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Interictal balance changes in migraine - A stabilometric and diffusion tensor imaging study

Cristiana P. Q. F. Góes,[1]* Ana Paula Fontana,[2] Isadora V. Silva[3]

Abstract

Cerebellar dysfunctions have been found in migraineurs, while ischemic lesions have been described to be more frequent in their posterior fossa. To examine balance abnormalities and anatomical cerebellar changes in migraine interictally, 48 patients (21 with aura — MWA; 27 without aura — MWoA) underwent an evaluation of their stance by computerized static stabilometry (CSS) and were compared with controls. The frequency and amplitude of swaying in both the anteroposterior and latero-lateral axes, as well as the area and velocity of oscillations were estimated with open and closed eyes. A subgroup of 10 individuals and 10 controls was also examined with MRI and diffusion tensor imaging. Fraction anisotropy (FA) was obtained in nine regions of interest at the posterior fossa. Clinical parameters (age, age at onset, timespan of disease and frequency of attacks) were correlated with FA and CSS data. Subclinical impairment with greater lateral axis oscillation, especially in MWA, was observed. MWA patients were more dependent on visual input to control lateral sway than MWoA subjects. The anatomy of the cerebellum, especially at the dentate nuclei and middle cerebellar peduncles was comparatively impaired in migraine sufferers, as estimated by FA.

1. Departamento de Especialidades Médicas, Faculdade de Ciências Médicas, Universidade Estadual do Rio de Janeiro, Rio de Janeiro, RJ, Brazil.

2. Departamento de Fisioterapia, Faculdade de Fisioterapia, Programa de Pós-graduação em Ciências da Reabilitação, Universidade Federal do Rio de Janeiro, Rio de Janeiro, RJ, Brazil.

3. Sala de Infusão, Universidade Estadual do Rio de Janeiro, Rio de Janeiro, RJ, Brazil.

*Correspondence address:

E-mail: cristiana.goes@gmail.com
ORCID: https://orcid.org/0009-0009-1920-8067

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Introduction

Migraine is a prevalent, paroxysmal, genetically driven, primarily neurological disease1 that compromises neurophysiology of a temporary or permanent nature. Although the most frequent and vigorous neurological symptoms of migraines, including headaches, occur during attacks, a series of interictal dysfunctions,[2] as well as possible structural changes,[3-5] have been reported.

Migraine may induce cerebellar and vestibular symptoms.[6] Around 2/3 of migraineurs are sensitive to motion and ¼ may present paroxysmal vertigo.[7] Migraine-associated dizziness has been correlated with vestibular dysfunction;[8] and a positive history of motion sickness with vestibular symptoms is relatively more prevalent in migraineurs.[9] Vestibular function

and balance are relatively more susceptible to visual influence in those migraine patients[10] whose the spatiotemporal function and visual motion processing are reported to be interictally abnormal.[11] Since the visual processing of migraine patients may be permanently impaired, it remains to be determined whether visual inputs play a role in some coordination and stance abnormalities associated with migraine. The cerebellum is particularly affected by infarct-like lesions in migraine patients,[5] which may theoretically lead to functional impairments. Hypermetria has been documented during finger-to-nose movements in migraineurs,[12] and stabilometric studies point to abnormal body sway in migraine.[13,14]

Fraction anisotropy (FA), a diffusion tensor imaging (DTI) magnetic resonance parameter that reflects the directionality of water diffusion due to cellular and subcellular components, may indicate the presence of lesions or dysfunctional tissues.[15] To further investigate migraine-induced cerebellar/vestibular abnormalities, the objective of the present study was to analyze interictal stance in migraine patients using Computerized Static Stabilometry (CSS) and correlate both balance and clinical parameters with fractional anisotropy (FA) values in cerebellar structures.

