

Inferences Regarding the Visual Precipitation of Seizures, Eye Strain, and Headaches

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In photosensitive epilepsy, seizures start in the visual cortex of one cerebral hemisphere or both hemispheres independently. The seizures occur when normal physiological excitation involves more than a critical cortical area, particularly when the excitation is rhythmic. Sodium valproate affects the discharge but leaves the trigger mechanism relatively unaffected.

Non-epileptic sensitivity to light, manifest as eye-strain or headaches, has similar mechanisms, although these mechanisms are less widespread and do not involve synchronisation.

There are many practical consequences of sensitivity to light for the design of the visual environment.

The following chapter will take each of the assertions in the above thesis in turn and describe the evidence upon which it is based. The evidence has been reported in a variety of publications by Binnie et al. (1981, 1985), Darby et al. (1985), Kesteleijn et al. (1982), and Wilkins et al. (1980, 1981, 1984, 1988, 1989).

Epileptic Sensitivity to Light

About 4% of patients with epilepsy are susceptible to visually induced seizures. In most of these patients, and in a few others, the EEG shows a photoconvulsive response to intermittent light (or more correctly, since there are no convulsions as such, a photoparoxysmal response). In about 30% of these patients, a similar EEG discharge will occur in response to stationary, continuously illuminated patterns

of stripes, provided the stripes have the appropriate spatial characteristics. If the stripes vibrate with the appropriate temporal characteristics, as many as 70% of photosensitive patients may be affected. Pattern sensitivity of this kind was initially reported as a rarity, first by Bickford et al. (1953), and subsequently by a number of investigators, notably Chatrian et al. (1970). It was only later, when more was known about the stimulus characteristics responsible, that the relatively high incidence of such a response was recognized (Stefansson et al., 1977).

Seizures Start in the Visual Cortex

There are five main lines of evidence that seizures start at the level of the visual cortex. The first four lines concern the fact that the stimulus characteristics of patterns responsible for the discharge are reminiscent of the response of neurons in the striate and prestriate cortex (see, for example, Hubel, 1988). The remaining evidence concerns the topographic characteristics of the EEG response the patterns evoke.

1. The longer the length of line contour within the pattern, the more likely a discharge is to occur (the pattern in Fig. 22.1a is less epileptogenic than that in Fig. 22.1b, and the pattern in Fig. 22.1b is less than that in Fig. 22.1c). The effect of the length of line contour is suggestive of a role played by neurons in the visual cortex, most of which respond to line contours.

