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Migrainous Vertigo

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Most neurologists are aware that Alfred Hitchcock's *Vertigo* centers around a woman who has height-induced agoraphobia, but consider the diagnosis in Paul Auster's *Mr. Vertigo*, the story of a 14-year-old boy who could walk on air.

> I felt a little light-headed, but it seemed that the crisis had passed. But then I stood up and it was precisely then that the headache returned, ripping through me with a blast of savage, blinding pain. I tried to take a step, but the world was swimming, undulating like a belly dancer in a fun-house mirror, and I couldn't see where I was going. By the time I took a second step; I had already lost my balance. If the master hadn't been there to catch me I would have fallen flat on my face again [1].

That migraine may manifest with attacks of vertigo has been known since the early days of neurology [2,3], but systematic studies have been undertaken only in the past two decades, starting with Kayan and Hood's classic paper [4]. The clinical features of the syndrome have been well delineated in several large case series [5–14], but knowledge of the pathophysiology and treatment of migrainous vertigo (MV) is scarce. As MV is an evolving entity, it is not surprising that terminology is confusing and that generally accepted diagnostic criteria are lacking. Various terms, including migraine-associated vertigo, migraine-associated dizziness, migraine-related vestibulopathy, vestibular migraine, benign recurrent vertigo, and basilar migraine, all have been applied to approximately the same patient population. The term, basilar migraine, however, should be

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restricted to patients who fulfill the diagnostic criteria of the International Headache Society (IHS) [15] for basilar migraine (see later discussion).

Dizziness and vertigo rank among the most common complaints in the general population and frequently are reported by patients who have migraine. Therefore, clinicians must determine if individual patients have MV (vertigo that is caused by migraine) or just a coincidence of migraine and dizziness of an unrelated cause. Somewhere in the middle are several vestibular and nonvestibular dizziness syndromes that statistically are related to migraine, even if a pathophysiologic link is not established, including Meniere's disease (MD), benign paroxysmal positional vertigo (BPPV), motion sickness, and orthostatic hypotension [16]. This article first addresses the core syndrome of MV and then the associated dizziness syndromes.

Epidemiologic associations of migraine and vertigo

Approximately 16% of the adult population is affected by migraine at some time in their lives [17] and the lifetime prevalence of dizziness (comprising vertigo and nonvestibular dizziness) is found to be 23% in a large population-based survey [18]. Thus, approximately 3% to 4% of the general population is expected to have migraine and dizziness by pure coincidence. There is evidence, however, that migraine and dizziness concur more often. In a recent study, the prevalence of migraine according to the criteria of the IHS [15] was 1.6 times higher in 200 patients in a dizziness clinic than in 200 age- and sex-matched controls in an orthopedic clinic (38% versus 24%, $P < 0.01$) [5]. Among patients who had unclassified or idiopathic vertigo, the prevalence of migraine is elevated [6,7,19]. Conversely, 53 of 200 unselected patients who had migraine reported vertigo, compared with 9 of 116 patients who had tension headache (27% versus 8%, $P = 0.01$) [4]. The association between migraine and vertigo was significant for vertigo with headache and for vertigo in the headache-free period. In another study, patients who had migraine reported approximately 2.5 times more vertigo and approximately 2.5 times more dizzy spells during the headache-free phase than controls who were not suffering from headaches [20]. In summary, the available data indicate a more than chance association of migraine with vertigo and dizziness.

Diagnostic criteria

Migrainous vertigo and the International Headache Society classification of migraine

Vertigo is not included in the IHS classification as a migrainous symptom in adults except in the framework of basilar migraine [15], which involves

vertigo in more than 60% of patients [21]. As an aura symptom of basilar migraine, vertigo should last between 5 and 60 minutes and be followed by migrainous headache. In addition, a second aura symptom from the posterior circulation should be reported (eg, dysarthria, double vision, or bilateral paresthesias). Less than 10% of patients who have MV in published case series fulfill the criteria for basilar migraine [5,11–13], which makes basilar migraine an inappropriate category for these patients. As a consequence, most adult patients who have MV cannot be classified with the current IHS criteria.