Methodology and resources

Twenty-one migraine patients with aura and 27 without aura who had been examined by neurologists experienced in the treatment of headaches, between April 2007 and July 2008, and diagnosed according to the 2004 International Headache Society criteria,16 randomly volunteered to undergo a CSS protocol between attacks. This procedure was also performed in 48 headache-free gender- and age-matched controls (HMC). Among these, 10 patients randomly selected and 10 matched controls were submitted to MR imaging. The age, age at onset, intensity of attacks as graded by the subjects from 1 (does not interfere with routine activity) to 4 (precludes activity since it is the most intense pain possible), frequency of attacks (as estimated by the subject), and timespan of disease in years were recorded for every subject. Inclusion criteria included: age between 18-65 years; presence of migraine for at least one year; and a minimum of two attacks per month. Concomitant tension-type headaches were permitted, provided their frequency did not exceed 15 attacks per month. Exclusion criteria were: presence of other forms of migraine; concomitant systemic or neurological diseases assessed by clinical examination; clinical evidence of vestibular diseases; overuse of analgesics — defined as intake in more than 4 days per week; pregnancy; breast feeding; and restrictions on MRI scans, such as claustrophobia or MR incompatible body implants. The study was approved by the ethics committee of the Hospital Universitário Clementino Fraga Filho, Universidade Federal do Rio de Janeiro (#004/06).

Computerized Static Stabilometry

Subjects were instructed to stand normally, barefoot, with arms relaxed alongside the trunk on a force platform device with a pressure sensitive sensor at each vertex. The apparatus was connected to a computerized recording system for data acquisition (LabView 5.0, National Instruments, Austin, TX) and analysis (MatLab 5.3, MathWorks). Signals were sampled (12-bit A/D conversion) and lowpass filtered with a second order Butterworth filter with a 5 Hz cut-off frequency. Centre of pressure (CP) displacements were used to evaluate the average amplitude of swaying both in the latero-lateral (x) and antero-posterior (y) axes, the total area of oscillation, the mean oscillation frequency in both axes and the total mean oscillation speed.

Two data acquisition blocks, each consisting of four successive one-minute periods, alternating closed eyes (CE) with open eyes (OE), were used to collect postural data. The two blocks were separated by a five-minute rest period, during which the subjects were allowed to sit and relax. The four acquisition steps within each block were separated one from another by 12-15 seconds, just sufficient for the examiner to ask the subjects to either close or open the eyes and activate the recording. The first acquisition was randomly chosen between the CE and OE situations and the first recordings with both OE and CE were discarded. Consequently, the final postural parameters were obtaining by averaging the 3 sets of data for both closed and open eyes. The following variables were considered for postural analysis: (a) for xSD and ySD, standard deviations of the amplitude of swaying in the x- and y-axis, respectively; (b) for VAvg, mean oscillation speed, all axes considered; (c) for Area95, area of sway after expurgation of the 5% outermost area; and (d) for FMx and FMy, mean oscillation frequency in the x- and y-axis, respectively. In addition, a Romberg Index (RI) was obtained from the division of the CE value by the OE value for each of the 6 stabilometric parameters.

Magnetic Resonance Imaging

Structural imaging: 3D reconstructions of the brain images were obtained using two high-resolution magnetization-prepared rapid acquisitions with gradient echoes (MP-RAGE) on a 3.0T scanner (Magneton Trio, Siemens, Erlangen, Germany) using an 8-channel head coil and the following parameters: 1.0x1.0x1.25mm; 128 slices; 256x256 matrix; echo time (TE)=3.44ms; repetition time (TR)=7.25ms; and flip angle=7°. The conventional MR imaging protocol also included axial FLAIR (2.0x2.0x5.0mm, TR=9950ms, TE=100ms, matrix 256x256 matrix, gap 35%) and T2-weighted images (2.0x2.0x3.0mm, TR=4410ms, TE=98ms, 320x320 matrix, gap 30%).

Diffusion Tensor Imaging: The DTI scans used a single-shot, twice-refocused echo planar sequence.17 Protocol parameters were: (a) forTR/TE, 9200/91ms, 2mm isotropic resolution, 64 slices, FOV 128x128mm, 1 average, 60 directions of diffusion encoding with b=700s/mm2, and 10 encodings with b=0s/mm2. Head motion was reduced by the use of padded clamps tightly attached to the head coil. Data were transferred to a workstation for off-line post-processing (Siemens Medical Solutions, DTI Task Card software, Massachusetts General Hospital).