2. If the two eyes see different patterns (e.g., the left eye sees the pattern shown in Fig. 22.1d

* In Memory of Lynda Bateman

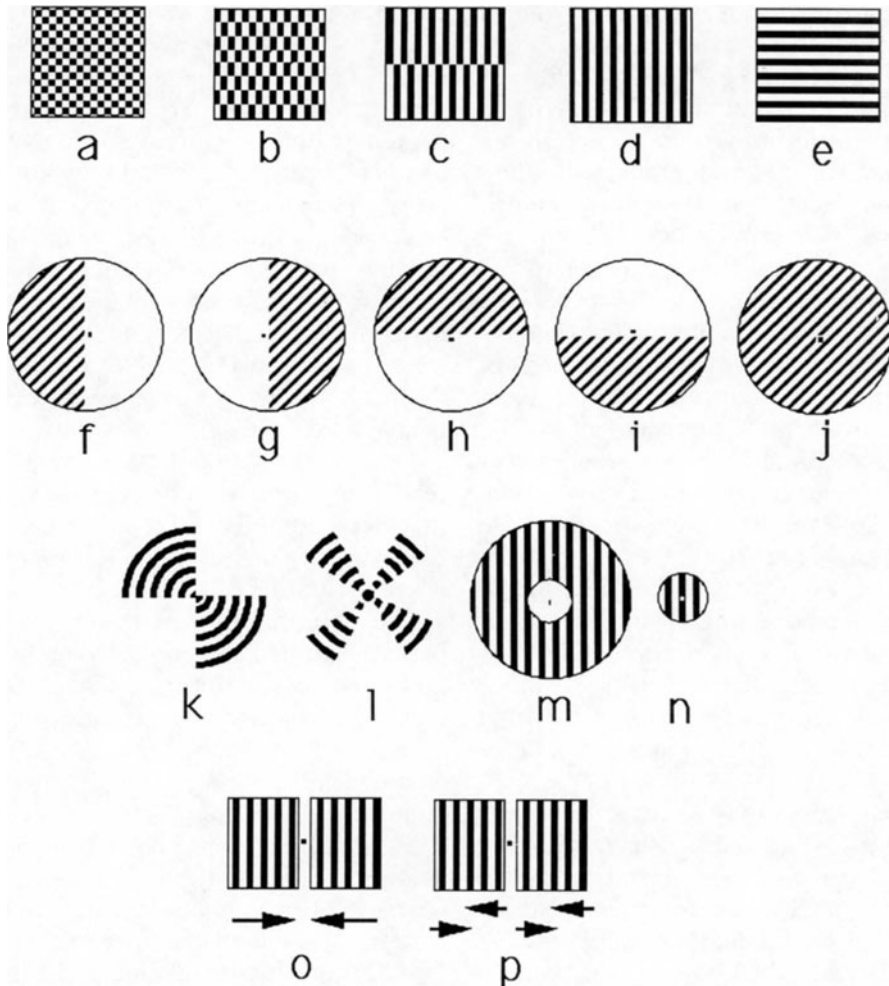


Figure 22.1. Schematics of patterns. A central fixation point was used. The patterns were increased in overall size until epileptiform EEG abnormalities were just detectable. For most patterns the size was increased by increasing the outer radius, but for pattern m the inner radius was also decreased; for patterns k and l the angle of the sectors was increased. The outer radius of the patterns therefore varied from about 2 to 28 degrees. The angular subtense of individual stripes was close to 15 minutes of arc. Most patterns were printed on a card and held against a tangent screen with the same space-averaged luminance as that of the patterns (about

300 cd/m²). The screen was lit with steady diffuse white light and viewed from 0.6 m. The Michelson contrast of the stripes (the difference in the luminance of the stripes divided by the sum of their luminances) was close to 70%. With a few exceptions, the patterns were viewed binocularly. The stripes in pattern o drifted in the direction of the arrows at velocities up to 14 degrees per second. The stripes in pattern p vibrated, abruptly reversing their direction of motion repeatedly after traveling the width of one stripe or two. During each phase the velocity was constant and similar to that of the stripes in pattern o.

and the right eye the pattern in Fig. 22.1e), discharge is less likely than when both eyes see the same pattern and binocular fusion occurs. Many neurons in the visual cortex respond to stimulation of either eye, and some respond

more vigorously to binocular than to monocular stimulation. In the lateral geniculate nucleus (a major subcortical visual structure), there is little binocular interaction (see, for example, Hubel, 1988).

3. Some patients are sensitive only when a pattern of stripes has a restricted range of orientations; most of these patients show little or no astigmatism (Chatrian et al., 1970; Wilkins et al., 1980). Neurones in the visual cortex respond selectively to line contours with a limited range of orientations (see, for example, Hubel, 1988).

4. When a pattern of stripes is moved to and fro in a direction orthogonal to the stripes, the temporal and spatial characteristics responsible for epileptiform activity are independent of one another. Epileptiform activity is most likely to occur when the frequency of oscillation is between 10 and 20 Hz and when two or three spatial cycles of the pattern subtend one degree at the eye. This remains the case whether the movement involves a displacement of one or two stripe widths. The independence of the temporal and spatial aspects of pattern sensitivity is consistent with, but not strongly supportive of, a trigger mechanism involving such neurons as complex cells that respond to lines of a certain width over a range of retinal positions.

5. The scalp topography, in response to patterns in one visual hemifield, is maximal over the contralateral posterior quadrant, and in some patients, the response to upper and lower fields shows a dissociation along the vertical midline (Wilkins et al., 1981). In other words, the topography of epileptiform activity follows that of the underlying visual cortex.

6. The response to intermittent light is often generalized, but when this response is suppressed with sodium valproate, focal occipital activity remains.

One Cerebral Hemisphere or Both Hemispheres Independently

Several lines of evidence suggest that the cerebral hemispheres act independently in the induction of the discharge.

In some patients, pattern-evoked epileptiform EEG activity may be more pronounced when the pattern is presented in one lateral visual field than the other (e.g., the pattern in Fig. 22.1f may be more epileptogenic than that in Fig. 22.1g). When the patient is examined in the conventional way with diffuse intermittent

light (i.e., when the stimulation is bilateral), the response shows a corresponding asymmetry: It is predominant over the hemisphere contralateral to the visual field showing the greater pattern sensitivity. This evidence for an unbalanced hyperexcitability of the two cerebral hemispheres can be found in patients with epilepsy of the primary generalized type.

Many patients do not show any cerebral asymmetry—both hemispheres appear to be more or less equally excitable—but even in these patients it is possible to infer that the discharge occurs independently in the two hemispheres as will now be shown.

First, as has already been mentioned, the response to a pattern presented in one hemifield has a contralateral topography.

Second, when the pattern is presented in one lateral visual field (see Figs. 22.1f,g) the response is far more likely than when the same pattern is turned and presented in the upper or lower visual quadrants (see Fig. 22.1h,i). Both hemispheres are then stimulated but each to a lesser extent. Perhaps a critical amount of excitation *within one hemisphere* is necessary to induce the discharge.

