MAD and Ménière's disease (MD) can manifest itself in two ways:

1. Differentiating early MD in a patient who has well-defined migraine disorder that could be causing spells of spontaneous vertigo, tinnitus, and perceived fluctuant hearing loss with or without mild sensorineural hearing loss.
2. Determining which disorder is the cause of current symptoms not responding to conservative MD treatment in a patient that has well-defined MD and likely MAD (definite migraineur).

In both of these dilemmas the distinction between MD and MAD is of importance as the management options are different for the two disorders (Boismier and Disher, 2001;

Thakar et al, 2002).

DEFINITIONS

For Ménière's disease the American Academy of Otolaryngology—Head and Neck Surgery Committee on Hearing and Equilibrium (1995) developed a revised set of characteristics that allow for the diagnosis of MD in the following categories: "certain," "definite," "probable," and "possible." In clinical practice the categories of "certain" and "definite" should be used to report an individual patient with Ménière's disease. "Probable" or "possible" Ménière's disease classifications are intended for use in group studies of the disorder. These criteria are widely used in the diagnosis of patients with Ménière's disease, especially in the reported scientific/clinical trials literature.

This same situation does not exist for the diagnosis of MAD (also known in the literature as "benign recurrent vertigo," "vestibular/vertiginous migraine," and "migraine-related vestibulopathy"). To begin to suggest a migraine-associated dizziness condition, one must first determine whether the patient has a migraine condition. A complete description of all the characteristics of migraines and the theories of the mechanisms that produce the various aspects is beyond the scope of this article, but a brief description is appropriate. Migraines are neurological events with the most common manifestation being that of a significant, unilateral headache associated with increased sensitivity to normal daily stimuli such as light and sound. That said, migraines can range from no pain to a form of dangerous, severe throbbing pain. The most common form of migraine event without pain involves visual hallucinations that can take the form of lightening bolts, jagged lines, gold/silver sparkles, or central visual areas that are opaque (central scotomas), and that last from 2–30 minutes in a classic format (Evans, 2003; Furman et al, 2003). Other reversible neurological events in addition to the visual occurrences such as paresthesia, numbness, muscle weakness, tinnitus, fluctuations in hearing, and various forms of spontaneous and head-motion-provoked vertigo, lightheadedness, and disorientation may occur (Evans, 2003; Furman et al, 2003). Collectively, these reversible neurological events are all referred to as "auras." When they present just before or during the head pain phase of the migraine, the condition is called "migraine with aura." If the headache takes place in the absence of an aura, it is termed "migraine without aura." When the aura occurs in the absence of the pain phase, the classification is "migraine aura without pain." The list of classifications of different forms of migraines continues with each form having specific characteristics that distinguish one form from another. The original classification system from the International Headache Society (IHS) (Olesen et al, 1988) was used by Neuhauser et al (2001) to develop proposed definitions of MAD. This classification system has been recently revised by the IHS (Olesen et al, 2004; found at http://216.25.100.131/upload/CT_Clas/ihc_II_main_no_print.pdf on the IHS general Web site). The distinctions between the new and older classification systems are not critical for the subject matter being discussed in this review. However, it is important to point out that in the second edition of the classification system, MAD was again not recognized as a migraine variant. The second edition classification, like the first, only recognizes two forms of migraine that have "dizziness" as a feature. The two types are the Basilar-type migraine and Paroxysmal Vertigo of Childhood (a migraine precursor). Therefore, the structure suggested by Neuhauser et al (2001) for a definition of MAD remains a critical step in the recognition of dizziness associated with migraines of classification other than the two indicated above. Also of importance is the realization that migraine headaches are a disorder diagnosed by the specific characteristics of the headache via history. There are no tests to determine migraine. The line of questioning used to determine if the headaches or other neurological events the patient experiences are migraines come directly from the characteristics given in the IHS classifications. An example of this schema (Olesen et al, 1988) as used by Neuhauser et al, (2001) is given in Table 1, providing the characteristics of "migraine without aura" (these have not changed in the second edition of the classification system by Olesen et al, 2004). It is against this basic set of characteristics that many other forms of migraine in the classification system are based. The interested reader is referred to Tusa (2000) for a basic introductory chapter

