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OCCASIONAL PAPER

Distinctive anatomical and physiological features of migraine aura revealed by 18 years of recording

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The mechanisms underlying the initiation and propagation of the migraine aura, and the visual percept that is produces, remain uncertain. The objective of this study was to characterize and quantify a large number of visual auras recorded by a single individual over nearly two decades to gain insight into basic aura mechanisms. An individual made detailed drawings of his visual percept of migraine aura in real time during more than 1000 attacks of migraine aura without headache over 18 years. Drawings were made in a consistent fashion documenting the shape and location of the aura wavefront or scotoma in the visual field at one minute intervals. These drawings were digitized and the spatial and temporal features of auras were quantified and analysed. Consistent patterns of aura initiation, propagation and termination were observed in both right and left visual fields. Most aura attacks originated centrally (within 10° eccentricity), but there were also other distinct sites of initiation in the visual field. Auras beginning centrally preferentially propagated first through lower nasal field (69–77% of all auras) before travelling to upper and temporal fields, on both sides. Some auras propagated from peripheral to central regions of the visual field—these typically followed the reverse path of those travelling in the opposite direction. The mean velocity of the perceived visual phenomenon did not differ between attacks starting peripherally and centrally. The estimated speed of the underlying cortical event (2–3 mm/min) was in the same range as has been previously reported by others. Some auras had limited propagation and spontaneously 'aborted' after a few minutes, despite being initiated in similar locations to those that spread throughout the entire visual field. The visual percept of the aura changed corresponding with the presumed propagation from the V1 to the V2 region of the occipital cortex. In some cases the visual percept disappeared for several minutes before reappearing in a distant location, providing direct evidence that the aura can be clinically 'silent'. These results indicate that there can be multiple distinct sites of aura initiation in a given individual and suggest that the spatial pattern of propagation in the occipital cortex is non-concentric with a variable extent of propagation. The visual percept of migraine aura changes depending on the region of the occipital cortex that is involved.

Keywords: migraine aura; visual aura; clinical case
Abbreviation: CSD = cortical spreading depression

Introduction

Migraine is the most prevalent neurological disorder (Lipton *et al*., 2007) and about a third of patients with migraine have attacks with aura (Russell *et al*., 1995), a usually transient clinical disturbance that can be attributed to brain dysfunction (Goadsby *et al*., 2002). In migraine with typical aura, the most prevalent aura symptoms are visual disturbances (Russell and Olesen, 1996).

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Individuals with migraine visual aura have been documenting the visual percepts of their auras for centuries (Schott, 2007). Lashley was the first to correlate the representation of the visual aura with its likely distribution on the occipital cortex (Lashley, 1941).

The temporal and spatial characteristics of the spread of the migraine visual aura were similar to those of cortical spreading depression (CSD), leading to the hypothesis that CSD is the underlying mechanism of the migraine aura (Leão, 1944).

Based on studies of CSD in animal models, the spread of activity in the occipital cortex underlying the migraine visual aura is commonly represented as a concentrically propagated, expanding wave. However, the pattern of the visual percept of migraine may not necessarily be consistent with this pattern of spread (Dahlem and Hadjikhani, 2009). The specific neuro-anatomical substrates of migraine aura initiation and propagation and their underlying cellular and neurochemical mechanisms remain poorly understood. Careful clinical observation continues to play a key role in expanding our understanding of migraine and its pathophysiology.

Here we present data from a single individual who made consistent and detailed drawings of his migraine visual auras over nearly two decades and created a database of 1000 aura attacks. This database includes detailed maps of his visual perception of hundreds of his migraine auras, noting the precise site of initiation and pattern of spread throughout his visual fields.

These data provide significant new insight into the patterns of visual aura, and suggest mechanisms by which the migraine aura begins and spreads in the visual cortex, and how it is perceived.

Materials and methods

The source of the aura collection (P.V.) is a 71-year-old non-smoking, right-handed male research engineer with frequent migraine auras without headache. His migraines started at the age of 14 as a classic migraine headache with typical visual aura. He was consistently having 20–24 attacks of migraine with aura per year except during two attack-free periods (6 months during military training in 1960 and for ~10 years, 1980–90). The last reported migraine headache meeting International Classification of Headache Disorders criteria (IHS, 2004) for migraine with aura was in 1992, but accompanying some of the auras presented here he experienced a sensation of pressure in the temple, an aching sensation in one eye, and/or nausea.

