11. Common forms of reflex epilepsy: physiological mechanisms and techniques for treatment

A. Wilkins  J. Lindsay

This chapter is dedicated to the memory of Patricia Ann Lane

Seizures can be induced by a variety of visual, auditory and tactile stimuli; they can even be induced by thought itself (Ames, 1982; Fenwick, 1982; Laidlaw & Richens, 1982). This chapter will not attempt a comprehensive review of the wide variety of reflex epilepsies but will concentrate instead on a few of the more common forms. It will describe recent techniques for treating them rather than therapy with anticonvulsant drugs. Discussion will centre on those epilepsies that are visually induced, but will not be confined to sensory mechanisms.

PHOTOSENSITIVE EPILEPSY

Photosensitive epilepsy is a relatively common form of reflex epilepsy in which seizures are precipitated by retinal stimulation. Over half the patients with reflex epilepsy described by Forster (1977) were photosensitive. About 3% of all patients with epilepsy are liable to visually-induced seizures (Jeavons & Harding, 1975). The seizures are often the result of the flicker from sunlight interrupted by road-side trees or reflected from the surface of a lake. The television is another common source of flickering light, and many patients experience their first seizure when, for one reason or another, they are close to the screen. These sources of epileptogenic visual stimulation are widely recognised. It is less well known that certain patterns of stripes, such as those on the metal stair tread of escalators, can also be highly epileptogenic. Sometimes patients are so sensitive to patterns of this kind that the stripes formed by successive lines of print prevent the patient reading books without the risk of seizures.

Because of the considerable advance in our understanding of the mammalian visual system it has been possible to use the properties of epileptogenic patterns to infer something about where and how the seizures are induced. The knowledge gained in this way has provided clues as to how to reduce the epileptogenic properties of the stimuli concerned.

Characteristics of epileptogenic visual stimuli: intermittent light

There are many parameters of a diffuse flickering field that are likely to affect its epileptogenic properties. Only a few have received thorough study.

Temporal frequency. As regards stimuli consisting of trains of brief flashes, there is general agreement that the frequency at which epileptiform EEG abnormalities are likely to be induced lies between 15 and 20 Hz in most cases (Jeavons & Harding, 1975). There are, however, individual patients for whom this is not the maximally
epileptogenic frequency (Wilkins et al, 1980), and many patients are sensitive to flash frequencies above and below this range (Fig. 11.1).

**Colour of intermittent source.** There are several studies that have investigated the effects of the colour of the intermittent source but those that have attempted to control the intensity of the source report little difference in the effects of different wavelengths, with the possible exception that blue light may be less epileptogenic (Jeavons & Harding, 1975) and red more so (Takahashi & Tsukahara, 1976; Takahashi et al, 1981).

Binnie and colleagues (personal communication) have shown that the differential sensitivity to light of different hues may be due to inhibitory interactions between the cones. Light from two sources was filtered and mixed so that wavelengths appropriate for the stimulation of the red cones alone were modulated whilst those appropriate for stimulation of both the red and the green cones remained constant. Patients were more sensitive to this stimulus than to light that was modulated in all the wavelengths concerned. Inhibitory interactions between the cones may explain why Takahashi & Tsukahara (1976) observed that the addition of steady blue light attenuated the photoconvulsive response to modulated red light, whereas the addition of steady red light did not. It would not appear to provide a ready explanation as to why these
authors report a beneficial therapeutic effect from the wearing of glasses with a blue filter.

**Diffusion of source.** Patterned light may be more provocative than diffuse, particularly if the patterns are grills and gratings (Jeavons & Harding, 1975). This suggests that to maximise the chances of detecting photosensitivity during the routine EEG examination it is essential that a patterned photostimulator be used. Jeavons and Harding’s original findings have twice been replicated (Stefansson et al, 1977; Takahashi, 1983). Engel (1974), however, failed to find any difference between patterned and diffuse stimulation, possibly because of a failure to equate the mean luminance of patterned and diffuse stimulation or because of an unfortunate choice of checkerboard pattern. As will be described later, patterns of stripes are generally more epileptogenic than checks.

**Whether the eyes are open or closed.** Intermittent photic stimulation given through closed eyelids differs in many respects from stimulation with the eyes open. The light is more diffused and has a different spectral composition. It has a lower luminance but stimulates a larger retinal area. The state of alertness of the patient may also be different in the two conditions. Nevertheless with the parameters of stimulation customarily employed, stimulation immediately after the eyes are closed is generally recognised as being the most epileptogenic (Panayiotopoulos, 1974; Jeavons & Harding, 1975; Kastelijn et al, 1982). Stimulation when the eyes are already closed is usually less epileptogenic than with the eyes open, although there are patients for whom the reverse is the case. In one such patient Wilkins et al (1980) reported that if the eyes were covered with a diffusing surface (thereby extending the area of retina stimulated and eliminating any patterned visual input during stimulation) the state of the eyes had no effect. In over 90% of photosensitive patients stimulation of one eye is less epileptogenic than stimulation of both eyes (Jeavons & Harding, 1975).

**Other parameters.** It is not known in what way the probability of epileptiform EEG activity is affected by the duration of the stimulus train, the angular subtense of the light source or the brightness of its surround. It is known that increasing the intensity of the source increases the likelihood of a photoconvulsive response, and that increasing the level of ambient illumination can reduce the likelihood (Jeavons & Harding, 1975; Newmark & Penry, 1979). With the intensity of light sources conventionally used in the EEG examination, however, the conditions of room illumination appear to have relatively little effect, although some patients are reportedly more sensitive with the room lights on and some with them off (Van Egmond et al, 1980).

Increasing the levels of ambient illumination has three principal effects: it changes the state of adaptation of the visual system; it reduces the depth of modulation of the source and it increases its time-averaged luminance (although with the high intensity sources conventionally used the increase is relatively slight). The authors had the opportunity in one patient to study the effects of the modulation depth of light that was sinusoidally modulated with a constant time-averaged luminance (i.e. the visual system remained adapted to a constant mean level of illumination throughout). It was found that the depth of modulation did not have to be very large in order for
Fig. 11.2 The probability of paroxysmal EEG activity as a function of modulation depth. The probability was estimated from the proportion of stimulus presentations on which paroxysmal activity occurred. Each point is based on a minimum of 11-10-second presentations of a diffuse field subtending 60 degrees, sinusoidally modulated at a frequency of 20 Hz. The time-averaged luminance remained constant throughout testing at about 100 cd/m². (Wilkins, unpublished data).

paroxysmal EEG activity to be induced: modulation in the order of 20% was quite sufficient (Fig. 11.2).

**Characteristics of epileptogenic visual stimuli: patterns**

If a photoconvulsive response is induced by intermittent photic stimulation there is a 30% chance that epileptiform EEG abnormalities will also be induced by observation of stationary patterns of striped lines with maximally epileptogenic parameters. There is no way of predicting pattern sensitivity from the response to intermittent light. Pattern-sensitive patients tend to have a slightly higher upper-frequency limit of sensitivity to diffuse intermittent light (Wilkins et al., 1980). The relationship is too weak to permit the identification of pattern-sensitive patients on the basis of the conventional EEG examination. It is therefore necessary to test for pattern sensitivity and Darby et al. (1980b) describe some suitable techniques.

The patterns that do and do not induce epileptiform abnormalities will now be considered.

**Spatial frequency.** Stripes are probably the most epileptogenic stationary patterns (Chatran et al., 1970; Wilkins et al., 1975, 1979a, 1980; Soso et al., 1980; Takahashi, 1983). The epileptogenic effects of the stripes are dependent on spatial frequency, defined as the number of cycles of the pattern in one degree of visual angle. In Figure 11.3 the pattern illustrated has a spatial frequency of 2 cycles/degree. The spatial frequency at which epileptiform EEG activity is most likely to be induced depends slightly on the subtense of the pattern, but is generally between 1 and 8 cycles/degree (Fig. 11.4) (Wilkins et al., 1980).

**Orientation.** Certain patients are sensitive only to a limited range of grating orientations (Chatran et al., 1970) even when there is no evidence of an astigmatism (Wilkins et al., 1980). The orientation selectivity, when present, shows little consistency from patient to patient.
**Fig. 11.3** A. Diagram to show the eye of an observer fixating the centre of a pattern of stripes subtending a visual angle of $2\theta$ at the eye. B. The luminance profile of the pattern shown in A. The profile is a square-wave with a spatial frequency of 2 cycles per degree visual angle.