Table 1. International Headache Classification of "Migraine without Aura" (older term was "common migraine")

- At least five headaches in your lifetime meeting the characteristics listed in 1–3 below:
  1. Length of the headache if untreated or treated unsuccessfully: 4 to 72 hours (2 to 48 hours in children <15 years of age)
  2. The headache carries at least two of the following characteristics:
     - ❑ Unilateral location
     - ❑ Throbbing or pulsating quality
     - ❑ Intensity level of pain that inhibits or prohibits continuation of daily activities
     - ❑ Exacerbation of symptoms by routine physical activity such as walking stairs
  3. At least one of the following should occur during the headache:
     - ❑ Photophobia and/or phonophobia
     - ❑ Nausea and/or vomiting
- One of the following must be met in addition to the above:
  1. History and physical examinations DO NOT suggest any other disorder to adequately explain the reported symptoms.
  2. History and physical examinations DO suggest other disorders that may explain the reported symptoms but have been ruled out by appropriate laboratory investigations.

on migraine and the association with dizziness.

Once it has been determined that the patient is a migraineur, the next step is to define the entity of MAD. Neuhauser et al (2001), through a prospective controlled study of migraine patients reporting dizziness defined a set of criteria that could be used to diagnosis migrainous vertigo (referred to in this article as MAD). The criteria put forth can be used for the diagnosis of definite versus probable MAD. Other authors have begun to use and recommend the use of these criteria as a consistent means of recognizing patients with dizziness and disequilibrium that is caused by a migraine disorder (Furman et al, 2003; Battista 2004). Since both MD and MAD are disorders diagnosed primarily on the basis of history and minimally by objective tests in the case of MD, having well defined criteria for the recognition of MAD and MD is of significant clinical assistance. Table 2 presents the criteria for MAD from Neuhauser et al (2001). Furman et al (2003) suggests the use of a structured interview incorporating the criteria presented by Neuhauser et al (2001). This logical questioning provides a systematic approach to determining if MAD should be in the patient's differential diagnosis.

In the older literature, a variant of Ménière's disease termed "Vestibular Ménière's" was purposed (Paparella and Mancini, 1985). The symptom presentation was very similar to that of both MD and MAD but with no loss of hearing sensitivity. Tinnitus and aural fullness was described as part of the symptom complex (Paparella and Mancini, 1985). This variant of Ménière's is no longer recognized by the current guidelines for Ménière's disease (American Academy of Otolaryngology—Head and Neck Surgery Committee on Hearing and Equilibrium, 1995). Others suggest that most likely this variant of MD was actually undiagnosed MAD (Rassekh and Harker, 1992; Neuhauser et al, 2001).

USE OF AUDIOMETRIC AND VESTIBULAR FUNCTION TESTS

In attempting to differentiate between migraine and Ménière's in the early stages of a diagnosis when a hearing loss may be either absent or only very mild, or when the two conditions coexist in a patient like that of the above example, are the audiometric and various vestibular function tests of use? As summarized by Furman et al (2003), the types of abnormalities seen during ENG, rotational chair testing and postural control assessment are no different than those noted

Table 2. Criteria for Definite and Probable Migraine-Associated Dizziness as Suggested by Neuhauser et al (2001)

DEFINITE MIGRAINE-ASSOCIATED DIZZINESS ("migrainous vertigo" was the term used in the Neuhauser et al [2001] publication)—each of the following four criteria must be met:

1. Migraine diagnosed by International Headache Society (HIS) criteria
2. Moderate-to-severe (intensity defined below), episodic symptoms of vestibular involvement including any of the following or combinations of the following:
   a. Spontaneous or positional rotational vertigo
   b. Other spontaneous or positional symptoms of self or object motion, not rotational vertigo (e.g., linear movements, falling, drifting, rocking, etc.)
   c. Head-movement-provoked symptoms of general intolerance, imbalance, self or object motion of a rotational vertigo, or nonrotational vertigo form
3. A minimum of one of the following migraine symptoms occurring during a minimum of two of the patient's reported episodes of "dizziness":
   a. Photophobia
   b. Phonophobia
   c. Visual or other auras (not including dizziness as one of the auras)
4. Rule out of other possible disorders that could explain the patient's reported symptoms of dizziness.

