Exploring the visual hallucinations of migraine aura: the tacit contribution of illustration

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The visual aura of migraine is a subjective phenomenon, and what the migraineur experiences is necessarily inaccessible to others. Fortunately, however, the sufferer can occasionally reveal what is being seen by means of graphic representation, enabling an otherwise closed ‘window’ to be opened on the transiently dysfunctioning brain. This article explores the unique contribution that illustration has made to understanding mechanisms subserving the visual aura. The most revealing illustrations are those made by the very few scientists who have recorded and analysed the scotomas, and in particular the expanding fortification spectra, experienced during their migraine attacks. It is solely through illustrations such as these that the uniform nature of many of these hallucinations has been demonstrated. As a result, it follows that there is likely to be a similarly uniform repertoire of processes that generate the hallucinations in the occipital cortex. The precise form of the zigzags that comprise the fortification spectrum, their shimmering appearance, and in particular the speed of the peripheral spread, all of which are entirely dependent on graphic display for their elucidation, enable conclusions to be reached about a number of the underlying pathophysiological mechanisms, including the involvement of spreading cortical depression, that likely occur. Illustration has been pivotal too in revealing uncommon and sometimes curious, if not bizarre, visual hallucinations, the forms of which suggest that extrastriate and temporal lobe involvement contributes to migraine aura in some instances. Illustration can also be valuable in differential diagnosis, depicting other forms of visual hallucination which result from a variety of non-migrainous causes. Illustration, particularly when made during the attack, provides an unusual, little used but powerful tool which uniquely allows the sufferer’s subjective visual experiences to inform objective analysis. In turn, this analysis leads to insights into some of the cerebral disturbances which subserve migraine aura.

Keywords: illustration; hallucination; visual aura; migraine

pivotal role of illustration when ‘surveying the inner wall of vision’ (Updike, 1964), and discusses its role in elucidating the processes subserving the visual aura of migraine.

As unusual and ingenious scientific devices for translating symptoms into objective information, the most revealing illustrations are those detailed graphic analyses made by the migraineur during the time the visual disturbance is actually being experienced. Considering the high prevalence of migraine both with and without aura, however, accurate illustrations are extremely few in number. It is all the more remarkable, therefore, that it is just these rare illustrations which have provided so much of importance for understanding the underlying cerebral disturbances. By comparison, illustrations made retrospectively, or at second hand, provide less accurate but none the less often valuable information.

It is obvious that the illustrations made by the migraineur depend on the ability to represent accurately what is seen. For example, children may have difficulty in expressing their visual disturbances, although illustrations have confirmed that even young children experience similar visual phenomena to adults during migraine attacks (Hachinski et al., 1973; Strafstrom et al., 2002). The ability to record what is being seen may also be compromised by debilitating accompanying symptoms such as impaired concentration and disturbed cognition (Aring, 1972). Fortunately, however, these disturbances may be minor or absent during the visual prodrome—contrasting with the more profound cognitive disturbances in drug-induced and various other hallucinatory states sometimes accompanied by visual aura.

On occasions, and not attributable to impaired ability or confusion, peculiar perceptual features appear in illustrations made by migraine sufferers. These pictures, which often also include features of distress or suffering, are well represented by contributions from the public to the various exhibitions of migraine art, and also by impressive examples of some professional artists’ output. Important as they may be to the sufferers, however, these illustrations, which are briefly discussed later, only occasionally provide insights into mechanisms. Thus it is to those few analytical illustrations made by scientists that most attention must turn when studying these visual phenomena.

The essential role of illustration in demonstrating uniformity

A prerequisite for any study of the mechanisms subserving migraine is that there should be at least some degree of constancy and uniformity in the phenomena experienced. If every sufferer’s experiences were entirely different, investigation would be almost impossible, and it would be equally impossible to envisage any common or unifying mechanism. It is, however, through illustration, rather than verbal description, that the uniform nature of the various hallucinations has been so unequivocally documented. In passing, it has also been possible to demonstrate that, in spite of what might be changing forms of verbal expression over the years and indeed centuries, this visual constancy can be shown to be uniform over time. Uniformity can readily be demonstrated in two particular examples of the visual aura of migraine: the scotoma and the fortification spectrum, and these are discussed in the sections that follow.

A comment on the use of the term ‘hallucination’ is apposite here. It should be clarified that the term is used throughout in a rather loose and umbrella fashion, following ffytche and Howard who deliberately had ‘not differentiated between hallucinated (without afferent sensory signals) and illusory percepts (false percepts with afferent sensory signals) nor’ . . . ‘differentiated between the perceptual experiences that are recognized as real by the patient and those that are not’ (ffytche and Howard, 1999).