Cafergot® (combination of ergotamine and caffeine) was tried as acute medication in the first few years, but only reduced nausea and was stopped. Sleep (when possible) was reported to relieve pain symptoms. Preventive treatment was never used and he is not currently on any medication. A validated questionnaire (Hauge *et al.*, 2010) indicated that the most reliable trigger factor for the auras was let-down after acute stress and >90% of attacks occurred within 24 h after relief from stress or following a period of time with a high level of mental concentration or cognitive effort.

Aura reports

Beginning in May 1995 migraine aura episodes were entered into a database, including detailed maps of his visual symptoms. He carried a stopwatch and marked the time of first awareness of migraine aura. The front of the advancing positive scintillating shapes ('fortification spectrum') or the dashed outline of a scotoma were traced at 1 min intervals and marked on paper (8.3 × 11.7 inches, 210 × 297 mm, A4). The fixation point was placed halfway down on the paper, and the eyeball to fixation point distance was held at 18 inches (46 cm). The maximum visual angle was 15–18° eccentricity from the centre. Auras commonly propagated beyond the angle in the visual field that could be recorded on A4 paper; in these cases he noted the time at which the visual percept disappeared. Larger pieces of paper were used for some auras, allowing for recording up to 50° eccentricity. All auras were recorded in a database that included date, time, laterality, and perceived point of origin.

All aura maps were scanned, digitized and quantitative measurements of drawings were made using ImageJ software (Rasband, 1997–2012).

We selected representative aura maps depicting auras drawn from the onset of the visual phenomenon and with duration of >15 min for mapping points of origin and end of aura pathways ($n = 216$) and for measuring velocity and direction of propagation ($n = 67$). Attacks with auras that showed limited propagation in the visual field ('aborts') were collected for subgroup analyses. The velocity of the visual percept was calculated based on the movement of the leading edge of the wavefront of positive visual symptoms. Results are presented as mean ± SEM. Statistical analyses were performed with SPSS 17.0 for Windows. Five per cent ($P \leqslant 0.05$) was chosen as the level of significance.

Imaging and visual field testing

MRI was performed on a single occasion with a 3-T unit (Magnetom Trio, Siemens Medical Solutions) using a 32-channel head coil. Anatomical T_1-weighted images were obtained using MP-RAGE sequence (Jack *et al.*, 2008), and magnetic resonance angiography was done as time of flight. These studies revealed age-related cortical atrophy as well as focal lesions in the left internal capsule, bilateral thalamus, and cerebellum consistent with lacunar infarcts. No lesions were observed in the occipital cortex.

Visual fields were tested with Humphrey 30-2 and 60-4 automated perimetry tests (Humphrey Field Analyser II, Carl Zeiss Meditec). This testing revealed a bilateral ring of reduced visual sensitivity centred at ~50°, but no abnormality within the areas of his visual fields that were represented by his visual auras (Supplementary Fig. 1).

Results

Aura characteristics

Visual auras occurred with an average frequency of 80 episodes per year (range 22–181/year). The mean number of visual auras per month was 6.2 ± 0.37 without any differences between months ($P = 0.87$). Some auras were perceived immediately upon awakening from sleep. On several occasions he experienced more than one aura on the same day. The auras originated in either left or right visual fields; left 54%, right 44%, and unknown 2%. On a rare occasion he experienced simultaneous aura propagation in both visual fields.