PROBABLE MIGRAINE-ASSOCIATED DIZZINESS (for patients not meeting the criteria for definite, but still considered as possible candidates for the diagnosis of MAD)—each of the following three criteria must be met:

1. Moderate-to-severe (intensity defined below), episodic symptoms of vestibular involvement including any of the following or combinations of the following:
   a. Spontaneous or positional rotational vertigo
   b. Other spontaneous or positional symptoms of self or object motion, not rotational vertigo (e.g., linear movements, falling, drifting, rocking, etc)
   c. Head-movement-provoked symptoms of general intolerance, imbalance, self or object motion of a rotational vertigo, or nonrotational vertigo form
2. Patient has one of the following:
   a. Migraine diagnosed by IHS criteria
   b. Migraine symptoms during at least two of the spells of "dizziness" (see above for list of symptoms qualifying)
   c. Two or more of the spells of "dizziness" that have responded to the use of antimigraine medications OR two or more spells provoked by migraine-specific triggers including any of the following:
      i. Particular foods or beverages
      ii. Hormonal changes
      iii. Sleep disruptions
      iv. Sudden exposure to bright light

Intensity of the "dizziness" for the purpose of these criteria is defined as:
Mild—no interference with routine daily activities.
Moderate—interfere with routine daily activities but do not prohibit their execution.
Severe—prohibit the execution of the routine daily activities.

in a variety of peripheral labyrinthine disorders. Of note abnormal asymmetrical caloric irrigation testing was reported in an average of 24.5% (ranging from 8% to 60%) of the 534 patients across the studies reviewed by Furman et al (2003). The magnitude caloric asymmetry has not been reported in the literature. Only the number of patients exceeding a criterion for abnormal asymmetry over a range of 22–30% reduced vestibular response (RVR) has been given when details were provided (Cutrer and

Baloh, 1992; Cass et al, 1997; Reploeg and Goebel, 2002). Therefore, it is difficult to use magnitude of reduced unilateral vestibular response as a criterion to help in the differentiation between the disorders. However, the explanation provided for mechanisms that could produce asymmetrical performance at the level of the periphery (Cutrer and Baloh, 1992) would not be likely to support a significant RVR of greater than a 40–50% magnitude, especially during an interval when migraine activity was not immediately apparent. The remainder of the parameters typically obtained on an ENG related to the strength of spontaneous and positional nystagmus is also not reported to be significantly different between MD and MAD. What would not be expected with a classic MD patient on ENG are ocular motor abnormalities suggestive of more central vestibular system involvement in brainstem or cerebellum. This is reported with varying prevalence in MAD patients related to impaired pursuit tracking or Optokinetic testing (Cass et al, 1997; Dieterich and Brandt, 1999).

Rotational chair abnormal asymmetry (sometimes referred to as a directional preponderance) is reported as a more frequent abnormality in MAD patients compared to the abnormal results seen on ENG (Cass et al, 1997; Furman et al, 2003). The most common combination of abnormal findings for the MAD patients is an RVR and rotational chair asymmetry. In an attempt to use the frequency and character of abnormalities related to rotational chair and caloric testing to differentiate between MD and MAD, Dimitri et al (2001) used multivariate statistics to improve the utilization of routine laboratory findings. In their work, the combination of rotational chair gain, time constant, asymmetry, and caloric RVR in an independent quadratic classification algorithm for discriminant analysis was able to achieve a 91% rate of correct differentiation between the well-defined MD and MAD patients compared against a control group. To make the criteria for the diagnosis of MAD clear, no patients with migraine aura without headache (IHS Criteria) were included. While showing promising results, the extensive analytical treatment of the routine data may limit use in other than sophisticated balance center environments.