Analyses of the FA maps were performed after correction for head orientation, eddy currents and susceptibilities. The same neuroradiologist, with 5 years of experience in this technique, defined nine regions of interest (ROIs) in the posterior fossa based on conventional MRI sequences and the FA maps: anterior middle cerebellar peduncle (0.4cm2, a-MCP); right (0.8cm2, r-MCP) and left (0.8cm2, l-MCP) middle cerebellar peduncles; right (0.2cm2, r-ICP) and left (0.2cm2, l-ICP) inferior cerebellar peduncles; right (0.3cm2, r-DN) and left (0.3cm2, l-DN) dentate nuclei; and right (3.0cm2, r-CWM) and left (3.0cm2, l- CWM) cerebellar white matter.

Statistics

The values were stored in a spreadsheet; Prism for Macintosh 4.0 (GraphPad Software, Inc.) and SPSS 12.0 (SPSS Inc. Chicago, Il, USA) for Windows were used for statistical analysis. The Student t-test and the Mann-Whitney test were used for group comparisons. FA values in all ROIs were correlated with the stabilometric parameters and the clinical data (linear regression), and statistical significance was considered for p-values < .05. Values are shown as mean ± standard deviation (min-max).

Results

Table 1 shows the demographic data. The MWA and MWoA patients were considered together in the FA analysis due to the small number of MR scans. The group of patients who underwent MR examinations was similar to the population as a whole, since no statistically significant differences in age, age at onset, timespan of disease, stabilometric and FA data were detected between this subgroup and the other patients. However, the 10 subjects with MRI examination reported more frequent headache attacks (p=0.0262).

Table 1. Demographic Data

Group		n	Age	A at O	Females	MTS	Freq
With MRI	MWA	3(6.25%)	*	**	3(100%)	***	****
	MHC	3(6.25%)	*****	-	3(100%)	-	-
	MWoA	7(14.5%)	28.8±6.6 (30, 16-40)	13.8±3.4 (14, 8-18)	7(100%)	15.0±6.3 (15, 2-22)	7.0±5.2 (4,2-16)
	MHC	7(14.5%)	28.2±7.5 (28, 12-42)	-	7(100%)	-	-
	Migraine	10(20.8%)	30.9±7.6 (30, 19-45)	14.4±5.2 (15, 6-24)	10(100%)	16.5±9.6 (15, 2-39)	6.5±4.5 (4,2-16)
	MHC	10(20.8%)	30.6±8.84 (29, 18-48)	-	10(100%)	-	-
	MWA	18(37.5%)	34.1±11.0 (31, 18-54)	17.6±9.3 (14, 7-35)	16(88.8%)	16.5±7.4 (17, 4-36)	7.8±10.1 (3. 0.2-30)
	MHC	18(37.5%)	34.6±11.7 (33, 19-55)	-	16(88.8%)	-	-
Without MRI	MWoA	20(41.6%)	35.0±11.8 (31, 21-62)	17.6±6.6 (17, 7-34)	16(90.0%)	17.4±13.0 (15, 1-40)	2.8±3.0 (2. 0.4-12)
	MHC	20(41.6%)	35.0±13.2 (30, 20-64)	-	18(90.0%)	-	-
	Migraine	38(79.1%)	34.5±11.3 (31, 18-62)	17.6±7.9 (16, 7-35)	34(89.4%)	16.9±10.6 (16, 1-40)	5.1±7.6 (2, 0.2-30)
	MHC	38(79.1%)	34.9±12.4 (32, 19-64)	-	34(89.4%)	-	-
Total Subjects	MWA	21(43.7%)	34.3±10.5 (32, 18-54)	17.3±9.1 (15, 6-35)	19(90.4%)	17.0±8.7 (17, 4-39)	7.4±9.4 (4, 0.2-30)
	MHC	21(43.7%)	34.8±11.3 (33, 19-55)	-	19(90.4%)	-	-
	MWoA	27(56.2%)	33.4±10.9 (30, 19-62)	16.6±6.1 (16, 7-34)	25(92.5%)	16.7±11.6 (15, 1-40)	3.9±4.0 (2, 0.4-16)
	MHC	27(56.2%)	33.3±12.3 (29, 18-64)	-	25(92.5%)	-	-
	Migraine	48(100%)	33.8±10.7 (31, 18-62)	16.9±7.5 (16, 6-35)	44(91.6%)	16.8±10.3 (15,.5, 1-40	5.4±7.1 (3, 0.2-30)
	MHC	48(100%)	34.0±11.8 (30, 18-64)	-		-	-