**Fig. 11.4** The probability of paroxysmal EEG activity in response to a striped pattern, expressed as a function of spatial frequency. Mean of 8 patients. The pattern had black and white stripes with square-wave luminance profile, contrast 0.7, mean luminance 330 cd/m²; it was circular in outline, centrally fixated, and subtended 24 degrees. (After Wilkins et al, 1979a).

*Duty cycle.* The relative width and spacing of the stripes is also important. The duty cycle of the pattern refers to the proportion of one cycle that one stripe occupies. In Figure 11.5 the probability of epileptiform activity is shown as a function of duty cycle, and it can be seen that the probability is maximal when the width of one stripe is approximately half the width of one cycle (Wilkins, unpublished data).

*Contrast.* The probability of epileptiform activity is shown as a function of pattern contrast in Figure 11.6. In this figure the contrast is measured by the expression

$$C(\%) = 100 \times (L_2 - L_1) / (L_2 + L_1)$$

where $L_2$ and $L_1$ are the luminance of the light and dark stripes respectively. This
expression, sometimes referred to as Michelson contrast, can in theory vary from 0 to 100%. In practice contrasts of 100% are never obtained because of the way in which light is scattered across the borders of a pattern. The sensitivity of the human visual system is such that certain patterns of stripes can be seen when the contrast across their borders is lower than 0.5%. The minimal contrast at which epileptiform activity is induced is generally in excess of 10%, a contrast well above that at which the pattern becomes visible. For contrasts in the range 10–30% epileptiform activity shows a rapid increase with contrast, but above 30% there is little further increase. These

Fig. 11.5 The probability of paroxysmal EEG activity in response to a striped pattern, expressed as a function of duty cycle. Mean of 4 patients. The pattern had black and white stripes of unequal width and spacing, spatial frequency 3 cycles/degree, contrast 0.7, mean luminance about 100 cd/m²; it was circular in outline, centrally fixated, and subtended 20 degrees. (Wilkins, unpublished data)

Fig. 11.6 The probability of paroxysmal EEG activity in response to a striped pattern, expressed as a function of the brightness contrast of the stripes. The pattern had black and white stripes, square-wave luminance profile, spatial frequency 2 cycles/degree, and mean luminance 300 cd/m²²; it was circular in outline, centrally fixated with an angular radius as shown. (After Wilkins et al, 1980)
functions may be compared with the curve shown in Figure 11.2. Both the functions for stationary patterns and for intermittent light are similar. They show that the probability of paroxysmal activity increases with spatial or temporal modulation maximally when the modulation is low.

*Luminance.* As might be expected the probability of paroxysmal activity is greatest when the mean luminance of the pattern is high. Figure 11.7 shows how the probability changes as a function of luminance, and it will be noted that in order to reduce the probability appreciably a reduction in luminance by a factor of 10 is usually

![Graphs showing the probability of paroxysmal activity vs. mean luminance of pattern](image)

*Fig. 11.7* The probability of paroxysmal EEG activity in response to a striped pattern, expressed as a function of the mean luminance of the pattern. The pattern had black and white stripes, square-wave luminance profile, spatial frequency 2 cycles/degree and contrast 0.7; it was circular in outline, centrally fixated, with an angular radius as shown. It was viewed with the natural pupil after adaptation had occurred, binocularly unless otherwise indicated. (After Wilkins et al, 1980)
required. This may have a bearing on treatment. Conventional sunglasses attenuate
the light by a factor of only two or three, and are therefore unlikely to be of much
help. Very dark glasses would be needed (about 80% absorption) and in order to allow
sufficient vision indoors as well as out of doors the glasses would need to be
photochromic.

Vibration. As mentioned above, about 30% of photosensitive patients are sensitive to
continuously-illuminated stationary patterns of stripes. If the stripes are vibrated in a
direction orthogonal to that of the stripes, the epileptogenic properties of the pattern
are greatly increased. About 70% of photosensitive patients are sensitive to patterns of
this kind: some patients being sensitive to vibrating patterns and not to static
(Stefansson et al, 1977).

In Figure 11.8 the probability of epileptiform activity is shown as a joint function of
temporal frequency of oscillation and the spatial frequency of the pattern. The
shading in the boxes is proportional to the mean probability of epileptiform activity
for a group of patients. All the patients who took part were sensitive to vibrating but
not to stationary patterns. It can be seen that, regardless of spatial frequency, the most
epileptogenic temporal frequency is about 20 Hz, a frequency close to that at which
diffuse intermittent light is maximally epileptogenic. The most epileptogenic spatial
frequency is about 2–4 cycles/degree, regardless of temporal frequency. The top
matrix describes the data for pattern oscillations of one half spatial cycle (one stripe
width) and the lower matrix data for oscillations twice this size. Over this range, the
amplitude of oscillation can be seen to have little effect. (Data from Binnie et al,
1979).

Physiological mechanisms of pattern sensitivity
There are many other parameters of pattern sensitivity. They have been reviewed
elsewhere (Wilkins et al, 1980; Meldrum & Wilkins, 1984) and there is no space to
describe them here. Suffice it to say that all the stimulus parameters strongly suggest
that the epileptiform activity is initiated within the visual cortex. This is because (1)
the probability of epileptiform activity is dependent on the linearity of contours rather
than their number, and cells in the visual cortex have linear receptive fields; (2) the
probability of epileptiform activity is greater when the patterns are binocularly-fused
than when different monocular images induce rivalry; cells in the visual cortex are
binocular and many show summation, firing maximally only when both eyes are
stimulated (Hubel & Wiesel, 1979).

Another source of evidence for a cortical mechanism is provided by the topography
of the EEG response to patterns of a size just below the threshold for inducing a
generalised discharge. If the pattern is presented in one lateral hemifield the response
is maximal over the contralateral hemisphere. If the pattern is presented in the two
upper quadrants the response can show a slightly lower distribution on the scalp than
if the lower quadrants are stimulated. In other words the EEG spikes are often
maximal in voltage over the region of visual cortex being stimulated, suggesting that
the discharges can be confined to and sustained within the visual cortex (Wilkins et al,
1981). (The response to diffuse intermittent light may have a less focal and more
variable locus of onset. Cells in the visual cortex of the cat do not respond strongly
Fig. 11.8 The probability of paroxysmal EEG activity in response to a pattern of vibrating stripes. The pattern had a square-wave luminance profile, a spatial frequency of 2 cycles/degree, a contrast of about 0.7 and a mean luminance of about 300 cd/m². It was 15 degrees square in outline and was centrally fixated. The pattern movement was in a direction orthogonal to the stripes. In A the stripes were vibrated through one half-cycle of the pattern, and in B through one cycle. The movement had an approximately constant velocity in each direction. The shading in the boxes is proportional to the probability of paroxysmal EEG activity. (After Binnie et al., 1979)

to diffuse flashes and the maximum temporal resolution is less than about 20 Hz; J. G. Robson, personal communication.)

The probability of epileptiform activity is critically dependent on the size of a pattern. There is a threshold pattern size which differs from patient to patient, below which no epileptiform activity is induced, and above which the probability of paroxysmal activity increases with the angle at the eye subtended by the pattern. If the
periphery of the retina is stimulated epileptiform activity does not appear until very large patterns are presented, but if the cortical magnification is taken into account the effects of central and peripheral stimulation are equivalent (Wilkins et al, 1980). There would thus appear to be a critical threshold of normal excitation of the visual cortex below which no epileptiform activity is induced and above which the probability of epileptiform activity increases with the extent of excitation.

The threshold is not necessarily the same for the two cerebral hemispheres. Often the threshold pattern size at which paroxysmal activity first appears differs for the two lateral visual fields. This asymmetry in threshold is mirrored in the response to diffuse intermittent light which can itself be asymmetric: maximal over the hemisphere contralateral to the visual field that exhibits the lowest threshold for pattern sensitivity (Wilkins et al, 1981; Binnie et al, 1981). This evidence of a difference in the hyperexcitability of the two cerebral hemispheres occurs in patients with primary generalised epilepsy as much as those with epilepsy of other types (Binnie et al, 1981).

Meldrum & Wilkins (1984) have argued that the epileptic discharge is precipitated independently in the two cerebral hemispheres when normal physiological excitation in the visual cortex of either hemisphere exceeds a critical level, regardless of the region of the visual cortex stimulated. This threshold is assumed to remain relatively constant, although it may vary slightly with circadian and other fluctuations (Kellaway & Frost, 1983), and may be elevated following a seizure. Meldrum & Wilkins (1984) believe that the distribution of cortical hyperexcitability is not uniform, even in patients with primary generalised epilepsy. It can evidently differ from one hemisphere to the other and may exist as a patchwork within the hemispheres.

