Stripes, Complex Cells and Seizures
An Attempt to Determine the Locus and Nature of the Trigger Mechanism in Pattern-sensitive Epilepsy

By
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Pattern-sensitive epilepsy is a rare disorder in which seizures are triggered by the viewing of certain patterns. It was first described by Bickford and his associates in 1953 and since then several cases have been reported (Bickford and Klass, 1962, 1964, 1969; Gastaut and Tassinari, 1966). The first intensive investigation of the phenomenon was undertaken by Chatrian and co-workers (Chatrian, Lettich, Miller and Green, 1970; Chatrian, Lettich, Miller, Green and Kupfer, 1970) who described 4 cases of pattern sensitivity and studied the conditions under which the discharge occurred. The present study benefited from their detailed description of the disorder, and attempted to determine the physiological nature of the mechanism by which the seizures were triggered and, in particular, the locus and type of the neurons involved.

Of 6 patients with pattern sensitivity followed by us, one was particularly suitable for a study of this kind. Her discharges were readily induced by patterns and were accompanied by brief clinical absence rather than generalized tonic clonic seizures. The present paper describes a study of this patient’s sensitivity. It is divided into four sections. The first provides a case history of the patient together with the clinical setting in which the study was performed. The second section contains the findings and the inferences which restricted the number of possible trigger mechanisms and suggested a method of treatment. The way in which the treatment was evaluated is described in the third section and the fourth and final section discusses a plausible trigger mechanism.

Clinical Background
M. T. is a 14-year-old girl born after normal pregnancy and prolonged labour lasting thirty hours. Forceps were used without producing apparent trauma and she weighed 8 lb. In the neonatal period she seemed normal; she sat up alone at 7 months, and walked at 13 months. She said words at 14 months but subsequently her language development was slow and this was attributed to the fact that she was learning to speak two languages. At the age of 3 she developed

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brief episodes of shaking and elevation of her arms followed by a fall. She also had infrequent brief absence attacks.

On examination she was found to be slightly unsteady but had no clear cerebellar syndrome nor other neurological signs. X-rays of the skull were normal. The EEG showed photosensitive generalized irregular high voltage bursts of spike and wave discharge lasting one to three seconds. Subsequent tracings showed very pronounced photosensitivity with multiple spike and wave discharges lasting two to three seconds, accompanied by facial or eyelid twitching and staring. Background activity was normal.

Her drop attacks ceased when she was treated with trimethadione, 600 mg and diphenylhydantoin, 75 mg per day, although her absence attacks continued. At the age of 6 her speech was immature, her movements slow and her comprehension limited. She lisped, substituted letters and tended to be repetitive in her speech. Her full scale IQ was 63 on the Stanford Binet Scale and she attended a special class.

When the child was 8 years old the mother noticed that her absence attacks were associated with looking at fences, grilles or blinds. She was treated with ethosuximide 1 g and acetazolamide 500 mg daily with a reduction in the frequency of her staring spells.

Two years later the mother convincingly demonstrated her daughter's pattern sensitivity to one of us (F. A.). She asked her to look at the grille of an air-conditioner and the child developed a brief absence attack with head nodding. This could be reproduced at will under conditions of normal illumination. Dark glasses were prescribed, with little reported effect on the frequency of staring spells.

It was at this time that a scoliosis was noted, with a primary curve in the thoracolumbar region centred at L2 and pointing towards the left side. There was a compensatory curve in the lower thoracic region. In order to exclude a systemic or neuromuscular disorder, EMG, conduction velocities and a muscle biopsy were done. These revealed no abnormality. Surgical correction with Harrington-rods was carried out.

In a further attempt to improve her absence attacks, treatment with nitrazepam 10 mg daily was started in addition to the ethosuximide and acetazolamide. The frequency of absence attacks diminished but she had a brief generalized seizure. Diphenylhydantoin, 50 mg twice daily, was added, the nitrazepam was stopped and there has been no recurrence of major attacks.

Psychometric evaluation was carried out at the age of 12 by Mrs. C. Strauss. On the WISC the verbal IQ was 53, and performance IQ 48 with a full scale IQ of 46. Reproduction of the Bender figures indicated severe perceptual motor problems with examples of distortion, rotation, substitution, etc. The patient now attends a special school.

