Migraine trait symptoms in migraine with and without aura
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ABSTRACT

Objectives: The aim of the study was to determine whether various transient sensory and neuropsychological symptoms (SNS) were associated with migraine using a custom questionnaire.

Methods: In this hypothesis-generating case-control study, the frequencies of transient SNS in 219 patients with migraine (149 without aura and 70 with aura) were compared with 161 age- and sex-matched healthy controls using a custom questionnaire. Patients from a tertiary academic headache center in Hamburg were contacted by regular mail. Healthy controls without a history of migraine were recruited by means of a screening questionnaire and consecutively approached by e-mail.

Results: The presence of both migraine and aura was associated with significantly higher frequencies of autokinesis, metamorphopsia, dyschromatopsia, cinematographic vision, illusionary visual spread, and synesthesia (for all comparisons: corrected $p < 0.05$). Double vision, inverted 2- and 3-dimensional vision, and altered perception of body weight and size were found more often in patients with migraine without aura than in those with aura. In contrast, aura was associated with the occurrence of visual splitting and corona phenomenon (for all comparisons: corrected $p < 0.05$). No relevant association with migraine was found for micropsia and macropsia, teleopsia and pelopsia, inverted vision, out-of-body experience, Doppelgänger phenomenon, complex visual hallucinations, and altered perception of body position in space.

Conclusions: The observed SNS seem to belong to a physiologic spectrum of multisensory phenomena. Some of these phenomena were significantly accentuated in patients with migraine and may therefore be termed migraine trait symptoms. However, these results will have to be confirmed in a prospective study with face-to-face interviews.

GLOSSARY

ICHD-II = International Classification of Headache Disorders, second edition; MA = migraine with aura; MIG = all migraine patients; MTS = migraine trait symptom; MwoA = migraine without aura; SNS = sensory and neuropsychological symptoms.

According to the current International Classification of Headache Disorders, second edition (ICHD-II), migraine with aura (MA) and without aura (MwoA) is a well-defined primary headache disorder. An increasing number of symptoms during migraine attacks that do not meet these criteria have been reported. Premonitory symptoms such as craving and yawning precede headache and aura by 2 to 48 hours. Other non–ICHD-II symptoms that have been termed migraine equivalents are inherent to migraine pathophysiology because they share their typical duration and paroxysmal nature without necessarily occurring in temporal relation to the headache, such as vertigo and cyclic vomiting. However, several sensory and neuropsychological symptoms (SNS) including transient visual illusions, hallucinations, and disturbances of higher cortical function have anecdotally been termed aura symptom or migraine-related, such as “Alice in Wonderland” syndrome. Only one controlled study found palinopsia to be more frequent in patients with aura (14.2%) than those without aura (6.6%) and compared with age- and sex-matched controls. Of interest, palinopsia was unrelated to the timing of headache or aura, thus migraine trait symptom (MTS) would be more appropriate. Elucidating their relationship with migraine and migraine aura might broaden our pathophysiologic and clinical understanding. We therefore conducted a retrospective study in a
clinically well-characterized sample of patients with MA and MwoA\(^1\) compared with a matched sample of healthy participants without a history of migraine headache to (1) determine the frequency of SNS and (2) classify these phenomena as related (i.e., MTS) or unrelated to migraine or migraine aura biology.

**METHODS** Study design. A retrospective, monocentric, hypothesis-generating, case-control study was conducted to determine the frequency of various putative MTS in patients with migraine (both with and without aura) compared with age- and sex-matched healthy controls (table 1).

Participants. Patients were recruited from the Headache Clinic of the University Medical Center Hamburg-Eppendorf, Germany, a tertiary-level center. Inclusion criteria were diagnosis of migraine with aura (International Headache Society classification 1.2.1) or migraine without aura (International Headache Society classification 1.1); exclusion criteria were history of drug abuse or epilepsy as stated in the medical records, any other severe neurologic disease including pain syndromes, relevant preexisting psychiatric comorbidity, and insufficient knowledge of German. Patients with relevant ophthalmologic conditions were not considered. All patients had initially been seen by a headache specialist (T.P.J., A.M.) confirming the diagnosis according to the current ICHD-II criteria\(^2\) and assessing psychiatric comorbidity as well as screening for concomitant painful conditions. All eligible patients (n = 381) were contacted by mail and received a custom questionnaire on the occurrence of various SNS. Patients who did not respond were contacted a second time by mail 2 months later.

