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ORIGINAL COMMUNICATION

The Dizziness Handicap Inventory does not correlate with vestibular function tests: a prospective study

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Abstract
The Dizziness Handicap Inventory (DHI) is believed to quantitate the handicap related to the presence or severity of underlying vestibular dysfunction. However, patients with chronic vestibular diseases may manifest various degrees of behavioural and physiological adaptation resulting in variances of the DHI. Our primary study objective is to evaluate the correlation between the DHI and measurable vestibular parameters. Secondarily, we compared DHI among different vestibular disorders (central, peripheral and functional), and different types of anatomic deficits (semicircular canal vs otolithic). We also correlated the DHI and posturography. We prospectively evaluated 799 patients with precise vestibular diagnoses using video head impulse testing (vHIT), caloric irrigation, and cervical/ocular vestibular-evoked myogenic potentials (c/oVEMP). Posturography was done for 84 patients. All participants completed the DHI. No significant correlation was found between DHI and (1) vestibulo-ocular reflex parameters: unilateral weakness $r=-0.018$, total calorics $r=0.055$, vHIT right $r=0.007$, vHIT left $r=-0.091$, vHIT asymmetry $r=0.013$; (2) otolith parameters: cVEMP amplitude right $r=-0.034$, amplitude left $r=-0.004$, asymmetry $r=0.016$; oVEMP amplitude right $r=0.044$, amplitude left $r=-0.007$, asymmetry $r=-0.008$. Patients with central vestibular disorders had higher DHI than those with peripheral ($z=-4.743$, $p=0.001$) or functional disorders ($z=-2.902$, $p=0.004$). DHI of patients with deficits of canal or otolith function did not differ significantly from those with no deficits ($z=2.153$, $p=0.541$). There was no significant correlation between DHI and postural sway on posturography. Therefore, the DHI does not correlate with vestibular tests, and neither reflects the presence nor severity of peripheral vestibular deficits.

Keywords Dizziness Handicap Inventory · Video head impulse test · Caloric testing · Vestibular-evoked myogenic potential

Introduction

The Dizziness Handicap Inventory (DHI) [1] is a validated scale of impairment widely used in clinical practice and many clinical studies [2–4]. The DHI is assumed to provide the clinician with an estimate of the severity of the underlying vestibular dysfunction. A high DHI score is associated with an increased level of handicap. Previous studies have demonstrated correlation of the DHI with posturography [5] and dynamic gait index testing [6]. However, there is no large study correlating semicircular canal and otolith function with the DHI in dizzy patients with central, peripheral or primary functional vestibular disturbances.

Peripheral vestibular deficits, and, to a lesser extent, central vestibular deficits, are amenable to central compensation [7–11] and sensory rebalancing to optimise gaze stability and balance function. The degree of compensation and the behavioural strategies used differ among individuals and may be influenced by age, medication use, socio-cultural background, cognitive resilience, physical activity, etc. Hence, the functional consequences of an underlying vestibular lesion may be variable. Therefore, we hypothesize that objectively measured vestibular deficits do not correlate with the patient-reported DHI.

Using data collected from a large outpatient cohort, our primary aim was to determine the correlation between DHI and (a) semicircular canal function, i.e. the angular vestibulo-ocular reflex (VOR) in the low-frequency (measured by

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caloric irrigation) and the high-frequency range (measured by video head impulse testing), (b) otolith function, measured with cervical and ocular vestibular-evoked myogenic potentials (VEMP). Our secondary aim was to compare the DHI in patient groups with (a) peripheral, central and primary functional (including psychogenic) vestibular disorders, (b) canal-type disorders and otolith-type disorders, and (c) to correlate the DHI with static posturography (a test of stance and somatosensory integration).

Methods

Study participants

The patients were prospectively recruited from the tertiary outpatient clinic of the German Vertigo and Balance Center at the University of Munich, Campus Grosshadern from 1 May 2014 to 31 April 2017.