Legend: n: Number of subjects (% of total population); Age: Mean age in years; A at O: Age at onset in years; Females: Number of female subjects (% of females within the group). MTS: Migraine timespan in years. Freq: Estimated frequency of headache days per month. MRI: Magnetic resonance imaging. MWA: Migraine with aura. MWoA: Migraine without aura; migraine: Total migraine patients, with and without aura. MHC: Matched healthy controls.

* Ages of the three subjects were 27, 35 and 45 years. / ** Ages at onset of the three subjects were 17, 24 and 6 years.

*** Migraine timespan in the three subjects was 10, 11 and 39 years. / **** Frequencies in the three subjects were 4, 8 and 4. / ***** Ages of the three subjects were 27, 33 and 48 years. Age, age at onset, timespan of migraine and frequency of attacks are shown as mean±SD (median, minimum value – maximum value)

Stabilometry

All 6 basic CSS parameters showed no differences between controls and migraineurs as a single group, with both closed and open eyes. With regard to the RI, the only significantly different variable was xSD, which was higher in the migraine group. Comparing the migraine subtypes, FMx was higher in MWoA than MWA with both OE and CE. The RIs showed no differences for all stabilometric parameters comparing all migraine subjects or MWoA with MHC; as well as MWA with MWoA. However, the RI was significantly higher for the variable xSD in MWA as compared with MHC. The stabilometric data are shown in Table 2.

Table 2. Stabilometric data (total n=48)

Stabilometric parameter	Mean	SD	Mean	SD	p value
MWoA			MWA		
Open eyes					
xSD	0.33	0.12	0.34	0.21	0.8503
ySD	0.45	0.14	0.40	0.17	0.3261
VAvg	0.94	0.22	0.83	0.31	0.1652
Area95	2.89	1.83	3.15	4.32	0.7798
FMx	0.22	0.05	0.18	0.05	0.0078
FMy	0.19	0.06	0.19	0.04	0.9876
MWoA			MWA		
Closed eyes					
xSD	0.35	0.12	0.35	0.24	0.9513
ySD	0.45	0.13	0.42	0.21	0.4888
VAvg	1.03	0.27	0.93	0.37	0.2824
Area95	3.05	1.71	3.43	5.02	0.7164
FMx	0.23	0.19	0.19	0.05	0.0210
FMy	0.19	0.20	0.20	0.05	0.2649
All patients			MCH		
Romberg Index					
xSD	1.06	0.19	0.99	0.20	0.0494
ySD	1.03	0.20	1.05	0.22	0.7047
VAvg	1.12	0.19	1.11	0.18	0.8660
Area95	1.08	0.24	1.05	0.33	0.6829
FMx	1.11	0.81	1.15	0.27	0.442
FMy	1.12	0.27	1.14	0.33	0.8114

Table 2. Stabilometric data (total n=48) (cont.)

Stabilometric parameter	Mean	SD	Mean	SD	p value
	MWA		MHC		
Romberg Index					
xSD	0.93	1.13	0.36	0.14	0.0172
ySD	1.04	1.26	0.47	0.17	0.8460
VAvg	1.09	1.19	0.89	0.20	0.6749
Area95	0.94	1.16	3.36	2.20	0.3687
FMx	1.18	1.00	0.18	0.05	0.5638
FMy	1.12	1.19	0.17	0.06	0.7357

Legend: SD: Standard deviation; MWA: Migraine with aura (n=21); MWoA: Migraine without aura (n=27); MHC: Matched healthy controls; xSD and ySD: Standard deviations of sway amplitudes in the x- and y-axis, respectively; VAvg: Mean oscillation speed, all axes considered; Area95: Area of sway after expurgation of the 5% outermost area; FMx and FMy: Mean oscillation frequency in respectively x- and y-axis; Significant p-values are shown in bold.