Healthy controls were recruited from a local electronic database, among the staff of the University Medical Center, and by an advertisement in a local newspaper. All responders were asked to complete a previously validated screening survey\(^5\) based on 4 questions online to ensure that they did not have headache with migrainous features. We ensured that volunteers were unaware that we looked for controls without migraine history. Only those who negated all questions and indicated fewer than 10 headache days during the last 3 months were eligible. Depending on the stratification procedure, subjects were then included and asked to complete a custom questionnaire on SNS. Exclusion criteria were defined as follows: previous diagnosis of epilepsy, use of illicit drugs (as stated by the subject), other severe neurologic disorders including other pain syndromes, or insufficient knowledge of German. All participants were asked about current medication. Details of the matching procedure are given below. Recruitment took place from January 2011 until March 2012.

Standard protocol approvals, registrations, and patient consents. All participants gave written informed consent before inclusion in the study and completion of the questionnaires. The study was approved by the local ethics committee (Hamburg, Germany, submission number PV3878) and in compliance with the Declaration of Helsinki from 2008.

**Matching.** Subjects were stratified according to age (divided into decades) and sex. Because inclusion of migraine patients preceded recruitment of healthy controls, controls fulfilling the inclusion criteria after the first questionnaire were prospectively allocated to the above strata, until the control sample was proportionally comparable to age and sex distribution.

**Questionnaire for detection of sensory and neuropsychological phenomena.** Data were collected using a custom questionnaire screening for various SNS (table 2). For each item, key clinical features (table e-1 on the Neurology® Web site at Neurology.org) characterizing the above-mentioned MTS were presented and participants were asked to indicate their presence with “yes” or “no” (whether they were experienced before or during attacks in patients with migraine). All symptoms were described in lay language, and visual symptoms were partly illustrated where deemed appropriate (see figure e-1 for examples). For all participants, age and sex were recorded as potential confounders. Patients with migraine were asked to indicate the average number of headache days per month for the last 3 months and the names of preventive drugs, if taken, as additional factors.

**Data analysis.** SPSS 20.0 (IBM Corp., Armonk, NY) was used for all data analyses. Frequency of SNS coded dichotomously was analyzed using \(\chi^2\) test (for group sizes less than 5, Fisher exact test was used).

<table>
<thead>
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<th>Epidemiologic details and results of statistical testing</th>
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<td></td>
<td>MIG</td>
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<tr>
<td>No.</td>
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<tr>
<td>Age, y</td>
<td>39.8</td>
</tr>
<tr>
<td>Sex</td>
<td>(\chi^2) test: MIG-control (p = 0.107;) MwoA-MA (p = 0.503)</td>
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<tr>
<td>Male</td>
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<td>Female</td>
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<tr>
<td>Headache days/mo</td>
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<tr>
<td>Topiramate intake</td>
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<tr>
<td>Sum score of sensory and neuropsychological phenomena</td>
<td>3.55</td>
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<tr>
<td>No sensory and neuropsychological phenomena present</td>
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</table>

Abbreviations: ANOVA = analysis of variance; MA = migraine with aura; MIG = all migraine patients; MwoA = migraine without aura; NA = not applicable. Regarded as significant: \(p < 0.05\).
used). For 3 × 2 tables, Freeman-Halton extension was used. A 3 × 2 \( \chi^2 \) test was applied to search for differences of frequency data among subjects with MwoA and MA, and controls. To account for multiple testing, these screening results were corrected with the Bonferroni approach (multiplication of \( p \) values with the number of tests; \( p \) values \( \geq 1.0 \) were set to 1.0). In case of significant screening results, post hoc tests (respectively, Fisher exact test) were done. Correlations between frequency of SNS and sex as well as the frequency of SNS and the intake of prophylactic medication and topiramate were calculated using \( \chi^2 \) tests (Fisher exact test for group sizes less than 5), and between SNS frequency and age using point-biserial correlations. To determine whether more than one SNS was present in participants, a sum score was calculated (the presence of an SNS was counted as 1) with a maximum of 32 points. Parametric data across all groups were analyzed by analysis of variance and \( t \) tests. The \( \alpha \) level was set to 5% unless reported otherwise; all tests were 2-tailed. Because missing data sets were rare and did not exceed 14 per SNS (pooled for all groups), no correction strategy was used.