An ophthalmological examination was performed in February 1974 by Dr. B. R. Younge and revealed a mild myopic astigmatism (measured by retinoscopy and checked subjectively) which did not require the use of glasses. Vision was correctable with refraction to 20/25 in each eye and the optokinetic response to a rotating drum containing pictures was normal.

The patient is sensitive to a wide variety of patterns in her environment, especially the grille on the family stereo, the father's striped shirts, escalators in department stores, and floor grilles at the entrances to buildings. According to the mother, all the patient's absence attacks can be attributed to the viewing of patterns and this observation is supported by the fact that no "spontaneous" attacks have been observed in the laboratory. She does not actively search for patterns to stare at, but is apparently unable to avoid looking at any which appear in her visual field.

**EXPERIMENTAL INVESTIGATION**

(a) **Intermittent Stimulation of Retinal Cells**

Some cases of pattern sensitivity have been responsive only to striped lines in one particular orientation, while others have been responsive to all pattern orientations
All cases reported to date have been sensitive to photic stimulation, that is, stimulation with stroboscopic light. It is conceivable that an intermittent stimulation of retinal cells similar to that produced by photic stimulation occurs when the image of a sharply contoured pattern is vibrated across the retina by physiological nystagmus. Nystagmus may have the effect of converting a spatial pattern on the retina into a temporal sequence of successive volleys in the visual pathway, in other words of producing a quasi-photic stimulation. Since physiological nystagmus is usually omnidirectional, quasi-photic stimulation is a rather unconvincing interpretation of the seizure trigger in those cases which, in the absence of any evidence of astigmatism, manifest a strong differential responsiveness to one particular pattern orientation. Such cases would require an abnormal nystagmus, and no clinical nystagmus was evident in the present case or has previously been reported in cases of pattern-sensitive epilepsy.

Given that preliminary investigations had established that M. T. was sensitive to a variety of symmetrical striped patterns, including concentric annuli and the Mackay (1961) figure of radiating lines, it was reasonable to investigate whether she was responsive only to patterns in a limited range of orientation.

Experiment 1.—Horizontal, vertical and oblique square-wave gratings, circular in outline, were back projected on a translucent screen by a carousel slide projector. The gratings were viewed at 63 cm, at which distance they subtended a visual angle of 18 degrees and had a spatial frequency of 2.5 cycles per degree. The mean luminance of the patterns was approximately 80 cd/m² and they had a contrast of 0.9. In two sessions, each containing a series of 24 randomly ordered presentations, gratings appeared for ten seconds with a ten-second interval between presentations. The patient's EEG was monitored, and she was simply instructed to look at the patterns.

The criterion for a response to a pattern was a discharge of generalized spike-and-wave such as that shown in fig. 1. Bursts of slow and sharp wave were not included. Table I provides a summary of the number of pattern presentations for which a response occurred. There is no significant difference between the incidence of discharge for any of the four patterns ($\chi^2 = 3.18, P > 0.5$). Studies which will be described later indicate that the failure to show a significant orientation specificity was not due to an insensitive response criterion or an inadequate number of trials. In fact, Experiment 4 confirms that the tendency for a less frequent paroxysmal response to vertical gratings was unreliable. Evidently M. T. was not sensitive

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<th>Table I.—Incidence of Discharge as a Function of Pattern Orientation</th>
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Fig. 1.—An example of a pattern-induced discharge. The recording is monopolar with the spinous process of C7 used as common reference. The horizontal bar indicates the duration of pattern presentation.
exclusively to a limited range of grating orientations and for this reason the hypothesis of quasi-photic stimulation was tenable in her case.

Experiment 2.—In an initial attempt to test whether quasi-photic stimulation was occurring, patterns were presented for a period too brief for nystagmus to displace the retinal image. The patient was seated in a darkened room for approximately ten minutes, after which a photographic print of a square-wave grating (18 × 18 cm square with 3-9 cycles/cm and contrast 0-9) was placed at a distance of 84 cm from the patient's eyes. A high-power flash gun illuminated the pattern momentarily, and although the patient reported seeing the after-image, it did not prove possible to obtain any seizure discharge using this technique. Since the failure to obtain a discharge may simply have reflected insufficient retinal stimulation, an alternative method of testing the hypothesis of quasi-photic stimulation was adopted.

