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 L. Excessive light sensitivity in papio, papio: its 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
	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 US 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

in chorionic material of the gene linkages for Duchenne dystrophy. Ten per cent of epilepsies have a defined genetic basis; the "epilepsy genes" are pleomorphic and outnumber their electroencephalographic phenotypes. We are instructed how to estimate the familial risks in various epilepsy syndromes. There is a helpful essay on the neuroimmunology of multiple sclerosis. MRI is discussed as a tool for both brain imaging and spectroscopic analysis of brain metabolism. Current reviews of Alzheimer's disease, cerebrovascular disease, infective peripheral neuropathies, including HIV and Lyme disease, myoclonus and inherited biotin-dependent diseases are provided.

The book blends useful if somewhat patchy clinical reviews with fundamental recent research. It is well produced and, on the whole, well written. Neurosciences have progressed rapidly in the last decade and the reviewer was filled with a sense of inadequacy in attempting to understand the details and the complexities of some modern techniques. A clinical neurologist seeking a concise refresher course, could fare worse than scrutinising these chapters. It is a book which if studied slowly and carefully, will more than repay the effort. But, it is not easy going.

JMS PEARCE

Recurrent and Chronic Psychoses (*British Medical Bulletin* Vol 43: No 3). Edited by TJ Crow. (Pp 766; £20.00.) Edinburgh: Churchill Livingstone. 1987.

The 17 chapters in this volume of the *British Medical Bulletin* have been assembled into the traditional pattern of medical texts, with sections on the history, diagnostic criteria, epidemiology, aetiology, clinical investigations, treatment and outcome of the psychoses supplemented by chapters pertaining to the social implications of these disorders.

The list of authors reads rather like a British who's-who in research into schizophrenia and affective disorders. The majority of the chapters are clearly written with the opinions of the authors openly expressed. It will be an invaluable source book for those who need authoritative statements to reproduce in postgraduate exams and for those whose research and clinical interests require them to keep abreast of recent developments.

The British Council and the committee who planned this volume are to be commended for producing such an informative, and

yet inexpensive text which should be on the shelves of all with an interest in mental disorders.

JANET TREASURE

Virus Infections and the Developing Nervous System. Edited by RT Johnson and G Lyon. (Pp 162; £35.00.) Dordrecht: MTP Press. 1988.

Twenty-nine authors contribute to this strange patchwork of a book. Dr Johnson's overview is very similar to the chapter of almost the same title in his marvellous single author book *Viral Infections of the Nervous System*. Reovirus, rarely a cause of disease in man, provides a useful model for studying invasion and neurotropism in mice. The molecular characterisation of the capsid protein, its gene and the receptor on the neuron, the interactions between which appear responsible for neurotropism, is well under way. JC virus, responsible for progressive multifocal leukoencephalopathy, appears, again in the mouse, to arrest the development of certain oligodendrocytes. This provides an attractive explanation for mechanisms of demyelination, as well as a model for its further exploration. Bovine leukaemia virus infection is offered as a suitable model for studying mechanisms of cell (lymphocyte) oncogenic transformation. Once again this is explored in gene molecular terms. The clinical and neuropathological features of HIV infection in children are described together with suggestions concerning its ability to produce immunodeficiency whilst remaining relatively latent. If the lymphocytic choriomeningitis (LCM) virus infects the perinatal mouse, virus replication ensues throughout a normal life span without damage to the host. In the adult, LCM infection usually causes illness and often death. This is a convenient model for examining the mechanisms allowing carrier status. Once again the T lymphocyte appears to play a vital role. Some apparently preliminary work of the susceptibility particularly of neuronal and muscle tissue to the murine leukaemia virus and of the feasibility of studying herpes simplex in foetal neuronal cultures is presented. A closely argued but short chapter suggests that the major envelope glycoprotein of the cytomegalovirus might provide a useful subunit vaccine against this important organism. An extensively referenced chapter on rubella virus, its structure and clinical effects follows. The biology of mumps and coronavirus infections are not forgotten in the final chapters.

There is no particular overall scheme or argument in this book. Each chapter stands on its own. Much of it is work in progress, using either in vitro or animal models. With the exception perhaps of Epstein-Barr virus and arboviruses most viruses of immediate clinical relevance are discussed. It is an expensive book for its size. Whilst it is of course work of this nature that will give us the tools for treating infections caused by these viruses, it is largely experimental virologists and well motivated (and funded) clinical virologists who will wish to buy this book.

RO ROBINSON

Facial Dyskinesias (*Advances in Neurology, Vol 49.*) Edited by Joseph Jankovic, Eduardo Tolosa. (Pp 560; \$121.50.) New York: Raven Press. 1988.

Five hundred odd pages on facial dyskinesia may seem a lot but there is no other book specifically on this subject. Such disorders are, of course, not rare, can be disabling and are almost always exquisitely embarrassing for the patient so their emergence as a subspecialty of movement disorders is justifiable. This volume starts with an excellent overview by Jankovic. This leads into a welcome review of the anatomy of the facial muscles, knowledge of which is crucial to the proper treatment of blepharospasm. The physiology of blinking is discussed and there is also an interesting chapter on ophthalmological causes of blepharospasm which will be welcome to neurologists; both emphasise the probable importance of the afferent system, otherwise easily and wrongly placed in a subservient position when discussing the movement disorders. There are several good chapters on blepharospasm, Meige's syndrome and lid apraxia with a welcome article from the patient's perspective. Hemifacial spasm, facial synkinesis, bruxism, head and voice tremors, epileptic facial movements and torticollis are all well and interestingly discussed. There is a delightful chapter by Lees on facial mannerisms and tics. There are reviews of the chronic known effects of neuroleptic treatment and discussions on dopaminergic and cholinergic aspects of dystonia. Treatment with botulinus toxin is a feature of several chapters and there is also a chapter on treating blepharospasm by myectomy. There is relatively little on possible aetiologies of these conditions though there is an interesting contribution by Jankovic