Third, if a pattern is presented in both visual fields (e.g., Fig. 22.1j), the probability of a discharge is not much more likely than when the pattern is presented in one half-field only (e.g., Fig. 22.1f,g). It is surprising that although the bilateral pattern is so much larger, the response to it is not appreciably greater than that to a unilateral pattern, even in patients who show pattern sensitivity equally in the two half-fields. Under other circumstances, doubling the size of a pattern greatly increases the probability of epileptiform activity (see the section on critical cortical area). The lack of a large difference in response to bilateral and unilateral patterns would be expected were the hemispheres acting independently and the independent probabilities of a discharge summed.

The above evidence that the two hemispheres participate independently in the induction of a discharge is quite consistent with the view that seizures arise in the striate and prestriate visual cortex. There are very few interactions between the hemispheres in posterior visual areas, at least in monkeys: It is

mainly in more anterior visual areas (e.g., in the temporal lobe) that both lateral visual fields are represented and that callosal fibers are widespread (see, for example, Zeki, 1978).

Normal Physiological Excitation

The inference that the excitation is normal is based on the observation that photosensitive patients usually have normal vision. It is rare to find ophthalmological abnormalities (although, as we shall see later, patients may complain of eye strain). When examined, patients usually have normal acuity, normal stereopsis, normal phoria, etc. What is more surprising is that contrast sensitivity is also normal. Contrast sensitivity refers to the ability to see very faint patterns, usually of stripes. We have examined contrast sensitivity using the Cambridge Low Contrast Gratings (Wilkins and Robson, 1987), a test developed for this purpose and subsequently found to be of use in detecting visual impairment in optic neuritis, multiple sclerosis, diabetes, and glaucoma when conventional ophthalmological tests fail to do so (Wilkins et al., 1988). The patient is forced to determine whether the grating is on the one or the other of two pages in a booklet, and as the pages are turned, the gratings become fainter and fainter. Although the test is an extremely sensitive measure of visual function, photosensitive patients tend to score well within the norms for their age (A.J. Wilkins, C.E. Darby, C. Neary, and D. Kasteleijn, unpublished data). This is despite the fact that the patterns are exactly those that would elicit epileptiform activity were they of higher contrast.

More Than a Critical Cortical Area

Any region of the visual cortex will induce epileptiform abnormalities provided a sufficiently large area is stimulated. We can infer this for two related reasons.

1. Provided the pattern is symmetrical so that it stimulates both lateral visual fields, it is immaterial which region of the retina is stimulated. Very different patterns of sectorized concentric annuli (see Fig. 22.1k,l) have a similar capacity to elicit epileptiform EEG abnormali-

ties, provided the total areas of the patterns are similar.

2. If the pattern stimulates only the periphery (see Fig. 22.1m), the probability of epileptiform activity is reduced: A large annulus has the same effect as a small pattern that stimulates the area of central vision (Fig. 22.1n). The probability of a discharge is then no longer predicted by the pattern area so much as by the area of the visual cortex to which it projects (as determined from the cortical magnification factor) (for example, see Drasdo, 1977). In other words, the discharge appears when more than a critical cortical area is stimulated. The discharge is extremely dependent on the size of the cortical area involved. If the size of the pattern is reduced slightly, then the discharge disappears. This has facilitated the study of epileptic patients with a minimum of risk: In our studies, pattern size was increased only until it was just sufficient to induce an EEG discharge with a recognizably epileptiform morphology.

Rhythmic Excitation

Inferences regarding the role of the temporal characteristics of excitation can be made from the response to various types of pattern movement.

If the gaze is directed at a central fixation point and a bilateral pattern of stripes drifts continuously towards that point (leftwards in the right visual field and rightwards in the left, see Fig. 22.1o), then nystagmus is avoided, and stable gaze can be achieved. Epileptiform activity is very unlikely to occur, regardless of drift velocity. Epileptiform activity, is however, very *likely* to appear if the characteristics of the pattern movement are altered so that the pattern moves to and fro in a direction orthogonal to the stripes, drifting through a distance of one or two stripe widths before changing its direction of movement (see Fig. 22.1p). The pattern is most epileptogenic when 10 to 20 changes of direction are made every second. The pattern is also highly epileptogenic if the phase of the pattern is repeatedly reversed (white stripes changing to black: black to white) 10 to 20 times per second. Static patterns are more epileptogenic than

drifting patterns and less epileptogenic than vibrating or phase-reversing patterns. In this case, the only retinal movement results from the small drifts and flicks that the eyes make during fixation.