Experiment 3.—A checkerboard pattern has a greater number of brightness contours than a striped pattern and any intermittent retinal stimulation as the result of nystagmus should therefore be at least as great for a checkerboard as for striped lines. It was consequently of some interest that, when presented for several minutes with a slide of a checkerboard in which the size of the checks was similar to the width of lines in previous patterns the patient's EEG showed hardly any sign of epileptic abnormality. In order to investigate further this differential sensitivity to checks and gratings, photographic prints of patterns shown in fig. 2 were used. All the prints had a contrast of 0-9. They were 18-5 cm square and were presented at a distance of 84 cm. The pattern in fig. 2b (line length/width = 64) was produced by dividing the grating shown in fig. 2a (line length/width = 128) into two sections in counterphase. Similar divisions produced patterns with line length/width ratios of 32, 16, 8, 4 and 2, for example, figs. 2c and 2d. The checkerboard pattern itself, fig. 2e, was obtained by juxtaposing four commercial prints. A series of trials comprised one presentation of each of eight patterns in a random order and at the end of each series the patient was given a rest of at least one minute. For 15 of the 17 series in Session 1 each pattern was presented for five seconds, separated by an interval of ten seconds. For the remaining series, the presentation time was seven seconds, with an eight-second interval; the longer exposure being introduced when responsiveness seemed to be decreasing. The mean luminance of the patterns was constant for a particular series but varied between series from 50 to 110 cd/m². The results of session 1 are shown in fig. 3, and it will be noted that there is a range of patterns for which responsiveness is a linear function of the logarithm of the ratio of line length to width. The responsiveness to the checkerboard (line length/width = 1) was probably spuriously low: in the commercial print used to produce this pattern, the area of dark squares was greater than that of the light squares.

Instead of attempting a direct replication of the above results it was decided to obtain some idea of their generality with respect to spatial frequency. All the prints except that of the checkerboard were cut along horizontal and vertical axes passing through the centre of the pattern, and one of the four resulting quadrants of each pattern was used in a second session run five weeks after the first. In this session,
Fig. 2.—Examples of the segmented patterns used in Experiment 3.
which comprised 12 series with a five-second presentation time and ten-second interval, the patterns were viewed at a distance of 42 cm, half the distance used in the earlier session. As a result, the area of retina receiving patterned stimulation was the same in both sessions, although the retinal width of the lines in the second session was twice the width used in the first. The results of session 2 are presented in fig. 3, and the degree of similarity between the two sessions indicates that the method of measuring seizure tendency was reliable.

![Graph showing incidence of discharges induced by segmented patterns expressed as a function of line length/width.](https://academic.oup.com/brain/article-abstract/98/3/365/281052)

**Fig. 3.**—Incidence of discharges induced by segmented patterns expressed as a function of line length/width.

The extent to which discharges are a linear function of the logarithm of line length/line width (or some concomitant of this variable) does not lend itself to a simple interpretation of pattern sensitivity in terms of a quasi-photic stimulation of retinal cells. It is difficult to see how the intermittent stimulation of retinal cells produced by nystagmus might decrease with the increase in brightness contours due to segmentation of stripes. The findings suggest an entirely different mechanism for the initiation of seizures, dependent on the length of line contour within the pattern, and thus on the spatial rather than temporal characteristics of stimulation. In short, it would appear that seizures are triggered by simple or complex cells (Hubel and Wiesel, 1962), cells which fire when their receptive fields are stimulated by lines. Presumably the longer the lines within a pattern, the more such cells are recruited.