If the cortex is excitable in a primary projection area, as would appear to be the case with pattern-sensitive patients, then adequate sensory stimulation in the appropriate sensory modality may evoke the critical mass of excitation necessary for epileptogenesis. If a neighbouring ‘association area’ is hyperexcitable, seizures may only occur when the patient thinks in a particular way. Wilkins et al (1982) described a patient in whom a range of tasks would precipitate epileptiform EEG activity. The tasks were those generally associated with impaired function following parietal lesions. The authors argued that, in the patient they described, a region of parietal cortex was hyperexcitable, and that paroxysmal EEG activity was triggered when physiological excitation within this region exceeded a critical mass.

**Television epilepsy**

The parameters of visual sensitivity described above would indicate that the patterns that are most epileptogenic are those that (1) are composed of stripes; (2) subtend a large area of the visual field; (3) have a spatial frequency between 1 and 8 cycles/degree; (4) have a contrast in excess of about 30%; (5) have a high luminance; (6) are vibrating with a temporal frequency between 5 and 30 Hz in a direction orthogonal to that of the stripes and (7) are viewed with both eyes. Unfortunately stimulation of this kind is present when television is viewed at close quarters.

*The nature of a television picture*

A television picture is created by variations in the brightness of a spot that scans the screen repeatedly from left to right, starting at the top and zig-zagging its way to the
bottom. It draws the odd-numbered lines and then flies back to the top and draws the even-numbered lines, thus continuously alternating odd and even. Each scan from the top to the bottom of the screen takes 1/50th sec, or 1/60th sec in some countries (such as the United States) which use System M. As a result of the zig-zag (or raster) scan the picture is composed of a pattern of stripes that alternates light with dark and dark with light 25 times a second (30 times per second in the case of System M). The pattern so generated is similar to a vibrating pattern. As can be seen from Figure 11.8, the frequency of vibration optimal for inducing epileptiform activity is close to 20 Hz. It is therefore not surprising that a television picture will induce seizures.

The pattern due to the interlacing lines can only be resolved when the viewer is close to the screen. At normal viewing distances the lines from which the picture is composed can no longer be resolved. The 25 Hz component is no longer visible because the odd and even lines cannot be resolved. Only the flicker generated as the spot flies from the top to the bottom of the screen can be seen, and since the spot flies from the top to the bottom in 1/50th second, this flicker has a frequency of 50 Hz (60 Hz in the case of System M). As can be seen from Figure 11.1, relatively few patients are sensitive to diffuse flicker at 50 Hz and very few at 60 Hz.

There may be a lower incidence of television epilepsy on the North American continent as a result of the different television system. As noted above, the diffuse flicker has a frequency of 60 Hz and the patterned line interlace a frequency of 30 Hz. Both components are less epileptogenic than their European counterparts. It might therefore be expected that comparatively fewer American patients will suffer seizures when the set is functioning normally. Those seizures that are induced will tend to be associated with the slow flicker that results from malfunction.

**Identification of patients at risk**

Jeavons & Harding (1975) have shown that the frequency limits at which a patient is sensitive to intermittent light remain relatively stable from one EEG examination to the next, unless anticonvulsant therapy is changed. It may therefore be possible at the time of examination to identify those patients who are liable to seizures at normal viewing distances. They will tend to be the minority of patients who are sensitive to flicker at 50 Hz. The majority of patients who are sensitive to flicker only at lower frequencies should not be at risk unless they approach the screen. If, however, they get close enough to the screen to see the line interlace they will be exposed to the effects of the 25 Hz pattern oscillation and will then be liable to seizure.

The above predictions concern the relationship between the minimal safe viewing distance and the frequency limits of the photoconvulsive response to diffuse intermittent light. The predictions were confirmed in a study in which photosensitive patients viewed television at progressively closer viewing distances until epileptiform EEG activity was evoked (Wilkins et al, 1979b). The threshold viewing distance was related to the upper frequency limit of the sensitivity to conventional diffuse intermittent photic stimulation, as shown in Table 11.1. (There was no relationship to the frequency limits of sensitivity to patterned intermittent light.) The angle subtended at the eye by the screen and its component lines changes with viewing distance. To investigate the relative contribution of the angular size of the screen and that of the component lines, three televisions were compared, one with a small (0.27 m; 10 inch) screen, one with a screen roughly twice the size (0.57 m), and one
with a similarly large screen masked with an aperture the size of the small screen. Eight patients were tested, as shown in Figure 11.9. In five patients (DW, HD, AS, GF and PS), the threshold viewing distance at which the epileptiform activity was induced was determined by the visual angle subtended by the lines. As can be seen from Figure 11.9, the threshold viewing distance at which epileptiform abnormalities were induced was the same for the masked screen and for the large screen, both of which had similarly-sized lines. The threshold distance for the small screen should have been half that for the other screens, and indeed for some patients (CT, GF and PS) this was the case. Other patients (HD and AS) were not sensitive to the small screen even at the closest viewing distance, presumably because at this distance the eyes were unable to accommodate and produce a clearly-focused image of the interlacing lines. For two patients (CT and SH) who were sensitive at conventional viewing distances the visual angle subtended by the screen was the determining factor. As can be seen from Figure 11.9 the threshold viewing distance for these patients was the same for the small screen as for the masked screen, and half that for the large screen. Presumably these patients were responding to the diffuse 50 Hz flicker. The patients taking part in this study were relatively few in number, but the findings have since been replicated by other workers (Binnie et al, 1980a; Soulrayrol et al, 1981).

The advent of computer video games has had undesirable consequences. Many computers provide a conventional video display with line interlace even though it would be simple and inexpensive to avoid interlacing the lines. Many computers are sold with UHF leads that are so short as to require the operator to sit within a metre of the television screen. Many of the games make use of visual effects that involve the flashing of large areas of the display. All these aspects of the games are unfortunate and unnecessary and have doubtless contributed to the photoconvulsive seizures reported recently in papers with fanciful titles such as ‘ZX81-epilepsy, a case for lumping’ (Sandercock & Warlow, 1982).

**Preventing television epilepsy**

An obvious conclusion from the studies mentioned above is that televisions with a small screen are far safer than those with screens of a more conventional size. If a patient is sensitive to intermittent light only at frequencies lower than about 50 Hz he may avoid the risk of a seizure altogether if he watches televisions with screens having a diagonal measurement less than about 0.25 m. Even if he gets close to screens of this size, the line interlace may not be resolved sufficiently to place him at risk.

Unfortunately it is not always possible for a family to change their set to one with a
The probability of paroxysmal EEG activity in response to television viewing, expressed as a function of viewing distance (column 1), the vertical subtense of the screen (column 2), and the subtense of the component lines (column 3). Data for each of eight patients are shown on separate rows. In order of the patients, sensitivity. Dotted lines show data for a television with a small screen; continuous lines data for a large screen. (After Wilkins et al., 1979b.)

<table>
<thead>
<tr>
<th>Viewing distance (m)</th>
<th>Vertical subtense of lines (degrees)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2 4 8 16 32</td>
<td>5 10 20 30 40 50 60</td>
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</tbody>
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Proportion of presentations inducing paroxysmal activity.
252 RECENT ADVANCES IN EPILEPSY

Monochrome or colour sets do not differ significantly in the likelihood with which they induce epileptiform EEG abnormalities. Nevertheless, in practice, a change to a monochrome set is sometimes found to be helpful (Jeavons, 1982a). This may be because the contrast in the picture is greater than that of the colour set and because the electronic image is more unstable. It may also be because the electronic image is less bright than is necessary. Only the television, grey or coloured parts of the picture result when the television is excited by the excitation of the phosphor. The black parts of the picture result when the television is switched off. They appear black both when the set is switched on and when the set is switched off.

In order to watch the television without having the picture unnecessarily bright it is essential to minimise the ambient light reflected from the outer surface of the phosphor. One possibility is to view the television in a darkened room. Until recently this was thought to increase the risk of a seizure. Patients were advised to view the television in a well-lit room with a lamp in front of the television. This study failed to separate the responses to the onset of the train and the flash from the end of the train. The effects of the flash from the end of the train may be more likely to cause a seizure when the brightness of the television is increased. More recent studies have shown that with increased brightness, the risk of a seizure is reduced.