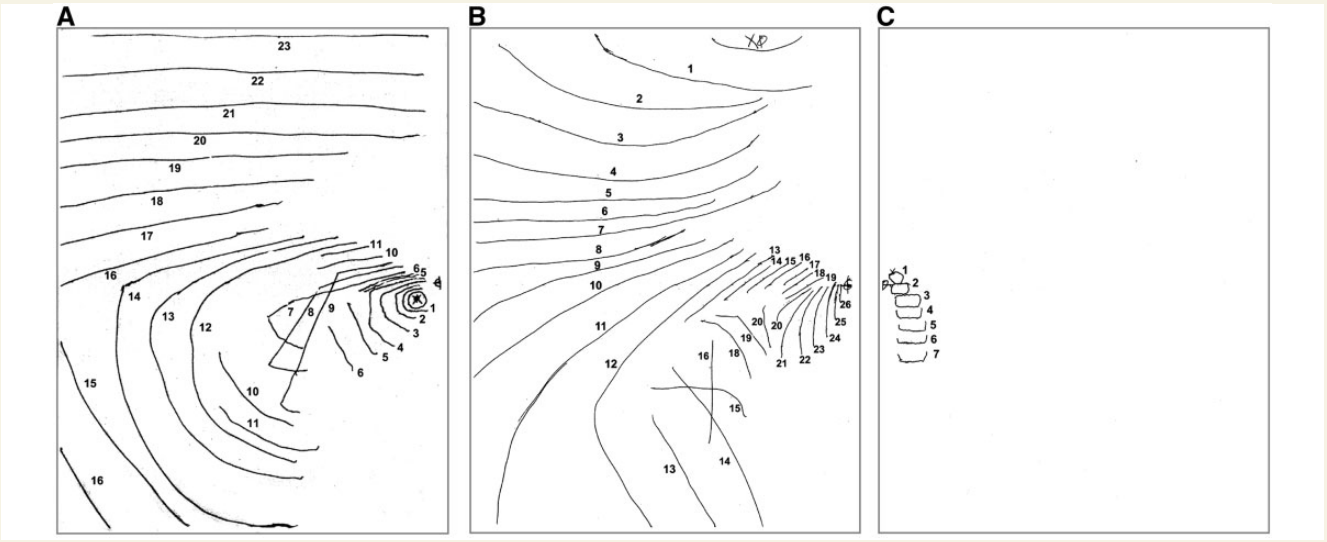

Figure 1 Original renderings of three typical patterns of propagation of visual symptoms from the database. Time indications were added digitally to original drawings for clarity. (**A**) Aura percept starts centrally in the left visual field, and moves along a typical pathway ending in the upper visual field after 23 min. (**B**) Aura percept starts peripherally in the upper left visual field and terminates close to the focal point after 26 min. Note that the visual percept in **A** and **B** displays similarities in pathways, shape and direction of convexity. (**C**) Aura percept originates centrally in the right visual field, but propagates for only 7 min before terminating.

Aura initiation and propagation

Several common patterns of aura initiation and propagation were observed, whereas other patterns occurred only rarely. Auras that originated peripherally and spread centrally generally followed the same path as those propagating in the opposite direction (Fig. 1).

In 800 attacks, he noted the polar coordinates of the origin of the auras. The majority of attacks, 683 (85%) originated within 10° eccentricity, with few auras originating along the vertical meridian (Fig. 2).

Auras propagated initially predominantly in the lower nasal fields bilaterally (69–77% of all auras), after which they spread to the upper temporal fields. Only a small fraction of auras (13–15%) terminated in the lower temporal fields (Fig. 3).

Interestingly, there were some regions where the aura wavefront had a similar shape and direction of convexity regardless of the direction of propagation, (Fig. 1A and B). In some cases an aura began close to the focal point, propagated peripherally, then later returned toward the focal point and propagated peripherally to (but not beyond) the initiation point.

The mean velocity of the percept in the visual field did not differ between attacks starting peripherally (17.6 mm/min ± 1.06 as drawn on paper) versus centrally (16.2 mm/min ± 0.59, $P = 0.41$).

ANOVA showed that the velocity of propagation of the visual percept changed over time, both for attacks starting peripherally and centrally ($P < 0.001$) (Fig. 4). Based on the assumption that the underlying cortical wave travelled in a direct path along the calcarine sulcus and crossed the entire primary visual cortex, from the occipital pole to the most rostral part of the striate cortex at the confluence of the calcarine and parieto-occipital sulcus (in this individual ~62 mm), the speed of the underlying cortical event was estimated to range from 2–3 mm/min.