Simple cells are responsive to lines within a restricted receptive field, whereas complex cells respond to lines over a larger visual field. They occupy intermediary positions on an hypothetical hierarchy of cells in the visual system (Hubel and Wiesel, 1962, 1965b). At the base of this hierarchy are cells with circular receptive fields. Simple cells are thought to receive projections from rows of these cells and to project to complex cells. Complex cells in their turn project to hypercomplex cells that respond to corners and "tongues." Whether or not such a hierarchy exists, it would seem that hypercomplex cells and cells with circular receptive fields were not responsible for triggering seizures in the present case, because such cells are likely to be more sensitive to checks than to stripes.
(b) Location of the Pattern-sensitive Trigger Mechanism

If line-detectors are indeed responsible for the initiation of M. T.'s seizures then available evidence would suggest a cortical locus for the trigger. This is because line-detectors have been found in the striate cortex of cats and monkeys (Hubel and Wiesel, 1962, 1968) but not elsewhere in the visual system. Little is known about the receptive fields of human geniculate cells, however, and it remains possible that these cells have linear receptive fields. Indeed, if it is assumed that some diffuse thalamic system is responsible for the spike and wave discharge (Bickford, Daly and Keith, 1953, p. 181) it is easier to conceive of a geniculate locus for the trigger mechanism than a cortical locus. Fortunately, current knowledge of the anatomy and physiology of the human geniculate is sufficient to provide a means of distinguishing these alternative loci. The geniculate is divided into layers of cells which respond to stimulation of one eye and not the other, in marked contrast to striate cortex in which the majority of cells are binocular and respond maximally only to stimulation in corresponding retinal fields of both eyes.

Experiment 4.—Previous investigators have noted that sensitivity to patterns viewed with one eye is less than that to patterns viewed binocularly (Bickford and Klass, 1964; Chatrian et al., 1970). This finding may simply be due to the fact that more retinal and geniculate cells are stimulated during binocular presentations. Alternatively the effect could be due to the fact that when binocular fusion occurs more binocular cells are stimulated in cortex. In order to control for stimulation of retinal and geniculate cells while manipulating the extent of cortical stimulation, a stereoscope was used to present binocular patterns that cannot fuse in addition to those which can. Monocular patterns were also presented with a view to assessing the prospects for treatment with monocular occlusion. It is worth noting that when a stereoscope is used to present monocular patterns it is difficult for the subject to tell which eye is being stimulated.

The patterns which were used are represented schematically in Table II. The full-size horizontal and vertical gratings were square in outline and comprised 16 cycles, each cycle subtending approximately 2 degrees with white and black bars of equal width. The half-size gratings (stimuli e, f, i, j, m) contained eight cycles and were obtained by dividing a horizontal full-size grating along a mid-line parallel to the lines of the grating. For all binocular stimuli, patterns were presented to corresponding retinal loci in the two eyes, with the exception of the combinative patterns (stimuli i and j) in which the two half-size gratings were vertically displaced so that the bottom line of the upper grating fused with the top of the lower grating. All the gratings had a contrast of 0.9.

In the first session of trials, stimuli were divided into two groups, one group comprising stimuli a, d, f, g, i and k, and the other group stimuli b, c, e, h, j and l. The mean luminance of the patterns was approximately 80 cd/m². Random series, consisting of stimuli from one group and then the other in alternating sequence, were presented for a total of 16 series, 8 from each group. Responsiveness was low, and so the criteria for a discharge were relaxed to include bursts of generalized sharp
and slow wave. The results, which are shown in Table II, indicate that monocular stimuli are considerably less effective in eliciting a discharge than binocular stimuli, and for this reason plans were made to attempt treating the patient’s seizure condition with monocularly occluding spectacles (see Treatment). Before treatment

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<th>Condition</th>
<th>Stimulus</th>
<th>Patterns</th>
<th>Discharge Incidence</th>
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commenced a brief session was run. It was designed to examine whether unfusible binocular patterns (stimuli g and h) were less effective in eliciting a discharge than binocularly fusible patterns (stimuli k and l), since the results of the first session were equivocal in this respect. Half-size patterns (stimuli i and j) were also included as a check on the effects of the amount of patterned stimulation. The patterns were brightly illuminated (approximately 100 cd/m$^2$) and the usual criteria for a discharge were sufficient. The incidence of a discharge in response to unfusible full-sized patterns (stimuli g and h) was considerably less than for full-size fusible patterns (stimuli k and l), and the difference is significant at the 5 per cent level on a Fisher exact test.