Unlike the routine vestibular tests of ENG, rotational chair, and posturography, the audiometric results in MD and MAD patients appear more distinctive over the progression of the disorders. When the results of hearing evaluations was summarized over seven different studies, plus their own results, sensorineural losses were found in an average 7.5% of 479 patients diagnosed with MAD (Battista, 2004). Aside from the much lower frequency of hearing loss in the MAD group, the prominent distinguishing feature of the MD patients is that of documented fluctuation of the hearing loss and progression of the loss over time. While this differentiation between the MAD and MD patient is quite distinct, it requires time to develop. Therefore, in the early stages of onset of MD, the hearing loss may not have declared itself to an extent that would allow for this distinction, and following the natural history of the disorder in a particular patient may be needed before final diagnosis can be made. As illustrated in the case example, when both disorders exist in the same patient, the issues surrounding hearing loss become less useful.

The use of click-evoked otoacoustic emissions (CEOAEs) and distortion-product otoacoustic emissions (DPOAEs) have both been investigated in MD patients. While the abnormalities seen were distinguishable from normal-hearing individuals, they were no different than the findings of other persons with hearing loss of other than MD origin (de Kleine et al, 2002). While no report of CEOAEs or DPOAEs in MAD patients was found, it is not likely that this tool would be of assistance in the differentiation between the disorders.

Electrocochleography utilizing the summating potential/action potential (SP/AP) ratio has been promoted for years as an investigative diagnostic test to assist in the diagnosis of Ménière's disease via identification of the condition of endolymphatic hydrops (Gibson et al, 1977; Chung et al, 2004). The implicit assumption in the use of the test for identification of MD is that there is a cause and effect relationship between endolymphatic hydrops and MD or that all cases of endolymphatic hydrops lead to clinically recognized MD. This assumption has been challenged on both an experimental and theoretical basis in the past (Ruckenstein and Harrison, 1998) and more recently via strong evidence from temporal bone studies (Merchant et al, 2005). The results of these

challenges is that endolymphatic hydrops, while an appropriate histological marker for Ménière's disease when appropriate clinical signs and symptoms were also present in the patient under review, it should not be considered in a cause and effect manner but a phenomenon caused by the source for MD. To add to the difficulty in the use of ECoG in the identification of MD are performance figures of sensitivity/specificity that vary from 20–95% for click stimuli (Campbell et al 1992) to 71%–96% (Chung et al, 2004) dependent on the clinical criteria used for determination of abnormal SP/AP ratio. If one was to apply the value of SP/AP ratio of >0.43 for click-evoked, extra-tympanic ECoG as recommended from a meta-analysis study by Wuyts et al (1997), sensitivity figure for most reported data would fall below the 75% figure. In patients with MAD, Johnson (1998) reported that five of eight patients tested with ECoG had positive findings. Therefore, while ECoG can be useful when taken in the context of other clinical findings and history, it is not one that would significantly assist generally in discriminating MD from MAD. If techniques to be able to recognize endolymphatic hydrops via imaging studies of MRI continue to progress, these together with the electrophysiologic data from ECoG may be able to provide an objective indication in the differentiation of the two disorders (Niyazov et al, 2001).

Vestibular evoked myogenic potential (VEMP) is a test using an intense, brief auditory stimulus that provides for an assessment of the saccule ipsilateral to the stimulus (Colebatch and Halmagyi, 1992). Interest in this objective measure as a means of assessing the saccular otolith organs has generated a significant increase in research activity associated with a large number of clinical entities. In particular, since Ménière's disease presents a histological picture of cochleosaccular hydrops, the effects of MD on these evoked potentials and the use of the VEMP to monitor or differentiate MD from other disorders has received attention (Rauch et al, 2004). Using toneburst stimuli, a threshold response tuning curve for frequencies from 250–1 kHz has been used to compare responses in MD patients to normal subjects (Rauch et al, 2004). This work has shown specific changes in the elevation of threshold at the most sensitivity frequency (between 500 and 1 kHz), and a widening of the threshold response curve in the MD ear versus a normal control group, and the MD patient's uninvolved ear. An additional finding was that the threshold response curve in the uninvolved ear of the MD patients also showed abnormalities when compared to the normal subjects. The implication is that if the changes in the response curve are unique to MD, then VEMP may serve as a diagnostic tool for the identification of MD, and given the results in the uninvolved ear, may suggest ears that are in some manner predisposed to the future development of MD (Rauch et al, 2004). If this line of work continues to progress in this fashion and contingent on VEMP results in MAD patients, this study may prove to be quite useful in the differentiation of MD and MAD patients. A recent prospective study of 20 Basilar-type migraine patients (by IHS criteria) indicated absent or delayed VEMP responses in 50% of the patients, all of whom also complained of vertigo (Liao and Young, 2004). After successful pharmacological treatment resulting in relief of both headaches and vertigo, the ten patients with abnormal pretreatment VEMPs were retested. Nine of these patients had their VEMP responses return to normal. While a specific pattern of abnormality in the VEMP response was not indicated, the recognition of aberrant VEMP findings and the ability to reverse those findings with migraine treatment present future possibilities for differentiation of MAD from MD.