Inclusion criteria

(1) Patients with persisting (i.e. longer than 3 months) vestibular signs and symptoms either due to a persisting vestibular deficit and/or due to recurrent attacks of vertigo/dizziness. (2) Patients fulfilling the diagnostic criteria for vestibular neuritis/unilateral vestibulopathy, bilateral vestibulopathy, vestibular migraine, benign paroxysmal positional vertigo (BPPV), Menière's disease, and vestibular paroxysmia (based on the International Classification of Vestibular Disorders) [12–15]. (3) Patients with central cause of dizziness due to stroke proven on MRI and do not have a concurrent diagnosis of any vestibular disorder listed in (2). (4) Patients with primary functional dizziness (who fulfil criteria for phobic postural vertigo [16] or chronic subjective dizziness [17], which is currently known as persistent postural-perceptual dizziness [18]), or psychogenic dizziness.

Exclusion criteria

(1) Patients with more than one diagnosis, e.g. concurrent Menière's disease and vestibular migraine. (2) Patients with "secondary" functional dizziness associated with a coexisting/previous vestibular diagnosis e.g. post-vestibular neuritis.

A total of 799 patients with a clear vestibular diagnosis were included: 320 females and 479 males (see Table 1). This study was conducted in parallel to routine clinical practice, and the patients gave verbal informed consent for their data to be used. As such, the hospital Ethics Committee approved the study with a waiver of formal written consent. The study was performed in accordance with the ethical principles laid down in the 1964 Declaration of Helsinki.

Patient examinations

All patients underwent a complete neurological, neuro-otological and neuro-ophthalmological examination during the first clinical consultation upon joining the study.

Vestibular function was assessed with the video head impulse test (vHIT), caloric irrigation and cervical/ocular VEMP testing. All the vestibular function tests, including posturography, were performed on the same day. It was not mandatory for all patients to undergo all the tests, but all

Table 1 Study participants' demographics and vestibular diagnosis

Sex	No. (*n*)	Mean age (years) ± SD
Female	320	60.0 ± 17
Male	479	62.5 ± 16
Vestibular diagnosis	No. (*n*)	Percentage (%)
BPPV	145	18.1
BVP	78	9.8
Central dizziness	39	4.9
Cerebellar ataxia	34	4.3
Downbeat nystagmus	19	2.4
Functional/psychogenic dizziness	125	15.6
Menière's disease	117	14.6
Unilateral peripheral vestibulopathy	147	18.4
Vestibular migraine	68	8.5
Vestibular paroxysmia	27	3.4
Total	799	100

SD standard deviation, *BPPV* benign paroxysmal positioning nystagmus, *BVP* bilateral vestibulopathy, *DBN* downbeat nystagmus syndrome

patients in the study had to fill out a DHI questionnaire. All the data were collected only once.

Vestibular function tests

Video head impulse test (vHIT)

For vHIT testing, the Otometrics® vHIT system was used. The horizontal angular VOR was tested. The method has been described in our previous paper [19]. Briefly, the patient sat 1 m in front of the wall with a small target affixed at eye level. The vHIT goggles were tightly strapped onto the patient's head. Rapid and unpredictable head impulses were applied in each direction i.e. left and right (to test the horizontal semicircular canal VOR) with the examiner's hands tightly holding the patient's head without touching the goggle straps. The procedure ended when the machine had registered seven correctly performed head impulses in both directions. The averaged positional gain for each direction was calculated according to the software algorithm.

Caloric irrigation

Caloric testing was done according to standard practice with the patient lying reclined with the head at 30° and wearing video oculography goggles with opaque lenses to prevent fixation. Binaural bithermal irrigations at 30 and 42 °C, respectively, were done in sequence with a 5 min delay between irrigations. The resultant vestibular nystagmus was recorded and de-saccaded to yield plots of the slow phase vestibular eye movements. The total caloric response and side difference in caloric responses (indicating unilateral weakness) was calculated by the software using the Jongkees formula.

Vestibular-evoked myogenic potentials

The recording of the ocular and cervical VEMPs was done as in our previous studies [20, 21].

Briefly, for the cervical VEMP, the recording electrode was attached to the belly of the ipsilateral sternocleidomastoid muscle, the reference electrode was attached to the ipsilateral sterno-clavicular joint, and the ground electrode attached to the midline forehead. Air-conducted 500 Hz, 120 dB SPL short tone bursts were used for monoaural ipsilateral stimulation of the saccule via earphone inserts. The patient lay on a couch with the head elevated 30°. During the stimulation phase, the patient was instructed to turn and lift the head in the opposite direction to the stimulated ear. The EMG activity was monitored and adjusted real time on the machine during the procedure. The ipsilateral p13–n23 peak-to-peak amplitude was derived from the average of 100 recordings.