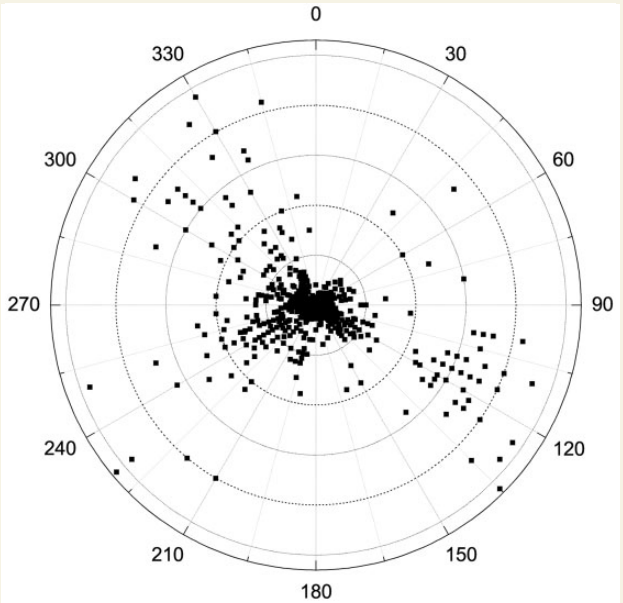

Figure 2 Points of aura origin of all recorded attacks. Figure showing the complete visual field up to 26.5° eccentricity. The right visual field of the patient is from (0–180°) and the left visual field is from (180–360°). $n = 800$.

Characteristics of visual percept

The predominant visual percept experienced during the visual aura was a curvilinear band of flickering (10–15 Hz) colourless brilliant-and-black gridlines, followed by advancing area of impaired vision. If represented statically, this percept is very similar to the

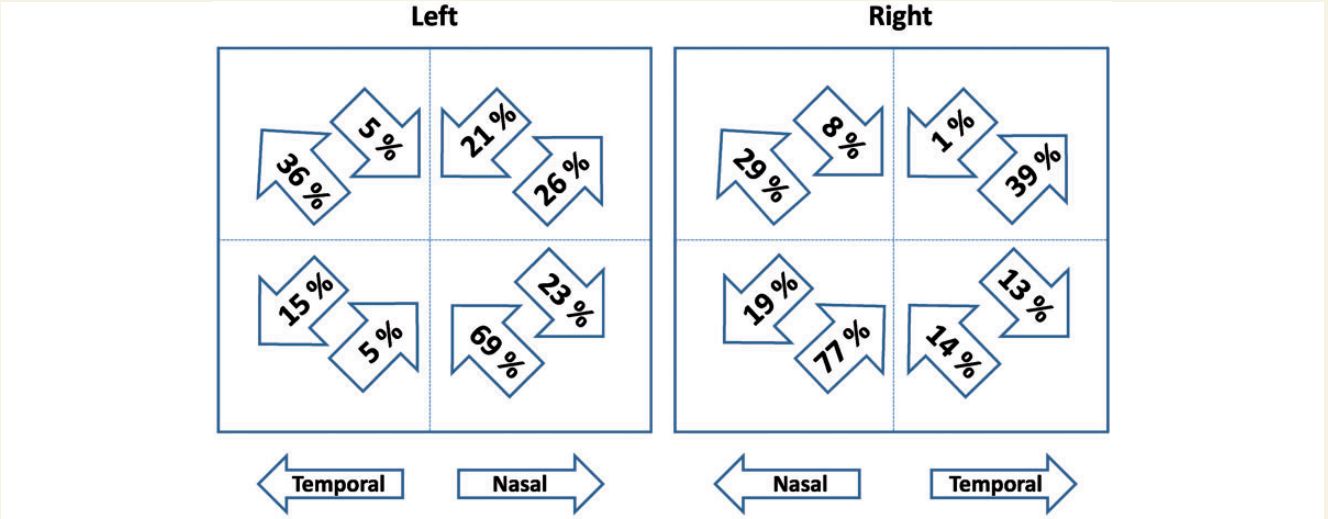

Figure 3 Origin and end of the visual aura. The numbers represent the percentage of all attacks originating in and ending in each of the four quadrants for each visual field.

