ELMER PRESS

Original Article

J Clin Med Res. 2024;16(2-3):63-74

Presence of Spontaneous Nystagmus, Benign Paroxysmal Positional Vertigo, and Tumarkin Fall in Patients With Primary Headache and Their Responses to Caloric and Video Head Impulse Tests

Ai Juan Zhang[a], Li Qun Yu[a], Li Zhou[a], Xian Zhu Cong[b], Qi Hui Liu[a], Wen Li[a], Ai Yuan Zhang[c, d]

Abstract

Background: Migraine, vestibular migraine (VM) and tension-type headache (TTH) are the most common disorders in dizziness and headache clinics, associated with dizziness or vertigo and postural imbalance, causing a substantial burden on the individual and the society. The objective of this research was to examine the presence of spontaneous nystagmus, comorbidity of benign paroxysmal positional vertigo (BPPV), and Tumarkin fall in patients; additionally, the study focused on assessing the patients' responses to bithermal caloric irrigation and video head impulse test (vHIT).

Methods: Consecutive patients diagnosed with migraine, VM, and TTH according to the International Classification of Headache Disorders, third edition (beta version (ICHD-3β)), who were referred to Dizziness and Headache Clinic were enrolled. BPPV and Tumarkin fall were assessed by questionnaires. The presence of BPPV was further evaluated through Dix-Hallpike or head roll maneuver, while spontaneous nystagmus was monitored using video-oculography during interictal period. Lastly, patients' responses to bithermal caloric irrigation and vHIT were analyzed.

Results: There was a significantly higher incidence of spontaneous nystagmus in VM compared to both migraine and TTH. The drop attack episodes were slightly more frequent in VM than in TTH and migraine, though not statistically significant. The prevalence of BPPV was significantly higher in VM than in migraine and TTH. Unilateral vestibular paresis was more common in the VM group than in migraine and TTH. There was profound unilateral weakness (UW) in VM patients than in migraine, but no significant difference was found between VM and TTH. In VM, the percentage of saccades along with reduced vHIT gain was significantly higher than in migraine. Lastly, the percentage of abnormal response in vHIT was significantly lower than the percentage of abnormal UW in caloric irrigation across all groups.

Conclusions: In VM patients, the prevalences of decompensated peripheral damage and BPPV were higher than in migraine and TTH patients as disclosed by the presence of peripheral spontaneous nystagmus and abnormal vHIT during the interictal period. Our findings suggest that the peripheral vestibular system acts as a significant mechanism in the pathogenesis of VM, and it might also be involved in migraine and TTH cases without vertigo symptoms.

Keywords: Migraine; Vestibular migraine; Tension-type headache; Caloric test; Semicircular canal; Spontaneous nystagmus; Vestibular drop attack; Benign paroxysmal positional vertigo; Video head impulse test; Horizontal canal

Introduction

Vestibular migraine (VM) has been known since ancient times, but extensive investigations only started around three decades ago. The term "vestibular migraine" firstly coined by Boenheim in 1917 and later revived by Dieterich in 1999 was accepted to describe the vestibular symptomatology related to migraine spectrum disorders [1]. In 2012, the International Headache Society and the Barany Society published the first consensus on diagnostic criteria for VM [2], which were subsequently included in the International Classification of Headache Disorders, third edition (beta version (ICHD-3β)) in appendix [3].

VM is the most common cause of spontaneous vertigo, and a recent multicenter study according to ICHD-3β criteria found the prevalence of VM to be 10.3% in migraine patients [3]. There is evidence that the occipital/back of the head location for headaches is more common in VM than migraine [4]. The 1-year prevalence of VM in the general population is 0.9% [5], with a general prevalence of 1-3%, which leads to VM's

Manuscript submitted December 6, 2023, accepted January 31, 2024
Published online March 16, 2024

[a]Department of Neurology, Weifang People's Hospital, Weifang 261041, Shandong, China
[b]Epidemiology and Health Statistics, Weifang Medical College, Weifang, Shandong, China
[c]Cardiovascular Intensive Care Unit and Rehabilitation Department, Weifang People's Hospital, Weifang, Shandong, China
[d]Corresponding Author: Ai Yuan Zhang, Cardiovascular Intensive Care Unit and Rehabilitation Department, Weifang People's Hospital, Weifang, Shandong, China. Email: zhangaiyuan@aliyun.com

doi: https://doi.org/10.14740/jocmr5088

responsibility for 10-30% of patients seeking dizziness treatments [6]. VM episodes often trigger great panic and anxiety [7, 8]. Although the mechanisms and pathophysiology are far under-understood, there is evidence that peripheral vestibular organs typically show no obvious abnormalities, suggesting VM primary involvement in the vestibular center. Functional magnetic resonance imaging (FMRI) showed increased thalamic activation during vestibular stimulation [9], and the 3.0-T scanner found selective gray matter volume increased in occipital and frontal regions compared to both controls and migraineurs [10]. However, symptoms like spinning vertigo and a variety of vestibular reflex disorders such as blurred vision (vestibular-ocular reflex), motion intolerance (vestibular-colic reflex), and disturbance of autonomic (central or peripheral) nerve (vestibular-vegetative reflex), etc. strongly imply the involvement of peripheral vestibular.

In the dizziness and headache clinic, the most alarming and dangerous symptom is a Tumarkin fall (vestibular drop attack (VDA)), while the most common comorbidity is secondary benign paroxysmal positional vertigo (BPPV). Spontaneous nystagmus, recorded by video goggles, is associated with acute vestibular imbalance, which is a hallmark of vestibular neuritis (VN) and a potential, though rare feature of interictal VM [11]. Migraine, VM, and tension-type headache (TTH) are lifespan disorders, and in this study, we aimed to assess the prevalence of spontaneous nystagmus, drop attack and BPPV in migraine, VM and TTH groups, with a focus on the differences in semicircular canal (SCC) responses to caloric stimulation and video head impulse test (vHIT).

The link between migraine and vertigo has been extensively explored in recent years, yet the relationship between TTH and vestibule remains understudied [12]. Oculo-vestibular signs and symptoms such as spontaneous nystagmus, BPPV, and vHIT have been documented in patients with VM and migraine [13-16]. Stabilometric abnormalities have been noted in VM and TTH patients [17]. Recent studies disclosed that the peripheral vestibular organ in VM patients was involved and became hypersensitive during interictal period [12, 18]. However, comparative studies examining peripheral spontaneous nystagmus, BPPV, Tumarkin fall, and horizontal SCC in the most common primary headache of migraine, VM, and TTH are lacking. Traditionally, Tumarkin fall was associated with Meniere's disease (MD) but primary headache. Additionally, there have been no reports on the severity of unilateral weakness (UW) in caloric irrigation in migraine, VM, and TTH patients.

Materials and Methods

Patients

Consecutive patients with migraine, VM, and TTH were recruited from the outpatients visiting the Headache and Dizziness Clinic of Weifang People's Hospital from December 2019 to September 2022. Patients were diagnosed by neurologists according to the ICHD-3β [3] by the Classification Committee of the International Headache Society and the Barany Society. Patients were divided into three groups: migraine (group 1), VM (group 2), and TTH (group 3). The study was approved by the Human Research Ethics Committees of Weifang Brain Hospital. The study was conducted in compliance with the ethical standards of the responsible institution on human subjects as well as with the Helsinki Declaration.