**RESULTS**

Sample characteristics. Screening results. Migraine. Of the 381 migraine patients, 166 (43.6%) completed the questionnaire. Of the remaining 215 patients, another 61 patients (28.4%) responded after a reminder 2 months later. This resulted in a total of 227 (59.6% of all patients) replies with 219 evaluable responses (57.5%). Controls. A total of 1,250 subjects were screened via the online survey; 396 (31.7%) qualified as control subjects and accordingly received the questionnaire by e-mail, of whom 163 (41.1% of eligible and 12.9% of all subjects) replied. After exclusion of 2 subjects for self-reported use of illicit drugs, 161 controls (40.7%) were finally included in the analysis (figure 1).

Demographic data. The age and sex distribution of the final study population did not differ between migraine patients and controls (for further demographic data, see Table 2).
see table 1). Among the patients with migraine, 70 (31.9% of included migraine patients) were diagnosed with MA and 149 (68.0% of included migraine patients) with MwoA. In the MA group, 64 patients (91.4%) reported visual aura symptoms, 14 patients (20.0%) recorded aphasic aura symptoms, and 16 patients (22.9%) recorded sensory aura symptoms. Six patients (8.7%) reported other symptoms associated with migraine aura. Seventeen patients (24.6%) had more than one aura symptom: 4 had visual and aphasic aura, 6 had visual and sensory aura, and 7 had visual, aphasic, and sensory aura.

**Main results. Frequency of MTS.** Most SNS were significantly more frequent among patients with migraine than healthy controls (table 2), but distribution frequencies of micropsia, macropsia, teleopsia, pelopsia, inverted vision, out-of-body experience, Doppelgänger phenomenon, altered perception of body position in space, and complex visual hallucinations did not reach statistical significance.

Patients with MA had a significantly higher frequency of autokinesis, cinematographic vision, metamorphopsia, dyschromatopsia, illusionary visual spread, synesthesias, corona phenomenon, and visual splitting than patients with MwoA (table 2, figure 2, and table e-2). However, only corona phenomenon and visual splitting were specific for migraine aura because their frequency in MwoA patients did not differ significantly from that in healthy controls. Double vision, inversion of 2D/3D vision, and altered perception of body size and weight were found significantly more often in patients with MwoA, while frequencies among MwoA and MA patients did not differ significantly (table 2).

For all participants, age did not correlate with the presence of SNS apart from a trend for micropsia ($r = 0.130; p = 0.01$), inversion of 2D/3D vision ($r = -0.102; p = 0.047$), and altered perception of body weight ($r = -0.162; p = 0.001$). Sex had no influence on the frequency of SNS except for illusionary visual spread ($p = 0.001$).

MwA = migraine with aura; MwoA = migraine without aura.
which occurred more often in women than in men.

Preventive medication taken by 30% of patients did not affect SNS frequency in the $\chi^2$ test ($p > 0.07$ for all SNS).

**Sum score of SNS.** The sum score was significantly higher among migraine patients than controls, with the highest ratings in the MA group (table 1). A weak correlation between the subject’s age and the number...
of SNS (sum score) was observed: the older the participants, the lower the sum score above all participants ($r = -0.107; p = 0.037$). However, within subgroups (all migraine patients [MIG], MA, MwoA, controls), no significant correlation was found ($p > 0.05$). The number of headache days increased with higher sum scores in all subjects ($r = 0.314; p < 0.001$) and also in the MIG group ($r = 0.143; p = 0.034$). The correlation was strongest in patients with MA ($r = 0.426; p < 0.001$). The number of aura subforms in the MA group did not correlate with the sum score ($r = 0.133; p = 0.271$). The intake of prophylactic treatment did not result in higher sum scores among the patients with migraine ($t$ test; $p = 0.430$).

**DISCUSSION** In this hypothesis-generating case-control study, we aimed to determine the occurrence of various sensory and neuropsychological phenomena in patients with migraine with or without aura compared with healthy controls. The fact that most examined SNS occur significantly more often in patients with migraine than controls and more often in MA than in MwoA suggests that they may be inherent to the biology of migraine and/or migraine aura, probably with a smooth transition between both phenotypes. Because they profoundly differ from both the ICHD-II criteria for MwoA and MA and the clinical picture of migraine equivalents, they should best be termed *migraine trait symptoms* (MTS). These MTS fit to the growing evidence that migraine pathophysiology may, in part, include dysfunction of cortical and subcortical structures. Our data suggest that the susceptibility to experience autokinesis, cinematographic vision, metamorphopsia, dyschromatopsia, synesthesias, and illusionary visual spread increases not only with the phenotype of migraine but particularly with the ability to experience aura symptoms as well (table 3).