In the present article this means the term can encompass the great variety of visual experiences that have been described during the aura, including reports of some extremely bizarre perceptual phenomena, (e.g. Podoll and Robinson, 1999, in which illustrative examples are included), and a number of which are discussed in a later section. Furthermore, some authors have reported that their visual aura was influenced by retinal illumination, (e.g. Fisher, 1999; Pöppel, 1973) this indicates that visual phenomena that originate within the brain can yet be modulated by such ‘afferent sensory signals’, thereby blurring the distinction between internally and externally triggered visual sensations. Nosological considerations aside, ‘hallucination(s)’ is a word now accepted and in widespread use throughout the literature on migraine aura, and indeed it even appears in titles of articles, (e.g. ffytche, 2004; Panayiotopoulos, 1994, 1999; Reggia and Montgomery, 1996; Wilkinson, 2004).

The uniformity of the scotoma

A striking example of uniformity, impressively revealed by graphic representation, is the illustration of certain scotomas, some of which precede, trail or replace the fortification spectrum. Interesting is the frequent use of the printed page, superimposed on which is displayed the central or paracentral and often jagged-edged scotoma, and examples showing this technique have spanned a century (Gowers, 1904; Jolly, 1902; Wilkinson and Robinson, 1985) (Fig 1). Using a fine detail and high-contrast background such as print, or more recently a dynamic random-dot noise pattern (Grüsser and Landis, 1991) (Fig. 2), with which to reveal the scotoma and its borders in greater detail is not surprising. This is because, as Ekbom noted, his scotoma was ‘no larger than the fraction of a letter’ when he was reading a newspaper (Ekbom, 1975), a visual field defect that would easily be missed in other circumstances. Sufferers often report the scotomatous area is grey and indistinct, rather than black, but colours may also be observed (e.g. Airy, 1870; Grüsser and Landis, 1991).
The uniform nature of the fortification spectrum

Arguably the most dramatic and revealing visual aura that accompanies an attack of migraine is the fortification spectrum. This term comes from the resemblance of the zigzags to the fortified, star-shaped bastions and ravelins of mediaeval town walls, and not to the appearance of the crenellations that surmount fortified or decorated walls. Another descriptive term, 'teichopsia', Greek for 'town-wall vision', was introduced by Hubert Airy (1870), and an account of the terminology of the fortification spectrum and related hallucinations has been given by Plant (1986), who also pointed out that 'spectrum' refers not to the coloured spectrum of the rainbow but to the spectral or ghostly apparition of the hallucination.

The fortification spectrum is so characteristic as to be almost diagnostic of migraine. Its homonymous jagged and shimmering, scintillating zigzags usually start central or more often just paracentral to fixation (e.g. Airy, 1870; Ekbom, 1975; Fisher, 1999; Grüsser, 1995; Pöppel, 1973); the zigzags then move outwards, expanding and becoming brighter as they proceed, and disappear in the periphery. As noted earlier, the sufferer often first becomes aware that there is something amiss when reading (e.g. Airy, 1870; Ekbom, 1975), finding one or more individual letters has disappeared, and the scintillating zigzags then develop soon after. These move when the point of gaze is moved, they affect both eyes simultaneously, and typically accompanying the zigzags is an inner, characteristically bean-shaped scotoma. The march of the hallucination takes ~20 min, and is often but not always followed by headache and the other typical components of migraine.

But if, in summarizing numerous sufferers’ descriptions, this account describes the fortification spectrum, it tells little else, and merely emphasizes that, in investigating the migraine aura, the 'main constraints are imposed by the subjective results from psychophysical recordings of the symptoms' (Dahlem and Müller, 2004). From the written description of this hallucination it would be impossible to envisage, let alone investigate, precisely what is being experienced; one instinctively reaches for a pencil.

Exemplifying why illustration proves an essential investigative tool, and one often far superior to the written account, are the supposedly earliest illustrations of a migrainous visual aura. These appeared in Scivias, the
mediaeval illuminated manuscript written around 1180 by Hildegard of Bingen (1098–1180). It has been claimed that this visionary German abbess suffered from migraine and that her illustrations ‘indisputably’ represented migrainous hallucinations (‘visions’), (e.g. Sacks, 1993). This view originated from the influential paper by the medical historian Charles Singer, who commented on the ‘often definite fortification figures’ (Singer, 1917). However, even cursory inspection of the illustrations shows they reveal features entirely different from those characteristic of migraine—rather, Hildegard’s ‘fortification figures’ resemble the castellated tops of battlements, which are depicted together with human figures. To the present writer, neither Hildegard’s illustrations, nor her descriptions, are at all suggestive of migraine aura, and others too have been sceptical (Levene, 1975–6; Plant, 1986; Rose, 2004). Here illustration has been instrumental in discriminating fact from fiction.

