events, we think that the evidence is sufficiently suggestive to prescribe drugs affecting platelet function such as aspirin and sulphinpyrazone. These observations emphasise the importance of thorough cardiovascular assessment in all patients with acute cerebral or ocular ischaemia, and we suggest that echocardiography should be part of the routine investigation of such patients.

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Television epilepsy and pattern sensitivity

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Summary

Properly functioning domestic television sets may induce seizures in epileptic patients (TV epilepsy). We investigated the effects of different types of visual stimuli on paroxysmal electroencephalographic (EEG) activity in 32 epileptic patients known to be sensitive to intermittent photic stimulation (stroboscopic light). We monitored sensitivity to patterns of horizontal and vertical lines, both stationary and vibrated (pattern sensitivity), and to normal broadcasts on a domestic, black and white (405- or 625-line) TV receiver (TV sensitivity). Twenty-three of the 32 patients were sensitive to pattern. Twenty-two were sensitive to vibrated patterns, and 11 to static patterns ($P < 0.01$). All patients sensitive to pattern were also sensitive to TV. The association between sensitivity to pattern and to TV was significant. Clinical history of TV epilepsy (16 out of 32 patients) and laboratory evidence of pattern or TV sensitivity were not significantly associated. The high incidence of pattern sensitivity among flicker-sensitive patients and its association with TV sensitivity suggests that linear patterns produced by the raster of a black and white set as it scans, or "line-jitter" produced by the raster in areas of low TV-signal strength may contribute to the epileptogenic effect of TV.

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Introduction

In patients with a history of epileptic seizures apparently induced by watching television (TV epilepsy) stimulation with stroboscopic light (or "flicker") induces paroxysmal electroencephalographic (EEG) activity.[1] Conventionally TV epilepsy is attributed to flicker, either the slow flicker that occurs when the TV picture slips[2 3] or the 50-Hz component present when the set works normally.[1]

Not all patients with TV epilepsy show EEG sensitivity to 50-Hz flicker, and we have rarely found any evidence that the set was malfunctioning at the time of the fits. We therefore decided to investigate other mechanisms by which TV might induce seizures, particularly the linear patterns which form the TV picture.

Patients and methods

We studied 32 patients found to be flicker-sensitive on routine clinical EEG investigation during a period of 16 months in 1975 and 1976. These patients formed a consecutive series, excluding 12 who attended when the necessary research facilities were not available. The mean age of the subjects was 13 years (range 6-31), with a 19:13 preponderance of women. Thirteen of the patients had had previous EEG and were known to be sensitive to light. All were referred because of known or suspected epilepsy, and 19 were receiving anticonvulsant treatment. Sixteen patients gave a history of major or minor seizures associated with TV viewing and in none of these were the attacks associated with known malfunction of the TV set. Only six of the 16 patients were close to the screen whenever seizures occurred. As most patients with known or suspected epilepsy who attend hospitals in the Southend and Basildon areas are referred to our EEG department for

investigation, no bias towards the selection of patients with TV epilepsy seems to have been likely.

Photic stimulation was usually carried out at the end of a routine EEG recording, which normally included overbreathing (performed by 30 out of the 32 patients). Only in seven did any activation of paroxysmal activity occur during overbreathing, and photic stimulation was not performed until the EEG had returned to its resting baseline. The photic stimulator was placed directly in front of the patient's eyes, about 300 mm from the nasion. The flash frequencies used varied, but always included stimulation at 18 Hz. The patient was asked to close his eyes and about six seconds later to reopen them. Stimulation began when the eyes closed and continued for about 12 seconds unless any paroxysmal activity occurred.

Pattern sensitivity—A patient found to be flicker-sensitive was asked to view patterns and television. We used two different methods of pattern stimulation. Initially the patient was asked to view a circular photographic print (diameter 165 mm) of a grating of black and white lines of equal width. At the viewing distance used (300 mm) the grating had a spatial frequency of 2 cycles/degree. One of us had already established this to be a suitable but not necessarily optimal spatial frequency for showing pattern sensitivity.[4] The patient watched the pattern with horizontal and vertical orientation of its lines, both when it was held steady and when it was vibrated orthogonal to the line orientation. The patterns were illuminated by a mixture of daylight and artificial light, and so the luminance varied considerably. About halfway through the series (after 15 patients had been tested) we introduced a different method. Patterns of horizontal or vertical stripes 200-mm square in outline with a mean luminance of about 2000 cd/m^2 and contrast of about 0·9 were projected on the rear of a translucent screen. They were viewed from 1 m, and again the patterns had a spatial frequency of 2 cycles/degree. A mirror in the projection system, connected to a galvanometer, was used to produce first a static image and then sinusoidal vertical movement of the image at 18, 10, 15, and 25 Hz (in that order) with an amplitude equal to one half-cycle of the pattern.