Proposed diagnostic criteria

Like migraine itself, MV can be diagnosed not by specific biologic markers but only on the basis of history. To date, there are no internationally approved criteria for the diagnosis of MV. A recent proposal from the authors' group uses operational clinical criteria modeled on the IHS classification of headaches [5]. Operational diagnostic criteria, however, are a trade-off between sensitivity and specificity. Therefore, two separate diagnostic categories seem to be useful: definite MV and probable MV (Table 1). The criteria for definite MV are stricter than the inclusion criteria used in most published case series of MV. A diagnostic interview applying this classification recently has been developed [22,23]. The prevalence of definite MV according to these criteria was 7% in a group of 200

Table 1
Diagnostic criteria for definite migrainous vertigo

Definite migrainous vertigo
- A. Episodic vestibular symptoms of at least moderate severity
- B. Current or previous history of migraine according to the 2004 criteria of the IHS
- C. One of the following migrainous symptoms during $\geq$ 2 attacks of vertigo: migrainous headache, photophobia, phonophobia, visual or other auras
- D. Other causes ruled out by appropriate investigations

Comment:

Vestibular symptoms are rotational vertigo or another illusory self or object motion. They may be spontaneous or positional. Vestibular symptoms are moderate if they interfere with but do not prohibit daily activities and 'severe' if patients cannot continue daily activities.

Probable migrainous vertigo
- A. Episodic vestibular symptoms of at least moderate severity
- B. One of the following:
  1. Current or previous history of migraine according to the 2004 criteria of the IHS
  2. Migrainous symptoms during vestibular symptoms
  3. Migraine precipitants of vertigo in more than 50% of attacks: food triggers, sleep irregularities, hormonal change
  4. Response to migraine medications in more than 50% of attacks
- C. Other causes ruled out by appropriate investigations

consecutive patients in a dizziness clinic and 9% in a group of 200 patients in a migraine clinic [5]. In a two-stage population-based study (n = 4869 adults) using screening interviews followed by expert telephone interviews, the lifetime prevalence of MV was estimated at 0.98% (95% confidence interval, 0.7–1.37) using the same criteria [24].

In accordance with most investigators, the proposed criteria conceptualize MV as an episodic vestibular disorder. Several reports, however, include patients who have nonspecific dizziness [9,11,12] or permanent symptoms [11,26]. There is little doubt that such variants exist, but for the sake of specificity it is useful to define the core syndrome first and then consider exceptions. The distinction between vertigo (which is a vestibular symptom) and dizziness (which is not) usually can be made by careful history taking: rotational vertigo or other illusory sensations of motion indicate vertigo (ie, vestibular symptoms), whereas a sensation of lightheadedness, giddiness, drowsiness, or impending faint suggests dizziness of nonvestibular origin. Nonspinning dizziness that occurs only during standing or walking usually indicates a neurologic gait problem rather than vestibular vertigo. A residual gray area remains, however, either as a semantic problem or because mild vestibular dysfunction may present with dizziness rather than vertigo.

Clinical features

Demographic aspects

MV may occur at any age [9,11,13]. It has a female preponderance with a reported female-to-male ratio between 1.5 and 5 to 1 [5,11,12,13]. Familial occurrence is not uncommon, probably based on an autosomal dominant pattern of inheritance with decreased penetrance in men [26]. In most patients, migraine begins earlier in life than MV [5,13]. Some patients have been free from migraine attacks for years when MV first manifests itself [13]. Not infrequently, migraine headaches are replaced by vertigo attacks in women around menopause. MV seems to occur more often in patients who have migraine without aura than in patients who have migraine with aura [4,10,12,13].

Migrainous vertigo in children

Benign paroxysmal vertigo of childhood is an early manifestation of MV that is recognized by the IHS classification of headaches. It is characterized by brief attacks of vertigo or disequilibrium, anxiety, and often nystagmus or vomiting recurring for months or years in otherwise healthy young children [27]. Many of these children later develop migraine, often years after vertigo attacks have ceased [28]. A family history of migraine in first-degree relatives is increased twofold compared with controls [29]. In a population-based

study, the prevalence of recurrent vertigo probably related to migraine is estimated at 2.8% in children between ages 6 and 12 [29].