In summary, the various audiometric and tests of vestibular function provide only modest assistance in differentiating between MD and MAD with tracking of hearing, over time, the strongest single indicator in the context of the historical features for initial diagnosis. Clearly, findings on ENG/rotary chair, EcoG, and possibly VEMP testing will help reinforce an initial diagnostic conclusion in different ways for a given patient. However, there does not appear to be a "typical" pattern of test results (other than progressive hearing loss) that differentiates the two disorders in the initial diagnostic stages. In the situation like that of the case example, the studies have little to provide that would differentiate which of the coexisting disorders is responsible at a given point in time for an exacerbation in symptoms. In this second dilemma the presenting features of the spells of vertigo together with diagnostic treatment

trials like that described lead the management process.

VESTIBULAR AND BALANCE REHABILITATION PROGRAM (VBRT) UTILITY

As illustrated in the case example, there are isolated roles for the use of VBRT in the management of MD. The driving force for use of a VBRT program is that it is symptom driven and as a result not seen as useful in the management of spontaneously occurring events of vertigo or vaguer symptoms of "dizziness" (Shepard and Telian, forthcoming). The program would find its most frequent use following aggressive, destructive surgical or chemical procedures when the spontaneous spells of vertigo have been controlled and residual symptoms of head-movement-provoked symptoms of vertigo, lightheadedness, and imbalance continue. Additionally, BPPV (benign paroxysmal positional vertigo) can present as a secondary event to other disorders that result in damage to the labyrinth including MD (Herdman and Tusa, 2000; Karlberg et al, 2000; Perez et al, 2002). BPPV is also more frequently associated with migraine than seen in the general population (Gizzi et al, 1998; Tusa, 2000). The treatment of BPPV and its residual symptoms by VBRT is very effective even if active MD or MAD is continuing. While infrequent, the use of VBRT techniques to reduce symptoms of head-movement sensitivity, gaze stabilization, and imbalance that is persistent between spontaneous events can be effective if the spontaneous events are two or more months apart. This time interval is suggested as a guide since most VBRT programs for stationary peripheral vestibular lesions achieve their major effects within six to eight weeks (Shepard and Telian, 1995).

It is of importance for the clinician to realize that VBRT also has a positive impact on patients with MAD. Therefore, the effectiveness of this type of management course would not be suitable as a differentiator between the two disorders. Studies on the application of VBRT to patients with MAD and those with vestibular and balance disorders unrelated to migraine (other than MD) but with concurrent history of migraine, show improvement on both objective and subjective measures, but do so to a lesser extent than the control patients with vestibular and balance disorders who have no history of migraine (Whitney et al, 2000; Wrisley et al, 2002).

CASE STUDY

The following case illustrates the problem of MD and migraine in the same patient and the dilemma that occurs in differentiating whether MD was actively causing her symptoms following a period of control, or whether it could be MAD was the origin of the current attacks of spontaneous vertigo. The patient is a 58-year-old female that reported the following history on her initial clinic visit.

