drugs as described by Okada *et al*² may have altered the pharmacodynamics of carbamazepine and explained the unusual nature of the dyskinetic movements.

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Accepted 27 May 1988

Autonomic neuropathy in systemic lupus erythematosus

Sir: In describing a case of acute autonomic neuropathy in association with systemic lupus erythematosus (SLE), Hoyle *et al* remarked that autonomic function had yet to be assessed in any series of patients with this disease. In as much as this remains the position regarding the English language literature, we now report our findings in 14 such cases.

The patients were participants in a wider study of autonomic function in Raynaud's phenomenon (RP), a detailed account of which is in preparation. Five had RP,² while the remaining nine were randomly selected from our SLE clinic population to serve as RP case controls. When enrolled, none displayed symptoms typical of autonomic insufficiency.³

Each patient fulfilled established criteria for the diagnosis of SLE.⁴ All were black females, their ages ranging from 21 to 58 (median 34) years. None had diabetes, renal failure or clinical signs of cardiovascular disease. All were non smokers and denied alcohol consumption. In no case was medication being taken that reputedly affects adversely peripheral autonomic or somatic nerve function.^{3,5,6} Specifically, 12 patients were

Accepted 24 December 1987

Sir: Two patients developed unusual idiosyncratic reactions to carbamazepine. *H.T. aged 59 years* had a past history of alcohol abuse and chronic pancreatitis. His medication included atenolol 100 mg/daily, isosorbide dintrate 10 mg/t.d.s. and Gaviscon (a combination of alginate acid, magnesium trisilicate, aluminium hydroxide and sodium bicarbonate). He developed blackouts associated with vomiting and was prescribed carbamazepine 200 mg/b.d. After receiving carbamazepine for 2 days he slept for 16 hours and on recovery exhibited bizarre, uncontrollable movements of both hands and arms, throwing any objects he grasped. The movements lasted 12 hours and no further carbamazepine was given.

J.T. aged 15 years had episodes of loss of consciousness which were later shown to be psychologically determined. On valproate he developed hypersomnia and was changed to carbamazepine 200 t.d.s.. He developed a rash and the dose was reduced to 100 mg/t.d.s. The rash cleared and he remained well for 5 months before complaining of tiredness and dizziness. A month later he started to walk slowly with small, shuffling, stiff limbed steps. His whole body was rigid and on examination he had repeated attacks of shivering involving most of his body. He recovered completely within a week of stopping his medication.

The shivering dystonia exhibited by J.T. is similar to the dose dependent dystonic movements induced by carbamazepine.¹ Ephemeral hemiballismic movements have not been reported with carbamazepine. Theoretically, interaction with atenolol or isosorbide could have raised the serum concentration of the carbamazepine; alternatively interaction with metal-containing

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