Exclusion criteria

Patients with any of the following conditions were excluded: previous cerebellar or brainstem stroke; severe heart diseases; severe renal or hepatic disorders; acoustic neuroma, or other posterior fossa lesions; previous VN; autoimmune diseases; obstruction of external ear canal; otitis media; temple bone surgery; profound hearing loss; vestibular paroxysm; MD; recurrent vertigo attack without headache; taking psychotropic drugs; unilateral tympanic membrane perforation [19]. For patients with mild hypertension, diabetes mellitus or hyperlipidemia, their blood pressure, blood glucose or lipids were controlled to normal range by conservative treatments. Smoking and alcohol consumption patients were not included in this study.

Examinations

Physical examination

All patients underwent ocular, ontological and general neurological examination. If the patient was suspected of central vestibular disorder, a brain MRI (3-T scanner, Siemens, Germany) was requested, including T_1/T_2/fluid attenuated inversion recovery (FLAIR)/diffusion-weighted imaging (DWI) sequence and magnetic resonance angiography (MRA), to exclude stroke and severe posterior vessel stenosis. The 24-h Holter monitor and echocardiography were performed to exclude malignant arrhythmia and myocardial ischemia that might cause syncope. Orthostatic hypotension was excluded by measuring blood pressure at supine and upright positions with reduced systolic pressure less than 20 mm Hg or reduced diastolic pressure less than 10 mm Hg at 30 s, 1 min, 2 min, and 3 min [20]. Routine blood tests, liver and kidney function, thyroid function, blood glucose, and lipids were examined to exclude severe disorders. Detailed medical history, especially before BPPV and the drop attack, was obtained by questionnaires.

Before the test, spontaneous nystagmus, primary position, or subtle spontaneous were observed, but sometimes these are detectable only by portable video goggles induced by side gazing. Peripheral vestibular nystagmus commonly beats away from the side of the lesion and increases when the eyes are turned in the direction of the quick phases (Alexander's law) [21]. The non-direction changing and positive suppression by visual fixation (peripheral type is horizontal, and can have torsional component) are also features of peripheral spontaneous nystagmus [22]. Nystagmus slow-phase velocity (SPV) was measured in degrees per second, and a minimum of three beats of nystagmus within 15 s of recording was required for analysis [23].

All patients underwent Dix-Hallpike or head roll maneuvers for typical positional nystagmus of BPPV [24]. The diag-

Table 1. Comparison of Mean Age and Sex Ratio

Group	n	Age (mean ± SD)	Median	Male, n (%)	Comparison between groups	P	
						Age (*t*)	Sex (χ^2)
1. Migraine	142	41.15 ± 15.69	38.00	42 (29.58%)	1 and 2	< 0.001	> 0.05
2. VM	226	47.95 ± 16.67	51.00	51 (22.57%)	1 and 3	< 0.001	> 0.05
3. TTH	140	54.41 ± 13.95	56.00	58 (41.43%)	2 and 3	< 0.05	< 0.01
Total	508	47.83 ± 16.42	51.00	151 (29.72%)			

SD: standard deviation; TTH: tension-type headache; VM: vestibular migraine.

nosis criteria of BPPV were: history of short-lasting (< 1 min) rotational vertigo provoked by head position changes, presence of nystagmus specific for the affected canal, with latency and fatigability, and attacks typically lasting from seconds to minutes [24].

Clinical diagnostic criteria for Tumarkin's otolithic crisis were: the characteristic of attacks is sudden drop attack, usually without warning, absence of aura and post-ictal signs, and no loss of consciousness unless head injury resulting from falling [25]. Physical examination and tests were performed to exclude other disorders [25], such as: 1) syncope resulting from vasovagal faint, carotid sinus syndrome, orthostatic syncope; 2) cardiac arrhythmias, structural cardiac and pulmonary causes; and 3) cerebrovascular disorders. The latter two categories could be fatal [26].

vHIT

The caloric irrigation and vHIT (ICS Impulse Type 1085, Denmark) were performed during the interictal period. Quantitative head impulse testing was performed using a video-oculography (VOG) device. Patients were sitting and asked to fixate a target at about 1.2 m distance. The target head velocity was between 150 and 200°/s and head displacement ranged between 10° and 20° for a duration of 150 - 200 ms [27].

Reduced vestibular ocular reflex (VOR) gain and overt or covert catch-up saccades are the two pathological signs of vHIT [28]. Patients with unilateral vestibular loss (UVL) have a reduced VOR gain of less than 0.7 during the head turn to their affected ear [29].

Bithermal caloric irrigation (AIRCAL Type 1079, Denmark)

Patients were positioned comfortably and briefly explained what the caloric test (Cal test) entails, having the subject supine with the head raised at 30° to make the horizontal SCC in the vertical plane [30]. Before the first irrigation, with the patient in the test position, spontaneous nystagmus was checked to exclude central origin. If spontaneous nystagmus was present, its direction was recorded.

The air temperatures at the outlet of the irrigator and the flow rate were: air (warm 50°, cold 24°) ± 0.4 °C, 8 ± 0.4 L in 60 s [31]. The four irrigations were carried out in the following order: right cold, left cold, right warm, left warm. The total recording time, following each irrigation, should be at least 60 s, allowing 5 min between irrigations for the inner ear temperature to stabilize before the next irrigation.

If the sum of the same ear response to cold and warm irrigation was less than 12°/s in magnitude for both ears, the result should be interpreted as evidence of bilateral canal paresis [32]. If the sum of the same ear response of cold and warm irrigation was equal to or exceeded 12°/s in magnitude, then we proceeded to calculate percentage canal paresis (UW) and directional preponderance (DP), which could be read from the recording equipment.

If the asymmetry between the responses for the left and right ears was ≥ 25% (UW ≥ 25%), the result was indicative of significant unilateral canal paresis. For DP, a difference between the right and left beating nystagmus of more than 30% (DP ≥ 30%) was considered pathological. The Cal test was considered normal when both canal paresis and DP were normal [33, 34].

Statistical analysis

Statistical analyses were completed using SPSS Statistics 26 software (SPSS Inc., Chicago, IL). The measurement data were analyzed using the Student's *t*-test, enumeration data using the Pearson's χ^2 test, and the rate of large samples using the U-test. A P-value of 0.05 was considered statistically significant.

Results

Comparison of sex and age for the three groups

The age averages were in the following order: migraine < VM < TTH. There was a significant difference among the groups (P < 0.001 between groups 1 and 2, groups 1 and 3; P < 0.05 between groups 2 and 3).

Gender distributions were similar between migraine and VM (P > 0.05, Pearson's χ^2), migraine and TTH (P > 0.05), but there was a significant difference between VM and TTH (P < 0.01). In VM, the female preponderance was 77.43%, while 70.42% in migraine, and 58.57% in TTH (Table 1).