MR and fraction anisotropy data

No lesion was noticed in conventional MR sequences in migraine and MHC. Table 3 shows the FA values in patients and controls. Patients had significantly lower FA values at the middle cerebellar peduncle and dentate nuclei bilaterally. On the contrary, the right cerebellar white matter showed significantly higher FA values in the group of patients. On the contralateral side with same ROI, FA was also higher in migraineurs, but the difference was not statistically significant (p=0,093).

Table 3. Fraction anisotropy (n=10)

ROI	Mean	SD	Mean	SD	p-value
	Migraine		MHC		
anterior-MCP	0.55	0.07	0.58	0.09	0.4309
right-MCP	0.60	0.08	0.69	0.06	**0.0109**
left-ICP	0.61	0.07	0.70	0.05	**0.0025**
right-ICP	0.57	0.05	0.61	0.04	0.1072
left-ICP	0.57	0.05	0.58	0.04	0.6876
right-DN	0.35	0.02	0.43	0.04	**0.0000**
left-DN	0.33	0.03	0.40	0.04	**0.0005**
right-CWM	0.29	0.01	0.26	0.03	**0.0028**
left-CWM	0.27	0.03	025	0.03	0.0930

Legend: ROI: Region of interest; SD: Standard deviation; MCP: Middle cerebellar peduncle; ICP: Inferior cerebellar peduncle; DN: Dentate nucleus; CWN: Cerebellar white matter; Significant p-values are shown in bold.

Clinical data correlations

Considering the group of migraine patients as a whole (n=48), the stabilometric parameters did not correlate with age in the OE situation. With CE, significant correlations were found between age and FMx (Pearson=0.30; p=0.033) and FMy (Pearson=0.38; p=0.0062). Both age and timespan of the disease correlated with the RI for VAvg (Pearson=0.33; p=0.02). Disease timespan also correlated positively with RI for FMx (Pearson=0.46; p<0.001). None of the parameters, with either CE or OE, correlated with age at onset and frequency of attacks. In MHC, age did not significantly correlate with any stabilometric parameter, with both OE and CE, and RIs.

The significant correlations between clinical data and stabilometry variables in the MR subpopulation (n=10) are shown in Table 4. No significant correlation in this subpopulation was found between any clinical parameter and FA.

Table 4. Clinical and stabilometric data correlations (n=10)

Variables correlated		Patients		Controls	
Clinical	**Stabilometric**	**Pearson**	**p-value**	**Pearson**	**P-value**
Age	VAvg OE	0.27	p=0.450	0.87	p=0.001
	VAvg CE	0.33	p=0.349	0.88	p=0.001
	FMx OE	-0.56	p=0.094	0.79	p=0.007
	FMy OE	0.61	p=0.060	0.90	p=0.001
	Area95 CE	0.51	p=0.131	0.67	p=0.034
	FMy CE	0.70	p=0.023	0.66	p=0.034
Timespan	FMy OE	0.08	p=0.010	-	-
	FMy CE	0.79	p=0.006	-	-
	Area95 CE	0.07	p=0.021	-	-
Frequency	ySD OE	-0.78	p=0.008	-	-
	FMx OE	0.07	p=0.039	-	-
	ySD CE	-0.67	p=0.027	-	-

Legend: xSD and ySD: Standard deviations of sway amplitudes in the x- and y-axis, respectively; VAvg: Mean oscillation speed, all axes considered; Area95: Area of sway after expurgation of the 5% outermost area.FMx and FMy: Mean oscillation frequency in the x- and y-axis, respectively.OE: Open eyes; CE: Closed eyes

Significant p-values shown in bold.

Stabilometry – fraction anisotropy correlations

Significant negative correlations were found between FA in different ROIs and various stabilometric parameters, particularly the r-DN with VAvg (OE and CE), l- CWM with xSD (OE and CE). In MHC, only two correlations were significant, in comparison with 8 in the migraine group (Table 5).