For the ocular VEMP, a Bruel and Kjaer Type 4810 minishaker (input signal: 2 ms clicks of positive polarity at a repetition rate of 2 Hz) provided the stimulation to the utricle. This was positioned on the patient's forehead in the midline (Fz). The recording electrode was attached approximately 3 mm below the lower eyelid in line with the pupil and the reference electrode just below the recording electrode. The ground electrode was attached to the patient's chin. The patient was asked to look upwards about 30° during the bone-conducted stimulation for maximum activation of the inferior oblique muscle. The procedure was completed after responses to 100 stimuli were obtained and averaged. The n10–p15 peak-to-peak amplitude was then calculated for each eye (and reflects contralateral utricular activation).

We defined the following as abnormal: vHIT gain < 0.7, side differences in caloric response $> 25\%$ (i.e. unilateral canal weakness), and/or total bilateral caloric responses $< 20°/s$, and VEMP amplitude asymmetry $> 35\%$.

Posturography

Static posturography was performed with the subject standing on a stabilometer platform system (Kistler, type 9261A) with the feet together but splayed outwards at an angle of 30°, and both arms hanging by the side. The total sway path (in the anterior–posterior and medio-lateral direction) was measured by the system for 30 s (sampling frequency 40 Hz) under two conditions: with the eyes open (EO), and eyes closed (EC). The data collected was analyzed off-line using customized computer software.

Statistical analysis

We identified the following vestibular test parameters for analysis: VOR right, VOR left, VOR asymmetry, unilateral (caloric) weakness, total caloric response, oVEMP amplitude and amplitude asymmetry, and cVEMP amplitude and amplitude asymmetry. The SPSS version 23 statistical package was used for all the analyses in the study. For the primary aim, multiple regression analysis was done for subjects in the study with a complete dataset (i.e. the "main" analysis, $n = 618$). The semi-partial (part) correlation between DHI and each vestibular parameter was derived. The analysis was performed again on patients with peripheral vestibular disorders only (i.e. the "sub-analysis", $n = 402$). The above two groups of patients were re-analyzed using each DHI subdomain (physical, emotional, functional) in turn as the dependent variable, resulting in eight separate analyses.

Next, the patients ($n = 799$) were grouped into ten disease groups (see Table 1) and the median group DHIs were compared using the Kruskal–Wallis test. A p value < 0.05 was defined as significant to reject the null hypothesis that there was no difference in the median DHI. The

Dunn–Bonnferroni post hoc analysis was used to determine the most significantly different group pairs.

The patients were further categorised into central (excluding vestibular migraine), peripheral and functional (including psychogenic) disorders, and the median group DHIs were compared. A p value < 0.05 was defined as significant to reject the null hypothesis that there was no difference in the median DHI.

Based on type of vestibular deficits, a subset of patients were classified into four groups: (1) isolated high-frequency VOR deficits (unilateral/bilateral abnormal vHIT), (2) isolated low-frequency VOR deficits (unilateral/bilateral abnormal caloric response), (3) isolated otolithic deficits (abnormal ocular and/or cervical VEMP amplitude or amplitude asymmetry), (4) no measurable vestibular deficits and no central lesions (i.e. BPPV and vestibular migraine). The median group DHI was analyzed using Kruskal–Wallis test. A p value < 0.05 was defined as significant to reject the null hypothesis that there was no significant difference in the DHI between groups.

Spearman's correlation test was used to analyze the correlation between the DHI and EC total sway, EO total sway and Rhomberg ratio (defined as EC sway divided by EO sway).

Results

In general, the DHI showed either no correlation or very weak correlation with each of the abovementioned vestibular test parameters: (a) vHIT VOR gain right (r (618) = 0.007, $p = 0.859$), VOR gain left (r (618) = − 0.091, $p = 0.024$), VOR gain asymmetry (r (618) = 0.013, $p = 0.740$), (b) total caloric response (r (618) = 0.055, $p = 0.173$), unilateral canal weakness (r (618) = − 0.018, $p = 0.656$), (c) cVEMP amplitude right (r (618) = − 0.034 $p = 0.395$), amplitude left r (618) = − 0.004, $p = 0.928$, amplitude asymmetry (r (618) = 0.016, $p = 0.696$), oVEMP amplitude right (r (618) = 0.044, $p = 0.267$), amplitude left (r (618) = − 0.007, $p = 0.856$), amplitude asymmetry (r (618) = − 0.008, $p = 0.836$). The regression model could only explain 2.7% of the variance seen in the DHI.