A month after the beginning of treatment with monocular occlusion it was possible to study the patient for a more lengthy session. In this third session, binocularly fusible half-size patterns (stimulus m) were introduced, facilitating an additional comparison of binocularly fusible patterns with those of similar size which cannot be fused (stimuli i and j). The results are shown in Table II and once again they demonstrate that the patient was primarily responsive to binocularly fusible patterns. The difference in responsiveness to fusible and unfusible binocular patterns is significant at the 0·1 per cent level for full-size patterns and 0·5 per cent level for half-size patterns, using a Fisher exact test.

The differential sensitivity to patterns which can and cannot fuse when the degree of retinal stimulation is held constant suggests that the trigger mechanism involves binocular cells and hence is cortical in locus. (An alternative interpretation of the effect of binocular fusion in terms of convergence nystagmus may be discounted on the grounds that the necessary preference for vertical gratings was not demonstrated.)

Taken together, the results of the above studies are consistent with the hypothesis that the trigger mechanism involves line detectors, that is, simple or complex cortical cells. There are, however, two reasons for favouring complex rather than simple cells. First, experiments on rhesus monkeys have indicated that most simple cells are driven by one eye, and could not therefore give rise to the effects of binocular fusion described above (Hubel and Wiesel, 1968). Secondly, complex cells receive projections from retinal ganglion cells which are particularly responsive to transient light (Maffei and Fiorentini, 1973). The fact that the patient was sensitive not only to patterns but also to transient light in the form of photic stimulation is therefore consistent with the notion that seizures were triggered by complex cells.

Experiment 5.—If complex cells do in fact provide the trigger, the patient should be sensitive to gratings which vary in brightness, and insensitive to those which vary only in hue. This is because relatively few cortical cells are responsive to one colour and not to others; the majority respond to light of any wave-length and are sensitive only to contours of brightness. To test the prediction a grating of alternate red and green lines with dimensions similar to that of the straight-line pattern used in Session 2 of Experiment 3, was prepared using paints of matched reflectance (Liquitex Modular Colour, value 4). The grating was presented to the patient at a variety of viewing distances (20–100 cm) for a period of approximately five minutes under conditions of
high incident illumination (mean luminance 170 cd/m²), and at no time while the pattern was viewed did a discharge occur in the EEG. Both before and after this presentation the patient was highly responsive to a black and white grating of similar dimensions.

**TREATMENT**

Chatrian *et al.* (1970) noted that monocularly viewed patterns were less epileptogenic than binocularly viewed patterns, and this observation has been confirmed in the present series of experiments (see Table II). Given the reduced sensitivity to monocular stimulation an obvious treatment for the seizure disorder is occlusion of one eye. Chatrian *et al.* gave one of their patients an eyepatch but unfortunately the patch was not worn consistently by the patient. Although the authors reported that the patient experienced fewer seizures while the patch was worn, no attempt was made to measure seizure incidence objectively. In the present study an alternative approach was used. The majority of binocular cortical cells, such as those apparently responsible for triggering the patient’s seizures, fire maximally only when binocular fusion occurs. It follows that any treatment which eliminates binocular fusion should decrease the level of activation of these cells and thus the number of seizures they trigger. The patient was therefore given a pair of plane spectacles, one “lens” of which was covered with translucent Scotch tape. The taped lens blurred the retinal image of the “occluded” eye and eliminated binocular fusion without, of course, reducing appreciably the light transmitted through the lens. The advantages of this treatment over the use of an eye-patch were twofold. First, the spectacles occluded binocular vision only in that part of the visual field for which fusion was possible, so that unnecessary occlusion was avoided and the patient was able to use peripheral vision in the “occluded” eye. Secondly, the spectacles were not socially obtrusive and were therefore acceptable to the patient.