Pattern contours pass through the overlapping receptive fields of cortical neurons, which induces firing. Some of these neurons respond more to one direction of motion than to another. Whilst the pattern is moving in one direction, contours flow into and out of overlapping receptive fields, which causes the neurons that are sensitive to that direction of motion to fire. Therefore the population of neurons as a whole presumably shows a sustained excitation. Some neurons are directionally sensitive. Every time the pattern changes direction, the population of neurons being stimulated will change. If the pattern changes direction rhythmically, as it does when it is caused to vibrate to and fro across the stripes, then the population of neurons as a whole will show rhythmic excitation. Similar rhythmic excitation should occur in response to a pattern that reverses phase. No rhythmic response should result from a drifting pattern—just a sustained excitation. The extreme difference in the response to drifting patterns, on the one hand, and to phase-reversing or oscillating patterns, on the other, suggests that rhythmic activity in the nerve net is important for epileptogenesis. Presumably the small movements of the eyes that occur whilst the eyes are fixating are sufficient to synchronize the action of some neurons and this synchronization may explain the response to stationary patterns. The effects of pattern movement, therefore, suggest that synchronization is critical at the very outset of epileptic activity as well as being a later consequence of the discharge.

Sodium Valproate Affects the Spread of the Discharge But Leaves the Trigger Mechanism Relatively Unaffected

We measured various characteristics of the pattern-induced discharge whilst valproate therapy was instituted. Patients' dose levels were changed over the course of about two

years and they had an EEG examination before each change in dose. We found that as the valproate dose increased, the duration of the discharge decreased, its voltage decreased, the number of electrodes involved in the discharge decreased, and the number of components in the discharge (spikes, sharp waves, slow waves) also decreased.

During each EEG examination, we showed patients patterns of stripes that gradually increased in size until a discharge occurred or until a large pattern had been presented without response. Although the discharge decreased in the manner described above, the critical pattern size just sufficient to induce a discharge showed relatively little change. The probability of a discharge was reduced, but when the discharge occurred it did so in response to a pattern similar in size to that necessary to induce a discharge before valproate therapy was initiated. This would suggest that although sodium valproate effects the spread of the discharge, the physiological mechanisms responsible for triggering the discharge are not so markedly affected.

Nonepileptic Sensitivity to Light

Nonepileptic sensitivity to light is best illustrated by observing the pattern in Figure 22.2. Most people find the pattern aversive: It induces illusions to which some individuals are more susceptible than others. The illusions may include colors, (red, green, blue, yellow), shapes (a faint rhomboid lattice or such distortions as bending of the lines) and movement (shimmering, flickering). We know very little about these visual effects, except that many of them do not appear to be readily attributable to peripheral ocular factors, and some have been interpreted in terms of cortical inhibition (see, for example, Georgeson, 1976, 1980).

Sensitivity Manifest as Eye Strain or Headaches

Curiously, the anomalous visual effects that people see in a grating, such as that in Figure 22.2, are related to the headaches and eye