Comparison of spontaneous nystagmus, drop attack and BPPV

There were significant differences for spontaneous nystagmus

Table 2. Comparison for Spontaneous Nystagmus, Drop Attack and BPPV

Group	n	Spontaneous nystagmus, n (%)	Drop attack, n (%)	BPPV, n (%)	Comparison between groups	P		
						Nystagmus	Drop attack	BPPV
1. Migraine	142	3 (2.11%)	1 (0.70%)	4 (2.82%)	1 and 2	< 0.01	> 0.05	< 0.02
2. VM	226	22 (9.73)	6 (2.65%)	22 (9.73%)	1 and 3	> 0.05	> 0.05	> 0.05
3. TTH	140	4 (2.86%)	2 (1.43%)	2 (1.43%)	2 and 3	< 0.02	> 0.05	< 0.01
Total	508	29 (5.71%)	9 (1.77%)	28 (5.51%)				

BPPV: benign paroxysmal positional vertigo; TTH: tension-type headache; VM: vestibular migraine.

Table 3. Comparison of Patients of Defect, Intolerance and Bilateral Vestibulopathy

Group	n	Invalid, n (%)	BVP, n (%)	Intolerance, n (%)	Comparison between groups	P		Residual cases (n)
						BVP	Intolerance	
1. Migraine	142	6 (4.23%)	16 (11.76%)	17 (12.5%)	1 and 2	> 0.05	> 0.05	103
2. VM	226	3 (1.33%)	35 (15.70%)	25 (11.21%)	1 and 3	> 0.05	> 0.05	162
3. TTH	140	2 (1.43%)	17 (12.32%)	12 (8.70%)	2 and 3	> 0.05	> 0.05	110
Total	508	11 (2.17%)	68 (13.68%)	54 (10.87%)				375

BVP: bilateral vestibulopathy; TTH: tension-type headache; VM: vestibular migraine.

between VM (9.73%) and migraine (2.11%) (P < 0.01), VM and TTH (2.86%) (P < 0.02). There was no significant difference in spontaneous nystagmus between migraine and TTH (P > 0.05).

The drop attack episodes in VM were higher (2.65%) than in TTH (1.43%) and migraine (0.70%), but there were no significant differences among the three groups (P > 0.05).

There were significant differences for BPPV between VM (9.73%) and migraine (2.82%) (P < 0.02), VM and TTH (1.43%) (P < 0.01). There was no significant difference in BPPV between migraine and TTH (P > 0.05) (Table 2).

Comparison of Cal test intolerance and bilateral weakness

There were a similar number of cases of Cal test intolerance and bilateral weakness among the three groups (P > 0.05, Pearson's χ^2). To exclude invalid cases (refusing the Cal test), intolerance and bilateral vestibulopathy (BVP), the remaining cases in three groups were 103 in migraine group, 162 in the VM group, and 110 in the TTH group (Table 3).

Comparison of cases with DP ≥ 30%

In VM, the number of cases with DP ≥ 30% (12.96%) was higher than in the migraine (8.74%) and TTH (8.18%), but there were no significant differences among the three groups (P > 0.05, Pearson's χ^2) (Table 4).

Comparison of UW

Comparison of UW ≥ 25% among the three groups showed that there was a significantly higher UW percentage in the VM group (47.92%) than in migraine (26.80%) (P < 0.005, Pearson's χ^2) and in TTH (35.05%) (P < 0.05), but there was no significant difference between migraine and TTH (P > 0.05) (Table 5).

Comparison of UW-ranked data

According to the severity of UW, the scale of the ranks is defined as follows: grade 1: UW < 15%; grade 2: 15% ≤ UW < 25%; grade 3: 25% ≤ UW < 50%; grade 4: UW ≥ 50%.

Comparison among groups in grade 4 (profound UW)

There was a significant difference in the percentage of the ad-

Table 4. Comparison of Cases With DP ≥ 30%

Group	n	DP ≥ 30%, n (%)	Comparison between groups	P
1. Migraine	103	9 (8.74%)	1 and 2	> 0.05
2. VM	162	21 (12.96%)	1 and 3	> 0.05
3. TTH	110	9 (8.18%)	2 and 3	> 0.05

DP: directional preponderance; TTH: tension-type headache; VM: vestibular migraine.

Table 5. Comparison of UW

Group	n	UW ≥ 25%, n (%)	Comparison between groups	P (χ^2 test)
1. Migraine	103	28 (26.80%)	1 and 2	< 0.005
2. VM	162	78 (47.92%)	1 and 3	> 0.05
3. TTH	110	39 (35.05%)	2 and 3	< 0.05

UW: unilateral weakness; TTH: tension-type headache; VM: vestibular migraine.

Table 6. Comparison of Ranked Data for UW Among Three Groups

Group	n	Grade 3, n (%)	Grade 4, n (%)	Comparison between groups	P	
					Grade 3	Grade 4
1. Migraine	103	26 (25.24%)	2 (1.94%)	1 and 2	< 0.05	< 0.01
2. VM	162	60 (37.04%)	17 (10.4%)	1 and 3	> 0.05	< 0.05
3. TTH	110	28 (25.45%)	9 (8.18%)	2 and 3	< 0.05	> 0.05

UW: unilateral weakness; TTH: tension-type headache; VM: vestibular migraine.

vanced UW of the response to bithermal Cal test between VM patients and migraine (10.49% vs. 1.94%, P < 0.01, U-test), but there was no significant difference between VM and TTH (10.49% vs. 8.18%, P > 0.05). The percentage of the advanced UW in TTH was significantly higher than that in migraine (8.18% vs. 1.94%, P < 0.05).

Comparison among groups in grade 3 (moderate UW)

The percentage of grade 3 in VM was significantly higher than that in migraine (37.04% vs. 25.24%, P < 0.05, U-test) and in TTH groups (37.04% vs. 25.45%, P < 0.05, U-test), but there was no significant difference between migraine and TTH (25.24% vs. 25.45%, P > 0.05) (Table 6).

Comparison of vHIT

The percentage of unilateral and multiple canal overt and covert saccades in the VM group was significantly higher than that of migraine group (11.06% vs. 4.23%, P < 0.05, U-test). There were no differences in the percentage of catch-up saccades between migraine and TTH (4.23% vs. 7.14%, P > 0.05), and between VM and TTH (11.06% vs. 7.14%, P > 0.05).

The total percentage of saccades plus reduced VOR gain in VM was significantly higher than that in migraine (13.27% vs. 4.93%, P < 0.01, U-test). There were no significant differences in the percentage of the sum of catch-up saccade and reduced VOR gain between migraine and TTH (4.93% vs. 8.57%, P > 0.05), and between VM and TTH (13.27% vs. 8.57%, P > 0.05) (Table 7).

Comparison of abnormal vHIT (overt or covert saccades plus reduced VOT gain) and abnormal Cal (UW ≥ 25%)

There was 4.93% abnormal vHIT and 26.8% abnormal UW in migraine patients, 13.27% abnormal vHIT and 47.92% abnormal UW in VM patients, and 8.57% abnormal vHIT and 35.05% abnormal UW in the TTH group. The abnormal UW was significantly higher than the abnormal vHIT (P < 0.001, U-test) in the same group (Table 8).