In order to evaluate this treatment the patient wore a miniature twenty-four-hour cassette tape recorder (Cashman and Stott, 1974) adapted for EEG use by the addition of small preamplifiers (Ives and Woods, 1975). Six EEG electrodes and a ground were hidden in the patient’s hair and they were connected to the tape recorder through a cable containing the preamplifiers passing down the back of the patient’s neck. Four channels of EEG were monitored: channel 1, F3–C3; channel 2, C3–P3; channel 3, F4–C4; channel 4, C4–P4. The patient wore the tape recorder in her everyday environment at home and at school and her father changed the cassette every twenty-four hours. An initial pilot trial and two ninety-six-hour clinical trials were run.

In the first of the clinical trials (18.00 hours Wednesday April 3 to 20.00 hours Sunday April 7) the patient wore spectacles that occluded the left eye on April 4 and 6 but no spectacles on the other days. At the end of the trial the taped EEG was played back on a Mingograf EEG machine at a paper speed equivalent to 2.5 mm/s, and despite the unusually slow speed, the spike and wave discharges could clearly be seen. To obtain an objective measure of the incidence of the discharges, the output
of channel 1 was filtered so that only frequencies in a range equivalent to 2.5 to 4 Hz were passed. Whenever the rectified output of the band-pass filter exceeded 50 μV for more than one second the EEG was visually inspected to screen out the few artifactual responses and non-generalized discharges. Provided these were absent the occurrence of a discharge was noted. Fig. 4 presents the number of spike-and-wave discharges for each hour of the four-day trial, together with the incidence of spike-and-wave on two days of the pilot run (March 20 and 21). The horizontal bars indicate the periods for which the glasses were worn, and the dotted columns represent sleep. The histograms within the sleep periods are not valid since it was impossible to discriminate spike-and-wave bursts from slow-wave sleep, even when the paper speed was increased.

![Fig. 4.—Incidence of spike-and-wave bursts on days with and without monocularly occluding spectacles. The horizontal bars indicate the periods for which spectacles were worn.](https://academic.oup.com/brain/article-abstract/98/3/365/281052)

The mean number of spike-and-wave bursts between 9.00 and 18.00 hours on March 21, April 5 and April 7 was 22.2 bursts/hour. The mean for equivalent periods on April 4 and 6 when glasses were worn was 2.5 bursts/hour, indicating that the glasses were effective in reducing the incidence of spike-and-wave by well over 80 per cent. The difference in the discharge incidence for days with and without
treatment is significant at the 1 per cent level, \( t(3) = 5.37 \). Given that in the laboratory seizures were only observed in the presence of line patterns, and that monocular occlusion was effective in reducing responsiveness to these patterns, the results are testimony to the large number of line patterns in the day-to-day environment.

After the above findings were obtained the patient was fitted with two pairs of spectacles, one pair which "occluded" the right eye, and the other which "occluded" the left. She changed the spectacles from day to day so as to alternate the eye "occluded."

Nine weeks after the beginning of treatment, a second trial was run (18.00 hours Wednesday June 12 to 20.00 hours Sunday June 16). Its purpose was twofold: first, to determine whether the effectiveness of the glasses would be reduced as a result of continuous wearing; secondly, to introduce a placebo control to check that the reduction in seizure incidence could be attributed to monocular "occlusion" and not to some more general effect. On June 13 the patient wore spectacles which "occluded" the right eye. The following day she wore placebo spectacles which occluded pattern vision in the upper and lower field. (Both "lenses" of these spectacles were "frosted" so as to leave a central horizontal strip of clear glass; the total area "occluded" was equal to the area of one lens.) On the third full day spectacles which "occluded" the left eye were worn, and on the fourth the patient wore no spectacles at all. The mean incidence of spike-and-wave between 9.00 and 18.00 hours was as follows: June 13—1.4 bursts/hour; June 14—8.3 bursts/hour; June 15—2.0 bursts/hour; June 16—12.2 bursts/hour. The difference in the incidence of spike-and-wave for days with and without monocular "occlusion" is significant at the 5 per cent level, \( t(2) = 4.2 \), indicating that monocular "occlusion" remained effective even after nine weeks of continuous treatment. The difference in the discharge incidence on Day 2 and Day 4 (an ostensible placebo effect) fails to reach significance when day-to-day variance is estimated from the previous clinical trial allowing for the higher mean values.