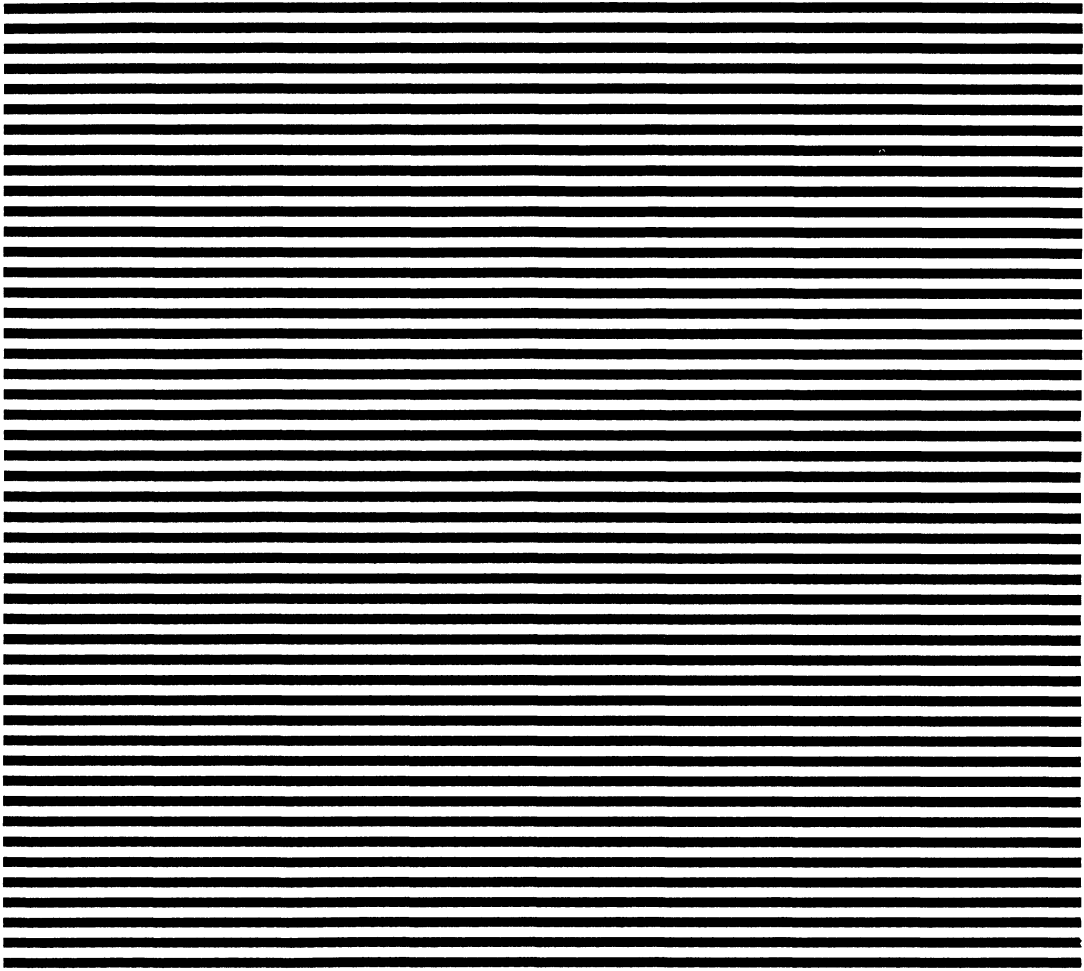


Figure 22.2. An example of an epileptogenic pattern. The Michelson contrast of the stripes is close to 80%. When viewed from a distance of 0.43 m, the pattern subtends 18 degrees and the stripes have a

spatial frequency of 3 cycles/degree (i.e., each stripe, white or black, subtends 10 minutes of arc at the eye).

strain they suffer. People who have frequent headaches tend to see more illusions (Wilkins et al., 1984). On days when they have a headache, they see more illusions (Nulty et al., 1987). If the headaches are unilateral, the illusions tend to predominate in one lateral visual field (Wilkins et al, 1984). People who have relatives with migraine tend to report more illusions than those who do not have migraine in the family (Wilkins, unpublished data). Finally, patients with classical migraine, when they are examined during headache-free periods, tend to report more illusions in the visual field in which their aura occurs (Khalil, personal communication).

The link between headache and sensitivity to light is seen not only in the characteristics of pattern sensitivity but also in the EEG response to diffuse intermittent light. Golla and Winter (1959) showed that in persons suffering episodic headaches, but not in controls, the amplitude of the steady-state evoked potential in response to intermittent light is greater at flash frequencies of around 20 per second than at lower frequencies. Their findings have been replicated by Jonkman and Lelieveld (1981) who used more up-to-date recording techniques and by Brundrett (1974) who showed that in a small sample of headache sufferers the decrease in amplitude with increasing fre-

quency was less than in controls. Evidently headache sufferers tend to be unusually sensitive to flicker, particularly at high frequencies (see Winter, 1987 for review).

Similar Mechanisms

Nonepileptic sensitivity to light, manifested as eye strain or headaches, has mechanisms similar to those that underlie epileptic sensitivity. Although little is known about the mechanisms of the illusions seen in patterns of stripes or the discomfort with which the illusions are associated, the patterns that induce these effects are very similar indeed to those that provoke epileptiform EEG abnormalities in photosensitive patients. As shown in Figure 22.3, the *length* of the contour, the overall *subtense* of the pattern, its *spatial frequency* (expressed in spatial cycles per degree visual angle and related to the size of the image of the stripes on the retina), *duty cycle* (the ratio of thickness to separation of the stripes), and *contrast* all have similar effects on illusions and epileptiform activity. This is also true of the effects of patterns such as those shown in Figure 22.1k,l,m,n): Both illusions in normal observers and epileptiform activity in patients with photosensitive epilepsy are dependent on the area of cortex stimulated and independent of the region of the retina being stimulated. A few observers with unilateral head pain report laterally asymmetrical illusions, which suggests that, as with epilepsy, there may be cerebral asymmetries in sensitivity.

Illusions of color, shape, and motion are also seen in response to diffuse intermittent light. Figure 22.4 shows the proportion of normal observers reporting illusions of shape as a function of frequency, and, for comparison, the proportion of patients with photosensitive epilepsy exhibiting a photoconvulsive EEG response (from Jeavons and Harding, 1975). The general shape of the functions is broadly similar. Presumably the response to pattern and flicker reflects the extent to which the visual system is activated by the stimulus.