Discussion

Relationship of migraine, VM, and TTH with the vestibular system, especially with peripheral vestibular system

Migraine, VM, and TTH are prevalent disorders with a well-established relationship to balance disorders, including both vertigo and postural instability. In 1873, Liveing published the first article that called attention to the association between vertigo and migraine [35]. Rossi et al recruited patients with migraine and TTH. The obtained stabilometric parameters indicated proprioceptive alterations, leading to balance disor-

Table 7. Comparison of vHIT in Three Groups

Group	n	Abnormal vHIT, n (%)				
		Saccades of single SCC	Saccades of multiple SCC	Total of saccades	Reduced VOR gain	Sum of abnormal
1. Migraine	142	4 (2.82%)	2 (1.41%)	6 (4.23%)*	1 (0.70%)	7 (4.93%)**
2. VM	226	17 (7.52%)	8 (3.54%)	25 (11.06%)	5 (2.21%)	30 (13.27%)
3. TTH	140	6 (4.29%)	4 (2.86%)	10 (7.14%)	2 (1.43%)	12 (8.57%)

*P < 0.05, **P < 0.01 between group 1 and group 2. SCC: semicircular canal; vHIT: video head impulse test; VOR: vestibular ocular reflex.

Table 8. Comparison in Sensitivity of vHIT and Cal Test in the Same Group

Group	Abnormal vHIT		Abnormal UW		P
	n	%	n	UW ≥ 25%	
Migraine	142	4.93%	103	26.8%	< 0.001
VM	226	13.27%	162	47.92%	< 0.001
TTH	140	8.57%	110	35.05%	< 0.001

Cal: caloric; UW: unilateral weakness; TTH: tension-type headache; vHIT: video head impulse test; VM: vestibular migraine.

ders, which were induced by cervicofacial muscle contraction of peripheral origin, in patients with migraine without aura. Alternations in optokinetic nystagmus (OKN) suggested impairments in the control of involuntary ocular motility, pointing to the central origin of these dysfunctions [36]. Asai et al showed that there were subclinical deviations of the subjective visual vertical (SVV) in migraine and TTH, which may be associated with their subjective imbalance [37].

Migraine is very prevalent, with population-based studies reporting in 4-6.5% of men and 11.2-18.2% of women in both the USA and Europe [38]. Neuhauser et al diagnosed migraine in 38% of 200 consecutive patients referred with complaints of dizziness [39]. Conversely, Bayazit et al reported dizziness in 30% and vertigo in 25% of migraineurs [40]. Migraine, rather than a headache in general, is uniquely linked to symptoms and signs of vestibulopathy such as vertigo, dizziness, unbalance, and blurred vision. Kayan and Hood reported vestibular symptoms in 54.5% of patients with migraine as opposed to 30.2% of patients with TTH [41]. The presence of co-morbid symptoms like vertigo with migraine predicts a longer lifetime duration of headaches and a poorer prognosis [41]. Hansson et al reported that phobic postural vertigo had the highest total score of dizziness handicap inventory (DHI) [42]; the vertigo frustrates patients more than headaches and may be referred for psychological counselling.

The epidemiology of TTH is less well-studied than that of migraine and is estimated in about 40% of people in the United States [43]. TTH is well defined as a primary headache in the ICHD classification [3]; although it is less severe, its association with instability draws the attention of the researchers [44]. Some evidence has shown significantly higher body sway during stance in TTH patients regardless of the visual conditions [36]. So et al used galvanic vestibular stimulation (GVS) to the mastoid processes of six patients with migraine, two with TTH and six healthy control subjects. All subjects exhibited balance problems, postural sway in the direction of the anode in response to the GVS perturbation. Headache subjects exhibited significantly larger peak responses to GVS [45]. These data showed for the first time that there is a relationship between postural instability in headache sufferers and changes in vestibular contribution [46].

Several works reported vestibular abnormalities in the form of involvement of peripheral or central vestibular pathways or both [47, 48]. Boldingh et al used a broad battery of bedside vestibular tests, a Cal test, and videonystagmography in migraine patients with or without vestibular symptoms, finding that 70% of the VM patients and 34% of the migraine patients showed abnormalities on one or more of the performed vestibular tests, including Romberg's test, smooth pursuit test, saccadic test, gaze-evoked nystagmus test, spontaneous nystagmus, positional test, horizontal headshake test, head impulse test, clinical testing of visual fixation suppression, subjective visual vertical, and bithermal Cal test. Mixed peripheral and central vestibular test abnormalities were found in 30% of VM patients and 13% of migraine patients; pure central vestibular signs were found in 25% of VM patients and 9% of migraine patients; pure peripheral vestibular signs were found in 16% of VM patients and 9% of migraine patients; no vestibular signs were found in 30% of VM patients and 66% of migraine patients [47]. Casani et al analyzed the results of a battery of neuro-otologic examinations in 22 migraine patients with the presence of vestibular symptoms. Group 1 included 22 migraine patients with the absence of vestibular symptoms, and group 2 included 22 normal subjects. Neurotologic abnormalities were observed in only 34% of the total, and the incidence was very similar in the two groups (36.3% versus 31.8%). Central vestibular involvement was observed in 18% of group 1 and 18% of group 2 patients. Peripheral vestibular involvement was demonstrated in 18% of group 1 patients and 16% of group 2 patients [48].

This study focused on the peripheral vestibular in migraine, VM, and TTH patients. Analyses aligned with Steiner's studies, showing that migraine predominantly afflicts young and middle-aged individuals [49]. The data revealed that the average age of VM patients is approximately 6 years older than those with migraine, and TTH patients are approximately 6 years older than those with VM. In VM and migraine, significant female preponderance was observed, while TTH showed no difference in occurrence between sexes.

Spontaneous nystagmus, VDA, and BPPV in patients with migraine, VM and TTH

Spontaneous nystagmus

Nystagmus is defined as an involuntary, rapid, rhythmic, and oscillatory movement of the eyes, characterized by a slow phase with a minimum amplitude of 1° [50]. The occurrence of spontaneous nystagmus indicates acute vestibular pathology without compensation of the central vestibular system. Therefore, the analysis of spontaneous nystagmus is crucial in assessing patients experiencing dizziness and vertigo.

Various central and peripheral patterns including spontaneous, positional, and mixed patterns of nystagmus have been reported during acute VM episodes [51, 52]. Less than 10% of

patients have demonstrated spontaneous interictal nystagmus [53, 54], with a slight increase when fixation was removed [55]. Published studies have reported interictal positional nystagmus in VM ranging from 0% to 28% [18, 53, 55]. Notably, central positional nystagmus, headshaking nystagmus, and saccadic pursuit have been observed in individuals with migraine without a history of vestibular symptoms [47, 48, 56], indicating the potential involvement of central and peripheral in these conditions.

Spontaneous nystagmus in the primary position, non-direction changing, and suppressible by fixation, typically indicates VN or peripheral vestibular lesions [22]. Pavlin-Premrl et al conducted a study with consecutive consenting patients experiencing acute vertigo from an emergency department and observed that several patients previously diagnosed with VN had complete resolution of all symptoms within 24 h. This rapid recovery of nystagmus raises the possibility of an alternative pathology, such as VM [11], indicating that peripheral vestibular system disorder may be the critical pathophysiological mechanism for VM [57].

In this study, a higher prevalence of peripheral spontaneous nystagmus was observed in patients with VM compared to those with migraine and TTH. There was no statistically significant difference in the occurrence of spontaneous nystagmus between migraine and TTH groups ($P > 0.05$). These findings indicated a greater degree of peripheral vestibular involvement in VM compared to migraine and TTH.

