Letters

Seasonal variation in the incidence of photoparoxysmal discharges among patients investigated after a single seizure

Sir: Among patients investigated after a single seizure, a recent study established that the incidence of photoparoxysmal discharges (PPD) was similar to that of clearly established epileptic patients suggesting that a single seizure was similarly associated with increased excitability of cortical neurons.[1] An earlier study observed a seasonal variation in the incidence of PPD among British epileptic patients.[2] Among known photosensitive patients, a seasonal variation has also been observed in the incidence of PPD.[3]

I report an observation of a seasonal variation in the incidence of PPD among patients investigated after a single seizure. The patients studied were the 408 consecutive patients investigated after a single seizure in the Department of Clinical Neurophysiology, The National Hospital for Nervous Diseases, Queen Square, London, between 1981 and 1985). The EEG of these patients was examined for evidence of photoparoxysmal response to photic stimulation (PPD). Details of methods of selecting these patients, photic stimulation during EEG and criteria for diagnosing PPD, have been described in a recent report on the same group of patients.[1]

The patients were grouped into four in accordance with the season of EEG recording: patients investigated; in Winter (December, January and February); in spring (March, April and May); Summer (June, July and August) and autumn (September, October and November). Each group was separately analysed and incidence of PPD obtained. The various incidences were compared with one another. The significance of any differences found in the incidence was tested using Chi-square test (with Yates correction) of 2 × 2 contingency table and Fisher's exact test for samples less than 40 and expected cell frequency less than 5. The result showed a seasonal variation in the incidence of PPD among the patients, the lowest occurring in summer and the highest occurring in winter. The result was consistent with the earlier observations in established epileptic patients.[2,3] The relative rarity of PPD in summer has been related to increased environmental sunshine during that season.[2,3] The inhibitory influence of sunshine on the incidence of PPD had been shown in experimental animals: Balzamo *et al* showed very low prevalence of PPD in Baboons (*Papio papio*) residing at the Eastern and Northeastern Savannah area of Senegal with abundant sunshine and a high prevalence of PPD in animals residing at the Southwest forest region with much rain and shaded environment.[4] Relative rarity of PPD has been shown in African patients with grand mal epilepsy compared with Caucasian patients with similar diagnosis, due probably to the relatively larger amount of sunshine in Africa compared with Europe.[5]

It has been suggested that PPD is a significant pointer to increased excitability of the cortical neurons.[6,7] The presence of PPD is therefore an expression of increased excitability of cortical neurons and the relative rarity of PPD among these patients in summer is a pointer to reduced excitability of their cortical neurons during that season, as a result of the increased environmental sunshine.

The mechanism for the inhibitory influence of sunshine on photoparoxysmal discharges, involves the influence of sunshine on the inhibitory neurotransmitters: adrenalin and dopamine. Details of the mechanism have been discussed in previous studies.[2,3,5]

This report suggests that the phenomenon of seasonal variability in photoparoxysmal discharges is not limited to epileptic patients alone but also occur in patients investigated after a single seizure. This reinforces the suggestion that this phenomenon is a reflection of seasonal variation in excitability of cortical neurons and probably bears no relationship to clinical expression of photosensitive epilepsy.

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Table *Seasonal variation in the incidence of photoparoxysmal discharges*

Season	*No of patients*	*No with PPD*	*Incidence (%)*
Winter	89	10	11·2
Spring	99	6	6·1
Summer	108	2	1·85
Autumn	112	8	7·3

The lowest incidence (1·85%) occurred in summer and the highest incidence (11·2%) occurred in winter. The difference in the two incidences was statistically significant ($\chi^2 = 5{\cdot}2$, $p < 0{\cdot}05$).

References

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Optic neuritis, confirmed by visual evoked response, and the risk for multiple sclerosis: a prospective survey.

Sir: Studies concerning the development of multiple sclerosis after uncomplicated optic neuritis reveal a great variability in percentages of that risk (8–78%). These great discrepancies are probably due to differences in the periods of follow up in those studies, (1–25 years).[1] This despite the fact that 83% of the patients studied by Nikoskelainen *et al*,[2] who developed multiple sclerosis, did this within a period of no more than 5 years.

Another factor which may influence the risk of multiple sclerosis after an uncomplicated optic neuritis is that of an incorrect diagnosis of the latter.[1,3] This indicates that previous studies on this matter, especially those before electrophysiology was introduced in neuro-ophthalmological diagnosis, may have involved other causes of visual loss than optic neuritis alone.

From the department of Electrophysiology (Eye Hospital, Rotterdam) 118 patients with sudden unilateral visual loss, seen from 1982–86, were reviewed. All patients underwent ophthalmological and