Although the sum score of SNS was highest in patients with MA compared to those with MwoA and controls, double vision, inverted 2D/3D vision, and altered perception of body weight and size were found more often in patients with MwoA (table 3). In contrast, the presence of aura in the migraine phenotype was associated with increased frequencies of visual splitting and corona phenomenon. Multiple SNS in one person were more frequent among patients with migraine than controls with the highest scores in MA. Some SNS were not associated with migraine or aura (table 3) and therefore should not be termed MTS.

However, no symptom was more frequent in controls than in patients with migraine, substantiating that MTS belong to a physiologic spectrum of multisensory phenomena but are significantly accentuated in patients with migraine.

Larger epidemiologic studies on SNS are scarce. Apart from one Italian study, a large Japanese study found dysmetropia in at least 9% of students with probable migraine supporting anecdotal reports on adult migraineurs. In contrast, our study could not detect any significant influences of migraine or aura on these phenomena, suggesting that they should not be termed MTS.

A retrospective uncontrolled study of 143 patients with migraine corroborated our findings of an altered color vision (figure e-1b) more frequently found in MA (up to 22%) than in MwoA (up to 11%). Likewise, in another cohort (n = 200) with “vascular headache,” 20 patients with presumable migraine reported various SNS.

The remaining putative MTS have been reported only anecdotal or in small case series, making comparison with our results difficult; among the symptoms was impaired color vision during and before migraine attacks, confirming that impaired color vision is indeed an MTS. Cone dysfunction in patients with migraine associated with impaired S-cone–mediated color vision (bluish light) could explain these findings.

Further single case reports describe the following symptoms: autokinesis, cinematographic vision, permanent cerebral diplopia, recurring episodes of double vision preceding migraine headaches, corona

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**Table 3** Classification of migraine trait symptoms after correction for multiple testing

<table>
<thead>
<tr>
<th>Migraine a</th>
<th>Aura &gt; migraine b, c</th>
<th>Aura c</th>
<th>No association</th>
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<tbody>
<tr>
<td>Double vision</td>
<td>Autokinesis</td>
<td>Corona phenomenon</td>
<td>Micro- and macroropsia</td>
</tr>
<tr>
<td>Inversion of 2D/3D vision</td>
<td>Cinematographic vision</td>
<td>Visual splitting</td>
<td>Telopsia, pelopsia, inverted vision</td>
</tr>
<tr>
<td>Altered perception of body size</td>
<td>Metamorphopsia, dyschromatopsia</td>
<td>Out-of-body experience, Doppelgänger phenomenon</td>
<td></td>
</tr>
<tr>
<td>Altered perception of body weight</td>
<td>Illusionary visual spread, synesthesias</td>
<td>Complex visual hallucinations, altered perception of body position in space</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: MA = migraine with aura; MwoA = migraine without aura.

*p MwoA-MA nonsignificant, p MwoA-control significant.

b All comparisons in table 2 significant.

c p MwoA-MA significant, p MwoA-control nonsignificant, p MA-control significant.
phenomenon,13 metamorphopsia,3,12,18 pelopsia, teleopsia,7,16 inverted vision,19 and illusory visual splitting.20 Complex visual hallucinations31 include recurrent illusory hallucinations during migraine attacks32 experienced as ego-dystonic and unreal. In another sample of 46 patients with migraine, 3 (6.5%) reported complex visual hallucinations, attributed to illicit drugs in one.18

While out-of-body experiences,23 Doppelgänger phenomenon, and autoscopy5,23 could not be classified as MTS, body-image distortion and dyssomatognosia have been associated with migraine3,8,10,23–25 including topiramate-induced cases15,26 and can be regarded as MTS. Because no relevant association between the intake of preventive medication and SNS frequency was found and only a few patients received topiramate, intake of preventive medication does not seem to explain altered SNS frequencies in our sample. Likewise, synesthesias27–29 have been linked to migraine, substantiating our observations.

It is important that several SNS are unspecific for migraine and can occur in other disorders,30 including virus infections,31 fever,7 brain lesions,32 schizophrenia, and epilepsy.3 Without a clear association with migraine headaches, further diagnostic testing (including brain imaging, EEG, and lumbar puncture) is mandatory.

