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 patients and observation by Balzamo et al in Papio papio, that animals living in areas with abundant sunshine had relative rarity of PPD compared with animals living in shaded environments.

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. 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 sunlight 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. 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 sun lighting 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 agree with the data presented by Dr Trenite et al, I believe we are studying different phenomena.

References
4 Danesi MA. Electroencephalographic manifestations of grand mal epilepsy in Africa: Observation of relative rarity of interictal abnormalities. Epilepsia 1985b;61:S216.

Book reviews


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