This advice was based on studies demonstrating that background lighting can reduce the sensitivity of the phosphor to intermittent light. The phosphor's exposure to intermittent light can be reduced by increasing the brightness of the television. This can be achieved by using a darkened room and by using a lamp in front of the television. The effect of increasing the brightness of the television is to reduce the sensitivity of the phosphor to intermittent light. The brightness of the television is increased by using a lamp in front of the television. This can be achieved by using a lamp in front of the television. The effect of increasing the brightness of the television is to reduce the sensitivity of the phosphor to intermittent light.

Another way of avoiding a bright television picture is to place a sheet of darkened perspex in front of the screen. This has the effect of increasing the apparent contrast of the screen (once to reach the outer surface of the phosphor, and again after reflection from the
phosphor). Light from the phosphor has to pass through the plastic only once. As a result the contrast of the picture is enhanced and the overall luminance reduced. The increase in contrast is unlikely to affect the epileptogenic properties of the television display. The contrast or modulation depth of the display is in excess of 30% and, as will be observed from Figures 11.2 & 11.6, the probability does not change with contrast in this range.

The main disadvantage of such a darkened ‘outer screen’ is that it can act as a mirror, reflecting images of the surroundings from its outer surface. For these reasons a fine wire mesh is sometimes used. It has the same effect of reducing the ambient light reaching the outer surface of the phosphor but is not prone to superficial reflections. It is sold mainly for installation on the screens of computer terminals.

Although we have received reports that such dark ‘outer screens’ are helpful, and in laboratory studies we have observed a slight reduction in the probability of epileptiform EEG activity when they are used, the screens are likely to be no more than an aid in reducing seizure susceptibility. There are indications that in most patients the intensity of intermittent light has to be reduced considerably before it ceases to be epileptogenic (see Fig. 11.7). There are no data one way or the other, but it would seem unlikely that the small reduction in luminance that such screens afford will be likely to effect a clinically-significant reduction in seizure susceptibility in many patients. The low cost of the screens justifies their use on a trial basis, however.

**Television glasses**

By minimising the size of the television screen, maximising the viewing distance, and reducing the luminance of the picture it may be possible to control seizures in the majority of patients, but there will remain those who are sensitive to the television at normal viewing distances. As described above, these patients may be identified with the help of the EEG: they will tend to be the patients who are sensitive to high frequency diffuse intermittent photic stimulation. For some of these patients anticonvulsant therapy may be inappropriate, in others it may be insufficient to control the seizures. In these cases it may be worth considering the following technique which takes advantage of the fact that over 90% of photosensitive patients are far less sensitive to monocular stimulation than they are to binocular (Jeavons & Harding, 1975). The technique simply prevents the light from the television reaching one of the patient’s eyes. It has not hitherto been reported in detail (cf. Wilkins et al, 1977) and so it will now be described at some length.

A sheet of polariser is placed over the television screen. This looks like darkened perspex and has the same effect as regards enhancing the contrast of the television picture. The patient wears a pair of spectacles which have the appearance of polaroid sunglasses. Unlike conventional sunglasses, one lens has an axis of polarisation orthogonal to that of the other. The light emitted from the television screen is polarised on passing through the sheet and will not then pass through one of the spectacle lenses, the lens with an axis of polarisation orthogonal to that of the sheet over the screen. As a result the patient can see everything normally with both eyes — apart from the television screen. The television screen appears black to one eye and normal to the other. In this way the glasses provide a selective and cosmetic form of monocular occlusion.

In Figure 11.10 the glasses are shown as having axes of polarisation oriented at 90
degrees to one another and at 45 degrees to the vertical. The polarised sheet also has an axis of polarisation 45 degrees to the vertical. As a result the eye occluded will depend on which side of the sheet is facing the observer. By turning the sheet round it is possible to change the eye occluded. This may be of use in avoiding discomfort. Unfortunately, because of the way the polariser is manufactured, it is more wasteful to cut the sheet with an oblique axis of polarisation, and hence the cost is greater.

With the set-up shown in Figure 11.10 the extent to which one eye is occluded depends on the angle of the patient’s head with respect to the sheet of polariser. If the slightly more expensive circular polarising material is used for both the sheet and the lenses, the occlusion is unaffected by the orientation of the patient’s head. Screens made of circularly polarising material are now manufactured for use on computer terminals.

The following case histories will demonstrate that polarised occlusion can be remarkably effective for patients with a compulsive attraction to the television in whom antiepileptic drugs are inappropriate or unsuccessful and for whom more conventional types of monocular occlusion are of no avail.

**Case histories**

AB was an 11-year-old girl, the youngest of three children. She was born prematurely after an uneventful pregnancy, and she weighed 5 lb 1 oz. Her developmental milestones were normal but she suffered frequent ear and throat infections with repeated antibiotic courses. (She underwent adenoidectomy, tonsillectomy and myringotomy at age 9 with little beneficial effect.)

When aged 4 AB was in the bathroom with her sister. The sister suddenly called ‘A. cannot see!’ and her parents arrived to find A. waving her arms and apparently blind. She then went limp and was unconscious for about a minute. After she recovered she remained blind for a short while. She was given medication (probably...
phenobarbitone) and she did not suffer a second seizure until 6 months later. Thereafter she suffered about one seizure a month. She was treated with phenytoin which affected her gums so badly than she had to be weaned off. Sodium valproate was substituted in doses up to 200 mg four times daily, reduced to three times daily because the parents did not observe any improvement as a result of the extra dose.

AB was impulsively attracted to the television. The family set had a 0.56 m (22 inch) screen. She felt drawn to the television every time she saw it. When questioned as to why, she was unable to give much information other than that it was the whiteness that attracted her, and that she ‘had to go there’. She would move towards the television and put her nose on it. Then (and only then) she would see a ‘blob’ of coloured scintillating spots, circular in outline, subtending about 4 degrees at a position about 20 degrees to the right of fixation. Her pupils would enlarge and her face would flush; she would experience a horrible feeling in both her stomach and her face, and she would go blind. During the blindness which usually lasted about 10 min she was quite unresponsive to visual stimuli. She would complain of feeling funny, would sit down, and she would often scream. She would then make swallowing movements and shake all over. Her head would flop to one side, usually to the right, and her eyes would roll up and to the right. This stage would last from 2–10 min. Afterwards the blindness would persist for 20–60 min, and be followed by sleep of 1 or 2 hours duration. When first admitted as an in-patient to the Park Hospital for Children, Oxford, these seizures were occurring about once a month.

In addition to the major attacks AB was subject to several ‘trances’ every day. She might be talking and stop in the middle of a sentence with her eyes ‘moving around’. At school these episodes were referred to as daydreams, and the teachers noticed that she would begin one activity and then begin a second with no recollection of the first. Despite its variability her school work remained low average. The WISC yielded a Full Scale I.Q. of 112, Verbal I.Q. 112, Performance I.Q. 109.

AB’s mother suffered one epileptic seizure after the birth of the first child and before she became pregnant with AB. She had approached the television because it needed adjustment and found that she could not see anything but ‘a picture in her eyes’. She then lost consciousness and had a tonic–clonic seizure in which she bit her tongue. The mother also suffered migraine attacks. She had a paternal aunt who was said to suffer fainting and dizzy spells and trouble with her eyes, and the daughter of a maternal aunt had tonic–clonic attacks. AB’s sister suffered migraine and developed epilepsy with photosensitivity.

During AB’s 10-week hospitalisation her valproate dose was increased to 200 mg four times a day without any noticeable improvement. For a few weeks she was given an eye patch but did not like to wear it. A skull X-ray revealed a marked mucosal thickening in both antra but no other abnormality, and the CT scan was normal. The EEG’s revealed bursts of spike and slow wave activity, both regular and irregular, with and without polyspike components. This activity was often preceded by a slow-closure of the eyes as described by Darby et al (1981) (see later). AB reported that her eyes got very tired, particularly in bright light, and that these slow closures produced a pleasurable feeling that ‘relaxed’ her eyes. Intermittent photic stimulation in the frequency range 8–40 Hz induced spike-wave activity. Patterns of stripes (oblique gratings with square-wave luminance profile, spatial frequency 2
cycles/degree, contrast 0.7, mean luminance 300 cd/m2) were presented in one or both visual fields. Patterns with a radius (or, in the case of the unilateral patterns, a hemiradius) of 6 degrees visual angle reliably evoked low voltage isolated slow waves or spike-slow wave complexes, maximal in the posterior temporal and occipital electrodes. These abnormalities were accompanied by ‘tightly feelings’ in the stomach. On one occasion A. was filmed in front of the television whilst the EEG was recorded. She sat extremely close and frequently leaned forward putting her face as close as possible to the screen for prolonged periods, often moving her eyes as if following something. On this occasion the previously normal background rhythms were replaced by irregular activity mainly in the theta range. On almost every occasion that the head was moved close to the screen a 2–10 second burst of high amplitude, fairly regular 3 Hz spike and wave activity occurred. When the television screen (0.11 m) was covered by a polarising filter and the patient wore the ‘television glasses’ polarised so that the television picture was visible only to the right eye the epileptiform abnormalities no longer occurred.