The mean incidence for the four days of the second clinical trial is less than half that for the first, indicating a considerable general improvement in the patient's seizure tendency irrespective of whether or not glasses were worn. This may conceivably be attributed to the fact that at the same time as treatment with the spectacles was instituted, phenobarbitone was added to the patient's medication in order to control the occurrence of infrequent myoclonic attacks. Phenobarbitone is not noted for its effect on absence attacks, however, and a more likely explanation of the improvement is that some neural adaptation—a negative "kindling" effect—occurred as a result of the lower seizure incidence.

Any reduction in seizure incidence must always be weighed against the possible adverse effects of treatment. In the present case little is known about the effects of prolonged alternate monocular occlusion. It is known to reduce the proportion of binocular cells in the cortex of kitten (Hubel and Wiesel, 1965a) but there is no evidence that any physiological change occurs in the final stages of development of an organism. The only known effect is a temporary impairment in stereoscopic acuity.
(Herman, Tauber and Roffwarg, 1974), and in the present case the risk of such impairment is at least commensurate with the benefit derived. Quite apart from the reduction in seizure incidence, there has been definite improvement in the patient’s arousal and attention. She is no longer tired at the end of the school day but goes out and plays with other children.

**DISCUSSION**

It has been shown that, in the present case of pattern-sensitive epilepsy, the neural events that culminated in generalized synchronous spike-and-wave discharge were triggered in the striate cortex. The experimental evidence consistently indicates a trigger mechanism involving complex cells and not cells of the simple or hypercomplex type (Hubel and Wiesel, 1965b, 1968).

In cat and monkey, and presumably also in man, complex cells are found principally in cortical layer II. They are organized in columns: neighbouring cells within a column have proximate receptive fields of similar orientation, and the orientation changes little from one column to its neighbour. This suggests that striped patterns are optimal stimuli for a cluster of closely spaced columns, and produce intense firing of cells within a small area of cortex. Complex cells fire maximally only when the image of the line is moved across the retina, and they often exhibit a preference for one particular direction of movement. The movement of the retinal image of a pattern by the physiological nystagmus of the eye therefore has the effect of synchronizing the firing of those cells which the pattern stimulates. One might speculate that when the synchronous firing of cells within a small area of hyperexcitable cortex (possibly in layer II) becomes sufficiently intense, cells in neighbouring areas of cortex are recruited in a rapidly cascading sequence, spreading from striate cortex to involve large areas of brain. As mentioned previously monocular “occlusion” is an effective treatment, purportedly because it reduces the average firing rate of binocular cells so that the critical level of neural activation is reached less frequently.

A mechanism of the above kind is capable of accounting for those cases of pattern sensitivity in which the paroxysmal discharge is present only for certain restricted pattern orientations. The trigger in these patients presumably involves only columns of complex cells with receptive fields of similar orientation.

The logical basis for the treatment reported in the present paper is that it reduced the general level of activation of cells which were thought to trigger seizures. This rationale has little in common with that underlying other ophthalmic treatments of light-sensitive epilepsy that control patients’ exposure to environmental photic stimulation (e.g. Harding, Drasdo, Kabrisky and Jeavons, 1969). Such control may provide a useful means of treating those cases of photosensitive epilepsy which have no localizable trigger. It is conceivable, however, that many cases of photosensitive epilepsy have a trigger mechanism in the striate cortex, even though it may involve a wide variety of cells, and the patients may not be obviously pattern-sensitive. Since the majority of cortical cells are binocular, reducing the level of neutral activity with
spectacles similar to those used in the present study might benefit these patients. Current work is aimed at determining whether patients who are photosensitive but not pattern-sensitive do in fact have a cortical trigger.

**SUMMARY**

The study concerns an epileptic patient whose absence attacks were contingent on the viewing of striped patterns. A series of experiments demonstrated first that seizures were not due to the intermittent stimulation of retinal cells produced as physiological nystagmus vibrated the image of the pattern, and secondly that seizures were triggered at the cortical level, probably by the firing of complex cells. Spectacles which occluded pattern vision in one eye were highly effective in reducing seizure incidence.

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