As has already been mentioned, headache sufferers differ from controls in that they show a greater susceptibility to illusions in patterns

of stripes and a greater amplitude of the evoked in potential in response to intermittent light at high temporal frequencies. A similar association between sensitivity to pattern and high-frequency intermittent light occurs in photosensitive epilepsy. The upper frequency limit at which a photoconvulsive reaction appears in response to intermittent light is higher in those patients who are pattern sensitive than in those who are not (Wilkins et al., 1980). It would therefore appear that both epileptic and nonepileptic pattern sensitivity is associated with a relatively greater response to intermittent light at high temporal frequencies.

The simplest way of interpreting the cohesion in the above data is to argue that certain visual stimuli are more effective than others at activating the visual system in some nonspecific way and that once activated, or indeed overactivated, many completely distinct neurological sequelae are possible. The limitation of such an interpretation is that it fails to account for the similarity in the neurophysiological and clinical responses the visual stimulation evokes. Goldensohn (1976) pointed out the similarities in the EEG response to intermittent light in epilepsy and migraine and proposed a link between headaches and cortical inhibitory processes. Kasteleijn-Nolst Trenité et al. (1982) reported that about 40% of patients with photosensitive epilepsy report headaches and eye strain in response to epileptogenic visual stimulation in the environment. Similar complaints are also commonly made by their relatives.

Mechanisms Are Less Widespread and Do Not Involve Synchronization

Nonepileptic sensitivity to light manifest as eye strain or headaches has similar mechanisms, although the mechanisms are less widespread and do not involve synchronization. As has already been described, epileptiform abnormalities are more likely when stimulation is confined to one lateral visual field than when similar stimulation occurs in upper or lower fields. This is not the case with respect to illusions: In most observers illusions are equally likely in upper, lower, and lateral visual fields,

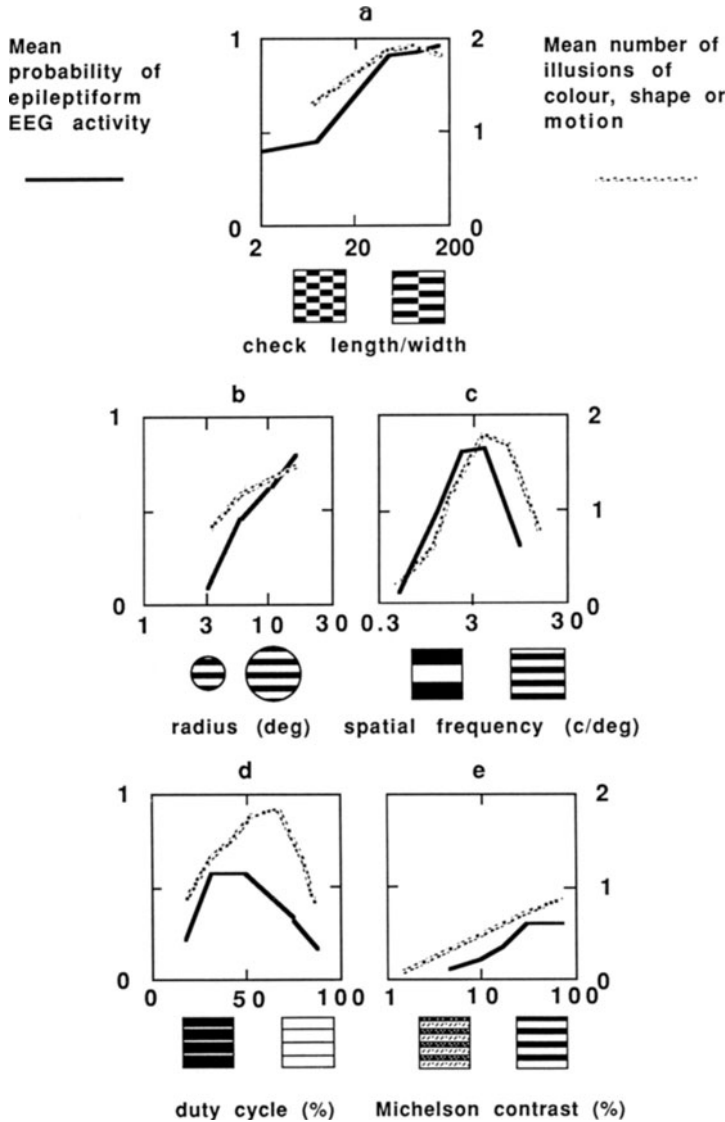


Figure 22.3. Patterns of stripes elicit epileptiform EEG activity in patients with photosensitive epilepsy, and illusions and discomfort in normal observers. The probability of epileptiform EEG activity and of illusions are expressed as functions of the spatial parameters of the pattern (after Wilkins et al., 1984). The curves were obtained by manipulating each parameter independently. The values of the other parameters were chosen arbitrarily, but as data were acquired it transpired that the chosen values were close to those for which illusions and epileptiform EEG activity were maximally likely.