TV sensitivity—The patient was also asked to view normal broadcasts on a 22-inch (560-mm) black and white domestic TV receiver. Initially we used a 405-line set but when we introduced the projected-pattern method this was replaced with a 625-line set. The TV set was first viewed at a distance of 3 m, but if this did not cause paroxysmal EEG activity to appear or increase the patient was gradually brought closer to the set until sensitivity was shown or until he was viewing the screen at a distance of 300 mm. Brightness and contrast controls were then varied throughout their ranges.

EEG analysis—The EEGs were divided according to the nature of the resting record. Thirteen patients did not have paroxysmal activity in the resting EEG. Seven had paroxysmal activity on closing the eyes, or while the eyes were closed, or both. Twelve had paroxysmal activity both when the eyes were open and when they were closed. We had little difficulty in assessing the records for sensitivity to visual stimuli except in the last group, for those patients with a substantial amount of paroxysmal activity in the resting EEG with eyes open. In these cases the records were accepted as showing sensitivity if (1) the percentage of time that paroxysmal activity occurred during stimulation was at least twice that which occurred with eyes open in the resting EEG, or (2) if the morphological or topographical characteristics of the paroxysmal activity during stimulation differed from those in the resting record. All the EEGs were assessed by three people who were unaware of the patient's clinical history.

Results

Twenty-three of the 32 patients were sensitive to pattern stimulation. All but one of the 23 were sensitive when the patterns were vibrated and 11 responded to static patterns. The difference in sensitivity to static and vibrating patterns is significant (McNemar's test; $P < 0·01$). The single patient who was selectively sensitive to static and not to vibrating pattern was tested with the photographic print. When we compared the data obtained in the first and second halves of the series we found the incidence of sensitivity to the photographic print was slightly less than to the projected patterns—namely, nine out of 15 patients in the second series (60%), as opposed to 14 out of 17 in the first (82%). The difference is not significant.

All the patients who were sensitive to patterns were also sensitive to TV. Of those patients who were not sensitive to pattern, four were sensitive to TV and five were not. The association between sensitivity to pattern and to TV is significant at the 0·1% level (Fisher's exact probability test). There was little difference between the incidence of sensitivity to the 405-line and to the 625-line TV sets, as 12 out of 15 (80%) and 15 out of 17 (88%) patients were sensitive to each of them respectively.

Fourteen of the 16 patients with a history of possible TV-induced seizures were sensitive to both TV and pattern; one was sensitive to TV alone. The association between clinical history of TV epilepsy and laboratory evidence of pattern sensitivity just fails to reach significance (Fisher's exact probability test; $P = 0·06$). The association between TV sensitivity and a history of TV-induced seizures is also non-significant ($P = 0·17$).

Discussion

PATTERN SENSITIVITY

Pattern sensitivity is said to be rare, not only among epileptics in general (Bickford and Klass[5] found the incidence in a series of 40 000 epileptics to be less than 0·25%), but also in relation to the convulsive effects of light (the same authors report that only about 5% of photosensitive subjects are pattern sensitive). We found 23 out of 32 photosensitive patients (72%) were sensitive to pattern. There are at least three possible reasons why we found such a high incidence of pattern sensitivity. Firstly, there was a considerable discrepancy between the clinical evidence of pattern-induced seizures and the laboratory evidence of pattern sensitivity. Only one of our 32 patients gave a clinical history implicating pattern. Evidently if testing is confined to patients with a positive history of pattern sensitivity many cases will remain undetected. The modern urban environment contains so many linear patterns that the possible effect of these stimuli may be unrecognised by the patient or relatives. Secondly, vibrating the patterns effectively doubled the incidence of pattern sensitivity. Nevertheless, 11 of the 32 patients were unequivocally sensitive to static patterns and this incidence was still considerably greater than expected. Thirdly, the criteria for accepting activity as paroxysmal inevitably differ between observers.

Wilkins *et al*[4] described a patient in whom striped patterns would readily elicit absence attacks, although the patterns were much less effective under conditions of monocular viewing. Spectacles with one frosted lens greatly reduced the frequency of "spontaneous" attacks as monitored by long-term EEG recording in the patient's normal surroundings. The high incidence of pattern sensitivity in the present series suggests that in photosensitive patients many seemingly spontaneous seizures may actually be induced by patterns.

