Current Neurology and Neuroscience Reports (2022) 22:601–609
https://doi.org/10.1007/s11910-022-01222-6

HEADACHE (R.B. HALKER SINGH AND J. VANDERPLUYM, SECTION EDITORS)

Vestibular Migraine

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Accepted: 23 June 2022 / Published online: 31 August 2022
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Abstract
Purpose of Review To explore recent developments in vestibular migraine (VM).
Recent Findings This review discusses the current diagnostic criteria for VM in the adult and pediatric populations, as proposed by the International Headache Society and Bárány Society. Recent VM studies confirm the prior findings and reveal new insights, including the wide range of vestibular symptoms, symptoms in the attack-free period, and triggers. Many patients experience persistent vestibular symptoms, even in the absence of acute attacks, which often significantly impact patients' quality of life. The syndrome of benign recurrent vertigo and its relationship to migraine, VM, and Meniere's disease is also discussed. There is a dearth of randomized controlled trials in VM treatment. Prospective and retrospective studies support the benefit of many migraine treatments are effective in VM, including neuromodulation, and calcitonin gene-related peptide monoclonal antibodies.
Summary VM affects almost 3% of the population, but remains under-diagnosed. Recent diagnostic criteria can help clinicians diagnose VM in adults and children.

Keywords Vestibular migraine · Migraine · Vertigo · Dizziness

Introduction

Vestibular migraine (VM) has a prevalence of 1 to 2.7%; it is the most common neurological cause of vertigo among adults [1•, 2•] yet remains under-diagnosed [1•, 2•, 3]. Only 1.8% of patients referred to a tertiary care center were correctly diagnosed with VM, compared to 20.2% who were finally diagnosed with this condition [3]. While a relationship between vertigo and migraine has been recognized since the time of ancient Greece, the term VM was only first used in 1999 [4], and consensus diagnostic criteria were only published in 2012, by the International Headache Society (IHS) and Bárány Society [5••], which have subsequently been adopted by the International Classification of Headache Disorders (ICHD-3) [6].

Diagnostic Criteria and Demographics

According to the consensus criteria, the diagnosis of VM can be made if a patient: (A) experiences at least 5 episodes of vestibular symptoms of moderate or severe intensity, lasting 5 min to 72 h, (B) with one or more migraine features accompanying at least 50% of the vestibular episodes, and (C) has a current or previous history of migraine with or without aura according to the ICHD. The migraine features may consist of headache (with at least two of the following characteristics: unilateral, pulsating, moderate or severe intensity, aggravated by routine physical activity), photophobia and phonophobia, or visual aura. Although not recognized by the ICHD, the Bárány Society recognizes the diagnosis of probable VM [5••]. These criteria are summarized in Table 1.

The IHS and Bárány Society have also collaborated on the diagnosis of VM in childhood. Benign paroxysmal vertigo of childhood (BPVC) affects approximately 3% of children up to the age of 18; it is characterized by spontaneous vertigo attacks accompanied by vomiting, pallor, fear, unsteadiness, ataxia, and/or nystagmus [7••]. In a recent publication [7••], the IHS and Bárány Society have proposed replacing the term BPVC with "Recurrent

This article is part of the Topical Collection on *Headache*.

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Table 1 Diagnostic criteria for vestibular migraine in adults [5••]

Vestibular migraine	Must fulfill criteria (A), (B), and (C): (A)at least 5 episodes of vestibular symptoms of moderate or severe intensity, lasting 5 min to 72 h; (B)with *one or more* migraine features accompanying at least 50% of the vestibular episodes. These include: 1.Photophobia and phonophobia; 2.Visual aura; or 3.Headache, with at least *two* of the following characteristics: i.Unilateral ii.Pulsating/throbbing iii.Moderate or severe intensity iv.Aggravated by routine physical activity (C)a current or previous history of migraine with or without aura according to the International Classification of Headache Disorders (ICHD)
Probable vestibular migraine	Must fulfill criterion (A), and (B) or (C): (A)at least 5 episodes of vestibular symptoms of moderate or severe intensity, lasting 5 min to 72 h; (B)with *one or more* migraine features accompanying at least 50% of the vestibular episodes. These include: 1.Photophobia and phonophobia; 2.Visual aura; or 3.Headache, with at least *two* of the following characteristics: i.Unilateral ii.Pulsating/throbbing iii.Moderate or severe intensity iv.Aggravated by routine physical activity (C)a current or previous history of migraine with or without aura according to the ICHD

Vertigo of Childhood" (RVC), as well as criteria for VM of childhood, and probable VM of childhood. The diagnosis of VM of childhood and probable VM of childhood can be made in children under the age of 18, who meet the same criteria applied to adults for VM and probable VM, respectively. The diagnosis of RVC is made in children (below age 18) who experience at least 3 episodes of vestibular symptoms of moderate to severe intensity, lasting 1 min to 72 h, but without any accompanying migrainous symptoms (as defined above), and who do not have a current or previous history of migraine [7••]. These criteria are summarized in Table 2.