Patients with only peripheral vestibular deficits also showed no correlation between the DHI and any of the vestibular test parameters (a) vHIT: VOR gain right (r (402) = − 0.007, $p = 0.877$), VOR gain left (r (402) = − 0.082, $p = 0.099$), VOR gain asymmetry (r (402) = 0.014, $p = 0.78$), (b) caloric irrigation: total caloric response (r (402) = 0.003, $p = 0.955$), unilateral canal weakness (r (402) = 0.03, $p = 0.541$), (c) VEMP: cVEMP amplitude right (r (402) = − 0.036, $p = 0.468$), amplitude left (r (402) = 0.0,1 $p = 0.839$), amplitude asymmetry (r (402) = 0.047, $p = 0.347$) and oVEMP amplitude right (r (402) = 0.27, $p = 0.587$), amplitude left (r (402) = − 0.008, $p = 0.877$), amplitude asymmetry (r (402) = 0.032, $p = 0.521$). The regression model could only explain 3.5% of the variance seen in the DHI.

Similarly, either no correlation or weak correlation was found between the DHI subscores and the vestibular tests from the main study group and the peripheral disorder subgroup (see Tables 2, 3).

As a group, the median DHI was significantly higher in patients with central disorders (excluding vestibular migraine) versus patients with peripheral disorders (Mann–Whitney U, $z = -4.743$, $p = 0.001$) (see Fig. 1). The DHI was also significantly higher in patients with central disorders compared to patients with functional disorders ($z = -2.902$, $p = 0.004$), but the DHI of patients with primary functional disorders was still significantly higher than the patients with peripheral disorders ($z = -2.008$, $p = 0.045$).

The DHIs differed significantly between the ten disease groups ($z = 46.19$, $p = 0.001$) (see Fig. 2). Post hoc testing

Table 2 Correlation between DHI subscores and vestibular test parameters for the main study group

DHI subcomponent	*P*		*E*		*F*	
Vestibular parameter	Correlation	Sig.	Correlation	Sig.	Correlation	Sig.
vorR	0.005	0.890	0.016	0.690	0.001	0.986
vorL	− 0.077	0.053	− 0.026	0.514	− 0.119	0.003
vorAsym	0.026	0.508	0.032	0.424	− 0.016	0.689
UW	0.010	0.799	− 0.034	0.395	− 0.019	0.639
Tcal	0.059	0.139	0.010	0.813	− 0.067	0.091
ovempR	0.080	0.046	− 0.005	0.910	0.044	0.265
ovempL	− 0.080	0.044	0.040	0.329	0.003	0.936
cvempR	− 0.071	0.073	− 0.001	0.974	− 0.017	0.677
cvempL	0.039	0.326	− 0.004	0.920	− 0.042	0.292
ovempAsym	− 0.006	0.890	− 0.006	0.879	− 0.011	0.776
cvempAsym	0.032	0.416	0.005	0.895	0.000	0.992

UW unilateral weakness, *Tcal* total caloric response, *vor* vestibulo-ocular reflex, *Asym* asymmetry

Table 3 Correlation between DHI subscores and vestibular test parameters for the peripheral vestibular disorders subgroup

DHI subcomponent	*P*		*E*		*F*	
Vestibular parameter	Correlation	Sig.	Correlation	Sig.	Correlation	Sig.
vorR	−0.001	0.986	0.003	0.959	−0.011	0.831
vorL	−0.064	0.199	−0.026	0.602	−0.113	0.023
vorAsym	0.030	0.554	0.040	0.428	−0.020	0.688
UW	0.023	0.652	0.023	0.644	0.028	0.576
Tcal	0.005	0.920	−0.016	0.748	0.012	0.806
ovempR	0.059	0.235	−0.012	0.807	0.035	0.477
ovempL	−0.053	0.291	0.017	0.731	−0.001	0.990
cvempR	−0.061	0.220	−0.021	0.675	−0.020	0.689
cvempL	0.073	0.4600	−0.004	0.936	−0.039	0.432
ovempAsym	0.008	0.866	0.009	0.860	0.047	0.338
cvempAsym	0.051	0.310	0.023	0.647	0.032	0.520