Revealing the characteristics of the fortification spectrum started in earnest in the 19th century. The consistently jagged outlines to scotomas, that recall the jagged periphery of the fortification spectrum, have been referred to earlier. However, probably the first but almost unknown illustration of the spreading scintillations of a migraine aura appears in a remarkable letter from Sir George Biddell Airy, the Astronomer Royal (Fig. 3, upper left). In 1865 he described his own spreading zigzags as resembling ‘a Norman arch’; the zigzags are deeper inferiorly, and they ‘tremble strongly’, again particularly inferiorly. Only one arch is seen at a time, it expands as it spreads outwards, and ‘finally passes from the visual field’, the duration of ‘this ocular derangement’ being 20–30 min (Airy, 1865).

However, undoubtedly the most famous and similarly detailed, but entirely different, form of representation of the fortification spectrum was published by G. B. Airy’s son, Hubert, in 1870 (Fig. 3, upper right), and this important
illustration is discussed in the following section. Later in the 19th century others, too, illustrated fortification spectra, another fine example being the numbered sequence of coloured hallucinations drawn by the sufferer, a professional etcher, and reproduced by Babinski in his paper on hysteria and migraine (Babinski, 1890) (Fig. 3, lower left). The visual hallucinations as depicted would be consistent with migraine aura, even if other features of the attacks were somewhat unusual.

Intriguingly, the patient was referred to Charcot in 1886, and a year later Charcot himself illustrated a fortification spectrum in his ‘Leçons du Mardi à la Salpêtrière’ for 22 November 1887 (Fig. 3, lower right). After interviewing ‘le malade’ who suffered from ‘la migraine ophthalmique’, Charcot not only illustrated the visual disturbance but also commented that it ‘ressemble à une plan de fortifications . . .’ (Charcot, 1887). Whether Charcot knew of the Airys’ papers and the various other descriptions of the fortification appearance is unclear, but Charcot evidently appreciated the analogy between the hallucination and its resemblance to the architectural features. Non-scientists, too, have left graphic records of what are probably their hallucinations, including Pascal, whose doodles in the margins of his letters are thought to show his own fortification spectra (Crichtley, 1967).

If these illustrations of the fortification spectrum, as with those of the scotoma, show how similar these hallucinations are, and thus strongly suggest an equally similar repertoire of underlying physiological processes, illustration was yet to prove far more powerful in understanding those physiological processes.

**Representing the march of the visual hallucination**

It would be all but impossible to investigate the dynamic cortical events taking place in the visual cortex without the detailed graphic depictions of the expanding aura made during the time of the migraine attack. As mentioned earlier, the best known illustrations of this spreading visual hallucination were those drawn in 1870 by Hubert Airy, son of Sir George Airy, both of whom suffered from migraine. Hubert Airy’s famous drawings, published by none other than his father in the *Philosophical Transactions of the Royal Society of London*, record the progress and expansion of his own visual disturbances (Airy, 1870), and are arguably the most beautiful scientific records of migraine aura ever made (Fig. 3, upper right). However, although Airy stated that his auras lasted ‘about 30 min’, his illustrations did not record the timing of the successive images.

A later, similar graphic representation of the outward rippling of the fortification spectrum was provided by Aring, who, although stating the phenomenon lasted ~20 min, again provided no further information (Aring, 1972). Thus the illustrations provided by the Airys (1865, 1870) and Aring (1972) documented the constant nature of the spreading and expanding disturbance with its time course of 20–30 min, and others during the past century have made similar graphic recordings of their self-observed spreading scotomas and fortification spectra, sometimes using a series of images, (e.g. Hare, 1966; Jolly, 1902; Pöppel, 1973). However, none of these illustrations, even though they sometimes included sequences, provided the detail necessary for further analysis of the temporal aspects.