Nevertheless, our data suggest that several SNS can indeed be termed MTS not routinely addressed by physicians. Patients may be hesitant to report these phenomena themselves because of fear of being considered psychogenic and may reveal them in paintings only.33,34

Although MTS are probably not “aura phenomena,” their facilitated perception seems to be part of the migraine and aura biology (table 3). Patients with migraine may experience such symptoms more easily, even more so in patients with MA. Migraine attacks are thought to originate from central brain disturbances, namely, of subcortical modulatory sensory systems.35,36 Thus, our data support the idea of a deficient cortical inhibition inherent to the underlying migraine biology as such, instead of merely being part of the headache attack. The increased responsiveness of the cerebral cortex to sensory information, which has been elegantly shown,37 may be the result of genetic vulnerability. If one has the genetic predisposition for migraine, the susceptibility to experience aura or MTS may depend on the degree of these changes alone or additional genetic and environmental factors.

This large study in a clinically well-characterized sample of patients with migraine explored the frequency of several putative MTS in migraine patients with and without aura. A retrospective approach seemed most feasible, because expected frequencies were low. Because our main intention was to screen for MTS, each symptom was covered by only one question and one item on the questionnaire, holding the inherent risk of overestimating the real frequency of MTS, especially in those with a rather diffuse clinical picture. Therefore, our results need to be confirmed in a prospective study with detailed face-to-face interviews focusing on MTS frequency over a defined period of time, their duration, their exact temporal relationship to both the migraine headache and the migraine aura, and the presence of additional accompanying symptoms. Although salient features of each MTS (table e-1) were presented, validation is challenging and we cannot exclude that the frequency of some MTS was underestimated.

A recall bias could have contributed to the high frequency of some MTS among the patients with migraine because headache attacks might have been associated more easily with MTS resulting in higher attention or retrieval. High neuroticism scores associated with anxiety about somatic syndromes found especially among patients with frequent migraine in specialized headache centers37 might accentuate this recall bias. Also, patients with migraine could be more suggestible than healthy controls resulting in an acquiescence bias. Because we wanted to keep that questionnaire as part of a pilot study as feasible as possible, we decided not to add a separate psychometrical inventory to quantitatively assess depressive-ness and somatization, which may have allowed us to correct for these dimensions more subtly. However, manifest psychiatric comorbidity was a strict exclusion criterion for all participants.

We note that different recruitment strategies including contact by regular mail with the patients and e-mail with controls carries the risk of a sampling bias—possibly reaching a different population regarding social and educational backgrounds.

Because patients were recruited in a tertiary headache center, a selection bias with the accumulation of more severely affected patients is possible. The higher the headache frequency, the more MTS occurred. This is plausible because rare symptoms are more likely to manifest themselves in more severely affected patients. However, we can exclude that the observed effects were only driven by the number of headache days, as the number of headache days was higher in the MwoA group and the sum score was higher in the MA group.

The surprisingly high frequencies of some MTS need confirmation in future trials with structured face-to-face interviews focusing on a small number of MTS with a more thorough clinical and temporal characterization regarding the relationship to headache and aura onset. The addition of a second control group with another chronic pain condition such as fibromyalgia could help to differentiate headache-specific contributions from more general pain-induced cognitive or emotional disturbances.

Our study revealed that various transient sensory and neuropsychological phenomena can be associated
with migraine and/or aura biology and can thus be termed MTS. The aura phenotype especially seems to predispose to the occurrence of multiple MTS. Recognition of these unusual symptoms as part of the complex disorder migraine might be helpful for a more elaborate clinical classification of migraine subtypes and could broaden our understanding of migraine pathophysiology.

AUTHOR CONTRIBUTIONS

T.P.J., L.H.S., and A.M. designed the study, outlined the methods, and drafted the recruitment strategy. T.P.J. and L.H.S. developed the questionnaire and implemented recruitment. L.H.S. collected the data. T.P.J. and L.H.S. had full access to all of the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis. T.P.J., L.H.S., and A.M. drafted the manuscript. All authors critically appraised the manuscript, revised where appropriate, and approved the final version of the manuscript.

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Migraine trait symptoms in migraine with and without aura (See p. 1416)

This podcast begins and closes with Dr. Robert Gross, Editor-in-Chief, briefly discussing highlighted articles from the April 22, 2014, issue of Neurology. In the second segment, Dr. Mike Sowell talks with Dr. Tim Jürgens about his paper on migraine trait symptoms in migraine with and without aura. Dr. Adam Numis reads our e-Pearl of the week about idiopathic normal pressure hydrocephalus. In the next part of the podcast, Dr. Lara Marcuse focuses her interview with Dr. Elinor Ben-Menachem on epilepsy therapeutics: Vigabatrin.

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