UW unilateral weakness, *Tcal* total caloric response, *vor* vestibulo-ocular reflex, *Asym* asymmetry

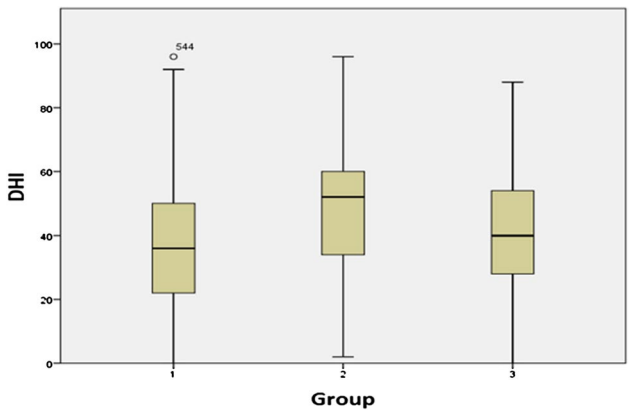

Fig. 1 Dizziness Handicap Inventory (DHI) scores of main groups of vestibular disorders. (1) Peripheral disorders, (2) central (excluding vestibular migraine) disorders, (3) functional (including psychogenic) disorders

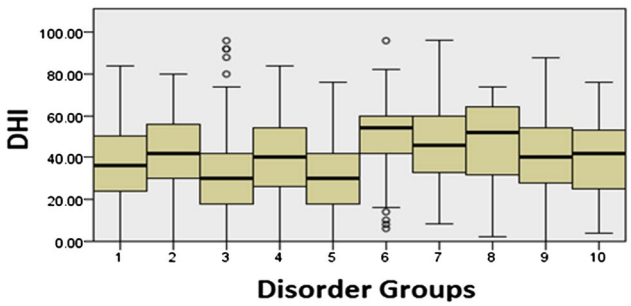

Fig. 2 Dizziness Handicap Inventory (DHI) scores grouped by etiological diagnosis. (1) BPPV, (2) BVP, (3) Menière's disease, (4) peripheral vestibulopathy (unilateral), (5) vestibular paroxysmia, (6) central dizziness, (7) cerebellar ataxia, (8) downbeat nystagmus syndrome, (9) functional/psychogenic dizziness, (10) vestibular migraine. The median DHI was significantly different ($z=46.19$, $p=0.001$) for group pairs 1–6, 5–6, 3–2, 3–4, 3–6, 3–7, 3–8, 3–9. BPPV, Menière's disease and vestibular paroxysmia had the lowest median DHI

showed the following group pairs were most significant: BPPV-central dizziness (pair 1–6), vestibular paroxysmia-central dizziness (pair 5–6), Meniere's disease-bilateral vestibulopathy (pair 3–2), Meniere's-unilateral vestibulopathy (pair 3–4), Meniere's-central dizziness (pair 3–6), Meniere's-cerebellar ataxia (pair 3–7), Meniere's-downbeat nystagmus syndrome (pair 3–8), Meniere's-functional disorder (pair 3–9) (see Table 4).

Subgroups of patients with isolated high-frequency VOR deficits ($n=14$), isolated low-frequency deficits ($n=73$), isolated otolithic dysfunction ($n=60$) and patients with no measurable deficits ($n=69$) were not significantly different in the median DHI ($z=2.153$, $p=0.541$) (see Fig. 3).

As a whole, the posturography results ($n=84$) showed that only the EO total sway correlated weakly with the DHI ($r=0.314$, $p=0.04$). In patients with central disorders [$n=48$, median DHI = 46 (95% CI 17.8–82.4)], only the EO total sway correlated weakly with DHI ($r=0.4$, $p=0.005$). The EC total sway ($r=0.216$, $p=0.141$) and Romberg ratio ($r=-0.081$, $p=0.585$) showed no correlation with the DHI. In patients with peripheral disorders [$n=36$, median DHI = 42 (95% CI 5–95)], no correlation with the DHI was found for all three posturography parameters. (EO $r=0.206$, $p=0.229$, EC $r=0.094$, $p=0.08$, Rhomberg ratio $r=-0.03$, $p=0.861$).