Recent publications into the demographic and clinical features of VM both support and add to the findings of previous studies (summarized in a comprehensive review [8••]). These studies confirm that VM predominantly affects women (1.5 to 5.6:1 female preponderance) in their late-30 s to mid-40 s [9•, 10•, 11, 12, 13•, 14•]. This higher female preponderance and later onset, compared to migraine, may be related to perimenopausal or menopausal hormonal changes. Most patients describe history of migraine headache (either with or without aura) [9•, 10•, 14•], and/or motion sickness [9•, 10•, 12, 14•, 15] prior to the onset of vestibular symptoms. Not infrequently, a headache-free period (sometimes lasting years) is experienced before the onset of vestibular symptoms [14•, 16]. Often, patients will report a family history of migraine, or episodic vertigo/dizziness [9•, 10•, 14•]. A family history of migraine or vestibular symptoms often predicts a lower age of migraine onset [9•, 14•]. The migraine-vertigo association in families is 4–10 times higher than the prevalence reported in the general population [17•].

Clinical Features

A very wide range of vestibular symptoms may be experienced by patients with VM. This can often be confusing to both the patient and physician and may explain why this disorder is often missed. The various vestibular symptoms that may occur during VM attacks include spontaneous vertigo [9•, 10•, 11, 13•, 14•], positional vertigo [9•, 10•, 13•, 14•], head motion-induced dizziness/vertigo [10•, 12], postural unsteadiness [9•, 10•], oscillopsia [9•, 10•], visually induced dizziness/vertigo [9•, 10•, 12], and directional pulsion [9•, 10•]. Leading to more confusion, most patients may experience more than one vestibular symptom during acute VM episodes [10•, 18•].

The inconsistent relationship of headache to vestibular symptoms in VM (which also adds to diagnostic confusion) was confirmed by recent publications. The proportion of patients who consistently experience a headache in relation to vestibular symptoms during VM attacks varies widely [10•, 11, 13•, 14•]. Furthermore, vestibular symptoms may occur before, during, or after the headache phase of a migraine attack [10•, 14•]. Younger patients (< 43 years) tend to experience more severe headache intensity, while older patients (> 41 years) report more severe vestibular symptoms [14•]. One study found that VM patients were three times more likely to experience occipital headaches

Table 2 Diagnostic criteria for vestibular migraine in children and recurrent vertigo of childhood [7••]

Vestibular migraine	Must fulfill criteria (A), (B), and (C): (A)at least 5 episodes of vestibular symptoms of moderate or severe intensity, lasting 5 min to 72 h; (B)with *one or more* migraine features accompanying at least 50% of the vestibular episodes. These include: 1.Photophobia and phonophobia; 2.Visual aura; or 3.Headache, with at least *two* of the following characteristics: i.Unilateral ii.Pulsating/throbbing iii.Moderate or severe intensity iv.Aggravated by routine physical activity (C)A current or previous history of migraine with or without aura according to the International Classification of Headache Disorders (ICHD)
Probable vestibular migraine	Must fulfill criterion (A), and (B) or (C): (A)At least 5 episodes of vestibular symptoms of moderate or severe intensity, lasting 5 min to 72 h; (B)With *one or more* migraine features accompanying at least 50% of the vestibular episodes. These include: 1.Photophobia and phonophobia; 2.Visual aura; or 3.Headache, with at least *two* of the following characteristics: i.Unilateral ii.Pulsating/throbbing iii.Moderate or severe intensity iv.Aggravated by routine physical activity (C)A current or previous history of migraine with or without aura according to the ICHD
Recurrent vertigo of childhood	At least 3 episodes of vestibular symptoms of moderate to severe intensity, lasting between 1 min to 72 h, without migraine features (as defined above), or a current/previous history of migraine

compared to migraine patients [19]. Instead of a painful headache, some may only describe a sensation of head fullness or pressure [10•].

