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Visual field loss after attacks of migraine with aura

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Visual fields were mapped with kinetic arc perimetry in 23 migraine with aura subjects and, for comparison, in 20 migraine without aura subjects and in 21 non-headache controls. Central vision on the Amsler eye chart and visual perception threshold on a computer task were also investigated. Measures were obtained at least seven days after an episode of migraine. In addition, 10 of the migraine with aura subjects and 10 migraine without aura subjects were studied the day after an attack. The day after migraine with aura, visual sensitivity in the periphery of the visual fields was depressed, central vision was blurred, and visual perception threshold was elevated. These visual disturbances had resolved 7 to 10 days later. With the exception of a minor increase in visual perception threshold, vision was normal after attacks of migraine without aura. Residual effects of the migraine aura could mediate the subclinical visual disturbances which persist for at least one day after attacks of migraine with aura. □ *Aura, migraine, perimetry, visual fields, visual perception*

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Persistent field abnormalities are surprisingly common in migraine sufferers. For example, Lewis et al. (1) reported recently that 21 of 60 migraine patients (35%) had some form of visual field loss. Generalized depression of the visual fields was the most common finding, but various forms of scotoma were also detected in some patients. The prevalence of visual field loss was greater with increasing age and history of migraine, suggesting that visual disturbances were a side-effect of recurrent migrainous episodes. Whether the prevalence of persistent visual field loss is similar in different forms of migraine has not yet been investigated. Since transient visual disturbances usually precede migraine with aura, the prevalence of persistent visual abnormalities in patients with migraine with aura could be greater than in patients suffering from migraine without aura.

To investigate this possibility, visual fields were mapped with perimetry in migraine with aura subjects and, for comparison, in migraine without aura subjects and non-headache controls. Visual perception threshold was also determined. In some subjects, the procedures were carried out the day after an attack of migraine and repeated 7 to 10 days later.

Method

Subjects

The headache sample consisted of 23 females with a history of migraine with aura, and 20 females with a history of migraine without aura (2). Ages ranged from 17 to 41 years (mean age 30 years), and the history of migraine ranged from 1 to 28 years (mean 11.9 years). Mean age and history of migraine were

similar in the two groups of migraine sufferers. For comparison, measures were also obtained in 21 females aged between 19 and 48 years (mean age 29 years) who reported six or fewer mild headaches per year.

All subjects had a visual acuity, corrected with glasses if necessary, of 6/6 vision or better on the Snellen Eye Chart. No subject had a history of head or eye injury, cerebral lesions, epilepsy, cardiovascular disease, drug or alcohol abuse. Subjects gave their informed consent for the procedures, which were approved by the Murdoch University Ethics Committee.

Procedures

To assess central vision, an Amsler eye chart was held 30 to 40 cm from the subject's eyes. The subject was asked whether any section of the chart looked blurred or incomplete.

Visual field boundaries were mapped with kinetic arc perimetry, separately for each eye (3). The perimeter was brightly illuminated (400 to 550 lux, measured with a Gossen Profi-lux light meter); background illumination in the laboratory varied between 200 and 250 lux. The visual stimulus was a white dot (3 mm diameter) which was moved slowly along the arc of the perimeter (radius 330 mm) until detected by the subject. The dot was then moved towards central vision to investigate areas of visual loss. To maintain a visual fixation point, the subject stared straight ahead at a reflection of their eye in a small mirror attached to the center of the perimeter. The other eye was covered. Since most subjects were inexperienced at visual field mapping, the procedure was practised until responses were consistent. The

test-retest reliability of the procedure was found to be satisfactory (see Results). Visual field boundaries were plotted at 15° intervals. The area of the visual field was later calculated from the coordinates plotted on the visual field chart (adapted to the "Meyrowitz" perimeter, Lafayette Instrument Company), and expressed as a percentage of the normal visual field shown on the chart.