Table 5. Fraction anisotropy and stabilometric data correlations (n=10)

Variables correlated		Patients		Controls	
FA (ROIs)	**Stabilometric**	**Pearson**	**p-value**	**Pearson**	**p-value**
right-DN	VAvg OE	-0.68	p=0.031	-0.06	p=0.578
	VAvg CE	-0.71	p=0.019	-0.15	p=0.680
left-CWM	xSD OE	-0.73	p=0.016	-0.06	p=0.852
	Area95 OE	-0.65	p=0.042	-0.03	p=0.450
	xSD CE	-0.76	p=0.011	-0.36	p=0.299
	VAvg CE	-0.66	p=0.037	-0.09	p=0.805
anterior-MCP	VAvg OE	-0.196	p=0.587	-0.65	p=0.044
	ySD CE	0.735	p=0.015	-0.36	p=0.303
left-MCP	FMx CE	-0.522	p=0.122	-0.65	p=0.040
right-ICP	ySD CE	0.712	p=0.021	-0.01	p=0.987

Legend: FA: Fraction Anisotropy; ROI: Region of interest; xSD and ySD: Standard deviations of sway amplitudes in the x- and y-axis, respectively; VAvg: Mean oscillation speed, all axes considered; Area95: Area of sway after expurgation of the 5% outermost area; FMx and FMy: Mean oscillation frequency in the x- and y-axis, respectively. OE: Open eyes; CE: Closed eyes. Significant p-values shown in bold.

Discussion

Stabilometry objectively measures stance performance, and changes in fractional anisotropy indicate changes in brain tissue. Testing migraine patients interictally and controls with both methods may provide information on the possible existence of subclinical stance impairment in migraine, and indicate possible anatomical bases for such dysfunction. Stabilometric parameters did not differ between migraine and controls in a head-to-head comparison. However, patients with MWoA showed significantly higher frequency of lateral oscillations compared with those with MWA. Focusing on the RI results, MWA subjects as well as the entire migraine group showed higher indices for the lateral sway amplitude parameter, a finding not present in MWoA. Thus, MWA subjects were more dependent than MWoA patients on visual inputs to control the amplitude of lateral swaying but showed less frequency of swaying in this axis. No impairments were found concerning anteroposterior oscillations. This finding contrasts with Mauritz *et al.*, who suggested that cerebellar lesions, particularly at the anterior lobe, would often induce an increased frequency of swaying in the antero-posterior direction.[18]

In a previous study, no differences were found using stabilometry in migraine patients in any of the parameters for the experiments with eyes open, but balance in migraineurs was worse than in controls with eyes closed. The RI of all parameters were mostly higher in migraineurs in comparison to healthy volunteers.[19] The parameters used in this study, however, were distinct from ours, suggesting that differences may be related to methodological differences. The extension and speed of swaying have been found to be higher in the migraine posturograms,[13] suggesting that balance impairment may be present in this condition. These studies, however, did not detect a preferable axis oscillation dysfunction.

FA values were significantly lower at the middle peduncle and dentate nucleus in the patient group. In contrast, a clear tendency to relatively higher FA values was seen at the cerebellar white matter bilaterally. Lower FA values have been detected at the cerebellar peduncles in spinocerebellar degenerative diseases[20] and various ataxia syndromes, including spinocerebellar ataxia type 1 and multiple system atrophy.[21] Since no abnormality in T1, T2 and FLAIR sequences were detected in our patients, FA changes are probably unrelated to the posterior fossa subclinical infarct lesions previously described to be more frequent in migraine.[22] Lower FA was previously found in regions involved with migraine pathophysiology, such as the trigeminothalamic tract and the ventrolateral periaqueductal gray region.[23] The DN, the most lateral and phylogenetically recent of the cerebellar nuclei, receives afferents from the cerebellar cortex and various other structures, including the trigeminal sensory nuclei.[24] It is possible that the reduction of FA in this nucleus seen in migraine patients is related to the same mechanism that reduces FA at the periaqueductal gray and the trigeminothalamic tract. The cerebellum has been found to be involved with non-motor functions, such as cognition.[25, 26]

In addition, somatosensory stimuli have been shown to activate the cerebellum,[27] including the DN.[28] The DN is functionally divided into a dorsal, microgyric portion and a ventral, macrogyric portion. This nucleus has developed in great apes and humans to a greater extent, particularly due to an expansion in the relative size of the ventral half, an area related to non-motor functions.[26] DN sensory functions may be particularly impaired in migraineurs.