Other migraine features also occur in VM, including photophobia, phonophobia [9•, 10•, 13•, 14•], and visual aura [10•, 13•, 14•]. Migrainous symptoms, not included in the diagnostic criteria, include nausea with/without vomiting [9•, 10•, 13•, 14•], osmophobia [10•, 13•, 14•], neuropsychiatric symptoms (e.g., fatigue, emotional lability, cognitive dysfunction, word-finding difficulty) [4, 10•], autonomic manifestations (e.g., pallor, dry mouth, diaphoresis, diarrhea) [10•], non-specific sensory symptoms [10•, 14•], visual symptoms (e.g., blurry vision, visual snow, palinopsia) [10•], and even Alice in Wonderland syndrome (AIWS) dysperceptions (e.g., micropsia, macropsia, teleopsia, macro-/micro-somatognosia, aschematia, depersonalization, derealization, out-of-body-experiences) [20]. Aural symptoms accompany VM attacks in over two thirds of patients, including tinnitus (most common), aural pressure, ear pain, and muffled hearing [9•, 10•, 13•, 14•, 21, 22]. Some unusual aural symptoms may be described as well, including bubbling, pulsating, or vibrations in the ear [10•].

The overlapping relationship between VM, migraine, and Meniere's disease (MD) is complex. The classic triad of MD (vertigo, tinnitus, and fluctuating hearing loss) only occurs in 40% of patients [23]; not infrequently, episodic vertigo without aural symptoms may occur early in the course of MD [24]. MD-related vertigo lasts between 20 min and 12 h [25•], falling within the attack duration of VM [5••]. Approximately one third of MD patients suffer from migraine [26]. Furthermore, headache and migraine symptoms frequently accompany MD attacks [26, 27, 28, 29], because vestibular activation can provoke migraine attacks [30]. Confounding matters even further, some patients suffer from both MD and VM [14•, 27, 31, 32]. The distinguishing characteristic of MD is unilateral, low-frequency, progressive, or fluctuating sensorineural hearing loss (SNHL). Audiometrically documented fluctuating low-frequency unilateral SNHL is the key to diagnosing MD [25•]. By contrast, VM-related SNHL has been described in up to 20% of patients but is usually mild, and symmetric, most likely representing presbyacusis [8••].

The diagnosis of migraine with brainstem aura (MBA) should also be considered in the presence of auditory symptoms. This diagnosis requires at least two posterior circulation symptoms (vertigo, diplopia, tinnitus, hypacusis, ataxia, or encephalopathy) lasting between 5 to 60 min (i.e., the aura), followed by a migraine headache [5••]. The rarity of this condition has led to the proposal of even stricter MBA diagnostic criteria, requiring at least three fully reversible brainstem symptoms (the aforementioned symptoms, simultaneously bilateral visual symptoms, and/or simultaneously bilateral paresthesiae) [33•]. In contradistinction to MBA, vestibular symptoms accompanied by other deficits are rarely limited to the aura phase in VM [5••].

Some patients experienced brief, isolated paroxysms of vestibular symptoms in the attack-free period [10•]. These attacks last seconds and are often described as dropping, falling, or ground-shifting sensations, comparable to the drop attacks of Tumarkin in MD. Some VM patients also describe feeling perpetually tilted to one side. These paroxysmal symptoms and tilt illusions may be due to dysfunction of the otolithic organs, which are responsible for detecting linear acceleration, including gravity. Compared to controls, people with VM make more errors in the perception of being upright [34, 35]. Vestibular myogenic-evoked potentials (VEMPs) measure otolithic organ function: cervical (c-) and ocular (o-) VEMPs for saccular and utricular function, respectively. The findings in VM have been mixed and contradictory. Some describe reduced [36, 37] or absent cVEMPs [38, 39]. Others report no cVEMP differences between VM and migraine without vertigo [40], and controls [13•, 41, 42, 43, 44]. While some report diminished oVEMPs in VM [42, 44, 45], others report no difference compared to controls [13•, 37, 41]. Abnormal cVEMPs, especially higher asymmetry ratios, may be more common in MD compared to VM and thus may potentially be useful in helping distinguish both disorders [41, 43, 44, 46].

VM attack triggers are similar to triggers described in migraine, and include stress, menses, sleep irregularities (lack of sleep, or sleeping more than usual), weather changes (typically rainy or windy weather), altitudes, missing meals, seasonal allergies, dehydration, physical exhaustion, bright lights, flashing lights, and loud noise [10•, 12, 14•]. Some people with VM also experience attacks precipitated by "typical" migraine food triggers like caffeine, alcohol (especially red wine), chocolate, and monosodium glutamate [10•]. Prolonged or excessive exposure stimuli that provoke visually induced dizziness/vertigo (e.g., going to department stores, computer games), as well as tasks that require excessive head movements, may also trigger VM episodes. 10•, 14•.