Discussion

The major findings of this study on 799 patients with a clearly defined vestibular diagnosis are: (1) the DHI and its subscores either do not correlate or correlate weakly with the tests of vestibular function; (2) the DHI reported by patients with *high-frequency VOR deficits, low-frequency* VOR deficits and otolithic deficits show no significant differences;

Table 4 Post hoc Dunn–Bonnferroni multiple comparisons testing

Group pair	Test statistic	Std. error	Std. test statistic	Sig.	Adjusted sig.
1–6	−147.719	41.612	−3.550	0.000	0.017
5–6	−217.450	57.754	−3.765	0.000	0.007
3–2	122.305	33.852	3.613	0.000	0.014
3–4	−95.125	28.668	−3.318	0.001	0.041
3–6	−214.303	42.654	−5.024	0.000	0.000
3–7	−166.421	44.445	−3.744	0.000	0.008
3–8	−196.840	54.672	−3.600	0.000	0.014
3–9	−108.978	29.675	−3.672	0.000	0.011

Only significant group pairs shown. (1) BPPV, (2) BVP, (3) Menière's disease, (4) peripheral vestibulopathy (unilateral), (5) vestibular paroxysmia, (6) central dizziness, (7) cerebellar ataxia, (8) downbeat nystagmus syndrome, (9) functional/psychogenic dizziness

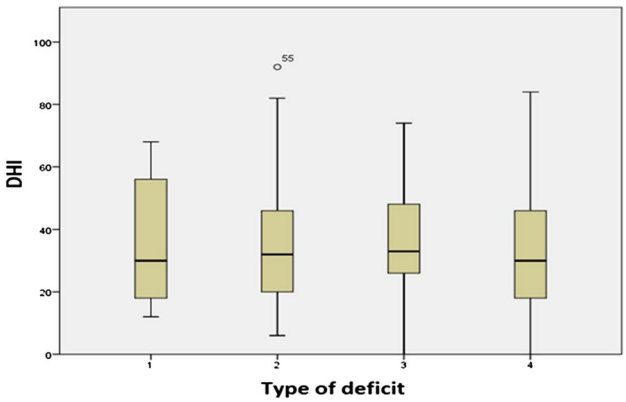

Fig. 3 Dizziness Handicap Inventory (DHI) scores grouped by type of vestibular deficits. No significant differences were seen in the median DHI of the four groups ($z=2.153$, $p=0.541$). (1) Isolated high-frequency VOR deficits, (2) isolated low-frequency deficits, (3) isolated otolithic dysfunction, (4) no detectable peripheral/central deficits

(3) patients with central vestibular disorders had the highest DHI; (4) patients with primary functional dizziness had significantly higher DHI than patients with peripheral disorders (even though they had no structural vestibular deficits) and (5) the DHI did not correlate with postural sway, measured by posturography.

Our study provides further evidence that objectively measured vestibular function alone cannot explain the variances in the DHI reported by patients with either persisting vestibular deficits (e.g. long-standing unilateral peripheral vestibulopathy) or episodically fluctuating vestibular symptoms (e.g. vestibular migraine). Overall, more than 96.5% of the variances in DHI was due to other unaccounted factors, most likely behaviourally related. Therefore, the functional repercussions of vestibular deficits do not correlate well with tests of structural integrity of the vestibular system. In patients with a previous vestibular insult, either central compensatory mechanism function to relieve the symptoms [22, 23] or additional maladaptive neurocognitive behaviours have become dominant (the same applies also to central vestibular disorders). Furthermore, vestibular compensation is a multi-modal (acting at different levels of the brainstem, cerebellum and cortex [24]) and multi-faceted process which could be influenced by the socio-cultural background of patients, brain cognitive reserve, coping mechanisms, emotional resilience to deal with the disorder, etc. In the dynamic situation of an episodic disorder, where vestibular dysfunction fluctuates too quickly to be reliably detected in routine clinical practice, the DHI provides collaborative information about the patient's functional difficulties (already evidenced by the patient's anamnesis), which may seem incongruent to the usually unremarkable vestibular testing. It is because of this lack of correlation with vestibular deficits that suggests the DHI may not be a sensitive nor specific indicator of underlying structural vestibular deficits.