The polarised ‘television glasses’ and screen were introduced 6 weeks after the increase in the dose of valproate. The polarising filter was fitted not only to the family’s television but also to the television at school. The major seizures and the impulsive attraction to the television both abruptly ceased. The incidence of the trances also decreased and her teachers noted a dramatic improvement in her concentration.

One year later she reported that she had not experienced any attraction to the television, had not gone blind or experienced any ‘horrible’ or ‘tightly’ feelings, despite the fact that on a few occasions she had watched television that were not covered with a polarising filter, for which the ‘television glasses’ afforded no monocular occlusion but only a reduction in luminance. She had also occasionally watched the television without her glasses but had not been adversely affected. Her visual acuity appeared to have improved: she was now able to attempt the 6/4 line of the Reduced Snellen chart (her limit 1 year previously was 6/6). Her stereoacuity was now better than the upper limit of the Titmus test (40 seconds of arc) whereas previously errors were made at angles of less than 80 seconds of arc. The lenses of the ‘television glasses’ had been fitted into new frames by an optician and the angle of polarisation of one lens had been slightly altered. As a result, A. had now to tilt her head slightly in order to obtain the monocular occlusion. It is significant that she chose to watch television in this way, rather than from a position in which both eyes would see the picture.

The ‘trances’ had continued, albeit at a decreased frequency, and so photochromic sunglasses were prescribed. Unfortunately AB was reluctant to wear them, and did so only during the summer months, and then only on sunny days.

Ten months later the parents said they had not seen any ‘trances’ for about a year.

CD was a 10-year-old girl, the second of three children. The pregnancy was normal although at birth she weighed only 5 lb. Her developmental milestones were normal until the age of 7 when she started being ‘drawn towards the television’. The family television had a 0.58 m screen. She would go close to the television, her face would go red, her whole body would ‘go still’ and sometimes she would fall to the floor. The attacks lasted no more than a minute or two and there was no postictal confusion. The

frequency of the seizures varied, although her mother had attributed this to general anxiety and diazepam. When her daughter was 11 months old she failed to return from the television viewing district and was withdrawn from the television viewing district. Her visual acuity had decreased from 6/5 to 0.5 D spherical equivalent and she was given +0.5 D sphere glasses which she wore continuously. This did not affect the seizures. The parents did not change their television and the seizures continued. Then she was referred to a neurologist for consultation without the television.

EF was a 5-year-old girl. In the early months of her pregnancy, she had had further maternal complications and was induced. She was born at term, normal birth weight, and was well for the months following. EF had a small right-sided cleft and little padding around the eye. The cleft lasted some months and was not deemed to be of any consequence, yet despite previous reports she was not required to wear glasses and was not of any adverse effect. EF had never been given amphetamine, but was described as being in the interquartile range on the Wechsler and chronological scores were consistent with this.

There were no reports of related siblings.

EF was a 12-year-old girl. She was initially seizure-free. However, she had had a seizure after being admitted to hospital for a viral illness the year before.

From her developmental milestones in the background were low-normal and she was not developmentally delayed. The seizures began shortly after the viral illness and were controlled by her usual medication. She was seizure-free during the day, although the frequency was increased at night. She was admitted to hospital...
frequency of the attacks varied; sometimes there were as many as two or three per week, although sometimes no attacks occurred for over a month. The patient’s mother had suffered from ‘petit mal epilepsy’ for which phenobarbitone, phenytoin and diazepam had been prescribed. She refused offers of anticonvulsant medication for her daughter. An EEG examination at the Dudley Road Hospital in Birmingham failed to reveal any abnormalities other than when intermittent photic stimulation evoked brief generalised poly-spike and slow-wave discharges. The range of sensitivity was 20–60 Hz. The patient was asked to watch a 0.56 m monochrome television whilst the EEG was recorded. A generalised discharge was elicited from a viewing distance of 1.7 m. The patient was short-sighted and wore a prescription of −0.5 D sphere in both eyes. She was given clip-on polarised lenses to wear over her glasses when watching television and a sheet of polariser to place over the television screen. The parents were advised to turn the polarised sheet from time to time so as to change the eye occluded. Both the compulsive attraction to the television and the subsequent attacks abruptly ceased and the patient remained seizure-free for 5 years. Then she was playing shove-halfpenny at a club within a few centimetres of a large television screen. She turned towards the screen and suffered a major attack. Her consultant then prescribed sodium valproate and she has remained seizure free without the glasses over the ensuing 2 years.

EF was a 9-year-old retarded boy, the second of three children. At the age of 31, in the early months of pregnancy, his mother took a combination antiemetic. The latter part of her pregnancy was complicated by high blood pressure and ankle swelling, but no further medication was taken for these complaints. Labour was overdue by 1 week and was induced. The baby weighed 9 lb 10 oz. The early milestones and growth were normal but as early as 1 month of age jerking movements were noted. At the age of 9 months EF suffered his first major seizure when he was sitting in bright sunlight in a little paddling pool. He suddenly fell forward and went into a major convulsion which lasted some 25 min. Thereafter he had an average of two or three seizures per week, despite phenobarbitone, phenytoin and nitrazepam. His frequent myoclonic jerks were treated with steroids but he became moon-faced within 6 weeks and the regime was stopped. His hyperactivity (noted from early childhood) was treated with amphetamine and haloperidol with no significant improvement and with an increase in the incidence of epileptic seizures. From the age of 7 he received carbamazepine and clonazepam and this was thought to be the best combination, although he continued to suffer one major seizure per week and many myoclonic jerks.

There was no family history of epilepsy; the mother’s father was diabetic. The two siblings were normal.

EF would ‘home in on the television like a moth’ and would end up in a major seizure. His mother could not even take him past a television shop without risking a seizure. His parents banned television at home, but whilst in residential care at a hospital school he was exposed to a considerable amount of television and his seizures increased.

From the age of 5, EEG recordings had noted an extreme photosensitivity. The background rhythms were slow and were interrupted by bursts of bilaterally-synchronous spike and wave activity. An EEG examination at age 7 at the Park Hospital for Children revealed an ill-organised irregular pattern in which medium to
high voltage rhythmic theta activity at 5 Hz was best defined in posterior regions, whereas a 1.5–3 Hz delta activity was prominent anteriorly. The latter was rather episodic and included frequent high-amplitude bilaterally synchronous sharp and slow waves. Generalised spiking was seen, sometimes with posterior emphasis when EF looked at striped patterns (square-wave luminance profile, spatial frequency 2–3 cycles/degree, Michelson contrast 0.7), provided the patterns subtended at least 12 degrees visual angle. He was sensitive to vertical but not horizontal stripes. Once EF’s attention had been gained by a pattern he exhibited a glazed expression and made a slow and seemingly deliberate movement towards the pattern. According to the parents this movement was similar to that towards the television. It was accompanied by generalised epileptiform EEG abnormalities. No EEG sensitivity was observed when the patient watched a 0.36 m television: one with a larger screen was not available at the time of testing.

When polarised ‘television glasses’ were issued (so that the right eye was selectively occluded) the compulsive attraction to the television ceased and the seizures were no longer associated with television viewing. Eighteen months later his parents reported that the glasses were being useful at the hospital school because they prevented the compulsive attraction and thereby enabled the other children to watch television without putting EF at risk of a seizure. At home, however, EF had a tendency to remove the glasses and the polarised screen on its own had not been sufficient to prevent seizures. Since EF did not derive any pleasure or benefit from television viewing his parents had found it simpler once again to prevent EF from viewing television at home.