Clinical presentation in adults

Adult patients who have MV typically report spontaneous or positional vertigo. Some experience a sequence of spontaneous vertigo transforming into positional vertigo after several hours or days [30,31]. This positional vertigo is distinct from BPPV with regard to duration of individual attacks (often as long as the head position is maintained in MV versus seconds only in BPPV), duration of symptomatic episodes (minutes to days in MV versus weeks to months in BPPV), and nystagmus findings (see later discussion). Altogether, 40% to 70% of patients experience positional vertigo in the course of the disease but not necessarily with every attack [4,8,26,32]. Head motion intolerance, similar to motion sickness (imbalance, illusory motion, and nausea aggravated or provoked by head movements) is a frequent additional symptom [11,20]. Visual vertigo (vertigo provoked by moving visual scenes [eg, traffic or cinema]) is another prominent feature of MV [11,25]. Nausea and imbalance are frequent but nonspecific accompaniments of acute MV. Duration and frequency of attacks vary between patients and in individual patients over time. The duration of vertigo ranges from seconds (approximately 10%), to minutes (30%), to hours (30%), and to several days (30%) [4,9,12,13,33]. For some patients, it may take weeks to recover fully from an attack. The attacks may occur days, months, or years apart in an irregular fashion. Some patients experience clusters of short attacks of vertigo, each one lasting from seconds to a few minutes. Such clusters can persist for hours to days and patients may experience head motion intolerance between the short attacks. Overall, between 10% and 30% of patients have vertigo with the typical duration of a migraine aura (5 to 60 minutes) [5,13].

MV often misses not only the duration criterion for an aura as defined by the IHS but also the temporal relationship to migraine headaches: vertigo can precede headache as is typical for an aura, may begin with headache, or appear late in the headache phase. Many patients experience attacks with and without headache [5,9,12]. Frequently, patients have an attenuated headache with their vertigo as compared with their usual migraine [12,14]. In some patients, vertigo and headache never occur together [5,9,12]. In these patients, diagnosis must be based on migrainous symptoms during the attack other than headache. Absence or attenuation of migrainous headache during MV attacks may be the result of an interaction of vestibular and trigeminal mechanisms, as suggested by a study showing disappearance of headache or a decrease in headache intensity after caloric activation of the vestibular system during migraine attacks [34].

Along with the vertigo, patients may experience photophobia, phonophobia, osmophobia, or visual or other auras. These phenomena are of

diagnostic importance, because they may represent the only apparent connection of vertigo and migraine (Table 2). Patients need to be asked specifically about these migrainous symptoms, because they often do not volunteer them. A dizziness diary is useful for prospective recording of associated features. Hearing loss and tinnitus are not prominent symptoms of MV but are reported in individual patients who have MV [4,11,12,35]. Hearing loss usually is mild and transient, without progression in the course of the disorder [12]. There are patients, however, who have severe and fluctuating hearing loss suggestive of MD and migrainous features during the attack, suggesting MV [4,12]. In this situation of diagnostic uncertainty, treatment can be tentatively directed at either condition. The frequency of cochlear symptoms and their temporal association with the vertiginous attacks has not been studied systematically. An increased prevalence of cochlear symptoms also is reported in vertigo-free patients who have migraine [4].

Asking for precipitants of attacks may provide essential diagnostic information. For example, vertigo attacks in women that occur regularly with menstruation are highly likely to be migrainous in origin, because migraine is influenced by hormonal changes, whereas other vestibular syndromes are not. Other typical migraine triggers include deficient or irregular sleep; excessive stress; the first day after a period of stress

Table 2
Clinical features of definite migrainous vertigo in 33 patients

Clinical features	%
Vestibular symptoms[a]	
Rotational vertigo	70
Other illusiory self or object motion	18
Positional vertigo	42
Head motion intolerance[b]	48
Duration of vestibular symptoms	
Seconds to 5 minutes	18
5 to 60 minutes	33
1 hour to 1 day	21
> 1day	28
Migrainous symptoms during vertigo	
Migrainous headache	94
Always	46
Sometimes	48
No headache	6
Photophobia	70
Phonophobia	64
Visual or other auras	36

[a] Several patients had more than one type of vestibular symptom.
[b] None of the patients had only head motion intolerance.

From Neuhauser H, Leopold M, v Brevern M, et al. The interrelations of migraine, vertigo and migrainous vertigo. Neurology 2001;56:684–6.

(weekends or beginning of a holiday); specific foods, such as matured cheese, red wine, and glutamate; and sensory stimuli, such as bright or scintillating lights, intense smells, and noise. The triggers are highly individual and each one applies to only a small percentage of the migraine population. Weather frequently is blamed but rarely proved as a provoking factor. Again, a headache and dizziness diary may be useful for identifying individual precipitants.

Sometimes, migrainous accompaniments and typical precipitants may be missing, but MV is considered the most likely diagnosis after other potential causes are investigated and seem unlikely. In this case, the broader term, benign recurrent vertigo [30,31], may be used and antimigraine drugs tried. A favorable response supports the suspicion of an underlying migraine mechanism. Apparent efficacy of a drug should not be regarded as a definite confirmation of the diagnosis, however, because spontaneous improvement, placebo response, and additional drug effects (eg, anxiolytic or antidepressant) have to be taken into account.