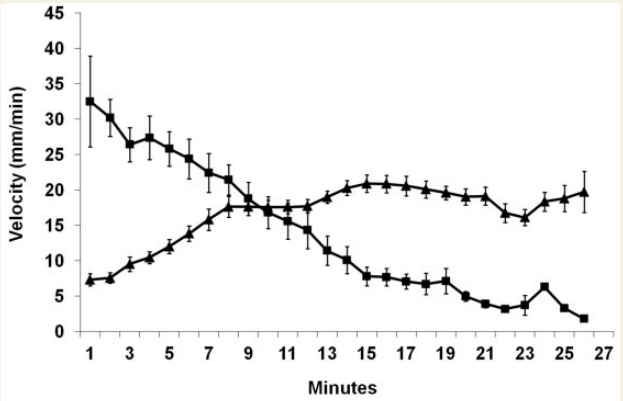

Figure 4 Velocity of propagation of aura percept in the visual field. Mean velocity (mm/min ± SEM) did not differ between attacks originating peripherally (squares) and attacks starting centrally (triangles). The former had a higher initial velocity ($P < 0.001$), whereas the latter had a higher terminal velocity ($P < 0.05$), consistent with a wave travelling with relatively constant speed over the visual cortex in which the central part of the field is 'magnified' (see Fig. 5).

'fortification spectra' described by others (Schott, 2007). In some events, however, the curvilinear positive wavefront disappeared for a few minutes, and in some cases was transiently replaced by an advancing circular scotoma, after which the flickering wavefront resumed (Fig. 5). The occurrence of the scotoma without a positive leading edge consistently occurred when the location of the visual percept neared or crossed the upper or lower vertical meridian. This location in the visual field corresponds with the transition between the V1 and V2 regions of the visual cortex (Fig. 5 and Supplementary Fig. 2).

Aborted attacks

Some attacks, described as 'aborts', began in the same regions as others but showed limited propagation and had a shorter than usual duration. These attacks, which comprised 20.4% (204/1000) of recorded attacks, lasted 1–10 min. The mean speed of propagation was lower in aborted attacks (7.9 mm/min ± 0.83) compared with the same time-span of the full attacks (13.5 mm/min ± 0.52, $P = 0.028$). There was no difference in eccentricity of origin between full and abortive attacks ($P = 0.51$) (Fig. 6), but the polar coordinate distribution differed between the two groups ($P < 0.01$), with the abortive attacks predominantly perceived in the left visual field, and thus originating from the right cortex.

Discussion

Among patients with migraine with aura, 99% report visual symptoms in some of their attacks (Kirchmann, 2006). The most prevalent visual disturbance of a typical migraine visual aura is a transient visual sensation, teichopsia or 'fortification spectra', although migraine patients also report blurred vision, scotoma, and non-propagating 'spots' in their vision (Russell *et al.*, 1994; Queiroz *et al.*, 2011). Individuals have been drawing and describing their migraine visual auras for centuries, and these drawings have provided key information about the phenomenon (Schott, 2007).

The characteristics of the auras presented here—visual percept, wavefront shapes and rate of propagation—show many similarities to those that have been described previously (Schott, 2007). The results reported here are, however, distinguished by a consistent method of recording in real time with excellent temporal and spatial resolution, allowing quantitative analysis that has not been