Benign Recurrent Vertigo (BRV)

BRV [47] or recurrent vestibulopathy [48] refer to a syndrome of episodic vertigo (lasting minutes to hours) accompanied by nausea and vomiting, but without any other neurological or aural symptoms. Prior to the publication of formal diagnostic criteria for VM, many patients with episodic vestibular symptoms were given the diagnosis of BRV, and as such, studies into this syndrome may have inadvertently included VM patients in their analyses. Publications on BRV since the publication of the VM diagnostic criteria have separated patients with BRV from those with VM and compared both groups. A more recent proposed term for BRV is recurrent vestibular symptoms not otherwise specified (RVS-NOS) [18•], but to date, there are no consensus criteria for diagnosing BRV/RVS-NOS.

Recent BRV publications highlight some differences and similarities with VM and MD. In contradistinction to the high female predominance in VM, the female preponderance in BRV is small to none [18•, 28, 49•, 50•]. One-third to half report a family history of migraine or recurrent vertigo episodes [49•, 50•]. The severity of vestibular symptoms in BRV is milder compared to VM and MD [18•]. About one-third experience attacks last less than a minute [18•], but there is a bimodal distribution of attack duration (1–5 min and 1–4 h) [28]. The variety of vestibular symptoms (even positional vertigo) in RVS-NOS is more similar to that of VM compared to MD [18•, 49•].

Compared to VM and MD, BRV causes less severe nausea and vomiting [18•]. Up to one-third describe aural symptoms (usually tinnitus, but sometimes aural fullness, or hearing loss) associated with the attacks [18•, 50•]. Up to one-fifth report migrainous symptoms (photophobia, phonophobia, or visual aura) [18•, 50•], and 20% of patients with BRV describe headaches that do not meet the criteria for migraine [18•]. Similar to migraine, BRV attacks are triggered by stress, menses, alcohol, and fatigue [50•]. BRV appears to have a stable clinical course. The prognosis of BRV appears favorable; over time, few transition to VM, and none progresses to MD [18•, 49•, 50•].

Persistent Symptoms and Co-morbid Disorders

Persistent vestibular symptoms, independent of VM attacks, is common in people with VM [10•, 12, 14•]. VM patients commonly complain of head-motion and visually induced dizziness [10•, 12] even when not experiencing an acute VM attack. Motion sensitivity is a very frequent complaint; VM patients often have heightened motion perception compared to controls [51, 52]. In addition, VM patients are much more prone to motion sickness [52, 53].

Not infrequently, people with VM also carry concomitant vestibular diagnoses that manifest with chronic, persistent symptoms. Persistent postural perceptual dizziness (PPPD), a functional otoneurologic disorder characterized by constant dizziness, is common in VM [10•, 54•, 55]. When associated with VM, PPPD is less likely to resolve [54•]. A vestibular disorder that causes a pervasive illusion of motion called mal de debarquement syndrome (MDDS) is less common than PPPD but is not infrequently encountered in VM [10•, 56•]. Chronic, constant vestibular symptoms often significantly impact a patient's quality of life and impair their ability to engage in job, school, social, family, and leisurely activities [10•, 54•, 56•]. In addition, the severity of persistent dizziness correlates with the degree of

cognitive impairment, a frequent complaint, in people with VM [21, 57]. In people with migraine, chronic dizziness is often associated with greater anxiety and depression levels, as well as depersonalization/derealization sensations [58]. Furthermore, the risk of falls is increased in VM patients with persistent dizziness [59].

The presence of persistent dizziness has led to the proposal of a subtype of "chronic VM," in which a person has more than 15 dizzy days per month [60], similar to the concept of chronic migraine. However, given the frequency and multitude of vestibular symptoms VM patients experience in the attack-free period, and the number of patients with comorbid PPPD or MDDS, many would be considered to have "chronic" VM and such a designation may not have much clinical value and may only serve to confound clinical trial data analyses. VM attack frequency may be a more clinically relevant method of stratifying VM severity for clinical studies, or evaluating the efficacy of a treatment intervention. The presence of comorbid vestibular disorders often indicates the need for a multidisciplinary approach.