The graphs show the effects of (a) length of contour, expressed as a ratio of the length to width of checks; (b) pattern size, measured in terms of the angle subtended at the eye by the radius of the pattern; (c) spatial frequency—the number of cycles of the pattern in one degree subtended at the eye; (d) ratio of bar width to separation; (e) Michelson contrast of the stripes—the difference between their luminances divided by the sum of their luminances. The icons beneath the x-axes show the effects of the x-variable on the appearance of the pattern.

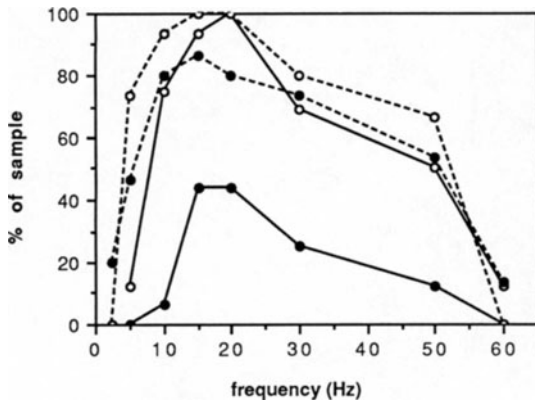


Figure 22.4. Illusions and epileptiform activity as a function of flash frequency. The solid curves show the percent of a sample of 16 patients with photosensitive epilepsy who exhibited a photoconvulsive response to the intermittent light from a xenon discharge lamp (Jeavons and Harding, 1975, p. 74). The broken curves show the percent of a sample of 15 normal observers who reported illusions of color or shape when the entire visual field was contourless and varying in luminance sinusoidally between a minimum of less than 0.01 cd/m^2 and a maximum of 25 cd/m^2 (C. Neary, unpublished data). The open points are for binocular presentation and the filled points for presentation to one eye, with light being occluded from the other.

perhaps because the mechanisms that are responsible do not involve spread within a cerebral hemisphere.

It will be recalled that the epileptogenic properties of a pattern are strongly influenced by pattern movement: Drifting patterns are most unlikely to evoke epileptiform abnormalities, whereas vibrating and phase-reversing patterns are. No such effects of pattern movement occur with respect to illusions of color (the other illusions have not been measured because of the difficulty in assessing illusions of movement when the pattern itself is in motion). Illusions of color are seen in stationary patterns but are more pronounced when the pattern is in motion. They depend on the contour velocity irrespective of whether movement is continuous or repeatedly changing direction.

The illusions of color have been attributed to retinal mechanisms but are usually associated

with illusions of form and motion for which retinal mechanisms are not easy to find. The fact that, in general, the illusions of color, shape, and motion are affected similarly by a wide variety of stimulus characteristics suggest that they may have a similar origin, at least in part. If, as has been suggested, some of the illusions have cortical mechanisms similar to those responsible for seizures, the links with headache and eye strain may prove to be instructive.

Summary

It would appear that the epileptic and non-epileptic responses to noxious visual stimulation are due to a massive excitation of the visual system. The epileptic response involves mechanisms dependent upon synchronization and the excitation spreads through the hemisphere. The nonepileptic effects do not depend on synchronization and are provoked by similar but more localized mechanisms that lead to a less specific neurological response.

Practical Consequences of Visual Sensitivity

The first cases of television epilepsy were reported almost as soon as television became widespread. It is now recognized that television is responsible for a substantial proportion of photosensitive seizures, at least in Europe, where the television picture is refreshed 50 times per second. Television can induce seizures at normal viewing distances in the minority of patients who are sensitive to intermittent light at frequencies as high as 50 Hz. In the majority of patients, seizures are likely only when the viewer is close to the screen where the 25 per second flicker from line-interlace can be seen. A variety of techniques for preventing television epilepsy have been described, including cosmetic and selective monocular occlusion using "television glasses" (Wilkins and Lindsay, 1984): A sheet of polarizer is placed over the screen and the patient wears polarized glasses, one lens of

which has an axis of polarization orthogonal to that of the sheet.

Fluorescent lighting has, like television, been associated with complaints of visual discomfort ever since it was introduced. The lighting pulsates in brightness, mainly at a frequency twice that of the electricity supply. Even though the pulsation cannot be seen as flicker, cells in the subcortical visual system respond to the fluctuation (Eysel and Burandt, 1984). A recent double-blind study (Wilkins et al., 1989) compared conventional pulsating lighting with a new steady form, outwardly indistinguishable. The mean incidence of headaches and eye strain under the two forms of lighting is shown in Figure 22.5. Evidently prolonged exposure to the high-frequency pulsations from fluorescent lighting can increase the incidence of these complaints. It is not known whether the incidence of seizures is similarly effected. Photosensitive patients do not complain of fluorescent lighting as much as do those with migraine (Debney, 1984). There may be a genuine difference in sensitivity, which perhaps reflects a difference in mechanism. The phase-locked response to the pulsating light reported by Eysel and Burandt was observed in the optic tract and lateral geniculate nucleus of the cat. It is unlikely that cortical cells show a similar phase-locked response because their temporal resolution is low (see,