In 1941 Lashley provided the first and remarkably detailed quantitative records of the nature and temporal spread of his own migrainous scotomas and fortification spectra. In his classic illustrations Lashley plotted in detail the movement of his hallucinations, enabling him to deduce that the speed of its spread over the cerebral cortex was ~3 mm/min (Lashley, 1941) (Fig. 4). Others came to a similar conclusion, and recently Wilkinson, using subjects who drew their auras under controlled conditions, projected these changes of spreading aura on to a map of the unfolded striate cortex, and confirmed a rate of spread of ~2 mm/min (Wilkinson, 2004).

Even more detailed and analytical, self-drawn illustrations of the evolving fortification-like patterns during migraine attacks have been made by a small number of scientists, including McCulloch (Bücking and Baumgartner, 1974), and Grüsser and his colleagues (Grüsser and Landis, 1991), and these illustrations have confirmed and developed Lashley’s observations. Once again, it is solely illustrations such as these, some examples of which are included in Fig. 5, which enable these fortunately uniform and consistent phenomena to be evaluated with precision, and considerable quantitative information to be derived.

Not only do these illustrations reveal a striking inter-observer consistency, but illustrations can reveal intra-observer consistency as well, as exemplified by Grüsser’s recordings of 11 of his own attacks over 15 years (Grüsser, 1995). From such recordings it has also been confirmed that the speed of movement of the spreading hallucination increases as the hallucination moves peripherally.

**Analysing the composition of the fortification spectrum**

An understanding of the composition of the fortification spectrum is almost entirely dependent on its graphic depiction. Drawings of the hallucination show that the characteristic features comprise short stacks of jagged, alternating black and white lines or bars, the stacks being set at angles to each other, and arranged in an arc surrounding a scotoma at the trailing border.

One of the most detailed early analyses resulted from the illustrations reported by Richards (1971), although these were not those recorded by the author during a migraine attack, but were those ‘seen by the author’s primary subject (his wife)’ and redrawn from her sketches (Fig. 6, left). Whilst acknowledging they are second-hand, these detailed illustrations...
of the ‘bars’ in the fortification arc show that they consist of two parallel bright lines with a dark gap between. The lines are described as shimmering and oscillating in brightness, ‘with all the inside lines ‘on’ when all the outside lines are “off”, and vice versa’, producing ‘a “boiling” or “rolling” motion’. This appearance suggested to Richards that there was a network of reciprocal inhibition, with depression of activity enhancing the spontaneous neural activity in adjacent regions.

The parallel lines of the fortification spectrum are ‘serrated’ or angulated, with an internal angle of $\sim 45^\circ$ near the centre of the visual field, increasing to $\sim 70^\circ$ at the periphery. Richards pointed out that his wife’s redrawn illustrations showed there were no radial lines perpendicular to the boundary of the fortifications, and that there were often gaps separating the ends of the lines (Fig. 6, right). The size of the individual phosphene ‘bars’ or ‘particles’, and the width of the scotoma, increase with increasing eccentricity.

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**Fig. 4** The first analytical drawings of the spatiotemporal evolution of spreading migraine hallucinations. Left: a scintillating scotoma, spreading at the intervals shown. The blindspot is shown as the dotted circle, and the fixation point as x; alternate outlines are solid and dotted to avoid confusion; scintillations were confined to the region above the line s-s. Right: successive forms of a scintillating scotoma, mapped at different time intervals as indicated; the fixation point is shown as x. From Lashley (1941). Copyright © 1941 American Medical Association. All rights reserved.

from the centre. This had first been quantified by Hare (1966), whose detailed self-observations were presented in a graph that showed the increasing vertical diameter of the visual disturbance as the attack progressed.

From Lashley’s drawings it has been possible to confirm not only the expansion and spread of the scintillating scotoma, but also that the detailed components of the fortification pattern remain unaltered, although Lashley noted that the pattern was coarser and more complicated inferiorly (Lashley, 1941) (Fig. 7). The difference in the appearance between the upper and lower parts of the hallucination strikingly recalls G. B. Airy’s observations of his own migrainous zigzags almost a century earlier (Airy, 1865), although such differences may simply relate to the distance of the fortification patterns from the fovea (Grüsser and Landis, 1991).

How many lines, their lengths, and the angles between them vary to some extent between different individuals, and because they move and shimmer, even the most detailed drawings are at best approximations as to what is being seen. Nevertheless, the reproducibility of these observations (e.g. Grüsser, 1995) again strongly suggests that there is likely to be constancy of pathophysiological mechanisms which generate the various components of the hallucinations.