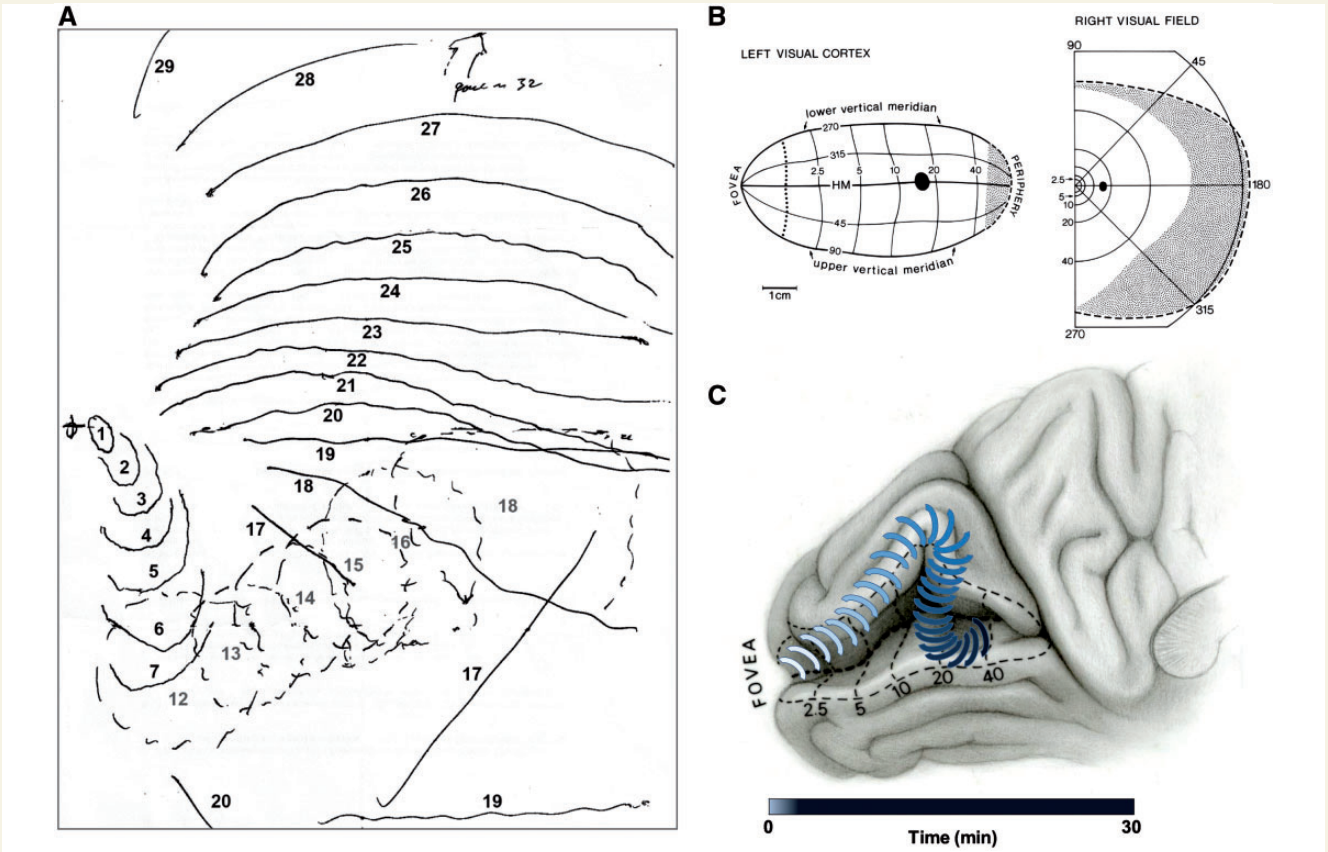

Figure 5 Possible retinotopic propagation of visual aura percept. (**A**) Scan of an original rendering of a typical visual aura in the database with times added digitally for clarity. Note that the visual phenomenon disappears between 7 and 12 min, and then reappears as a scotoma (indicated by dotted lines and grey time stamp) that moves across the visual field until the scintillating wavefront reappears at 17 min. (**B**) Schematic retinotopic map of the human striate cortex (V1). To the right is the map flattened artificially. Dark oval = blind spot, stippled zone = monocular crescent. (**C**) A hypothetical course of a cortical wave that could produce the visual symptom in (**A**) is mapped out onto the left occipital lobe. The main part of the striate (V1) cortex is buried in the calcarine fissure, and on this figure the fissure is opened, with superimposed gridlines indicating the horizontal and vertical meridians as well as the distance (eccentricity) from the fovea (centre of gaze) marked in degrees. Note that as the wave crosses the V1/V2 transition, the visual percept disappears and then reappears as a scotoma. When the cortical wave re-enters V1, the scintillation resumes. Maps in **B** and **C** provided by Horton (2013) and adapted with permission.

possible based on previous reports. This analysis provides some important new insights into this fundamental brain event.