In summary, the clinical presentation of MV is variable in many respects and the connection to migraine can be subtle. The key to the diagnosis is the repeated concurrence of migrainous symptoms and vertigo, migraine specific precipitants, and, sometimes, response to antimigraine drugs.

Clinical and neurotologic findings in patients who have migrainous vertigo

In most patients, the general neurologic and otologic examination is normal in the symptom-free period [9]. Approximately 10% to 20% of patients who have MV have unilateral hypoexcitability to caloric stimulation and approximately 10% have directional preponderance of nystagmus responses [9,13]. Such findings, however, are not specific for MV, because they also are found in migraine patients who do not have vestibular symptoms [36,37] and in patients who have many other vestibular syndromes. Neuro-ophthalmologic evaluation may reveal mild central oculomotor deficits in the absence of other brainstem or cerebellar signs [13].

A neurotologic study of 20 patients during the acute phase of MV shows imbalance with increased sway on tandem Romberg testing or tandem walking in all patients but one. Fourteen patients had pathologic nystagmus: a peripheral type of spontaneous nystagmus was observed in three patients, a central type of spontaneous nystagmus in three, a central positional nystagmus in five, and a combined central spontaneous and positional nystagmus in three. Unlike benign paroxysmal positional nystagmus, MV positional nystagmus always was persistent as long as the provoking position was maintained and usually was not beating in the plane of positioning. A unilateral deficit of the horizontal vestibulo-ocular reflex was observed in three patients; one of them did not recover peripheral function on follow-up [38]. Saccadic pursuit during the acute attack was

noted in two patients. Overall, findings during acute MV pointed to central vestibular dysfunction in 10 patients (50%), pointed to peripheral vestibular dysfunction in three patients (15%), and were inconclusive with regard to the involved structures in seven patients (35%) [38].

In clinical practice, history usually provides more clues for the diagnosis than vestibular testing, because there are no abnormalities that are specific for MV. Therefore, in patients who have a clear-cut history, no additional vestibular tests are required. Vestibular testing in the interval, however, can be useful to reassure patients and doctors that there is no severe abnormality, such as a complete canal paresis, which suggests another diagnosis. Sometimes, minor findings on vestibular testing may help to explain residual symptoms in between attacks. Alternatively, testing during or shortly after an attack can reveal more profound peripheral or central abnormalities that should improve or disappear within a few weeks.

Pathophysiology

The pathophysiology of MV is obscure, as systematic investigations based on proper methodology and patient identification are just beginning. Several hypotheses have been put forward [22]. Spreading depression, which is the presumed mechanism of the migraine aura, may play a role in patients who have short attacks [9]. Spreading depression is a cortical mechanism, which can produce vestibular symptoms when the multisensory cortical areas become involved that process vestibular signals, which are located mainly in the posterior insula and at the temporoparietal junction [39]. Several findings, however, during the acute stage of MV, including canal paresis and complex positional nystagmus, cannot be explained by cortical dysfunction [38].

Several neurotransmitters that are involved in the pathogenesis of migraine (calcitonin–gene-related peptide, serotonin, noradrenaline, and dopamine) also modulate the activity of central and peripheral vestibular neurons and may contribute to the pathogenesis of MV [9,11,12,40]. Speculatively, unilateral release of these substances—analogous with the often unilateral location of headaches—might cause a static vestibular imbalance leading to rotatory vertigo, whereas bilateral release would cause a state of altered vestibular excitability leading to a motion sickness type of dizziness. Recently, a synopsis of potential pathophysiologic links between migrainous and vestibular mechanisms has been presented [22]. Such interactions may involve the vestibular nuclei, the trigeminal system, and thalamocortical pathways.

In the past decade, genetic defects of ion channels have been identified as the cause of various paroxysmal neurologic disorders. The finding of an abnormal voltage-gated calcium-channel gene in familial hemiplegic migraine (FHM) and episodic ataxia type 2 (EA-2) [41]—both of which

can have vertigo and migraine headache as prominent symptoms—has prompted the search for a susceptibility gene for MV in the same region. So far, however, no such genetic defect is identified [26,42].

Treatment

In many patients, MV attacks are severe, long, and frequent enough to warrant acute or prophylactic treatment. Unfortunately, there are as many opinions about the most effective substances as there are dizziness experts around the globe. This state of confusion reflects the lack of solid data derived from placebo-controlled trials. Apart from one small and inconclusive study on the use of zolmitriptan for acute MV [43], no proper study has been undertaken.