Cortical spreading depression (CSD): a likely mechanism revealed through illustration

In 1944, Leão described a new phenomenon: the wave of depression of cortical electrical activity that follows electrical or mechanical stimulation of the cerebral cortex (Leão, 1944). This spreading depression is a wave of short-lasting neuronal and glial depolarization that moves across the cortex in all directions at a speed of 3–5 mm/min. It is accompanied by a brief, marked increase in cerebral blood flow, which is followed by a more prolonged phase of hypoperfusion (Lauritzen, 1994). It is now known that CSD can also be induced by other stimuli, including high concentrations of potassium ion and glutamate, and, at least in animals, it can be blocked by NMDA antagonists. The interruption of cortical function is associated with profound changes in cortical ion homeostasis, in particular intracellular loss of potassium and hydrogen ions, and entry of sodium, calcium and chloride ions together with water, but involvement of a host of other chemicals and up-regulation of a variety of genes are also known to occur (Sanchez-del-Rio and Reuter, 2004), though the precise mechanisms mediating cortical spreading depression remain unclear.

Fig. 6  
Left: Redrawn sketch of Richard’s wife’s spreading fortification spectrum; ordinarily one arc is seen at a time; the dot is the fixation point. Right: analysis of its component bars, showing the inner angle of the serrations are ~45° near the centre, increasing to ~70° at the periphery; the radial lines, which are never seen, have been added to show the postulated underlying honeycomb pattern and hence latticed structure of the visual cortex. From Richards (1971). Reproduced with permission of Jerome Kuhl.

Fig. 7  
The scotoma and different components of the fortification outline, with the coarser and more complex features represented in the lower part. See text for further details. From Lashley (1941). Copyright © 1941 American Medical Association. All rights reserved.
A year after his first paper on CSD, Leão himself had speculated in passing that it might be associated with epilepsy

‘and the presumably related condition of migraine. The latter disease . . . is suggestively similar to the experimental phenomenon here described’ (Leão and Morrison, 1945).

It was Lashley’s illustrations of his migraine aura, however, and his conclusion concerning the speed of spread of the hallucination, that subsequently enabled Milner to more clearly envisage the possible link between CSD and migraine. In a prescient comment, he stated:

‘In brief, the writer thinks that attention should be drawn to the striking similarity between the time courses of scintillating scotomas and Leão’s spreading depression because, if there is a true correspondence between these phenomena, there is hope that some of the work done on spreading depression can be brought to bear on the problem of migraine’ (Milner, 1958).

For two reasons the implications of Milner’s comments proved a subject of much debate. First, theories that postulated a primarily vascular basis for migraine and its aura vied with theories invoking a primarily neural basis (Spierings, 2004), even interictally (Kennard, 1996). Second, although the involvement of CSD in migraine had, as indicated earlier, been long proposed, and indeed Lauritzen had noted ‘It appears that CSD displays sufficiently important similarities to the migraine attack that it can be considered a disease model’ (Lauritzen, 1994), it was unclear whether the experimental findings in animals that underpinned the concept of CSD also obtained in humans—which it has recently been confirmed they do (e.g. Fabricius et al., 2006).

This suggestion has been indirectly supported by numerous imaging techniques including xenon, SPECT and PET scanning, and MRI, and by magnetoencephalography (MEG) (Hadjikhani et al., 2001). However, further and compelling evidence was recently provided by means of functional MRI studies on three migraineurs. One of the subjects induced two of the attacks, and two other subjects had three spontaneous attacks. Admittedly, the spreading visual aura was not a fortification crescent, ‘like TV snow’, that progressed peripherally in a similar fashion and was followed by a central scotoma. Admittedly, too, the illustration of the spreading hallucination was not drawn by the migraineur during the scanning but was mapped by noting the times of onset and cessation of the scintillations (Fig. 8). Nevertheless, it was crucially the graphic display of the migraineurs’ hallucinations during the aura that allowed correlation with the changes in the blood oxygenation level-dependent (BOLD) signal (Hadjikhani et al., 2001).

Hadjikhani and colleagues (2001) were able to confirm that there was an initial increase in BOLD signal, possibly due to vasodilatation, which progressed slowly and contiguously at a rate of ~3.5 mm/min, and this was congruent with the visual aura. Following this progression was diminution in the BOLD signal, perhaps due to vasoconstriction. These and other data enabled the authors to conclude that the visual aura does indeed have characteristics typical of CSD, and the vascular changes appear to be secondary. Whilst both areas are retinotopic, in one of their patients the aura appeared to arise from the extrastriate area V3 rather than V1, and extrastriate and temporal lobe involvement in migraine aura has also been detected by MEG (Hall et al., 2004)—an aspect commented on later.