Neuropsychiatric comorbidities are frequently encountered in VM [10•, 14•, 61], consistent with the relationship between vestibular symptoms and mood disorders, especially anxiety [62]. A history of anxiety and depression also portend a higher risk of developing VM [2•]. Sleep disturbances are frequently experienced by VM sufferers [10•, 63, 64]. Because sleep irregularities are a well-recognized migraine trigger, recognizing and addressing sleep disorders in VM patients are important. Children with vertigo are at higher risk of attention deficit disorder, learning disability, emotional disturbances, trouble focusing, and behavioral difficulties, and are more likely to utilize special education services [65]. Functional neurological disorders (particularly non-organic gait dysfunction) may also be present, particularly in those languishing with long-standing symptoms without a clear diagnosis [10•]. Patients complaining of vestibular symptoms and exhibiting anxiety are at risk of being dismissed as having a "psychogenic" or "psychiatric" disorder only. Furthermore, some VM patients experience bizarre AIWS dysperceptions [20] which may be mistaken for psychosis.

Diagnosis

VM is a clinical diagnosis, based on the aforementioned diagnostic criteria. There are no specific examination or test findings that point to the diagnosis of VM. A wide range of non-specific neuro-otologic abnormalities is commonly encountered in VM patients, both during attacks and the interictal period. Unfortunately, no particular pattern of abnormalities is specific to VM. Central and peripheral patterns of spontaneous, positional, and mixed patterns of nystagmus may occur during acute VM attacks [13•, 14•, 66•, 67]. In some, the nystagmus may even change during the course of the attack [13•]. Interictally, positional nystagmus appears to be the most frequent neuro-otologic abnormality [10•, 13•], but findings vary widely and include spontaneous nystagmus, gaze-evoked nystagmus, spontaneous nystagmus with fixation removed, saccadic pursuit, head shaking-induced nystagmus, skull vibration-induced nystagmus, and hyperventilation-induced nystagmus [10•, 11, 13•, 14•, 68]. Underscoring the frequency but lacking specificity of these neuro-otologic signs, central positional nystagmus, head-shaking nystagmus, and saccadic pursuit have been observed in patients with migraine but no vestibular symptoms [8••]. However, a thorough otoneurologic examination and appropriate tests (e.g., audiogram, brain MRI, rotary chair, VEMPs) may help identify or exclude other conditions that may co-exist with, or mimic, VM.

Treatment

There is a stark paucity of randomized controlled trials in the acute treatment of VM. One small randomized, double-blind, placebo-controlled clinical trial showed zolmitriptan-reduced vertigo at 2 h but was underpowered and the results were inconclusive [69]. One retrospective study reported benefit with sumatriptan (both oral and intramuscular) [70]. Rizatriptan diminishes visually induced motion sickness in VM patients [71]. In my experience, triptans are an effective treatment option for VM attacks. In cases of triptan failure or contraindication, benzodiazepines, meclizine, or dimenhydrinate may be offered for acute treatment but may be more sedating.

Neuromodulation provides safe, drug-free, and effective rescue therapy for VM attacks. A retrospective observational study showed that noninvasive vagus nerve stimulation (NVNS) was an effective rescue treatment for acute VM attacks, reducing the severity of vertigo by almost 50% in the majority of patients treated with no side effects aside from platysma muscle contraction [72•]. A case series of acute VM episodes showed that NVNS ameliorated both vertigo and nystagmus associated with the VM attacks [67]. It is possible NVNS treats acute VM episodes by modulating activity within both the trigeminal and vestibular systems via the nucleus tractus solitarius, as well as other brainstem nuclei, including the dorsal motor nucleus of the vagus nerve, the rostro-ventro-lateral medulla, reticular formation, locus coeruleus, and nucleus intercalatus [73•]. External trigeminal nerve stimulation (ETNS) relieved both vertigo and headache associated with acute VM attacks by more than 50% in a retrospective observational study [66•]. While altering vestibular system activity via the trigeminal input is a known phenomenon [66•], the effect of ETNS on VM

may also support the central role for the trigeminal system in both the pathophysiology, and treatment of VM. In fact, a recent animal study suggests that changes in the trigeminal nuclear complex may lead to sensitization of vestibular nuclear neurons [74].

All VM patients should be counseled about lifestyle modifications: trigger avoidance (if possible), proper sleep hygiene, regular meals, exercise, and stress management. A migraine-friendly diet (e.g., avoiding trigger foods, limiting caffeine, regular eating habits) alone may be effective in controlling VM in some [8••]. A prospective study confirmed that lifestyle modification (especially proper sleep) significantly improved both vestibular symptoms and headache in VM [75]. Nutraceuticals like riboflavin, magnesium, and coenzyme-Q10 are safe and effective migraine prophylactics, and should be offered in VM as well [8••].