A few case reports suggest that medication used for migraine prophylaxis may be effective, including propranolol [44], metoprolol [13], tricyclic antidepressants [45], pizotifen [14,25], and flunarizine [13] (not licensed in the United States). The carboanhydrase inhibitors, acetazolamide [46] and dichlorphenamide [47], which normally are not used for migraine prophylaxis, also have been applied successfully (Table 3). All these reports are difficult to interpret, however, in the absence of controls and a well-documented pretreatment period, because frequency and duration of attacks vary considerably in the natural course of the disorder [9]. Expected side effects, such as orthostatic hypotension with β-blockers or weight gain with pizotifen, influence the selection of the drug. Patients should monitor their attacks in a diary. Treatment response should be evaluated after 3 months. A greater than 50% reduction in attack frequency is a realistic goal.

Table 3
Prophylactic treatment of migrainous vertigo

Drug	Daily dose	Side effects
Propranolol [44]	40–240 mg	Fatigue, hypotension, impotence, depression, nightmares, bronchial constriction
Metoprolol [13]	50–200 mg	Fatigue, hypotension, impotence, depression, nightmares, bronchial constriction
Amitriptylin [45]	50–100 mg	Sedation, orthostatic hypotension, dry mouth, weight gain, constipation, urinary retention, conduction block
Pizotifen [14,25] (not available in the United States)	1.5–6 mg	Weight gain, sedation
Flunarizine [13] (not available in the United States)	5–10 mg	Weight gain, sedation, depression, reversible parkinsonism
Acetazolamide [46]	250–750 mg	Paresthesia, nausea, sedation, hypokalemia, hyperglycemia
Dichlorphenamide [47]	17.5–75 mg	Paresthesia, nausea, sedation, hypokalemia, hyperglycemia

Treatment of acute MV with acute migraine medication can be attempted with triptans [48,49] and vestibular suppressants, such as promethazine, dimenhydrinate, and meclizine [48]. A retrospective study finds that the effect of triptans on vertigo is correlated to its effect on headache [50]. Multicenter trials with triptans administered subcutaneously, lingually, or nasally for fast relief of MV clearly are needed.

Nonpharmaceutic approaches in the treatment of MV should not be neglected and may be more effective than drugs in individual patients. A thorough explanation of the migrainous origin of the attacks can relieve unnecessary fears. Regular sleep, meals, and exercise and avoidance of identified triggers have a firm place in migraine prophylaxis. Selected patients may profit from vestibular rehabilitation [51].

Migraine and Meniere's disease

That MD and migraine may be associated was noted as early as 1861 by Prosper Ménière himself [52]. Sporadic accounts of headaches as an additional symptom in typical Meniere attacks provided further reason to suspect a link between MD and migraine [53,54]. Subsequent studies of the prevalence of migraine in MD produced conflicting results [55–57], but a recent controlled study provides well-documented evidence for such an association [58]. The study compared 78 patients who had idiopathic unilateral or bilateral MD according to the criteria of the American Academy of Otolaryngology [59] with age- and sex-matched controls. The prevalence of migraine according to the IHS criteria was almost twice as high in the MD group than in the control group (56% versus 25%, $P < 0.001$). Furthermore, 35 (45%) of the patients who had MD always experienced at least one migrainous symptom (migrainous headache, photophobia, or aura symptoms) along with their MD attacks. The study illustrates that there are patients who have migraine and recurrent vertigo for whom it is not possible to differentiate with certainty if they have MV or MD. In most patients, however, the distinction between the two can be made, considering that hearing loss is an occasional, mild, and non-progressive feature in MV [12], whereas it is a regular accompaniment of MD progressing to severe hearing loss within a few years. The two conditions may share pathophysiologic mechanisms, such as neurotransmitter imbalances or ion-channel dysfunction, that lead to a spectrum of migrainous, vertiginous, and cochlear symptoms.

Migraine and benign paroxysmal positional vertigo

BPPV is the most common cause of recurrent vestibular symptoms in unselected patients [13,60] and in migraineurs [5] presenting to a dizziness clinic. Although clinically they are two separate entities, there is evidence for

a link between migraine and BPPV. Migraine is found three times more common in patients who have idiopathic BPPV than in patients who have BPPV secondary to trauma or surgical procedures [61]. Moreover, migraine is two times more common in patients who have idiopathic BPPV than in age- and sex-matched controls [62]. Genetic factors and vascular damage to the labyrinth are discussed as pathogenetic mechanisms linking the two conditions [61].