VDA or Tumarkin fall

In 1936, Tumarkin first documented sudden drop attacks in patients with recurrent vertigo episodes [58]. These attacks involve sensations of being pushed to the ground, leading to falls without loss of consciousness, this phenomenon has been known as Tumarkin's otolithic crisis or VDA [27]. VDA was thought to occur due to sudden changes in endolymphatic fluid pressure stimulating the end organs (probably deformation of the utricular or saccular macula) with a reflex-like vestibulospinal loss of postural tone. This unusual stimulation arising from the otolithic organ is conducted via the vestibular nucleus to the vestibulospinal tract ipsilaterally, and then to the motor neuron of the lower limbs occurs bilaterally [59]. This loss of balance between the excitatory signals to the extensor muscles and inhibitory signals to the flexor muscles leads to a sudden drop fall [60]. Drop attacks are typically associated with MD in approximately 72% of the patients during the Tumarkin phase [61]. However, to date, there have been no reports of such attacks in patients with VM, migraine, and TTH patients.

VDAs manifest in a range of severities, from mild symptoms like gait disturbances and needs for support due to postural instability, to more severe cases resulting in falls, often referred to as "drop attacks" [61, 62]. These attacks cause considerable alarm for the patients, as they could potentially lead to injury, reduction in general health-related quality of life, and higher anxiousness scores.

Ray et al demonstrated that the interaction between vestibular sympathetic reflex and baroreflex is additive in humans [63], where the dyssynergia between them could lead to vestibular syncope. Vestibular syncope is a reflex condition mediated by neural pathways, where vertigo-induced hemodynamic changes lead to presyncope with blurred vision, syncope with loss of consciousness postictal sweating, pallor, and fatigue, which is attributed to dyssynergia of the vestibulo-sympathetic reflex and baroreflex system [64, 65].

Syncope has been associated with Tumarkin attacks, migraine, a history of ischemic heart disease, and a history of cerebrovascular disease [66]. In this study, we enrolled patients with VDA, excluding vestibular syncope, and syncope deriving from cardiac and cerebral disorders. We found that the drop attack in VM patients was higher (2.65%) than in TTH (1.43%) and migraine (0.70%). However, the differences among these three groups were not statistically significant ($P > 0.05$), suggesting the need for further studies with a larger sample size.

BPPV

BPPV is diagnosed based on a history of transient episodes of vertigo, typically lasting less than 1 min. The Dix and Hallpike provided the provocative maneuver necessary for the diagnostic confirmation of the condition [67]. If nystagmus lacks latency and fatigability, a central origin of the symptoms may be indicated [11].

BPPV may occur primarily without causative factors or secondarily with vestibular pathology. A review of 1,932 articles published between 1975 and 2018 identified 17 potentially causative factors for BPPV, including aging, migraine, MD, trauma, chronic head and neck pain, vestibular or SCC pathology, and others. Migraine patients have a higher incidence of BPPV (6%) compared to 3.2% in control subjects [66, 67]. BPPV can manifest at any time from childhood to senility, but the idiopathic form is primarily affecting elderly patients, peaking in the sixth to seventh decades. In a previous study, 415 patients with VM were evaluated by using a structured questionnaire in addition to clinical examination, showing a 12.3% BPPV prevalence during the interictal period [68]. The importance of BPPV at the population level is still underestimated due to low recognition rates in primary care [69].

In our study, all outpatients underwent the Dix-Hallpike test and roll test. We observed significant differences in the BPPV incidence between VM patients and both migraine and TTH groups. There was no significant difference in the prevalence of BPPV between the migraine and TTH groups.

To sum up, the mechanism of VM may differ from that of migraine and TTH. Our study suggests a more profound peripheral vestibular involvement, which leads to spontaneous nystagmus, Tumarkin drop attack, and BPPV.

Cal test and vHIT for patients with migraine, VM and TTH

Cal test

The thermal caloric irrigation test, first introduced by Robert

Barany [70], is a tool for the assessment of the function of one single vestibular end organ-horizontal SCC, at a time. Since then, the test has become a mainstay of the clinical assessment of patients with dizziness. It remains the gold standard for detecting unilateral hypofunction of the horizontal SCC in acute vestibular syndrome or chronic vestibulopathy [71] through low-frequency stimuli, around 0.002 - 0.004 Hz [33].

In this study, the invalid cases (refusing the Cal test), intolerance, and bilateral vestibule-pathology (BVP) individuals were excluded from the Cal test. The remaining participants included 103 in migraine group, 162 in the VM group, and 110 in the TTH group. The incidence of abnormal DP (DP ≥ 30%) was similar across all three groups.

There was a significantly higher prevalence of UW in the VM group than in migraine and TTH groups, while the difference between migraine and TTH groups was not statistically significant. There was a significantly more profound loss of horizontal canal (HC) function (UW% ≥ 50%) in VM patients than those with migraines, while no significant difference was found when compared with TTH. Additionally, the incidence of profound HC loss in the TTH group was significantly higher than that in the migraine group. The incidence of moderate impairment of HC function (25% ≤ UW% < 50%) in VM was significantly higher than that in migraine and TTH groups, while no significant difference was found between the migraine and TTH groups.

These results showed a hierarchy in unilateral HC hypofunction at low frequencies in the following order: VM > TTH > migraine. To date, we have found no reported studies addressing the degree of HC UW for low frequency in these conditions.

vHIT test

In 1988, impulsive testing of SCC function at high frequency (about 5 Hz) measured with scleral search coils was introduced, proving its capacity for accurate and reliable detection of SCC function. However, this method was costly, complicated, and cumbersome [72]. In 2009 and 2013, MacDougall came up with a system using a high-speed head-mounted camera mounted on tight-fitting goggles with head velocity sensors, coupled with software for precise objective measurements of the head and eye velocities [73]. Since then, this innovation has become a widely used vHIT in dizzy laboratory [74].

Eye movement responses to small, brief, fast, unpredictable head turns (head impulses) are valid indicators of SCC function. The "overt" or "covert" catch-up saccade observed by clinicians serves as the signs of canal paresis [74]. However, this assessment is subjective, leading to the introduction of the objective parameter of VOR gain. VOR gain calculates the ratio of the eye velocity curve area to the head velocity curve area during the head impulse [75, 76].

In our vHIT test, the rate of unilateral and multiple canal overt and covert saccades in the VM group was significantly higher than in migraine, while no statistically significant differences were observed between the migraine and TTH groups or between VM and TTH groups.

The total percentage of saccades plus reduced VOR gain in the VM group was significantly higher than in the migraine group. There were no significant differences between the migraine and TTH groups or the VM and TTH groups. These results indicated that VM had a greater degree of SCC paresis for high frequency, while migraine had the least involvement.

Sensitivity of Cal and vHIT tests

In studies with data collected from vHIT and Cal tests in patients diagnosed with MD, the dissociation for these tests has been reported to be 30-91%, most commonly showing normal vHIT gains but canal paresis on Cal test [77, 78]. These discrepancies may vary according to the active or inactive phase of the disease, or the compensation status of peripheral vestibulopathy. For example, during the acute phase of VN, 78-95% of patients exhibit low vHIT gains and unilateral hypofunction in the Cal test [79, 80], with only 5-10% showing dissociated results [81-83]. As vHIT gains recover at a faster rate than Cal UW, it is possible to obtain Cal hypofunction even in compensated VN while the vHIT appears normal [79].