The threshold of visual perception was assessed by a microcomputer-controlled task, developed from the lexical-detection procedure described by Marcel (4). At the start of each trial, a fixation point (a cross) appeared in the centre of the computer screen for 1.5 sec. The cross was then replaced by a word (the visual stimulus) or a blank screen. Shortly afterwards, the word or blank screen was masked by a row of 11 hash signs. The subject's task was to decide whether a word had appeared on the screen. If necessary, the computer increased the length of presentation of the word in 10 msec steps from a baseline of 60 msec, until the subject made one or no mistakes in a block of 8 trials. The computer then decreased the stimulus duration in 10 then 5 msec steps until the subject made mistakes on 10 or more trials in a block of 16 (the visual perception threshold). The procedure was repeated two more times and the detection thresholds were averaged.

Twenty subjects were studied within 26 h of a typical attack (10 migraine with aura and 10 migraine without aura), and again 7 to 10 days later. For comparison, measures were repeated after 7 days in 5 control subjects. The other control subjects and migraine sufferers were studied only once, at least 7 days after an attack (range 7 to 137 days, mean 25 days).

Statistical procedures

Differences in visual field area and visual perception threshold among the migraine and control groups were investigated with analyses of variance. For measures taken at least a week after an attack, analyses had one between-groups factor (migraine with aura versus migraine without aura, versus controls). In migraine subjects studied twice, analyses of variance had factors of group (migraine with aura versus migraine without aura) and time (one day versus 7 to 10 days after the attack). Significant interactions were investigated with Student's *t*-test. The number of subjects with blurred vision on the Amsler eye chart was compared among groups with chi-square tests.

Results

At least 7 days after migraine

Neither visual field area, visual perception threshold, nor the proportion of subjects with

Table 1. Visual measures at least 7 days after migraine.

	Aura (<i>n</i> = 23)	Mean ± SD Without aura (<i>n</i> = 20)	Controls (<i>n</i> = 21)
Central vision blurred (<i>n</i>)	2	1	0
Visual field area (% normal)	87 ± 8	93 ± 16	94 ± 12
Perception threshold (msec)	20.2 ± 6.3	20.2 ± 7.0	19.8 ± 7.2

Scores did not differ significantly among the groups for any of the measures.

blurred central vision, differed among the migraine and control groups (Table 1). Previous experience with the procedure did not influence visual field area. However, visual perception threshold was lower in migraine sufferers who had been tested previously than in subjects who were tested only once [17 ± 3 msec versus 23 ± 8 msec, $F(1, 41) = 7.46$, $p < 0.01$]. Visual field area for the left and right eyes were similar for individual subjects [$r(63) = 0.95$, $p < 0.001$], indicating that the measure was reliable.

One day after an attack

Each of 10 migraine with aura subjects reported that the attack was preceded by visual disturbances (scintillations and progressive visual field loss) lasting 10 to 30 min. The aura was reported to be unilateral in 7 subjects, contralateral to the side of headache in 3. Six of the 10 migraine with aura subjects reported that central vision was blurred after their attack (Table 2). For all but one subject blurred vision had resolved 7 to 10 days later. Central vision was clear in all 10 migraine without aura subjects.

Visual fields were smaller the day after migraine with aura than 7 to 10 days later (Fig. 1). In general, visual sensitivity in the periphery of the visual fields was depressed, but a homonymous defect in the upper right quadrant was also detected in one subject. This quadrantic deficit had resolved 7 days later. In most cases of migraine with aura the visual field area of both eyes was reduced after an attack (Fig. 2). In contrast, the area of the visual fields was similar a day and a week after an attack of migraine without aura (Fig. 1) [difference in area not statistically

Table 2. Blurred central vision after migraine and 7 to 10 days later.