- She had experienced a vestibular crisis style event 12 years prior to this clinic visit with vertigo, nausea, and vomiting slowly changing from continuous to head-movement provoked over seven days. Over the subsequent two months her symptoms had improved to minor symptoms provoked only by pitch plane (vertical) movements of her head. She denied any auditory symptoms at the time.
- One year prior to this initial clinic visit, she began having spontaneous spells of vertigo with nausea and vomiting lasting one to six hours. These had been as frequent as one time per week, but she had no spells reported for the six weeks prior to the initial visit.
- Tinnitus, aural fullness, and fluctuant hearing were all reported in the left ear starting with the onset of the spells of vertigo. A temporal relationship between increased tinnitus and decrease in hearing sensitivity just prior to each of the spontaneous events was noted.
- Her past medical history was significant for migrainous headaches with aura (by International Headache Society criteria), with the condition improving as she had progressed into the later stages of menopause.
- Family history was positive for migraine headaches temporally associated with spells of vertigo in her son. She reported that her mother had been born deaf

bilaterally.

- She presented with a contrast MRI study that was well within normal limits for the cerebellar pontine cistern region. Balance function studies inclusive of ENG and postural control assessment were normal (Shepard and Telian, 1996). Audiometric evaluation showed a left side moderate-to-severe, sensorineural, low-frequency hearing loss that was shown over several studies to fluctuate in the low frequencies. A mild flat sensorineural loss of sensitivity was noted on the right. Speech recognition testing was 92% on the right and 68% on the left.
- Direct clinical examination (Walker and Zee, 2000) gave normal findings excepting a positive left Hallpike indicating left posterior canal BPPV canalithiasis.

Working diagnosis for this patient was:

- Most likely a vestibular neuritis event at the onset of symptoms with development of recurrent benign paroxysmal positional vertigo (BPPV) indicated by history.
- Currently active left side posterior semicircular canal BPPV of a canalithiasis type.
- Ménière's disease versus migraine aura without pain (IHS criteria) as the cause for the spontaneous events of vertigo that had been occurring prior to the last 6 weeks. Ménière's was considered more likely given a recent improvement in her migraine headaches and the degree of hearing loss together with the temporal relationship between the auditory symptom exacerbations and the spells of vertigo.

Initial treatment recommendations were:

- Use of Canalith repositioning maneuver with home self-maneuvers for treatment of left BPPV.
- Use of general vestibular and balance rehabilitation therapy program (VBRT) for mild residual imbalance continuing in association with her BPPV.
- Continuation on conservative medical management for Ménière's disease of a low sodium diet and mild daily diuretic. Since the spontaneous spells had gone into resolution, watchful waiting was suggested.
- Continuation of dietary changes to reduce the triggers for migraine (primarily the elimination in her diet of caffeine, chocolate, red wine, and smoked meats).

At her two-week and then four-week follow-up visits, the left BPPV was resolved, and the general imbalance with standing and walking was resolved. She continued to be in a condition of resolution regarding the spontaneous spells of vertigo; however, she was persistent with documented fluctuations in hearing, tinnitus, and aural fullness on the left.

She returned in six months with reported recurrence of the spontaneous spells of vertigo lasting hours multiple times per month. At the same time, she had a significant recurrence of migraine headaches one to two times per week, but not temporally related to the spells of vertigo. The auditory symptoms were no longer related to the occurrence of a spell but continued with fluctuations. Because of the simultaneous reappearance of migraine headaches, the spells of vertigo and the report that she was experiencing photophobia with some of the vertiginous events (a migraine symptom that is not associated with Ménière's disease), there was a strong possibility that the current spells of vertigo were migraine in origin and not an exacerbation of her Ménière's disease. Since the next step in Ménière's treatment would be an aggressive, destructive approach with transtympanic gentamicin injections, it was decided that aggressive treatment for migraine would be a more reasonable management path to determine the source for the recurrence in symptoms. A combination of strict dietary changes for reduction in migraine triggers and the use of a prophylactic migraine medication were used over the subsequent two months. This treatment course successfully eliminated her headaches. However, no changes occurred in the characterization of the spells of vertigo other than an increase in frequency to onetime per week. It was concluded that the vertiginous events were related to her Ménière's disease since the control over the migraine condition did not produce an effect in the control of the vertigo. Consequently, she underwent

transtympanic gentamicin treatment over the course of three weeks with complete resolution of her spontaneous spells of vertigo.