Migraine and motion sickness

Motion sickness occurs more frequently in patients who have migraine (30% to 50%) than in controls who have tension headache or in controls who are headache-free (approximately 20%) [4,20]. The association is more pronounced in children [63] and in patients who have migraine with aura [20]. Findings from a study of crewmembers in a yacht race suggest an influence of the menstrual cycle on the occurrence of motion sickness and headache [64]. Migraineurs also report more visual vertigo while looking at spinning objects [20]. Headache, scalp tenderness, and photophobia may be provoked by optokinetic stimulation, according to a recent study of migraine. Patients who have migraine were more nauseated and had longer-lasting headache and photophobia than controls [65]. In individual patients, it may be difficult to differentiate between episodic motion sickness and attacks of MV induced by motion stimuli. The distinction can be made regarding the type and duration of symptoms. Nausea and dizziness improving after cessation of the motion stimulus point to a diagnosis of motion sickness, whereas rotational or positional vertigo persisting after the motion stimulus ends suggests MV. Chronic MV [25] may be explained by a constantly lowered threshold to motion stimuli.

Migraine and cerebellar dysfunction

Cerebellar dysfunction causes imbalance that patients may experience as dizziness. Some families who have FHM, a rare subtype of migraine, develop progressive cerebellar ataxia and nystagmus [66]. Mutations in the CACNA1A gene coding for the α_{1A} subunit of a neuronal Ca^{2+} channel, which is heavily expressed in the cerebellum, has been identified not only in FHM but also in EA-2 [67] and spinocerebellar ataxia type 6 [68]. EA-2 is characterized by short bouts of cerebellar ataxia, often with vertigo, and interictal nystagmus. Approximately half of patients who have EA-2 have migraine [69]. FHM and EA-2 are associated with typical symptoms of basilar migraine [69,70].

In more common types of migraine, cerebellar symptoms usually are not present, but subclinical hypermetria and other subtle subclinical cerebellar signs in patients who have migraine with or without aura recently have been

reported [71]. The investigators suggest dysfunctional Ca^{2+} channels as a possible cause. This hypothesis relies on findings of an involvement of the CACNA1A gene region in some families who have nonhemiplegic migraine with and without aura [72]. Another possible link between migraine and cerebellar dysfunction is the mild oculomotor deficits of cerebellar origin observed in patients who have MV [13].

Migraine and nonvestibular dizziness

Patients who have migraine report not only more vertigo but also significantly more dizzy spells than controls (32% versus 13%) [20]. These usually are attributed to nonvestibular causes. Mild vestibular dysfunction, however, may present with dizziness rather than vertigo.

Migraine, orthostatic hypotension, and syncope

Syncope during migraine attacks is reported in 5% of 500 unselected migraineurs [73]. Orthostatic symptoms were more frequent in students who had frequent headaches than in controls who had rare or no headaches [74] and were considerably more common in patients who had migraine than in controls (68% versus 8%) [75]. Orthostatic hypotension can be induced by small doses of dopamine agonists, such as bromocriptine and piribedil, and counteracted by the dopamine antagonist, domperidone, in migraineurs but not in controls, suggesting hypersensitivity to dopaminergic stimulation as the underlying mechanism [76,77].

Migraine and dizziness resulting from a comorbid psychiatric disorder

The interrelations of migraine, dizziness, and certain psychiatric disorders are intricate. There are bidirectional associations of migraine with major depression and panic disorder, with migraine being a risk factor for first-onset major depression and panic disorder and vice versa [78,79]. Dizziness is the second most common symptom of panic attacks, after palpitations [80], and can be a symptom of major depression. To complicate things further, patients who have panic and anxiety have an increased rate of vestibular test abnormalities [81], which may reflect an elevated risk of patients who have vestibular disorders developing an anxiety disorder [82]. Likewise, anxiety or panic disorder was reported by 14 of 100 patients who had MV [11].

Dizziness resulting from antimigraine medication

Dizziness is listed as a side effect of many medications, some of which are used in the treatment of migraine. It is useful, therefore, to elicit a detailed drug history and ascertain the onset of dizziness in relation to changes in medication. β-Blockers and calcium channel blockers, like most

antihypertensive agents, can cause orthostatic hypotension, particularly at the beginning of treatment. Long-term treatment with β-blockers also can cause lightheadedness and fatigue. Antidepressants, in particular tricyclic antidepressants, which are used in the prophylactic therapy of migraine, can cause sleepiness, blurred vision, lightheadedness, and postural hypotension.

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