The middle peduncle is the main structure for projection to the cerebellar cortex from pontine nuclei, which receive input from the cerebral cortex, including visual areas.[25] In monkeys, there is a dense projection to the pons from the dorsal visual stream of extrastriate visual areas, where most of the neurons are motionsensitive; and there are few or no inputs from areas in the ventral stream of cortical visual areas, where cells are involved with higher visual processes, such as face recognition and form discrimination. Visual motion perception is known to be impaired in migraine interictally,[29] an abnormality not caused by lack of attention.[30] The influence of vision in reducing the amplitude of lateral sway was detected by a significantly higher RI in MWA. In addition, the frequency of lateral oscillations was comparatively shorter in MWA as compared with MWoA, but not in relation to controls. Dysfunctions in visual perception, possibly more impactful in MWA concerning lateral sway during stance, may explain the present findings. Speculatively, cortical-ponto-cerebellar dysfunctional fibers originating in the migraineur's visual areas could contribute to the FA changes at the middle peduncle.

FA reduction does not necessarily imply a tissue lesion. From a functional perspective, an increase in axonal diameter secondary to over-functioning, as previously speculated, could lead to reduced FA.[23] On the contrary, if this assumption is correct, functional changes in an opposite direction could explain FA increases with decreases in axonal diameter, such as found at the cerebellar white matter in migraineurs. However, the mechanisms that govern such reactions and their reasons remain obscure. Taken together, the CSS and the FA data seem to indicate dysfunctions of output/input to the cerebellum involving visual and trigeminal pathways, rather than direct cerebellar lesions. In the right DN, but not the left, a negative correlation was found between the mean oscillation speed and FA in migraineurs. This suggests that increased anatomical dysfunction is associated with higher oscillation speeds. Asymmetrical activation of the DN, greater at the right side, was noted with both bilateral28 and left27 somatosensory stimulation. Overstimulation of the sensory pathways involved in migraine pathophysiology could therefore lead to asymmetrical dysfunction of the DN, causing a significant correlation with FA-sway speed only on the right side. In migraineurs, negative cor-

relations were also found between the left cerebellar white matter and the amplitude of lateral sway as well as the area of sway (situation OE) and the oscillation speed (situation CE). The reasons for such findings are unknown.

Clinical parameters were correlated with both stabilometric (total patients, n=48 and the MR subgroup, n=10) and FA (n=10) data. Findings seemed scattered and puzzling. The MR subgroup showed significant correlations concerning frequency of attacks for three stabilometric variables (negative for ySD with OE and CE; positive for FMx), but frequency correlations were not significant in tests of the population as a whole. This may be explained by the fact that, with regard to frequency, the MR subgroup was by chance not representative of the whole group because of more frequent headache attacks. Both the total migraineur and the MR subgroup showed positive correlations between age and stabilometric parameters, a finding not present in MHC, when the total patient group is considered but present for some parameters in the MR subgroup. These findings are inconsistent and must be regarded as the result of chance after multiple comparisons. The timespan of the disease showed positive correlations with 5 parameters in the MR subgroup, but only 2 when considering the whole group of migraineurs; variables distinct from the 5 were found to correlate with the timespan of the disease in the smaller group. Until proven otherwise, the correlation of clinical parameters with stabilometric data should not be considered as relevant. Similarly, no correlation was found between FA and clinical data. FA was shown to vary with age.[31] In our population, the lack of correlation between FA and age may be explained by the relatively small population sample and by the fact that migraine tends to be concentrated in young patients.[32]

Conclusion

In conclusion, our data suggest that migraine may interfere with the anatomy of the cerebellum, especially the DN and middle cerebellar peduncles, as estimated by diffusion tensor MR imaging. DN abnormalities with tendency to be mild dysfunctional changes may result in interictal subclinical imbalance characterized by impairment of oscillation in the lateral axis, especially in MWA patients, who may be more dependent on visual input to control lateral sway than MWoA subjects.

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