The implications that structural elements of the brain, or what would now be considered the specific cytoarchitectonic composition of the visual cortex, might be crucial for understanding the visual hallucinations in migraine, was correctly foreshadowed over a century ago. Sir John Herschel, another famous 19th century astronomer, speculated that his migraines might be due to ‘. . . a kaleidoscopic power in the sensorium to form regular patterns by the symmetrical combination of causal elements . . . ’ (Herschel, 1866). Over the ensuing 150 years, as a result of detailed analyses of the illustrations of the aura made during migraine attacks, a number of likely neural processes at the cellular level have been elucidated (for reviews, see, e.g. Grüsser and Landis, 1991; Grüsser, 1995; Dahlem et al., 2000; Dahlem and Chronicle, 2004; Silberstein, 2004). In brief, a continuous and smooth wave of excitation probably propagates across the primary visual cortex (Dahlem et al., 2000); the zigzag pattern has been related to the functional micro-structure of the hypercolumns in area V1 (Richards, 1971; Grüsser, 1995; Wilkinson, 2004); the flickering phosphenes have been attributed to spreading waves of cortical hyperactivity, with the scotoma at the trailing edge of the fortification spectrum being attributed to neural hypoactivity (Lashley, 1941; Grüsser and Landis, 1991); and the peripheral spread of the hallucination has been attributed to spreading diffusion of a variety of ions and neurotransmitters, including potassium ions or glutamate, or both, along the extracellular space along the stripe of Gennari—the disappearance of the hallucination perhaps being because this process stops at the area V1/V2 border (Grüsser, 1995).

Furthermore, computational models of travelling waves of neural activation as seen in CSD can generate corresponding visual features that strikingly resemble the hallucinations in migraine aura (Fig. 9). Such models also satisfactorily predict an exponentially increasing speed of the visual hallucinations as they spread peripherally (Reggia and Montgomery, 1996).

Thus, in summary, it is now generally accepted that neural processes which give rise to CSD are indeed likely to account for the spreading aura of migraine (Silberstein, 2004; Teive et al., 2005): Leão and Milner were almost certainly correct. This link could only have been made by means of illustration, and it is striking that so many scientific articles, including those dealing with theoretical
aspects, necessarily incorporate one or more of the illustrations discussed in this review.

**Illustrating the effect of haemodynamic changes on the migraine visual aura**

Hare plotted his own migrainous hallucinations in 12 attacks (Hare, 1966). On six occasions during three attacks he was able to show that inhalation of ‘small doses (3–5 breaths)’ of amyl nitrite temporarily reduced the vertical diameter of the spectral pattern by $5\%-15\%$ of the expected diameter. Larger doses of amyl nitrite had no effect. He speculated that the march of the visual disturbances was either related to a steady state of vasoconstriction with increasing spread of anoxia, or that there was a gradually increasing spread of vasoconstriction throughout the march. As an aside, he also speculated in a later study whether ergot, presumably through its vasoconstricting properties, altered the form of the visual hallucination.

Hare’s graph recorded an effect that had previously been shown but in a different form by observations made by Cahan. During two episodes of migraine, Cahan made ‘accurate and rapid observations of his own visual field defects’; he displayed his observations by means of serial plots of his visual fields after inhaling amyl nitrite, comparing the visual changes after an amount insufficient to induce hypotension with an amount producing symptomatic hypotension (Fig. 10). Schumacher and Wolff, who reported these studies, concluded that cerebral vasodilatation without hypotension caused the scotoma to disappear, but hypotension that presumably resulted in decreased cerebral blood flow exacerbated the visual disturbances (Schumacher and Wolff, 1941). Neither Hare, nor Schumacher and Wolff, took account of the neural substrates that might also be involved and which are considered today to be crucial.

**Illustrating neurophysiological influences on the fortification spectrum**

Although Pöppel had reported that eye movements shifted his fortification patterns by the angle of eye movement (Pöppel, 1973), more extensive studies were published by...
Jung (1979). Whilst confirming Pöppel's observation, Jung also investigated the influence of induced nystagmus on the fortification spectra seen during his own and a colleague's migraine attacks. He found that there was translocation of the phosphene in the direction of the slow phase of the nystagmus induced by optokinetic stimulation, but—in contrast to voluntary eye movements—the phosphenes induced did not cross the midline. Furthermore, he depicted the effects of nystagmus induced by a rotating chair: on rotation to the right, the phosphene moved to the left in the direction of the slow phase of the nystagmus, and became more elliptical. On stopping rotation, the phosphene moved to the right in the direction of the slow phase, became flattened, and moved transiently beyond its initial position.