GH was a 12-year-old boy. The pregnancy and birth were uncomplicated apart from a 24-hour labour. There was no family history of epilepsy. Developmental milestones were normal but at the age of 6 months the child suffered a reaction to a whooping cough/diphtheria/tetanus vaccination. After the injection he slept for half an hour then woke up screaming, lost consciousness and went blue. He was ‘given an injection’ and recovered after 10 min. Subsequent development was normal. At the age of 5 he was watching television when he ‘went quiet’, his mouth became blue and he was unresponsive. After about 10 min he recovered and had no memory of the incident. He then slept for 2 hours. Subsequent attacks also occurred during television viewing and two were associated with vomiting. They occurred about once every 2 weeks until phenobarbitone was prescribed (30 mg twice daily). In the ensuing 7 years only two attacks occurred, both of them associated with a failure to take the medication. At the age of 10 the dose was reduced to 30 mg once a day.

An EEG was recorded in the family home using a transportable 8-channel machine whilst GH watched the family’s 0.3 m monochrome television from his customary position 1.6 m from the set. The set was receiving normal broadcast programmes and the mean luminance of white sections of the picture averaged 300 cd/m². The television was viewed for a succession of 100 second periods, 12 with and 11 without the polarising filter and glasses occluding the left (non-dominant) eye. Frequent discharges were observed, consisting mainly of multiple spike and slow-wave activity. Under normal viewing conditions there were an average of 1.64 discharges per 100 seconds. Paroxysmal activity was present for 1.8% of viewing time. When the polarising filter and glasses were used the incidence of discharges was reduced to 0.33
per 100 seconds and paroxysmal activity was present for only 0.2% of viewing time.1

Discharges exceeding about 2.5 seconds in duration were associated with a slowing of motor responses, a reduction of awareness, and a subjective sensation of daydreaming. No such discharges occurred when the polarised occlusion was used.

Over the subsequent 3 months GH found the glasses uncomfortable to wear and eventually ceased to use them.

**Implications**

Sensitivity to television is determined by the size of the screen, the viewing distance and the frequency range of the patient’s sensitivity to diffuse intermittent light. The EEG findings obtained in the laboratory apply in the home environment (see Patient GH).

The impulsive attraction to the television appears to have been ictal in origin in all the patients whose histories we have reported. Had the behaviour been learned as a result of feelings experienced when close to the screen, it should have taken time to extinguish. It should have taken the patients time to learn that the behaviour no longer resulted in pleasurable sensations. Denied of these sensations patients might have been tempted to remove the glasses and then approach the set. Only one of the patients did so and in all patients there was an abrupt cessation of the impulsive behaviour.

The impulsive attraction is not invariably ictal in origin. Andermann (1971) described a patient who would self-induce seizures by making the television picture unstable. She would threaten to give herself a seizure in order to get her own way. We have seen a boy with television epilepsy who would get up early in the morning in order to watch the television, replacing the fuse that his parents had removed. It seems highly unlikely that these complex preparatory behaviours were ictal in origin. They were presumably motivated by feelings that, if not directly pleasurable, were reinforcing, perhaps because they served to relieve stress.

The history of patient AB suggests that the repeated induction of epileptic discharges by television viewing can result in frequent seizures, and that when the convulsive properties of television are reduced by the wearing of glasses for a prolonged period an extensive recovery can ensue.

We have routinely examined the stereoaucity of patients to whom the glasses have been issued and have never observed any adverse change as a result of the monocular occlusion.

One obvious practical problem with the glasses is that both patients and their televisions require ‘treatment’. Arrangements have to be made to ‘treat’ not only the domestic television but also any others with which patients are likely to come into prolonged contact; for example, the televisions at school.

The glasses are not invariably useful. Many patients find them unpleasant to wear and will not tolerate them. The glasses would seem to be most effective for patients who experience unpleasant sensations as a result of the epileptic discharges that are induced when watching television. The pleasantness of the discharges may depend on

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1 The reduction of over 80% in the incidence and total duration of paroxysmal EEG activity was significant at the 1% level by two-tail Fisher exact probability tests.
EPILEPSY: PROVOKED BY READING

The term "reading epilepsy" is reserved for those patients who suffer seizures when they read. If the seizures are exclusively associated with reading, the term "primary reading epilepsy" is sometimes used. Although the seizures often have a stereotyped form, being preceded by myoclonic movements of the jaw, the seizure pattern is quite variable. In this section, it will be argued that many different types of seizure trigger may be involved when seizures are induced by the act of reading and that the patients may suffer seizures when they read. Some distinct types of epilepsy are induced by the act of reading. Reading is a complex skill involving the comprehension of the eyes across a detailed visual scene. All the component skills that may be involved when seizures are induced by reading are considered first.

Visual parameters

The successive lines of printed text form a pattern of stripes that can have parameters similar to those of epileptiform EEG abnormalities in pattern-sensitive patients. Groups of normal volunteers were asked to position a selection of books at a comfortable reading distance and the distance from their eyes to the page was measured. The selection of books included those with text that was judged to be "clear" and those with less clear text. When the separation of the lines of text was more than 4 mm, the patients reported reading difficulties.
text was measured it was possible to calculate the spatial frequency of the grating formed by the text. It turned out that people positioned the book so that the (fundamental) spatial frequency remained roughly constant regardless of the separation of the lines, perhaps because books with widely separated lines tended to have larger print which could be read at a greater distance. The mean spatial frequency for the group as a whole was 1.4 cycles/degree (range 0.6 to 2.9). Figure 11.4 shows the way in which the probability of paroxysmal EEG activity varies as a function of spatial frequency and, as can be seen, the frequency of the lines of text lies on the low frequency arm of the curve.

The duty cycle of the grating formed by text can be estimated by measuring the height of the body of the letters (x-height) and expressing this as a ratio of the distance from one line to the next. (The ascenders and descenders contribute little to the pattern of stripes.) The duty cycle was estimated in this way for books with ‘clear’ and those with ‘less clear’ print, and it was found to vary considerably, ranging from 46% to 73%. Much of this range is epileptogenic, as can be seen from Figure 11.5 which shows the probability of paroxysmal EEG activity as a function of duty cycle.

The contrast of the lines of text was more difficult to measure than either of the two above parameters, being determined not only by the contrast of the ink on the page but also by the width and separation of the strokes from which the letters are composed. Optic fibres were used to channel light from a narrow slot onto the page and the reflected light was collected by other intermixed optic fibres. When the slot was positioned parallel to the lines of text and moved across them the device gave an output that indicated contrasts that ranged from 14% to 22%. The values are likely to be conservative because of mirror-like reflections from the surface of the paper. They are nevertheless sufficiently high as to be in the epileptogenic range for the majority of patients (see Fig. 11.6).

Of course, the lines formed by printed text are not continuous but composed of elements that have many orientations, some of them orthogonal to the axis of the stripes. It has already been shown that the epileptogenic properties of a striped pattern are reduced when mixed with another striped pattern having an orthogonal orientation (Wilkins et al, 1979a). It was obviously essential to determine whether, as suggested by Mayersdorf & Marshall (1970), the striped properties of printed texts can indeed be epileptogenic. To this end, pattern-sensitive patients were asked to read whilst their EEG was monitored. Only two pattern-sensitive patients have so far been tested, but the results appear to confirm the predictions based on the parameters of visual sensitivity. In both patients there was a significant increase in the incidence of paroxysmal abnormalities when reading, compared with the incidence when the patient was resting. This increase could have been due to a number of non-specific factors. It is therefore of interest that when the patients used a mask that darkened the lines of text above and below those being read, allowing three lines to be seen clearly, the incidence of paroxysmal activity was significantly reduced (Table 11.2). The mask used in these studies is now being manufactured as the ‘Cambridge Easy Reader’ by Engineering and Design Plastics in Cherry Hinton, Cambridge (Wilkins & Nimmo-Smith, in press). It consists of two sheets of darkened perspex measuring 170 mm wide and 55 mm high. They are covered with a matt antireflection coating and attached at one end to a magnetic slide so that the separation of the sheets can be adjusted.
Table 11.2 Incidence of paroxysmal EEG activity (discharges/minute) at rest and when reading. Data for two pattern sensitive patients (MN and IJ)

<table>
<thead>
<tr>
<th>Patient MN</th>
<th>Patient IJ</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.70</td>
<td>0.00</td>
</tr>
<tr>
<td>1.48</td>
<td>2.67</td>
</tr>
<tr>
<td>0.73</td>
<td>0.58</td>
</tr>
</tbody>
</table>

The clinical history of one of the patients who contributed the data shown in Table 11.2 will now be described. The history is interesting in that the patient was asymptomatic for seizures at the time of testing but reported difficulties with reading.