Current findings from testing patients with VM display considerable variability, which could be attributed to whether the testing occurs during ictal or interictal phases, as well as the duration of the VM condition [47]. It appears that Cal tests tend to yield more abnormal results in VM compared to the vHIT tests [84]. This pattern implies that the Cal is more sensitive in detecting abnormalities in peripheral vestibular function in VM patients [85]. DP on Cal has also been described as approximately 8-15% for migraine [47, 78, 86]. Kang et al retrospectively reviewed 81 patients with VM during their interictal phase. They found that the abnormal results in both vHIT and Cal tests revealed decompensated SCC dysfunction in VM [87]. We reported comparative results of a DP (≥ 30%) of 12.96% in VM, 8.74% in migraine, and 8.18% in TTH.

Our study showed 4.93% abnormal vHIT and 26.8% abnormal UW in migraine patients, 13.27% abnormal vHIT and 47.92% abnormal UW in VM patients, and 8.57% abnormal vHIT and 35.05% abnormal UW in TTH patients. Notably, the rate of abnormal UW was significantly higher than that of abnormal vHIT ($P < 0.001$) within the same group. These findings indicated the presence of a few decompensated SCC dysfunction during interictal, and that the Cal test was more sensitive than vHIT for SCC hypofunction detection.

Conclusions

Our study indicates a higher prevalence of peripheral vestibular nystagmus in VM patients compared to migraine and TTH patients. VDAs occurred slightly more frequently in VM than in migraine and TTH while not significant. BPPV prevalence was significantly higher in VM than in migraine and TTH. VDAs were also observed in primary headaches, not just in MD. VM patients showed more pathological findings in low-frequency hypofunction of the horizontal canal and high-frequency hypofunction in the SCC than in patients with migraine or TTH. The Cal test was more sensitive than the vHIT test in detecting SCC hypofunction. These results

suggest that the peripheral vestibular system of SCC could be the critical mechanism and pathophysiology of VM, and its involvement in migraine and TTH cases without vertigo. Further research is required to understand migraine's impact on the inner ear.

Acknowledgments

The authors express their gratitude to Su Zhen Wang, Department of Statistics, Weifang Medical College.

Financial Disclosure

This study was supported by the grant from the Bureau of Science and Technology of Weifang (grant No. 2021YX059).

Conflict of Interest

The authors declare no conflict of interest.

Informed Consent

Written informed consents were obtained from all participants.

Author Contributions

AJZ: study design, cases collection, literature retrieval, manuscript writing and final revision; LQY: vestibular function test; LZ: cases collection, patients management; XZC: statistical analysis; QHL: vestibular function test; WL: vestibular function test; AYZ: literature retrieval, dynamic ECG and cardiac color ultrasound analysis; cardiovascular falls screening.

Data Availability

The authors declare that data supporting the findings of this study are available within the article.

References

1. Furman JM, Balaban CD. Vestibular migraine. Ann N Y Acad Sci. 2015;1343:90-96. doi pubmed
2. Cho SJ, Kim BK, Kim BS, Kim JM, Kim SK, Moon HS, Song TJ, et al. Vestibular migraine in multicenter neurology clinics according to the appendix criteria in the third beta edition of the International Classification of Headache Disorders. Cephalalgia. 2016;36(5):454-462. doi pubmed
3. Headache Classification Committee of the International Headache Society (IHS). The international classification of headache disorders, 3rd edition (beta version). Cephalalgia. 2013;33(9):629-808. doi pubmed
4. Wattiez AS, Sarah A. O'Shea SA, Eyck PT, et al. Patients with vestibular migraine are more likely to have occipital headaches than those with migraine without vestibular symptoms. Headache. 2020;1-11.
5. Neuhauser HK, Radtke A, von Brevern M, Feldmann M, Lezius F, Ziese T, Lempert T. Migrainous vertigo: prevalence and impact on quality of life. Neurology. 2006;67(6):1028-1033. doi pubmed
6. Goadsby PJ, Holland PR, Martins-Oliveira M, Hoffmann J, Schankin C, Akerman S. Pathophysiology of migraine: a disorder of sensory processing. Physiol Rev. 2017;97(2):553-622. doi pubmed pmc
7. Chari DA, Liu YH, Chung JJ, Rauch SD. Subjective cognitive symptoms and dizziness handicap inventory (DHI) performance in patients with vestibular migraine and Meniere's disease. Otol Neurotol. 2021;42(6):883-889. doi pubmed
8. Zamyslowska-Szmytke E, Politanski P, Jozefowicz-Korczynska M. Dizziness handicap inventory in clinical evaluation of dizzy patients. Int J Environ Res Public Health. 2021;18(5):2210. doi pubmed pmc
9. Russo A, Marcelli V, Esposito F, Corvino V, Marcuccio L, Giannone A, Conforti R, et al. Abnormal thalamic function in patients with vestibular migraine. Neurology. 2014;82(23):2120-2126. doi pubmed
10. Messina R, Rocca MA, Colombo B, Teggi R, Falini A, Comi G, Filippi M. Structural brain abnormalities in patients with vestibular migraine. J Neurol. 2017;264(2):295-303. doi pubmed
11. Pavlin-Premrl D, Waterston J, McGuigan S, Infeld B, Sultana R, O'Sullivan R, Gerraty RP. Importance of spontaneous nystagmus detection in the differential diagnosis of acute vertigo. J Clin Neurosci. 2015;22(3):504-507. doi pubmed
12. Teggi R, Colombo B, Bernasconi L, Bellini C, Comi G, Bussi M. Migrainous vertigo: results of caloric testing and stabilometric findings. Headache. 2009;49(3):435-444. doi pubmed
13. Benjamin T, Gillard D, Abouzari M, Djalilian HR, Sharon JD. Vestibular and auditory manifestations of migraine. Curr Opin Neurol. 2022;35(1):84-89. doi pubmed pmc
14. Du Y, Liu X, Ren L, Wang Y, Wu Z. Analysis of video head impulse test saccades data in patients with vestibular migraine or probable vestibular migraine. J Otol. 2022;17(4):197-202. doi pubmed pmc
15. Momtaz S, Hajiabolhassan F, Togha M, Jalaie S, Almasi A. Quantitative oculomotor findings in migrainous patients. Iran J Neurol. 2014;13(4):250-252. pubmed pmc
16. Corrado M, Demartini C, Greco R, Zanaboni A, Bighiani F, Vaghi G, Grillo V, et al. Oculo-vestibular signs in experimentally induced migraine attacks: an exploratory analysis. Neurol Sci. 2022;43(11):6561-6564. doi pubmed pmc
17. Lee SU, Choi JY, Kim HJ, Kim JS. Recurrent spontaneous vertigo with interictal headshaking nystagmus. Neurology. 2018;90(24):e2135-e2145. doi pubmed
18. Beh SC, Masrour S, Smith SV, Friedman DI. The spec-