Jung used illustrations to demonstrate the effects of these vestibular influences on the spontaneous peripheral spread of the fortification spectrum, and concluded that the vestibular system has inhibitory effects on what would otherwise be more marked movements of the visual hallucination. However, the illustrations themselves lack detail and have limitations; for example, it is unclear how they were drawn, the distance of the screen from the migraineur, and the timing of the normal outward drift of the phosphene in relation to the induced nystagmus. The illustrations therefore provide in some but not exhaustive detail what would otherwise be unrecordable physiological phenomena.

**Illustrating visual perceptual changes in migraine**

Contrasting with the illustrations discussed earlier, and which contribute much to the scientific analysis of the migraine aura, are the numerous illustrations which simply express distress. However, there are yet other illustrations that reveal much more unusual perceptual visual disturbances, the processes causing them being ill understood. Various disturbances of visual perception in migraine have been reported over the past century, and 40 years ago Klee and Willanger reviewed the available literature and added eight cases of their own (Klee and Willanger, 1966)—although their observations are limited by the lack of graphic representation of these disturbances. It was, however, and unexpectedly, through the advent of exhibitions of migraine art that the extent of the visual phenomena experienced by migraineurs first became evident.

In the first British exhibition of migraine art in 1981, 32 of the 207 entries showed metamorphopsia or alterations of shape or objects with distorted contours (Wilkinson and Robinson, 1985). From larger numbers of pictures available through further exhibitions, even more unusual perceptual disturbances have been identified; for example, 7 illustrations from a total of 562 pictures were recorded as showing out-of-body features—including hallucinations of a duplicate or parasomatic body (Podoll and Robinson, 1999). Sufferers’ drawings of a corona around objects, Lilliputian hallucinations, illusory splitting, and macro- and microsomatognosia are other examples in which illustration has been the sole means by which these visual auras became recorded and characterized, and even digital image reconstruction of such disturbances has been employed (Kew et al., 1998).

Unfortunately there is little if any information on when the pictures were made in respect of the timing of the migraine attack, and often specific elements of the visual aura and expression of distress are combined in an interesting but scientifically less useful way. Nevertheless, despite their subjective nature, ‘the large number of similar representations probably confirms their organic nature’ (Wilkinson and Robinson, 1985). Temporal lobe dysfunction during the visual aura of migraine in particular may be relevant (Podoll and Robinson, 1999), and temporal lobe changes detected by MEG (Hall et al., 2004) and by functional MRI (Hadjikhani et al., 2001) have been referred to earlier.
The differential diagnosis of the visual aura of migraine

Countless illustrations have recorded the great variety of visual hallucinations in many non-migrainous disorders. These disorders have been classified into those involving the visual pathways, and those involving the brainstem/cholinergic system. The former group is seen in ocular disease, occipital infarcts and epilepsy, as well as migraine; the latter group is variously attributable to anticholinergic drugs, Parkinson’s disease, narcolepsy and peduncular lesions. Some disorders, for example dementia, psychoses and delirium, may implicate both groups. In ‘matching syndrome to aetiology’, it has been argued that ‘the palette of hallucinations in a given patient thus reflects the location and extent of their susceptible cortex’ (ffytche, 2004).

Particularly when visual hallucinations result from disorders of the visual system, the disorder may be far more clearly identified when revealed by self-illustration. For instance, in another irritative cortical phenomenon, the occipital epilepsy of childhood, Panayiotopoulos argued that, whilst not invariable and whilst epilepsy and migraine

Fig. 10 Cahan’s self-observations on his migrainous field defects when inhaling a small (upper figure) or larger (lower figure) amount of amyl nitrite. In the former experiment, when blood pressure was maintained, the migraine scotoma disappeared, whereas in the latter, when hypotension developed, the scotoma increased. See text for further details. From Schumacher and Wolff (1941). Copyright © 1941 American Medical Association. All rights reserved.
can coexist, colours and circular or spherical shapes are typically encountered in the former, and jagged, and often non-coloured hallucinations in the latter (Panayiotopoulos, 1994, 1999). Although comprising pictures made by children after, rather than during, their epileptic attacks, Fig. 11, left, shows the appearance of visual hallucinations which clearly are quite different from the usual hallucinations experienced during migraine aura.