Lashley (1941) provided the first quantitative recording of the temporal spread of the migrainous scotomas and fortification spectra. The change in perceived velocity in the visual field is consistent with propagation of a wave of activity with constant velocity though occipital cortex (Grusser, 1995) due to the 'cortical magnification' with magnified representation of central versus peripheral fields. Since the original description of CSD by Leão (1944) soon thereafter, it has been hypothesized that CSD is the underlying mechanisms of the migraine visual aura (Lauritzen, 1994).

Although this hypothesis has still not been definitively proved, its widespread acceptance has shaped thinking about the mechanisms of aura. A number of assumptions about the migraine aura are based on animal studies of CSD.

In animal models, CSD can be evoked by a number of stimuli, including local application of potassium, glutamate agonists, pinprick and micro-emboli (Charles and Brennan, 2009). In humans, spreading depolarizations closely resembling CSD have now been well documented following ischaemic stroke, intracerebral haematoma, subarachnoid haemorrhage, and brain trauma (Strong *et al*., 2002; Drenckhahn *et al*., 2012; Woitzik *et al*., 2013). The specific events that trigger the migraine aura are still not well understood.

In lissencephalic (i.e. lacking gyri and sulci) animal cortex, CSD spreads as a concentric wave in all directions from a site of focal initiation (Leão, 1944). Using functional MRI, spreading blood oxygen level-dependent events in the visual cortex can be linked to visual aura symptoms in humans (Hadjikhani *et al*., 2001). Cerebral blood flow studies during migraine aura showed broad, concentric spreading oligaemia moving from posterior to anterior brain areas (Olesen *et al*., 1981; Woods *et al*., 1994). The results presented here, however, indicate that these haemodynamic changes may not be reflective of the pattern of spread of the underlying CSD in the human cortex.

In the individual described here, migraine auras that began in the vertical midline generally migrated first to the inferior nasal

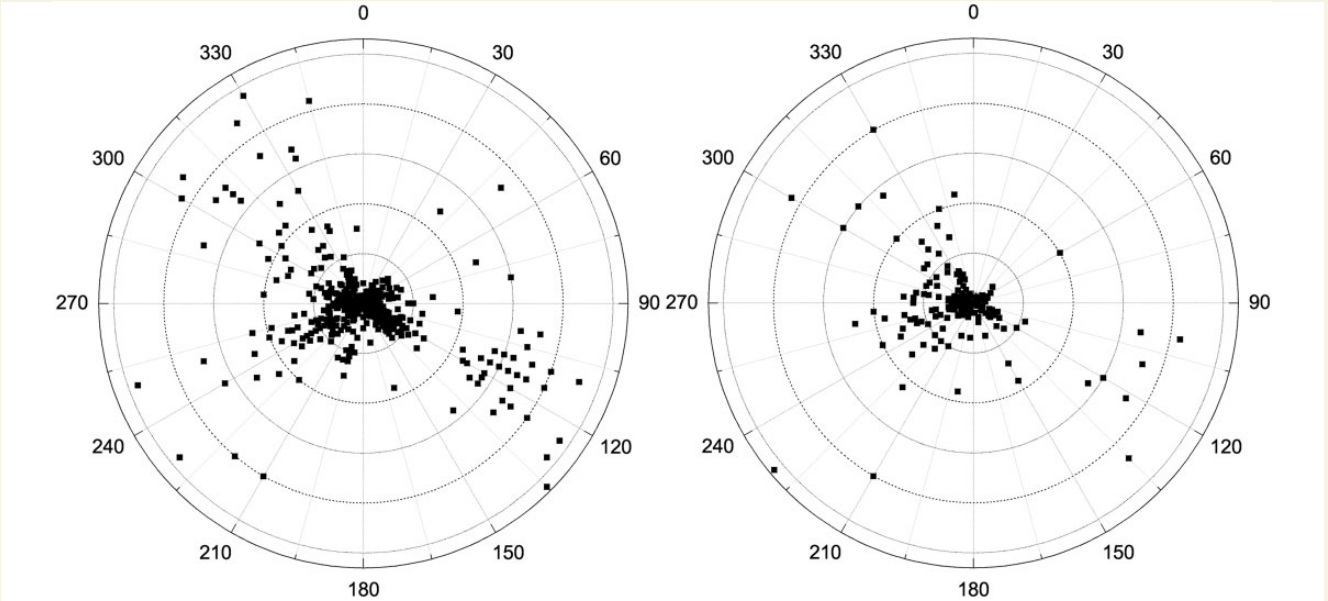

Figure 6 Points of aura origin of full (*left*) and abortive attacks (*right*). Figure showing the complete visual field up to 26.5° eccentricity. The right visual field of the patient is from (0–180°) and the left visual field is from (180–360°). Of the full attacks ($n = 596$) 84% originated within 10° eccentricity, and of the abortive attacks ($n = 204$) 89% originated within 10° eccentricity.