This perceived deficiency of the DHI has resulted in attempts by various investigators to revise the DHI subscale structure (using factor analysis) into new clusters which may provide a more valid representation of the functional handicap faced by patients. For instance, Kurre et al. proposed a three-factor model (using factor analysis) which informs about (1) the effects of dizziness and unsteadiness on emotion and participation, (2) the specific activities or movements which causes dizziness and unsteadiness, and (3) the situation-dependent self-perceived walking ability [25]. Additionally, we performed a post hoc analysis of our data, and derived a similar three factor solution (see Table 5), and we further analysed all our vestibular test parameters in a multiple regression using each derived factor in turn as the dependent variable. Even with this novel method of analysis, there was no significant correlations found, except between "ovemp Left" and "factor 1", which was weak ($r=0.11$, $p=0.046$, see Table 6).

Canal disorders affecting the angular VOR result in dysfunction of gaze stabilization, while otolithic disorders cause postural imbalance and perceptual tilts. Theoretically, these

Table 5 The three-factor solution from principal component analysis

	Factor		
	1	2	3
E23	0.785		
E22	0.711		
F6	0.674		
E18	0.664		
E2	0.617		
F24	0.614		
F3	0.594		
E21	0.546		
E9	0.493		
F7	0.459		
E20	0.394		
E10	0.292		
P13		0.786	0.313
F5		0.688	
P11		0.658	
P25		0.605	−0.337
P1		0.387	
P17			−0.729
F19			−0.631
F16			−0.583
E15			−0.572
P4			−0.533
F14			−0.523
F12			−0.491
F8		0.287	−0.432

Extraction method: principal component analysis, rotation method: Oblimin with Kaiser normalisation. Values of factor loadings are results of the pattern matrix

E emotional subscale, *F* functional subscale, *P* physical subscale of the DHI

different mechanisms pose different functional challenges, which may result in differences in the DHI. Surprisingly, we found no difference in the median DHI severity in patients with isolated high- or low-frequency canal deficits, isolated otolithic deficits and symptomatic patients selected from the study population (i.e. vestibular migraine/BPPV subjects) with no measurable peripheral vestibular deficits and no central lesions. One explanation may be that our study subgroup consists of patients with stable and fixed vestibular deficits who have adapted in various ways to their deficits, making them feel less handicapped and functionally more similar to the symptomatic group with no measurable vestibular deficits.

Of the patients with peripheral vestibular disorders, those with bilateral vestibulopathy scored highest on the DHI. We believe that bilateral de-afferentation could have posed great difficulty for vestibular compensation, requiring a switch to alternative somatosensory rebalancing strategies as the only way to cope with the continuous symptoms.

Our study showed that patients with central vestibular disorders (mainly stroke affecting the brainstem-cerebellar structures) had a higher DHI, both as a group (Fig. 1) (central versus peripheral) and when separated by disease category (Fig. 2) (in the grouped analysis, we excluded vestibular migraine because it could have both central and peripheral components [26–29]). This finding is consistent with a study on the rehabilitation of central disorders [30] which found that central disorders compensate less well. One explanation could be that there is damage to critical central vestibulo-cerebellar structures which are involved in the "repair" process [31], as compared to peripheral disorders where the central repair mechanisms are intact.

Interestingly, patients with primary functional dizziness scored even higher DHI than patients with peripheral disorders. We believe that their heightened sense of bodily sensation and pre-occupation with their continuous/fluctuating

Table 6 Correlation between derived factors and vestibular test parameters

Factor	1		2		3	
Vestibular parameter	Correlation	Sig.	Correlation	Sig.	Correlation	Sig.
vorR	−0.015	0.786	0.076	0.164	−0.101	0.054
vorL	0.016	0.777	−0.104	0.057	−0.101	0.056
vorAsym	0.051	0.352	0.028	0.603	0.045	0.387
UW	0.017	0.759	0.016	0.765	0.010	0.847
Tcal	0.015	0.784	0.042	0.445	0.036	0.495
ovempR	−0.018	0.737	0.089	0.103	0.066	0.210
ovempL	0.110	0.046	−0.099	0.072	0.010	0.851
cvempR	−0.019	0.727	−0.081	0.137	−0.086	0.103
cvempL	0.030	0.591	0.067	0.220	0.022	0.670
ovempAsym	−0.002	0.973	−0.063	0.249	−0.024	0.651
cvempAsym	−0.017	0.757	−0.006	0.909	−0.061	0.242

UW unilateral weakness, *Tcal* total caloric response, *vor* vestibulo-ocular reflex, *Asym* asymmetry

symptoms, could be responsible for their high levels of distress.