When reviewing the various hallucinatory phenomena encountered in eye disease, ffytche and Howard (1999) commented on the similarity of certain forms of hallucinations. For example, these authors drew attention to ‘the consistency’ of tesselloptic hallucinations in patients with a right occipital infarct, under the influence of LSD and mescaline, ‘patients with eye disease’, and migraine. The term ‘tesselloptic’ is derived from ‘tessellopsia’ (from the Greek ‘tesseres’ or tessares’, ‘four’ as in four-sided, and thence a small tile used in mosaic); it was proposed by ffytche and Howard (1999) to describe the patterns of regular, repeating forms seen in brickwork, lattices, mosaics, crazy paving, etc., and recalls what Klüver called ‘form constants’ when describing the similar, often geometric visual hallucinations experienced by those under the influence of mescaline (Klüver, 1966). Acknowledging there are often angulated features to the drawings, to the present writer examination of the graphic appearances often reveals dissimilar features. This dissimilarity is hardly surprising, since it is likely that the different disorders are subserved by equally different underlying pathophysiological processes. Indeed, numerous detailed illustrations testify to the very different visual hallucinations recorded by patients whose disorders range from psychotic disturbances (Guttmann and Maclay, 1937) to the effects of hallucinogenic drugs (Maclay and Guttmann, 1941); and to ophthalmic conditions which affect structures as anterior as the lens (Hu and Scotcher, 2005) and retina (Burke, 2002), and as posterior as the occipital cortex.

Exemplifying the value of illustration in revealing different underlying causes is an example of mescaline-induced hallucinations, described as comprising ‘spreading of zigzag lines’ (Maclay and Guttmann, 1941) (Fig. 11, right). Were these hallucinations migrainous? Recalling the discrepancy between the written and visual accounts of Hildegard’s auras, although the description might suggest a migrainous disturbance, the illustration made during mescaline intoxication immediately reveals a pattern of hallucinations that is easily distinguishable from that occurring in migraine—including the lack of a hemianopic distribution and the far less regular and angulated form of fortification pattern.

Illustration has also allowed detailed analysis of experimentally induced visual hallucinations, including those following stroboscopic (Smythies, 1959, 1960), electrical (Brindley and Lewis, 1968), combined electrical and drug (Knoll et al., 1963), and transcranial magnetic (Cowey and Walsh, 2000) stimulation. Again, whilst the written accounts may variously refer to tesselloptic hallucinations, including those similar to the hallucinations reported with hallucinogenic drugs (Klüver, 1966), the graphic records show that experimentally-induced visual hallucinations are in reality entirely different from those occurring during migraine.

**Conclusions**

Probably the first scientific illustrator of the migrainous visual aura, G. B. Airy rightly, although at the time controversially, concluded that ‘the seat of the disease is the brain’ (Airy, 1865), and graphic representation can provide invaluable and sometimes the only method for elucidating those underlying processes occurring in the brain. Nevertheless, there have been remarkably few
accurate and detailed illustrations—and nearly all of them have been made during the past 150 years by scientists, notably first by astronomers and then by neuroscientists. The paucity of scientifically useful illustrations contrasts with the increasing numbers of illustrations of migrainous phenomena provided by the public. It also contrasts with the output of professional artists such as de Chirico (Fuller and Gale, 1988); in these artists, migraine—diagnosed with varying degrees of certainty—merely seems to ‘expand the repertoir of visual forms’ available to them, rather than reveal underlying mechanisms (Chatterjee, 2004).

Illustration, particularly self-made during the attack, remains a unique but neglected scientific method in an age which prizes objectivity, and in the study of the visual aura of migraine the use of illustration has enabled subjective experience to inform objectivity and analysis. To extend these studies further, Pöppel (1973) and later Grüsser (1995) pleaded for migraine sufferers with visual auras to communicate their observations to the respective authors, and recently it has also been possible for patients to compare their auras with computer-generated animated simulations of fortification spectra. Since these simulations can be ‘alarmingly realistic’ (Dahlem and Chronicle, 2004), they may perhaps prove a valuable tool for further research.

Finally, the ancients from the time of Plato to the albeit wavering believer, Leonardo da Vinci, with whom this account began, were sometimes right (Kemp, 1977): in certain circumstances, what we see indeed emanates from within rather than from without.

Acknowledgement
I am most grateful to Professor Ian McDonald for his comments and encouragement.

Note added in the proof: This contribution is respectfully dedicated to the memory of Professor Ian McDonald, who died as the paper was going to press.

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