As with most patients treated successfully with gentamicin injections, they are then left with head-movement-provoked vertigo or lightheadedness and imbalance. She was placed back into the VBRT program with a combination of exercises to be used to help with gaze stabilization and symptoms provoked by head movement and the general imbalance. At her three-month follow-up and subsequent 12-month return visit, she was well controlled for migraine headaches and was free of spontaneous spells of vertigo.

This case illustrates several points critical to the issue of overlap of the disorders of migraine and Ménière's disease. First, as indicated above, the literature and especially the work by Radtke et al (2002) suggest that the situation just described, where the two conditions coexist in a single patient, is not that unusual. Second, while evidence to date in the literature would support the magnitude of this patient's hearing loss as clearly related to her Ménière's disease, the source for the spontaneous spells of vertigo could have been either condition or a combination. At the historically reported start of the spontaneous spells, their characteristics with temporal relationship to hearing changes and no other unusual symptoms appeared to be more likely Ménière's disease in origin. The exacerbation of spontaneous spell temporally related to the re-appearance of significant migraine headaches and lack of temporal relationship to hearing changes are features that begin to suggest that migraine or a combination of the disorders may be the source of the spontaneous events. Third, the presence of migraine symptom of photophobia during the spells of vertigo is another of the factors highly suspicious for the vertigo being of migraine origin. Finally, when faced with the possible use of aggressive treatment options for Ménière's disease, which are not reversible, it is critical that the origin of the spontaneous spells of vertigo be labyrinthine. In a situation where either migraine or Ménière's may be causing the symptoms, aggressively treating migraine to secure a change in not only migraine headache symptoms but also the vertigo was the more conservative approach. When changes in migraine headache were clearly evident and no changes occurred in the spells of vertigo, then it was rational to precede in an aggressive manner with the Ménière's treatment options given the failure of the conservative management for Ménière's.

SUMMARY

The significantly greater incidence of migraine in a population of Ménière's disease patients, and the greater incidence of complaints of "dizziness" and MD in a migraine population than is seen for either in the general, unselected population has caused many to suggest the possibility of a common link in the pathogenesis between migraine and Ménière's disease. From a clinical perspective in the diagnosis of migraine-associated dizziness versus Ménière's disease, significant confusion can result given the large overlap in symptoms and clinical test findings produced by both disorders. The realization that it is not unlikely to have both conditions coexist in the same patient produces a different clinical dilemma in deciding on the management path based on which disorder may be the cause of the symptoms being expressed at that point in time. The following would be suggestions for assistance in resolving the differentiation between MD and MAD in the two situations discussed:

- The use of a consistent and verifiable definition of MAD is important for consistency and comparison across facilities and clinical trials regarding diagnosis and treatment (see Neuhauser et al, 2001, table 2). This provides the criteria for MAD to be considered as part of the differential diagnostic options. Other aspects of the patient's presentation are needed to push the diagnosis toward this entity or to MD.
- In the patient's history, the report of only very short (seconds to <15 minutes) or prolonged (>24 hours) spontaneous spells of vertigo are more likely the work of migraine rather than Ménière's disease. Also if the spontaneous spell of vertigo is associated with migraine features like photophobia, paresthesia, visual disturbances other than blurring (scintillating lights, visual hallucinations, and other descriptors consistent with visual auras),

migraine as the source for the vertiginous events is again likely.

- For MD there should eventually be a documented hearing loss that is progressive in nature.
- In MAD while audiometric and vestibular test findings can be found, they are typically mild in magnitude and more likely to be stable over time rather than fluctuant.
- When there is a strong possibility that MAD may be at the source of the patient's complaints, even though MD has not been ruled out, the use of conservative treatment for migraine as a diagnostic trial may be useful. If controlling other indicators of migraine controls the dizziness, then MAD is likely.
- As in the case example, when both migraine and MD coexist and aggressive treatment is indicated, unless it is very clear that MD is the source of the exacerbations, starting first with aggressive approach to migraine presents a more conservative approach.

MAD is a diagnosis of exclusion based on the historical diagnosis of the patient having a migraine disorder that could be causing the dizziness.

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