Exercise and vestibular rehabilitation therapy (VRT) are also beneficial in VM patients. Low-impact physical exercise (20 min daily, 3 times a week for 6 weeks) reduced the number of vertigo attacks and pro-inflammatory markers in a small study [76]. A randomized, single-blind study comparing resistance to relaxation exercises confirmed that both were effective for controlling VM symptoms and markers of inflammation, although resistance exercises was significantly superior [77]. Vestibular rehabilitation therapy (VRT) is especially beneficial for VM patients suffering with constant dizziness [11, 78].

To date, very few randomized controlled trials in VM prophylaxis have been conducted. The Prophylactic Treatment of VM with Metoprolol (PROVEMIG) trial sought to compare metoprolol to placebo in people with VM but unfortunately did not clearly show superiority of metoprolol; the study was terminated early due to poor patient accrual, and lack of funding [79••]. However, the PROVEMIG trial provided several important learning points for the development of future clinical trials. One randomized study showed that flunarizine improved vertigo frequency and severity, but not headache [80]. Another showed that venlafaxine and propranolol reduced vertigo attacks and dizziness, but venlafaxine was superior at addressing comorbid mood disorders [43].

Several small unblinded prospective studies provide insight into VM prophylaxis. One unblinded, prospective study showed improvements in vertigo and headache frequency in patients randomized to 5 weeks of treatment with acetazolamide, amitriptyline, flunarizine, propranolol, and topiramate; while all treatment groups showed similar improvements, a high incidence of side effects was observed with flunarizine. Unfortunately, out of the original 125 patients recruited, only 31 were included in the final sample due to exclusions, failure to attend follow-up visits, and medication non-compliance [81]. Verapamil reduced the frequency of vertigo and headaches at 3 months in one unblinded, prospective study of patients with both VM and MD [82]. Two prospective studies showed that topiramate successfully controlled the frequency and severity of vertigo and headache [83], and auditory symptoms [84]. Propranolol and venlafaxine improved vertigo attack severity and frequency in an open-label, prospective parallel group study [85]. Another open-label, prospective study showed that venlafaxine, flunarizine, and valproic acid decreased vertigo attacks and overall dizziness in VM [86]. A small observational study comparing lifestyle modification to preventive therapy using a combination of cinnarizine and dimenhydrinate significantly reduced the number of vertigo and headache episodes in the medication group [87].

Based on several retrospective cohort studies, a reduction in duration, intensity, and frequency of vertigo attacks is observed with well-known migraine prophylactics, including anti-epileptic drugs (e.g., topiramate, lamotrigine, valproic acid), beta-blockers (e.g., propranolol, metoprolol), calcium-channel blockers (e.g., verapamil, cinnarizine, flunarizine), tricyclic antidepressants (e.g., amitriptyline, nortriptyline), selective serotonin-reuptake inhibitors, selective norepinephrine-reuptake inhibitors (e.g., venlafaxine), acetazolamide, methysergide, and cyproheptadine, as well as benzodiazepines [8••, 11, 12, 56•].

The potential of calcitonin-gene related peptide (CGRP) inhibitors in VM treatment certainly adds to our therapeutic arsenal. One small retrospective study reported that CGRP monoclonal antibodies were effective VM preventives [88]. Several clinical trials of CRGP inhibitors in VM treatment are taking place, at the time this manuscript was written. In chronic migraine rat models, CGRP expression is increased in the vestibular nucleus [74, 89] and results in vestibular dysfunction [74]. CGRP receptor blockade not only reduced the density of dendritic spines in the vestibular and trigeminal nuclei but also improved mechanical allodynia, thermal hyperalgesia, and vestibular dysfunction [89]. The benefit of CGRP blockade in VM treatment is perhaps unsurprising in view of the role CGRP plays in the vestibular system, which is summarized in an excellent review by Balaban and colleagues [90••].

Conclusion

In conclusion, VM is a common but under-recognized neurological cause of vestibular symptoms in adults and significantly affects quality of life and economic productivity. Neurologists, especially headache specialists, will often encounter VM in clinical practice (up to one third of patients in headache and neuro-otologic centers may have vestibular migraine [1•, 2•]), and as such, familiarity with the potential and common symptoms, and comorbidities of VM is important. Treatments for VM are similar to those for

migraine, and may significantly improve patients' symptoms and quality of life.

Declarations

Conflict of Interest The authors declare no competing interests.

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