trum of vestibular migraine: clinical features, triggers, and examination findings. Headache. 2019;59(5):727-740. doi pubmed
19. Starkov D, Strupp M, Pleshkov M, Kingma H, van de Berg R. Diagnosing vestibular hypofunction: an update. J Neurol. 2021;268(1):377-385. doi pubmed pmc
20. Wieling W, Kaufmann H, Claydon VE, van Wijnen VK, Harms MPM, Juraschek SP, Thijs RD. Diagnosis and treatment of orthostatic hypotension. Lancet Neurol. 2022;21(8):735-746. doi pubmed pmc
21. Serra A, Leigh RJ. Diagnostic value of nystagmus: spontaneous and induced ocular oscillations. J Neurol Neurosurg Psychiatry. 2002;73(6):615-618. doi pubmed pmc
22. Hotson JR, Baloh RW. Acute vestibular syndrome. N Engl J Med. 1998;339(10):680-685. doi pubmed
23. Younga AS, Rosengrena SM, Souzaa MD, Bradshawc AP, Miriam S. Welgampola Nystagmus characteristics of healthy controls. Journal of Vestibular Research. 2020.
24. Kattah JC. Update on HINTS plus, with discussion of pitfalls and pearls. J Neurol Phys Ther. 2019;43(Suppl 2):S42-S45. doi pubmed
25. Black FO, Effron MZ, Burns DS. Diagnosis and management of drop attacks of vestibular origin: Tumarkin's otolithic crisis. Otolaryngol Head Neck Surg. 1982;90(2):256-262. doi pubmed
26. Benditt DG, van Dijk JG, Sutton R, Wieling W, Lin JC, Sakaguchi S, Lu F. Syncope. Curr Probl Cardiol. 2004;29(4):152-229. doi pubmed
27. Alhabib SF, Saliba I. Video head impulse test: a review of the literature. Eur Arch Otorhinolaryngol. 2017;274(3):1215-1222. doi pubmed
28. Du Y, Ren L, Liu X, Guo W, Wu Z, Yang S. The characteristics of vHIT gain and PR score in peripheral vestibular disorders. Acta Otolaryngol. 2021;141(1):43-49. doi pubmed
29. Halmagyi GM, Chen L, MacDougall HG, Weber KP, McGarvie LA, Curthoys IS. The Video Head Impulse Test. Front Neurol. 2017;8:258. doi pubmed pmc
30. Coats AC, Smith SY. Body position and the intensity of caloric nystagmus. Acta Otolaryngol. 1967;63(6):515-532. doi pubmed
31. Recommended procedure. British Journal of Audiology. 1999;33:179-184.
32. Stockwell CW. Bilateral weakness. ENG report. ICS Medical. 1993;73-75.
33. Shepard NT, Jacobson GP. The caloric irrigation test. Handb Clin Neurol. 2016;137:119-131. doi pubmed
34. Burnette E, Piker EG, Frank-Ito D. Reevaluating order effects in the binaural bithermal caloric test. Am J Audiol. 2018;27(1):104-109. doi pubmed
35. Liveing E. Observations on Megrim or Sick-Headache. Br Med J. 1872;1(588):364-366. doi pubmed pmc
36. Rossi C, Alberti A, Sarchielli P, Mazzotta G, Capocchi G, Faralli M, Ricci G, et al. Balance disorders in headache patients: evaluation by computerized static stabilometry. Acta Neurol Scand. 2005;111(6):407-413. doi pubmed
37. Asai M, Aoki M, Hayashi H, Yamada N, Mizuta K, Ito Y. Subclinical deviation of the subjective visual vertical in patients affected by a primary headache. Acta Otolaryngol. 2009;129(1):30-35. doi pubmed
38. Lipton RB, Stewart WF, Diamond S, Diamond ML, Reed M. Prevalence and burden of migraine in the United States: data from the American Migraine Study II. Headache. 2001;41(7):646-657. doi pubmed
39. Neuhauser H, Leopold M, von Brevern M, Arnold G, Lempert T. The interrelations of migraine, vertigo, and migrainous vertigo. Neurology. 2001;56(4):436-441. doi pubmed
40. Bayazit Y, Yilmaz M, Mumbuc S, Kanlikama M. Assessment of migraine-related cochleovestibular symptoms. Rev Laryngol Otol Rhinol (Bord). 2001;122(2):85-88. pubmed
41. Guidetti V, Galli F, Fabrizi P, Giannantoni AS, Napoli L, Bruni O, Trillo S. Headache and psychiatric comorbidity: clinical aspects and outcome in an 8-year follow-up study. Cephalalgia. 1998;18(7):455-462. doi pubmed
42. Kayan A, Hood JD. Neuro-otological manifestations of migraine. Brain. 1984;107(Pt 4):1123-1142. doi pubmed
43. Schwartz BS, Stewart WF, Simon D, Lipton RB. Epidemiology of tension-type headache. JAMA. 1998;279(5):381-383. doi pubmed
44. Saylor D, Steiner TJ. The Global Burden of Headache. Semin Neurol. 2018;38(2):182-190. doi pubmed
45. Day BL, Severac Cauquil A, Bartolomei L, Pastor MA, Lyon IN. Human body-segment tilts induced by galvanic stimulation: a vestibularly driven balance protection mechanism. J Physiol. 1997;500(Pt 3):661-672. doi pubmed pmc
46. So CW, Bent LR. Increased vestibular contribution to posture control in individuals with chronic headache. J Vestib Res. 2009;19(1-2):49-58. doi pubmed
47. Boldingh MI, Ljostad U, Mygland A, Monstad P. Comparison of interictal vestibular function in vestibular migraine vs migraine without vertigo. Headache. 2013;53(7):1123-1133. doi pubmed
48. Casani AP, Sellari-Franceschini S, Napolitano A, Muscatello L, Dallan I. Otoneurologic dysfunctions in migraine patients with or without vertigo. Otol Neurotol. 2009;30(7):961-967. doi pubmed
49. Steiner TJ, Stovner LJ, Vos T, Jensen R, Katsarava Z. Migraine is first cause of disability in under 50s: will health politicians now take notice? J Headache Pain. 2018;19(1):17. doi pubmed pmc
50. Eggers SDZ. Approach to the examination and classification of nystagmus. J Neurol Phys Ther. 2019;43(Suppl 2):S20-S26. doi pubmed
51. von Brevern M, Zeise D, Neuhauser H, Clarke AH, Lempert T. Acute migrainous vertigo: clinical and oculographic findings. Brain. 2005;128(Pt 2):365-374. doi pubmed
52. Polensek SH, Tusa RJ. Nystagmus during attacks of vestibular migraine: an aid in diagnosis. Audiol Neurootol. 2010;15(4):241-246. doi pubmed
53. Cass SP, Furman JM, Ankerstjerne K, Balaban C, Yetiser S, Aydogan B. Migraine-related vestibulopathy. Ann Otol Rhinol Laryngol. 1997;106(3):182-189. doi