fields before crossing over to superior temporal fields. This path is not consistent with concentric propagation, but rather with preferential propagation along the superior aspect of V1 region in the visual cortex before crossing over to the inferior aspect of V1, suggesting that the cortical disturbance causing these symptoms is travelling in a non-concentric manner across different cortical regions (Dahlem and Hadjikhani, 2009). In some regions of the visual field, the convexity of the advancing visual percept was consistent regardless of the direction of propagation (Fig. 1A and B), indicating that the curvature of the visual percept can be determined by the functional anatomy of the visual cortex rather than by the curvature of the underlying physiological phenomenon. Thus, the visual percept of a concentrically expanding wave could be produced by a straight wave with constant width travelling on the cortical surface, rather than by a concentrically expanding CSD event as is commonly represented.

The auras presented in this report commonly changed from an advancing positive wavefront to an advancing scotoma at the upper or lower vertical meridian, corresponding with the transition from V1 to V2 in the visual cortex. It was previously proposed that the aura disappeared at the V1/V2 transition because of cellular barriers to diffusion of $K+$ (Grusser, 1995). The results presented here indicate that rather than an interruption of the aura wave at the V1/V2 transition, the change in percept is a function of the cortex through which the wave is continuously propagating (Fig. 5 and Supplementary Fig. 2). This observation underscores the concept that the same physiological process may result in completely different clinical expression (or none at all) depending on the cortical region involved. If CSDs are indeed happening within brain regions that are clinically silent, CSD or CSD-like events may therefore also be present in migraine without aura (so-called 'silent auras'). Migraine preventive medications that have proven efficacy for both migraine with and without aura inhibit CSD in animal models (Ayata *et al.*, 2006), raising the possibility that such 'silent auras' could be a therapeutic target.

Although auras generally propagated throughout the entire visual field, the occurrence of auras that were spontaneously aborted with limited propagation indicates that the cortical mechanism underlying the aura is not an all or none event. It is possible that these events had an inciting stimulus that was weaker than events that propagated more extensively, or that the cortex was not permissive to aura spread.

Regardless, this observation raises the possibility that the propagation of an aura could be suppressed under certain conditions, including in the setting of acute or preventive therapies.

The results presented here indicate that the aura can be initiated in multiple sites in the visual cortex in a given individual. Their bilateral occurrence and their initiation both centrally and peripherally indicate that migraine auras are not initiated by a single hyperexcitable focus.

The recurrent initiation at specific individual sites does, however, indicate that there are distinct regions of the occipital cortex that have a higher propensity toward initiating an aura. Even though auras having similar points of origin often showed similar patterns of spread, this was not always the case, indicating that these patterns are not hardwired but subject to modifying factors.

The fact that some auras seemed to be already in progress upon awakening from sleep suggests that visual input is not essential as part of the initiation of migraine aura, although it is possible that different patterns of visual stimulation could influence the site of initiation of the aura.

The migraine aura is a unique phenomenon where spontaneous activation of the cortex propagates slowly in a continuous fashion, resulting in a sensory percept in the absence of a sensory stimulus.

It therefore represents an unusual opportunity to map the function of the visual cortex, and potentially other cortical regions, when studied in conjunction with structural and functional imaging. It may also provide important insight into basic cortical physiology and pharmacology. Whether the migraine aura is a direct cause of migraine headache remains controversial, but the aura is a common component of a migraine attack in many individuals. Identifying conditions that enhance or suppress the aura are likely to identify new therapeutic approaches to this extraordinarily common disorder.

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Supplementary material

Supplementary material is available at *Brain* online.

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