Letters

Carbamazepine-induced choreoathetoid dyskinesias

Sir: Choreoathetoid and choreiform dyskinesias have been attributed to antiepileptic drug intoxication, especially with phenytoin.¹⁻⁶ Isolated cases of choreoathetoid movements also have been observed during intoxication with other anticonvulsants, including ethosuximide, methsuximide,⁷⁻¹¹ keto-carbamazepine,¹² phenobarbitone,^{13 14} primidone,¹⁵ and sulthiame.² The dyskinesias caused by simultaneous administration of phenytoin and sulthiame or succinimides may have been provoked by an increase in the serum level of phenytoin. Very few cases of involuntary movements after administration of carbamazepine have been reported so far. One patient of Lefevre and Gablain¹⁶ developed dyskinesias similar to a "flapping tremor" as a symptom of carbamazepine intoxication. Wendland¹⁷ described three patients with myoclonic jerks in connection with carbamazepine intake. A patient of Gruska *et al*¹⁸ taking 20 g of carbamazepine in a suicidal attempt developed "incoordinate ballistic or cramp-like movements of all extremities". Several authors¹⁹⁻²¹ have reported dystonia in patients treated with carbamazepine. Troupin *et al*²² also observed a dyskinetic eye movement disturbance as a symptom of carbamazepine intoxication.

A 71-year-old female patient, without any family history of involuntary movements, was referred because of choreoathetoid dyskinesias. The patient developed generalised epileptic seizures of unknown aetiology (pneumencephalography was normal) at the age of 53 years, and subsequently was treated with 250 mg of primidone daily. Because of the development of partial seizures with elementary symptomatology, additional anticonvulsant therapy with carbamazepine was started four years ago. The daily dose was increased to 800 mg of carbamazepine over four days. Her husband reported the sudden onset of distal choreoathetoid dyskinesias of the upper and lower limbs on the third day of administration of carbamazepine, which also caused an impairment of gait and a confused state of mind with visual and acoustic hallucinations. On neurological examination, there were intermittent distal choreoathetoid dyskinesias of the extremities. The speech was

slurred and the gait ataxic, but nystagmus was not present. The EEG was characterised by a 8-9/s background activity, interspersed with theta and 2-3/s delta waves. Epileptic discharges and focal activity were not observed. Serum levels of the antiepileptic drugs (method: Emit Immunoassay) were: carbamazepine 80.4 µmol/l (19 µg/ml), phenobarbitone 61.6 µmol/l (14.3 µg/ml), and primidone 17 µmol/l (3.7 µg/ml). The carbamazepine value was confirmed by high pressure liquid chromatography. The dose of carbamazepine was gradually reduced and completely withdrawn within 7 days, and the choreoathetoid dyskinesias disappeared. Two months later the patient was re-examined and showed no involuntary movements or psychosis.

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Post-traumatic tremor due to vascular injury and its treatment by stereotactic thalamotomy

Sir: We have elsewhere given an account of the development of "peduncular" tremor in patients surviving severe head injuries, and its responses to stereotactic thalamotomy.¹ The clinical and radiological evidence in those cases pointed to a midbrain lesion, probably at the level of the superior cerebellar peduncle, as the cause of the disabling postural and action tremor usually of the upper limb. We have recently had the opportunity to study and treat a patient in whom tremor developed