

Danesi replies:

I wish to react to the comments of Dr Trenite and colleagues on my paper on the phenomenon of seasonal variability in photosensitivity in epileptic patients. They gave impressive figures from their study and concluded that their data did not corroborate my findings.

Perhaps the misunderstanding arose from the fact that Dr Trenite *et al* and I have been studying different phenomena. They have been studying photosensitivity and its clinical implication. My study mainly investigated the influence of environmental sunshine on excitability of cortical neurons. Photoparoxysmal discharge (PPD) was studied as one of the indices of hyperexcitable neurons. This study was inspired by our earlier observation of relative rarity of PPD in Nigerian epileptic patients¹ and observation by Balzamo *et al* in *Papio papio*, that animals living in areas with abundant sunshine had relative rarity of PPD com-

pared with animals living in shaded environment.²

I had earlier looked at the EEG records of a large number of epileptic patients recorded from 1977 to 1984 at the National Hospital for Nervous diseases, London, the results of which were presented at the World Congress in 1985.³ Table 1 shows the distribution of photoparoxysmal discharges according to the season. The lower incidence of PPD in summer compared with winter was not a spurious one. It consistently occurred yearly as shown in table 2. The diagnosis of photosensitivity in my study was mainly a laboratory one since I was studying PPD (and spike and wave discharges) as indices of cortical excitability. The study showed that cortical excitability was reduced in summer compared with winter. The patients were not selected in any form and they were not studied on the basis of history of photosensitive epilepsy.

Dr Trenite *et al* made the observation that my hypothesis leads to the prediction that chronic exposure to high levels of ambient lighting may have adverse effects on people with photosensitive epilepsy. I do not agree with this observation. My hypothesis suggests that chronic exposure to high level of ambient sunlighting reduces excitability of cortical neurons and thereby susceptibility to photoparoxysmal discharges among epileptic patients. The relative rarity of PPD in Africans may be due to the high level of ambient sunlight in Africa. Recently, relative rarity of spike and wave discharges have also been demonstrated in African patients with grand mal epilepsy perhaps due to reduced cortical excitability.⁴ I have data (not yet published) which show that incidence of generalised spike and wave discharges

among epileptic patients was lower in summer compared to winter. I have not studied the clinical implication of this phenomenon of reduced cortical excitability associated with exposure to high level of sunlight. However, I certainly do not predict that chronic exposure to sunlighting would have adverse effects on people with photosensitive epilepsy. By reducing cortical excitability such exposure should have beneficial effects on the patients.

While I do not disagree with the data presented by Dr Trenite *et al*, I believe we are studying different phenomena.

References

- 1 Danesi MA, Oni K. Photosensitive epilepsy and photoconvulsive responses to photic stimulation in Africans. *Epilepsia* 1983;24:455-8.
- 2 Balzamo E, Best J, Menini CH, Naquet R. Excessive light sensitivity in papio, papio: variations with age, sex and geographical origin. *Epilepsia* 1975;16:269.
- 3 Danesi MA. Geographical and seasonal variations in the incidence of epileptic photosensitivity. *Electroencephalogr Clin Neurophysiol Abstr* 1985b;61:S216.
- 4 Danesi MA. Electroencephalographic manifestations of grand mal epilepsy in Africans: Observation of relative rarity of interictal abnormalities. *Epilepsia* 1988;29:446-50.

Table 1 Seasonal variation in the incidence of photoparoxysmal discharges among 4,569 epileptic patients.

Season of recording	Incidence of photoparoxysmal discharges		
	No of patients	No with PPD	Incidence (%)
Spring	1116	56	5.0
Summer	1273	35	2.8
Autumn	1160	66	5.7
Winter	1020	90	8.8

The lowest incidence of PPD occurred in summer and the highest incidence occurred in winter. The incidence in summer was significantly lower than the incidence in winter ($p < 0.001$).

Table 2 Summer-winter differences in the incidence of photoparoxysmal discharges yearly, over a period of 8 years.

Year tested	Tested in summer			Tested in winter			Significance of difference % Sum < wint
	No	PPD	%	No	PPD	%	
1977	157	2	1.3	126	7	5.6	$p < 0.01$
1978	179	6	3.4	128	8	6.3	N.S.
1979	175	7	4.1	132	13	9.8	$p < 0.05$
1980	122	4	3.3	139	14	10.2	$p < 0.01$
1981	128	3	2.3	123	13	10.6	$p < 0.01$
1982	172	3	3.5	114	11	9.6	$p < 0.05$
1983	192	4	2.1	134	12	9.0	$p < 0.01$
1984	148	3	2.0	124	12	9.6	$p < 0.01$
Total	1273	35	2.8	1020	90	8.8	$p < 0.01$

No = Number of patients.

PPD = Number of patients with PPD.

% = Incidence of PPD.

%Sum < wint = Incidence in summer is lower than winter.

The incidence of PPD was consistently lower in summer compared to winter.

Book reviews

Current Neurology Vol 8. Edited by Stanley H Appel. (Pp 360; £49.00.) Chicago: Year Book Medical. UK Distrib: Wolfe Medical, 1988.

The *Current Problems* series provides much valued original articles by experts aimed at lightening our heavy reading load. In this 8th neurological collection, the editor leans heavily on colleagues from Baylor College, Houston and from Henry Ford Hospital, Michigan. And, though many current USA big names are thereby not to be found here, the result is a most useful collection of contemporary essays. The emphasis throughout is to try to expound basic science work, to explain it and to educate the workaday clinician.

Recent advances in molecular biology have improved the diagnosis and detection