	Aura (<i>n</i> = 10)	Without aura (<i>n</i> = 10)	Fisher's exact test
1 day after migraine	6	0	$p < 0.05$
7 to 10 days later	1	0	—

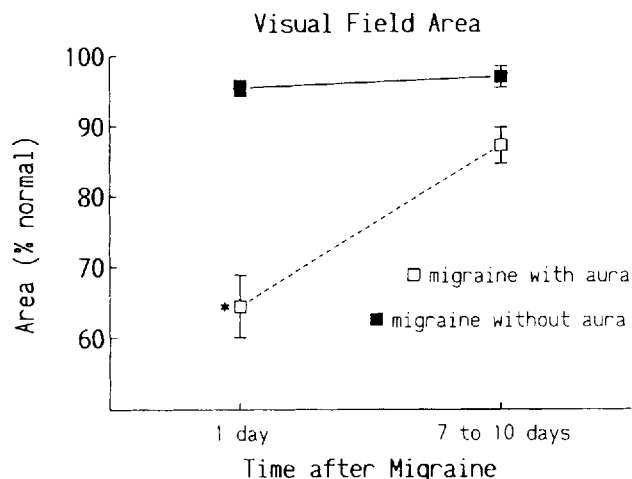


Fig. 1. Visual field area after an attack of migraine and 7 to 10 days later. * In migraine with aura subjects, visual field area was significantly smaller after an attack than 7 to 10 days later [$t(9) = 5.79, p < 0.001$], and significantly smaller than in migraine without aura subjects [$t(9) = 5.59, p < 0.001$].

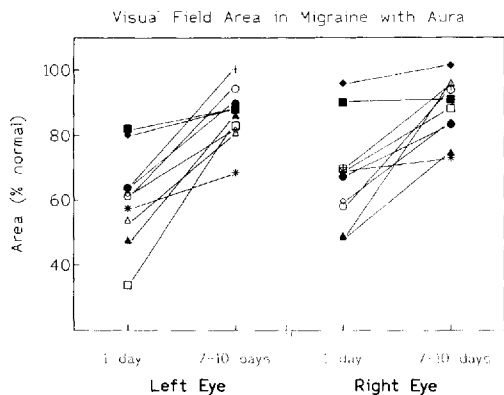


Fig. 2. Visual field area, plotted separately for each of 10 migraine with aura subjects, one day and 7 to 10 days after an attack. Each symbol represents a different subject.

significant; test-retest reliability, $r(9) = 0.64, p < 0.05$]. The interaction between groups and time for visual field area was statistically significant [$F(1, 18) = 32.01, p < 0.001$], primarily because of the decrease in visual field area the day after attacks of migraine with aura (Fig. 1). Visual field area did not change in 5 control subjects retested after 7 days [mean area 94% on both occasions; test-retest reliability $r(4) = 0.99, p < 0.001$].

Visual perception threshold was greater the day after migraine than 7 to 10 days later [$F(1, 18) = 20.75, p < 0.001$], particularly for attacks of migraine with aura [interaction between groups and time $F(1, 18) = 4.34, p = 0.052$] (Fig. 3). However, even in migraine without aura subjects, visual perception

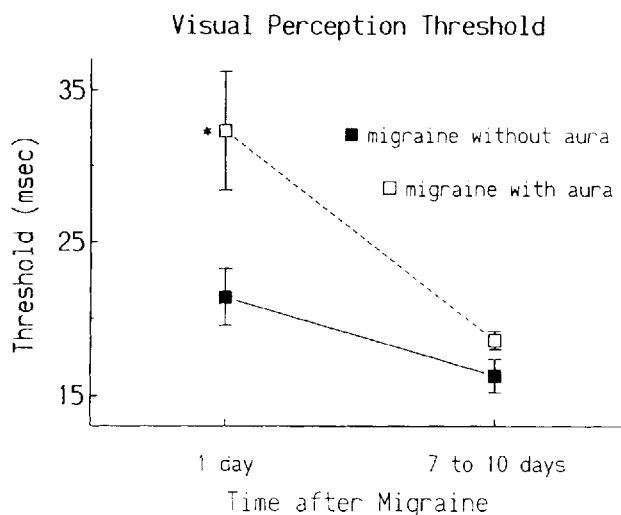


Fig. 3. Visual perception threshold after an attack of migraine, and 7 to 10 days later. * In migraine with aura subjects, visual perception threshold was higher after the attack than 7 to 10 days later [$t(9) = 3.57, p < 0.01$], and higher than in migraine without aura subjects [$t(9) = 2.55, p < 0.05$].

threshold was greater the day after migraine than 7 to 10 days later [21 msec versus 16 msec, $t(9) = 3.38, p < 0.01$]. In contrast, the visual perception threshold did not change in 5 control subjects retested after 7 days (17.2 ± 2.6 msec versus 17.4 ± 2.9 msec, no significant difference).