pubmed
54. Neugebauer H, Adrion C, Glaser M, Strupp M. Long-term changes of central ocular motor signs in patients with vestibular migraine. Eur Neurol. 2013;69(2):102-107. doi pubmed
55. Radtke A, von Brevern M, Neuhauser H, Hottenrott T, Lempert T. Vestibular migraine: long-term follow-up of clinical symptoms and vestibulo-cochlear findings. Neurology. 2012;79(15):1607-1614. doi pubmed
56. Harno H, Hirvonen T, Kaunisto MA, Aalto H, Levo H, Isotalo E, Kallela M, et al. Subclinical vestibulocerebellar dysfunction in migraine with and without aura. Neurology. 2003;61(12):1748-1752. doi pubmed
57. Altunay ZO, Ozkarakas H. Comparison of the efficacy of video head impulse and bi-thermal caloric tests in vertigo. Ear Nose Throat J. 2021;100(10):742-748. doi pubmed
58. Tumarkin A. The otolithic catastrophe: a new syndrome. Br Med J. 1936;2(3942):175-177. doi pubmed pmc
59. Brandt T, Daroff RB. The multisensory physiological and pathological vertigo syndromes. Ann Neurol. 1980;7(3):195-203. doi pubmed
60. Ozeki H, Iwasaki S, Murofushi T. Vestibular drop attack secondary to Meniere's disease results from unstable otolithic function. Acta Otolaryngol. 2008;128(8):887-891. doi pubmed
61. Kentala E, Havia M, Pyykko I. Short-lasting drop attacks in Meniere's disease. Otolaryngol Head Neck Surg. 2001;124(5):526-530. doi pubmed
62. Baloh RW, Jacobson K, Winder T. Drop attacks with Meniere's syndrome. Ann Neurol. 1990;28(3):384-387. doi pubmed
63. Pyykko I, Manchaiah V, Zou J, Levo H, Kentala E. Association between syncope and Tumarkin attacks in Meniere's disease. J Int Adv Otol. 2019;15(1):135-140. doi pubmed pmc
64. Kwon E, Lee JY, Kim HJ, Choi JY, Kim JS. Can dyssynergia of vestibulosympathetic and baroreflexes cause vestibular syncope? The Hypothesis Based on the Velocity-Storage Function. Cerebellum. 2022;21(2):244-252. doi pubmed
65. Kwon H, Kwon E, Kim HJ, Choi JY, Kim JS. Vestibular syncope: clinical characteristics and mechanism. Ann Clin Transl Neurol. 2022;9(10):1616-1625. doi pubmed pmc
66. Pyykko I, Manchaiah V, Zou J, Levo H, Kentala E. Vestibular syncope: A disorder associated with drop attack in Meniere's disease. Auris Nasus Larynx. 2018;45(2):234-241. doi pubmed
67. Yetiser S. Review of the pathology underlying benign paroxysmal positional vertigo. J Int Med Res. 2020;48(4):300060519892370. doi pubmed pmc
68. Celebisoy N, Kisabay Ak A, Ozdemir HN, Gokcay F, Durmaz GS, Karti DT, Toydemir HE, et al. Vestibular migraine, demographic and clinical features of 415 patients: A multicenter study. Clin Neurol Neurosurg. 2022;215:107201. doi pubmed
69. von Brevern M, Lezius F, Tiel-Wilck K, Radtke A, Lempert T. Benign paroxysmal positional vertigo: current status of medical management. Otolaryngol Head Neck Surg. 2004;130(3):381-382. doi pubmed
70. Barany R. Physiologic and Pathologic des Bogengangapparates beim Menschen. Deuticke, Vienna, Australia. 1907.
71. Baloh RW. Robert Barany and the controversy surrounding his discovery of the caloric reaction. Neurology. 2002;58(7):1094-1099. doi pubmed
72. Halmagyi GM, Curthoys IS. A clinical sign of canal paresis. Arch Neurol. 1988;45(7):737-739. doi pubmed
73. Weber KP, MacDougall HG, Halmagyi GM, Curthoys IS. Impulsive testing of semicircular-canal function using video-oculography. Ann N Y Acad Sci. 2009;1164:486-491. doi pubmed
74. MacDougall HG, Weber KP, McGarvie LA, Halmagyi GM, Curthoys IS. The video head impulse test: diagnostic accuracy in peripheral vestibulopathy. Neurology. 2009;73(14):1134-1141. doi pubmed pmc
75. Macdougall HG, McGarvie LA, Halmagyi GM, Curthoys IS, Weber KP. The video Head Impulse Test (vHIT) detects vertical semicircular canal dysfunction. PLoS One. 2013;8(4):e61488. doi pubmed pmc
76. Cleworth TW, Carpenter MG, Honegger F, Allum JHJ. Differences in head impulse test results due to analysis techniques. J Vestib Res. 2017;27(2-3):163-172. doi pubmed
77. McGarvie LA, Curthoys IS, MacDougall HG, Halmagyi GM. What does the dissociation between the results of video head impulse versus caloric testing reveal about the vestibular dysfunction in Meniere's disease? Acta Otolaryngol. 2015;135(9):859-865. doi pubmed
78. Yilmaz MS, Egilmez OK, Kara A, Guven M, Demir D, Genc Elden S. Comparison of the results of caloric and video head impulse tests in patients with Meniere's disease and vestibular migraine. Eur Arch Otorhinolaryngol. 2021;278(6):1829-1834. doi pubmed
79. Redondo-Martinez J, Becares-Martinez C, Orts-Alborch M, Garcia-Callejo FJ, Perez-Carbonell T, Marco-Algarra J. Relationship between video head impulse test (vHIT) and caloric test in patients with vestibular neuritis. Acta Otorrinolaringol Esp. 2016;67(3):156-161. doi pubmed
80. Bartolomeo M, Biboulet R, Pierre G, Mondain M, Uziel A, Venail F. Value of the video head impulse test in assessing vestibular deficits following vestibular neuritis. Eur Arch Otorhinolaryngol. 2014;271(4):681-688. doi pubmed
81. Jung J, Suh MJ, Kim SH. Discrepancies between video head impulse and caloric tests in patients with enlarged vestibular aqueduct. Laryngoscope. 2017;127(4):921-926. doi pubmed
82. Hannigan IP, Welgampola MS, Watson SRD. Dissociation of caloric and head impulse tests: a marker of Meniere's disease. J Neurol. 2021;268(2):431-439. doi pubmed
83. Shugyo M, Ito T, Shiozaki T, Nishikawa D, Ohyama H, Fujita H, Yamanaka T, et al. Comparison of the video head impulse test results with caloric test in patients with Meniere's disease and other vestibular disorders.

Acta Otolaryngol. 2020;140(9):728-735. doi pubmed
84. Blodow A, Heinze M, Bloching MB, von Brevern M, Radtke A, Lempert T. Caloric stimulation and video-head impulse testing in Meniere's disease and vestibular migraine. Acta Otolaryngol. 2014;134(12):1239-1244. doi pubmed
85. Yoo MH, Kim SH, Lee JY, Yang CJ, Lee HS, Park HJ. Results of video head impulse and caloric tests in 36 patients with vestibular migraine and 23 patients with vestibular neuritis: a preliminary report. Clin Otolaryngol. 2016;41(6):813-817. doi pubmed
86. Waissbluth S, Sepulveda V. Dissociation between caloric and video head impulse tests in dizziness clinics. Audiol Res. 2022;12(4):423-432. doi pubmed pmc
87. Kang WS, Lee SH, Yang CJ, Ahn JH, Chung JW, Park HJ. Vestibular function tests for vestibular migraine: clinical implication of video head impulse and caloric tests. Front Neurol. 2016;7:166. doi pubmed pmc