Although posturography assesses somatosensory integration, rather than vestibular function per se, our study showed either no or poor correlation of postural sway with the DHI. In a different study, paradigm from ours (which included more balance conditions being tested), a correlation between the DHI and the results of computerized dynamic posturography was found, although it was slight [5]. In contrast, in a study on acute vestibulopathy where patients presented within 1 week of symptom onset, no correlation was found between the DHI and the Romberg test performed either with the eyes opened or closed. [2]. All these studies suggest that either there are other factors e.g. cognitive resilience, dynamic walking conditions, visual influences, etc. which may differentially combine to modulate somatosensory processing in affected individuals (even at an early stage of illness), or that conventional platform posturography was not an appropriate predictor of functional handicap, as measured by the DHI, when patients are required to move about in their natural environment.

The strength of the study is its large sample size and large number of very relevant diagnostic categories commonly seen in everyday practice, allowing meaningful analysis and sub-analysis of the data.

The limitations of our study are that all the patients either have stable central or peripheral deficits or have episodic active disorders with little peripheral deficits. We have not assessed patients with acute vestibular loss (e.g. acute vestibular neuritis) who would certainly have both very high DHI and very severe peripheral deficits. We only tested the horizontal angular VOR without testing the vertical semicircular canals. This was because most of the disorders affected the horizontal canals and/or superior division vestibular nerve with possible additional involvement of the vertical canals/inferior division vestibular nerve. Isolated involvement of the vertical semicircular canal/inferior vestibular nerve was thought to be rare and separately testing for them would not contribute much to our correlation study. The study cohort consists of predominantly European/German patients who may report subjective impairments differently from cohorts elsewhere e.g. Asia/Middle East, because of socio-cultural differences and attitudes towards dizziness. It would be interesting to perform a similar study in these regions for comparison.

In conclusion, this large study provides further validation that the DHI and vestibular function tests really measure two different aspects of the patients' vestibular disorder, which do not go hand in hand. The lack of correlation between the DHI and vestibular function tests (and posturography) may not be unexpected, given that there are mitigating factors linking the two, mostly at the socio-behavioural level. This concept is especially relevant to physicians practicing in the current increasingly test-focused culture, where the tests may appear rather unremarkable and yet the patients are suffering from significant handicap with high DHI. Vice versa, the well-adapted patient may report a very low DHI in the presence of a severe measured vestibular deficit. Patients with chronic well-compensated isolated canal or otolithic disorders reported similar DHIs to patients who did not have any fixed vestibular deficits. Patients with bilateral vestibulopathy and functional dizziness reported high DHIs for different reasons as discussed. Patients with central dizziness have the highest DHIs, and represent a great challenge for treatment. Finally, based on these findings in a large group of patients with various vestibular disorders examined with state-of-the-art methods, the clinical and scientific applications and usefulness of the DHI has to be re-evaluated.

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Author contributions CWY wrote the manuscript, collated the data and performed the statistical analysis. MS conceptualised and designed the study, collected the data, and helped revised the manuscript.

Compliance with ethical standards

Conflicts of interest CW Yip reports no conflict of interest relevant to the manuscript. M. Strupp is Joint Chief Editor of the Journal of Neurology, Editor in Chief of Frontiers of Neuro-otology and Section Editor of F1000. He has received speaker's honoraria from Abbott, Actelion, Auris Medical, Biogen, Eisai, GSK, Henning Pharma, Interacoustics, MSD, Otometrics, Pierre-Fabre, TEVA, UCB. He acts as a consultant for Abbott, Actelion, AurisMedical, Heel, IntraBio and Sensorion.

Ethical standards This study was approved by the Hospital Ethics Committee and performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments. All participants gave their informed consent prior to their inclusion in the study. There are no details in this manuscript that might disclose the identity of the participants.

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