Letters

oidal bones as well as irregular bone destruction superiorly in the vault. There was also considerable encroachment on the left hemisphere with oedema of the white matter, compression of the left lateral ventricle and displacement of the ventricles to the right. After contrast an enhancing mass was demonstrated in the area of dysplastic bone (fig). At surgery a grey gelatinous tumour was found beneath the left parietal bone compressing the cerebral cortex, which was shown histologically to be a meningothelial meningioma infiltrating bone. There were no features of a sarcoma.

The association between fibrous dysplasia and meningioma does not seem to be recognised and this may be the first report. The direct juxtaposition of the dysplastic bone and the tumour make it most likely that the link is causal. Neither previous craniectomy nor acrylic prostheses are known to be associated with meningioma and the patient had never been treated with radiotherapy. Although bone histology was not available in this case, the radiological appearances, the normal serum biochemistry and the age of

onset in late childhood make fibrous dysplasia the only reasonable diagnosis. The bony changes could not be explained on the basis of the known hyperostosis which is associated with meningiomas both because many cranial bones were involved radiologically which were distant from the tumour and secondly no meningioma was present at his original craniectomy 22 years previously.

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References

- 1 Lello GE, Sparrow DC. Craniofacial polyostotic fibrous dysplasia. *J Maxillofac Surg* 1985;13:267-72.
- 2 Albright F, Butler AM, Hampton AO, Smith PH. Syndrome characterised by fibrosa disseminata, areas of pigmentation and endocrine dysfunction, with precocious puberty in females: report of five cases. *N Engl J Med* 1937;216:727-46.
- 3 Edgerton MT, Persing JA, Jane JA. The surgical treatment of fibrous dysplasia. *Ann Surg* 1985;202:459-79.
- 4 Dabska M, Buraczewski J. On malignant transformation in fibrous dysplasia of bone. *Oncology* 1972;26:369-83.
- 5 Halawa M, Aziz AA. Chondrosarcoma in fibrous dysplasia of pelvis. A case report and review of the literature. *J Bone Joint Surg [B]* 1984;66-B:760-4.

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Matters arising

Seasonal variations in the incidence of photoparoxysmal response

Sir: The phenomenon of seasonal variability in photosensitivity of epileptic patients, reported by Danesi¹ is at variance with the findings from two other British centres² and is not corroborated by our data from the Netherlands.

Since 1980 we have conducted a prospective study of photosensitivity and its clinical implications in 100 photosensitive patients (Kasteleijn-Nolst Trenité, thesis in preparation) and no such consistent seasonal variability has been found. Table 1 lists the

Table 1 *Relation between the incidence of newly identified photosensitive patients and the number of new EEG referrals per season*

Season	1980-1983	
	Newly identified photosensitive new EEG patients	Total number of new EEG referrals
Spring (Mar, Apr, May)	20	586
Summer (Jun, Jul, Aug)	20	492
Autumn (Sep, Oct, Nov)	37	609
Winter (Dec, Jan, Feb)	23	655
Total	100	2342

number of newly identified photosensitive patients against the total number of new EEG referrals for EEG investigation of epilepsy per season. Apart from a slight autumnal excess in incidence of photosensitivity ($p < 0.1$, chi-square), there is no seasonal difference. Moreover, unlike Danesi, we quantified the degree of sensitivity by measurement of the photosensitivity range.³ From Danesi's model one might expect not only the prevalence but also the degree of photosensitivity to be greater during winter time, yet when our 100 patients are divided into two equal groups according to their photosensitivity ranges at the time of the first examination, no such seasonal effect is present (table 2).

Dr Danesi's hypothesis of increased photosensitivity during the winter season is thus not supported by the findings of three other centres. On the contrary, in our

material a weak seasonal effect appears to result from patients presenting with visually-induced seizures during the summer who then attend for EEG investigation in the autumn. This issue is not of purely academic interest as Dr Danesi's hypothesis leads to the prediction that chronic exposure to high levels of ambient lighting may have adverse effects on people with photosensitive epilepsy.

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Table 2 *Relation between degree of photosensitivity and season in 100 photosensitive patients*

Season	Photosensitivity range		
	++	+	Total
Spring	10	10	20
Summer	9	11	20
Autumn	20	17	37
Winter	11	12	23
Total	50	50	100

References

- 1 Danesi MA. Seasonal variations in the incidence of paroxysmal response to stimulation among photosensitive epileptic patients. *J Neurol Neurosurg Psychiatry* 1988;51:875-7.
- 2 Scott DF, Thurlong PF, Moffat AM, Harding GFA. Is sunshine protective in photosensitive epilepsy? *Electroencephalogr Clin Neurophysiol* 1985;61:S216-7.
- 3 Jeavons PM, Harding GFA. *Photosensitive Epilepsy*. Heinemann, London, 1975:21 pp.

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