TELEVISION EPILEPSY

Most flicker-sensitive patients are sensitive to stroboscopic stimulation at frequencies near 16 Hz.[1] TV occasionally produces flicker at around this frequency when grossly malfunctioning (for example, when frame synchronisation is lost); or when stroboscopic effects are deliberately used in popular music programmes; or conceivably when a televised pattern of horizontal stripes and the raster that generates its image visibly interact.[1] Nevertheless, we did not find any of these sources of flicker was necessary to induce paroxysmal EEG activity among our patients, nor did the clinical details of the TV seizures suggest that these factors were important in the evocation of fits.

Approximately half of flicker-sensitive patients respond to 50-Hz stimulation,[1] and the diffused light from a European TV receiver fluctuates at this frequency. Nevertheless, an explanation of TV epilepsy based on flicker alone does not explain the failure of Gastaut *et al*[6] to show television sensitivity in patients with a history of TV-induced seizures, nor the difference in sensitivity to black and white and colour television found by Connell *et al*.[7]

Our finding of a high incidence of pattern sensitivity in flicker-sensitive patients and its association with TV sensitivity

suggest that pattern may contribute to the epileptogenic effect of TV. The most obvious linear pattern stimulation from a black and white television set is provided by the raster as it scans across the screen. Half frames of alternate lines are emitted at 50 Hz; the lines interlace and thus give an effective displacement of the retinal image similar to that produced by horizontal lines vibrating at 25 Hz. On black and white TVs in areas of low signal strength there is also a small up and down motion of the raster producing so called "line jitter." This last phenomenon may have some relevance to our study as Runwell Hospital lies in a valley where signal strength is low. Aberrations such as line jitter are accepted as normal by domestic viewers but are not seen on studio-quality equipment and may account for the discrepancy between the high incidence of TV sensitivity reported by us[8 9] and by others,[7] and the failure of Gastaut *et al*[6] to show television sensitivity in the studios of the French national broadcasting organisation.

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Cardiovascular and sympathetic response to exercise after long-term beta-adrenergic blockade

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Summary

The response to dynamic exercise was investigated in 21 patients receiving long-term treatment with beta-adrenoceptor antagonists and 22 controls. An electrocardiogram (ECG) and blood pressure were recorded before and after treadmill exercise, and plasma dopamine-β-hydroxylase (DBH) activity was measured as an index of changes in sympathetic activity. Heart rate and blood pressure were lower at rest and throughout exercise in treated patients, although the pressor effect of exercise was not reduced. The ECG P-R interval was lengthened, and in addition the Q-T interval was prolonged. After exercise, plasma DBH activity was significantly increased in controls but not in treated patients. We conclude that long-term administration of beta-adrenergic blockers increases myocardial repolarisation time and reduces sympathetic nervous activity. These actions may contribute to the antiarrhythmic and hypotensive effects of long-term beta-blockade.

Introduction

Beta-adrenoceptor antagonists are widely used in hypertensive and ischaemic heart disease, yet despite their proved clinical value their therapeutic mode of action is not fully understood. The mechanism by which they lower blood pressure is the subject of continuing controversy,[1] and, more important, the basis of their protective action after myocardial infarction[2] remains to be established. Most investigations of the actions of beta-adrenergic antagonists in man have centred on their short-term effects: they cause an immediate reduction in cardiac output but arterial blood pressure is unchanged.[3] In contrast, long-term administration of beta-blockers produces a well-documented fall in blood pressure, which may be maximal only after several weeks[4] and is associated with a reduction in peripheral resistance.[5] Acute beta-adrenergic blockade produces reflex sympathetic hyperactivity,[6] but studies on animals have suggested that long-term beta-blockade may reduce sympathetic nervous system activity.[7] Long-term treatment also prolongs the repolarisation time of the cardiac action potential in animals,[8] an effect that is known to be antiarrhythmic.

We therefore decided to evaluate in patients the effects of long-term beta-adrenergic blockade on sympathetic function and cardiac repolarisation time. Changes in plasma activity of the noradrenaline-synthesising enzyme dopamine-β-hydroxylase (DBH) after exercise were used as an index of sympathetic activity. The effects of prolonged beta-blockade on the electrocardiogram (ECG) and on blood pressure at rest and after exercise were also investigated.

Patients and methods

Two matched groups of patients were studied—21 aged 44-60 years who had been receiving beta-adrenergic blocking drugs for at least three weeks, and 22 aged 31-78 years who were not being treated and served as controls. The two groups were closely similar clinically (table I) and were matched for exercise tolerance, the mean maximal exercise time being the same in both groups (see Results). All the patients had been referred to the cardiac department for diagnostic maximal treadmill exercise testing. Table II gives the beta-blocker regimens in the treated group. No differences were observed between

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