Case history
IJ was a 13-year-old right-handed girl, the youngest of three children. Her mother’s pregnancy was complicated by pre-eclamptic toxaemia and she was born 2 weeks post mature. The birth and neonatal period were both unremarkable. She suffered an isolated febrile convulsion in the second year of life and from the age of 7 or 8 years was observed to have brief absences usually lasting no longer than 2 or 3 seconds. (The mother refused offers of anticonvulsant medication for her daughter.) At the time of examination the attacks had not been seen for several months.

IJ was inclined to urticaria which seemed to be provoked by certain foods, particularly soft fruit, and she suffered from hayfever. She also suffered from frontal headaches which were associated with a difficulty in finding words, with nausea, and, once, with vomiting. Her mother suffered from migraine.

A previous ophthalmological examination had noted no errors of refraction although there was some limitation of movement and blurring on moving the eyes to the left. Ophthalmic exercises and glasses with a plano prism in the right eye had been prescribed to control ‘poor binocular coordination’. The glasses were not worn for more than a few months.

A routine EEG examination at Runwell Hospital revealed a responsive symmetrical alpha rhythm and bilateral posterior temporal slow waves. Brief runs of delta waves
often occurred. Intermittent photic stimulation with eyes open elicited a photoconvulsive response at frequencies of 6 and 50 Hz but not at frequencies above and below.

Post-central spike and wave discharges were elicited when the patient looked at patterns of black and white striped lines subtending as little as 6 degrees at the eye. (The patterns were gratings with a square-wave luminance profile, Michelson contrast 0.7, mean luminance 100 cd/m², spatial frequency 2 cycles/degree, circular in outline and centrally fixated.) Larger patterns (subtending 12 degrees) elicited generalised spike-wave discharges. Patterns with a hemiradius of 3 degrees were presented in random succession in the left and right visual fields and elicited post-central paroxysmal activity, maximal in voltage over the contralateral hemisphere. There was a marginal tendency for the discharges to be evoked more readily by stimulation of the right visual field. As might be expected from the previous section, the discharges were also evoked by television.

The patient was asked to read a book with and without a mask that darkened and blurred the lines of text above and below those being read, allowing three lines of text to be visible: (the ‘Cambridge Easy Reader’; Wilkins & Nimmo-Smith in press). The mean incidence of paroxysmal activity at rest and when reading is shown in Table 11.2. The data were obtained at a conventional reading distance of 40 cm when the grating formed by the lines of text had a spatial frequency of 1.9 cycles/degree. IJ had a tendency to read at a distance of only 20 cm from the page, a distance at which the rate of paroxysmal discharges was lower, presumably because at this distance the spatial frequency of the stripes was considerably reduced. The patient spontaneously reported that the reading mask helped her concentration.

When last seen, 7 months later, IJ reported that she found the reading mask helpful, and that she was still using it, mainly for textbooks in which the print was closely spaced.

**Eye movements**

It is known that eye movements, notably those involved in convergence, can induce seizures (Vignaendra, 1978). Alajouanine et al. (1959) described a patient with reading epilepsy who was liable to seizures when she followed a light moving in a pattern similar to that made by the eyes during reading. We have recently observed that the pattern of eye movements from one letter to another is influenced by the lines of text. Normal volunteers were asked to move their eyes repeatedly between two letter ‘e’s embedded in neighbouring words in a line of text. The number of high velocity eye movements (saccades) required to get from one target to the other was measured. When the task was performed on the centre line of three lines of text, 20% fewer saccades were required than when the same three lines appeared in the centre of a page of text. These data would suggest that the reading mask facilitates the movements of the eyes across text. The patient whose case is now to be described had difficulty with eye movements and found the reading mask of benefit for this reason.

**Case history**

KL was a 10-year-old right-handed girl, the younger of two children. She was referred as a result of difficulties with reading. In the first week of life she had been a victim of proteous meningitis. A subcutaneous reservoir was inserted through a right frontal burr hole for intraventricular antibiotic administration. At the age of 6 years (and
possibly also before then) she began to suffer focal seizures, usually when awakening from sleep or just before. A Jacksonian seizure affecting the left hand and arm would sometimes spread to involve the whole of the left side of the body, sometimes with a loss of consciousness. The seizures were never generalised and were not followed by Todd’s paralysis. Initially the attacks occurred about once every 3 months, and were treated with carbamazepine, subsequently discontinued because of a cardiac arrhythmia. Phenobarbitone and phenytoin resulted in allergic reactions. She then received sodium valproate 500 mg daily and suffered no Jacksonian seizures for 4 years. She continued to suffer episodes in which her eyes would spontaneously deviate to the left when she was tired. There was no obvious impairment of consciousness associated with these episodes.

CT brain scans revealed a porencephalic area in the right-frontal region. The only neurological abnormality on detailed examination was a difficulty with eye co-ordination. When her eye movements were examined by an infra-red reflective method the saccades were revealed to be slow and hypometric in both directions. Pursuit movements, particularly to the right were inaccurate and broken up, and were not improved by an eye-hand co-ordinating task. Hearing and visual acuity were normal.

An EEG revealed an alpha rhythm that occurred intermittently, most obviously in the right parietal region. Delta waves of high amplitude were seen in the right frontal quadrant and these were mixed with independent sharp waves and spikes. High voltage delta waves and theta rhythms were also seen in the right posterior temporal area in single episodes or in clusters of waves, spreading occasionally to involve right mid-temporal and parietal electrodes. This activity was seen most often when the eyes were shut. There were no clear responses to pattern stimulation. Intermittent photic stimulation elicited a fundamental following response, and the delta and theta activity from the right posterior temporal electrode increased in amplitude.

KL was asked to read various passages of text both aloud and silently, and on every occasion that she did so the incidence of slow waves over the right temporal region increased by a factor of about ten. She was then asked to read using a mask that partially obscured the lines of text above and below those being read (the Cambridge Easy Reader). When conventional print was read and three lines of text remained unobscured there was no appreciable change in the incidence of the slow-wave abnormalities. When KL read her own book with large print and the device was adjusted to reveal only a single line of text the incidence of slow waves was reduced by one third. Passages of text from the Neale Analysis of Reading Ability with equivalent levels of difficulty were then alternately administered, with and without the reading aid. There was a 9 month improvement in reading accuracy and a 12 month improvement in comprehension when the aid was used. The improvement with the reading mask was obtained with printed text that was too large to comprise a striped pattern with epileptogenic properties.

The mask appeared to have its effect because of the way it guided visual search. When KL was asked to count the ‘i’s in an inverted page of text the incidence of slow waves was similar to that during reading. When she attempted non-visual spatial tasks (describing from memory the route around an outline of the letter ‘F’, recognising unseen objects palpated by the left hand) the incidence of slow waves was similar to that occurring at rest.
Linguistic comprehension

Forster (1977) described eleven patients whose reading epilepsy did not appear to be due to the patterned properties of the text they were reading nor the movement of their eyes across the text. Pattern sensitivity is known to be reduced or eliminated under conditions of monocular viewing and in all the patients epileptiform EEG activity was induced by monocular presentation of the text. In all patients the activity occurred when the text was presented in vertical format, one word on a line. Such a format is likely to result in the use of different oculomotor strategies from those customarily employed. The seizure activity also occurred whether the patients were reading aloud or silently, with or without movements of the lips or tongue. Finally, the activity tended not to occur when the patients read material they had memorised. In these patients, and in another patient reported by Ramani (1983), it would appear to have been the comprehension of the written material that was responsible for evoking the seizures. A cognitive trigger mechanism of this kind has been described in circumstances other than reading (Geschwind & Sherwin, 1967; Tsuki & Kasuga, 1978).

A complex of precipitating factors

If seizures are triggered only by reading they tend to have a stereotypical form. They usually occur only after prolonged reading and, as mentioned in the introduction to this section, they are often preceded by myoclonic movements of the jaw. It is perhaps because of the stereotypical nature of the seizures and the question of their heritability (Matthews & Wright, 1967; Rowan et al, 1970; Daly & Forster, 1975) that this form of reflex epilepsy has been regarded by some as a discrete clinical entity, and been referred to as primary reading epilepsy. The classification of patients whose seizures occur when reading has, however, been associated with more than the usual controversy. Even in patients with the stereotypical seizure pattern the EEG may show focal (Critchley et al, 1959; Baxter & Bailey, 1961; Bennett et al, 1971; Forster & Daly, 1973; Daly & Forster, 1975; Forster, 1977) or generalised (Lemmi & Farris, 1970) paroxysmal abnormalities and treatment with a variety of anticonvulsants has proved effective, including clonazepam (Hall & Marshall, 1980; Saenz-Lope & Herranz-Tanarro, 1982) and valproic acid (Vanderzant et al, 1982; Harrington et al, 1983).