Discussion

Blurred central vision and depression of visual sensitivity in the periphery of the visual fields were detected in most subjects the day after an attack of migraine with aura. In contrast, the visual fields were intact after episodes of migraine without aura. The scintillations and expanding scotoma of the migraine aura usually resolve before the onset of headache. However, vision often blurs during headache (5), and most of our migraine with aura subjects described "cloudy" vision the day after their attack.

The migraine aura is associated with a reduction in cerebral blood flow, spreading forward from the posterior part of the brain (6). The reduction in cerebral blood flow outlasts overt neurological symptoms by several hours, persisting well into the headache phase of the attack (7). Hyperperfusion then develops and frequently outlasts headache (7). Residual effects of the migraine aura (e.g. focal oedema) could be responsible for the persistence of visual field abnormalities detected the day after an attack. Most of these abnormalities had resolved 7 to 10 days later. Cerebral hyperperfusion also subsides within a week of an attack of migraine with aura (7). Interestingly, abnormalities in visual evoked poten-

tials gradually resolve over several days after attacks of migraine with aura (8).

The screening tests used in our study did not provide enough detail to locate the source of the visual disturbance. In general, however, the depression of visual sensitivity in the periphery of the visual fields was consistent with cerebral deficit (3). More accurate charting of the visual fields with computerized static perimetry might help to resolve this issue.

Blurred central vision or visual field loss may have contributed to the increase in visual perception threshold the day after an episode of migraine with aura. Part of the increase in the perception threshold was non-specific, because a small increase was also detected after attacks of migraine without aura. Migraine sufferers generally feel tired or "washed out" after an attack (9); thus, concentrating on the perceptual task may have been more difficult than usual. The decrease in threshold the week after the attack may have resulted from previous experience with the test; however, control subjects did not show a practice-effect.

Lewis et al. (1) detected persistent, minor visual disturbances in 35% of their sample of migraine patients, and Salerni et al. (10) reported scotomas or homonymous quadrant defects in 9 of 20 (45%) of migraine with aura patients studied when headache-free. Our perimetry procedures were not sensitive enough to detect minor visual disturbances with any precision but, nevertheless, small areas of visual loss were observed in 10 of 23 migraine with aura subjects studied at least 7 days after an attack. Visual fields were intact in all migraine without aura subjects and non-headache controls. The pathogenesis of these persistent minor visual disturbances is uncertain, but the association with the visual aura of migraine suggests a common mechanism.

Major focal neurological deficit induced by stroke is a rare complication of migraine (11). Migraine-related stroke usually develops in patients with a past history of migraine with aura (12), although stroke in migraine without aura has also been described (13). Welch and Levine (14) proposed that platelet aggregation might contribute to the pathogenesis of migraine-induced stroke; sluggish flow through cerebral vessels during and after the migraine aura could further increase the risk of cerebral thrombosis. Cerebral blood flow decreases to a mildly ischaemic level during the migraine aura, and rarely may decrease to a level which causes irreversible neurological deficit (15). The mechanisms which mediate migraine-related stroke might contribute to

the persistent, albeit minor, visual field loss after attacks of migraine with aura. Whether subjects with persistent subclinical visual disturbances have an increased risk of migraine-related stroke requires further investigation.

In conclusion, the present findings indicate that minor visual deficit outlasts attacks of migraine with aura by at least one day. Neurovascular disturbances associated with the migraine aura probably mediate this residual visual deficit.

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