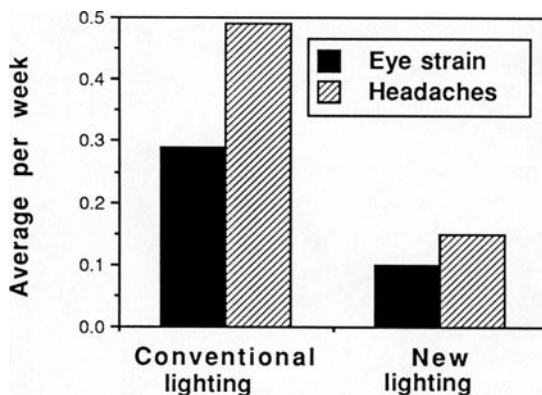


Figure 22.5. Incidence of headaches and eye strain under conventional fluorescent lighting and under a new form of lighting, outwardly identical but incorporating a solid-state circuit reducing 100-per-second light pulsation.

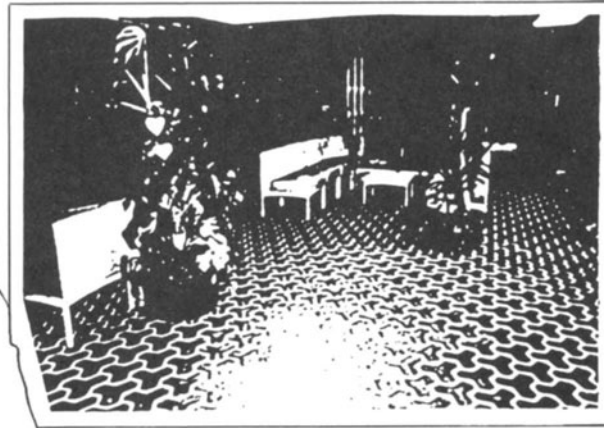
for example, Movshon et al., 1978). Fluorescent lighting may therefore produce relatively little synchronization at a cortical level, and for this reason provoke eye strain but not seizures. In the short term, photosensitive patients do not show epileptiform EEG abnormalities when exposed to normally functioning fluorescent lighting, even when the lamps are made to increase their flicker (Binnie et al., 1979), but it is not known whether the incidence of EEG abnormalities is increased with long-term exposure.

An increase in the incidence of EEG abnormalities as a result of prolonged exposure to mildly epileptogenic stimulation can sometimes be demonstrated even when short-term recordings would suggest that the patient is not sensitive. For example, when pattern-sensitive patients are asked to read there is often no obvious change in the EEG, even though the text they are reading resembles a pattern of stripes with characteristics appropriate for the induction of illusions and seizures (Wilkins and Nimmo-Smith, 1987). At the time the recording is taken, the occurrence of epileptiform abnormalities appears spontaneous but by calculating the long-term average, the incidence can be shown to increase during reading. Covering the lines of text above and below those being read reduces the incidence (Wilkins and Lindsay, 1984). Such a mask also reduces headaches and eye strain in those who are susceptible to these complaints when they read (Wilkins and Nimmo-Smith, 1984).

The increasing use of visual display terminals has been associated with complaints of visual discomfort. On the basis of the data outlined above, the discomfort may be partially attributable to the intermittent illumination of the screen (Laübli et al., 1981; Harwood and Foley, 1987) and partly to the stripes of text that are displayed. The intermittent illumination of the screen also has effects that may be quite independent of those pertaining to epilepsy: It affects ocular motor control (Wilkins, 1986) and accommodation (Neary, 1989) even when the frequency is above that at which flicker is perceived.

The modern urban environment contains a

A



B



Figure 22.6. Examples of patterns in the environment. (a) a carpet in the Stonebow Psychiatric Centre of Hereford County Hospital that gave rise to complaints of headaches and eye strain. Note that the design is such as to produce stripes when viewed from a distance; (b) metal stair treads on an escalator, one of the many commonly occurring striped patterns that, when viewed from a typical distance, have characteristics close to those for which illusions and seizures are maximally likely; (c) a tessellation that does not produce stripes at any viewing angle or spatial scale.

C



large number of striped patterns with characteristics appropriate for the induction of illusions and seizures. As often as not, the spatial periodicity results from the way in which the objects are constructed from smaller components. The tessellations such construction involves often result in periodic patterns that have characteristics within the epileptogenic range (see Fig. 22.6a,b), but it is quite possible to construct surfaces with small components in such a way that spatial periodicity is avoided (see Fig. 22.6c).

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