It is obvious from the above sections that the several perceptual, motor and cognitive skills involved in reading may each be separately responsible for inducing seizures. In addition, a few individual patients will, by chance, be susceptible to seizures as a result of more than one of the above contributing factors. If the patient is sensitive to all of them the epilepsy may never be recognised as being reflex in nature: the seizures may be triggered by many activities other than reading and therefore appear to occur at random. If, on the other hand, patients are minimally sensitive to each of the component skills, the sensitivity may demonstrate itself only during the performance of a task such as reading that simultaneously demands all those skills. This may provide one explanation why there are patients who suffer seizures only when they read, patients for whom the observation of patterns, the movement of the eyes, and the comprehension of linguistic information do not, of themselves, appear to be epileptogenic. Another possible reason is that there are few other everyday tasks

eating. They are also light-sensitive and may

all also increase. It is perhaps for this reason that, in a patient with reading epilepsy, White (1970) was able to demonstrate photosensitivity after sleep deprivation but not before. In view of the cases histories reported above, it is of interest that the reading mask has proved an effective treatment in patients who suffered seizures that had the stereotyped form described above and which were associated only with reading. Patents who are demonstrably sensitive to both pattern and picture stimulation rarely report seizures as a result of reading, and this might be taken as evidence that such seizures are rare. However, patients only if they notice them. There are many epileptogenic patients described above, and many are also other precipitants. On the other hand, the two photosensitive patients whose data are shown in Table 11.2 were more pattern sensitive than is often the case. It is however only when pattern were more pattern sensitive than is often the case. It is however only when pattern sensitivity is pronounced that the pattern, in form by text, is sufficient to induce seizures. As has been noted above, printed material varies considerably in the extent to which approximates an epileptogenic pattern, and its visual parameters are never as epileptogenic as those of the most provocative patterns. When text is electronically displayed on the surface of a cathode ray tube, the epileptogenic properties of flickering light are added to those of the pattern of text. Usually the display is refreshed 50 times per second and often there is no 25 Hz line interface. There are yet been electrographic studies of the epileptogenic properties of these displays. Before completing this section it may be worth emphasizing the following points:

(a) If patients are sensitive to diffuse intermittent photic stimulation there is a 30% chance that they will also be sensitive to patterns (see above).

(b) If they are sensitive to patterns, patients may have difficulty reading because of seizures (possibly subclinical) induced by the successive lines of text. This may be one of the many reasons for the poor reading skills of patients with epilepsy (Stokes & Hart, 1976).

(c) If the lines of text above and below those being read are partially obscured, patients with seizures induced by reading may be enabled to read without the risk of reading difficulties are attributable to eye movements, those who suffer seizures which involve mydriatic movements of the jaw, and which are triggered exclusively by reading.

SEIZURES INDUCED BY THE PATIENT

Patients use a variety of techniques to induce seizures. Many of the techniques appear to take advantage of their sensitivity to light. As has already been described, patients...
may cause the television picture to go unstable and then stare at the flickering light that it produces. Alternatively they may stare at patterns (Andermann et al, 1962; Panayiotopoulos, 1979) or direct their gaze upwards, close the eyes and wave the outstretched fingers of a hand across the forehead, thereby producing intermittent retinal stimulation. These obvious behaviours are rare (Forster, 1977; p 39). Other forms of self-induced seizures may be more common, more subtle, and (in contradistinction to the above) rarely associated with mental retardation (cf. Andermann et al, 1962). About 20% of photosensitive patients close their eyes for about 2 seconds, rolling up the eyes and attempting to keep the eyelids closed (Darby et al, 1980; Binnie et al, 1980). When the eyes are rolled upwards and the eyelids are closed it is easy to produce a very rapid flutter of the eyelids across the pupil. This stereotyped movement of the periorcular musculature may be learned. In collaboration with colleagues we have seen several patients who used to wave their fingers across their eyes, but ceased to do so when the habit led to teasing at school. Within a short while they began to make the slow closure of the eyes with eyelid flutter (C. D. Binnie, personal communication).

Close television viewing, handwaving or slow closure of the eyes with eyelid flutter are all behaviours that result in epileptogenic stimulation by patterns or intermittent light. Although stimulation of this kind may be the mechanism whereby seizures are initially induced, there is evidence that the non-visual consequences of the associated behaviours may eventually come to induce seizures. The processes whereby this might occur are not well understood although some form of learning might be involved. Gastaut et al (1956) found that if a word was repeatedly associated with the presentation of intermittent light and a photoconvulsive response induced, the word itself would eventually elicit paroxysmal EEG activity. It is perhaps as a result of such learning that behaviours that would ultimately produce an intermittent stimulation of the retina will sometimes continue to be associated with paroxysmal EEG abnormalities even in darkness. Thus Darby et al (1980a) found that when the room was in darkness, fewer slow eye-closures were made and they were rarely accompanied by paroxysmal abnormalities. Such abnormalities were not, however, completely eliminated in darkness, presumably because some of the discharges were triggered by afferents that were not visual. Wastell et al (1982) found that the EEG power in the low frequency range prior to the closure of the eyes distinguished those closures that were to be followed by paroxysmal EEG activity from those that were not. This result is difficult to interpret. Perhaps the EEG reflected a non-visual sensorimotor contribution to the induction of the discharge, one that preceded the intermittent retinal stimulation. Periorcular afferents are known to potentiate the photoconvulsive effects of intermittent light in the baboon Papio papio (see Naquet et al, 1975, for review) and it is possible that similar afferents contributed to the induction of seizures by eye closure. Alternatively, it is also possible that the eye-lid flutter is part of the seizure, described by Jeavons (1982) as ‘eyelid myclonia with absences’. Of course, it is difficult, if not impossible, to disentangle cause and effect in complex systems that involve feedback, and the induction of seizure activity may involve sensory augmentation in just such a feedback system. This difficulty of attributing cause is also illustrated in the controversy as to whether, in patients who wave their fingers in front of their eyes, the movement of the hand is (Livingston & Torres, 1964; Ames, 1974) or is not (Forster, 1977) part of the seizure.
OTHER TECHNIQUES FOR TREATMENT

This chapter has concentrated on a few of the more frequent forms of reflex epilepsy, their physiological mechanisms, and some recent techniques for treating them. These techniques can be classified as ways of avoiding or modifying the provocative sensory stimulation. The chapter would not be complete without a description of methods that involve desensitisation. Far from avoiding provocative stimulation these methods repeatedly expose the patient. Since the methods have received extensive description in the work of Forster (1977) only a brief mention will be made here. The techniques involve the repeated presentation of provocative stimulation that is below the convulsive threshold in the hope that a rise in threshold will generalise to more provocative stimulation. In the case of photosensitive epilepsy, for example, the patients receive presentations of monocular stimulation, stimulation with a frequency outside the photoconvulsive range, or stimulation against a steady intense background illumination. Forster (1967) reports successes with these methods but Braham (1967) and Harding et al (1967) report failures. Unfortunately those studies that have reported the success of conditioning techniques have not described the details of the behaviours antecedent to paroxysmal EEG activity.

In the case of reading epilepsy, patients can be instructed to make some overt response such as hitting the thigh with the hand each time a certain letter occurs. This activity disrupts the process of comprehension and may reduce seizure susceptibility for this reason. Apart from the work of Forster (Forster et al, 1969; Forster, 1977) we know of only one case study reporting success with this method, although in this instance the success may have been due as much to the patient’s decision to give up reading for longer than 15 min at a time, as to any effect of conditioning (De Weerdt & Van Rijn, 1975).

If patients are warned of an imminent seizure they can sometimes (if they wish to) take steps to avoid the seizure. This appears to apply even if the warning is ictal in origin. It might be in this context that behavioural techniques have the greatest contribution to make (Efron, 1957; Zlutnic et al, 1975; Ince, 1976; Mostofsky & Balashak, 1977).

CONCLUSION

This chapter has described a variety of techniques for controlling seizures induced by television and reading. The techniques have been derived from a detailed study of the sensory parameters that elicit paroxysmal EEG activity in patients with epilepsy. This study has not only suggested techniques for avoiding seizures, it has indicated ways in which the electrophysiological contribution to diagnosis might be improved.

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