facilitate a more in-depth discussion than the modest contributions of Geoffrey Jones and myself have permitted.

BUCHANAN, A. (1994) Brain, mind and behaviour revisited. *The Journal of Forensic Psychiatry*, **5**, 232–236.

DEPARTMENT OF HEALTH AND HOME OFFICE (1994) *Report of the Department of Health and Home Office Working Group on Psychopathic Disorder*. London.

H. PRINS

1 Home Close Road
Houghton on the Hill
Leicester LE7 9GT

OGS or tardive dystonia?

SIR: Tan *et al* (*BJP*, September 1994, **165**, 381–383) suggest that the delayed onset oculogyric spasm (OGS) observed in their study may be a form of tardive dystonia although there was no associated tardive dyskinesia. This appears unconvincing.

Burke & Kang (1988) note that the dystonic movements of acute and tardive dystonia are 'indistinguishable, with the exception that oculogyric crisis occurs in acute dystonia but not tardive dystonia'. Very few cases of OGS which may be regarded as a form of tardive dystonia have indeed been reported (Fitzgerald & Jankovic, 1989); in these, having developed after prolonged exposure to neuroleptics, and associated with other tardive movement disorders, the chronic and disabling OGS did not respond readily to anticholinergic agents, and persisted for months to years after neuroleptics were stopped.

Another rare form of delayed onset OGS has been described (Sachdev & Tang, 1992; Thornton & McKenna, 1994). The abrupt appearance of OGS along with other dystonic movements was accompanied by psychotic symptoms, catatonic phenomena and autonomic disturbances, reminiscent of post-encephalitic crises. This 'complex' acute dystonic reaction responded to intravenous procyclidine, but an oral anticholinergic agent was ineffective; and recurrences tended to persist for months after the termination of neuroleptic or switching to clozapine.

The OGS attacks observed in Tan *et al*'s study were of acute onset, and in all 34 cases promptly reversible with the use of oral benzhexol or intramuscular promethazine. There was no associated tardive dyskinesia. They showed no features suggestive of a form of tardive dystonia or the complex variant of acute dystonic reaction. They were no different from the usual form of acute dystonic reaction, apart from their late occurrence; all the 34 patients had received neuroleptics for more than five months.

Although acute dystonic reactions usually occur soon after a neuroleptic is started, they may develop in the course of therapy following sudden increases in dosage (Lees, 1985). Some patients who are receiving depot neuroleptics have recurrent acute dystonia a few hours after each injection. It is noted that eight of the 16 patients with recurrent OGS in Tan *et al*'s study were on fluphenazine decanoate, a depot neuroleptic prone to induce acute dystonia. Other possible causes include the use of neuroleptic as required (p.r.n.) on top of maintenance medication for mental deterioration or disturbing behaviour; and poor drug compliance – the patient restarting treatment after a period of self-prescribed abstinence.

The delayed onset of OGS Tan *et al* observed may well be related to these 'simple' causes, which were not excluded in their study.

BURKE, R. E. & KANG, U. J. (1988) Tardive dystonia: clinical aspects and treatment. In *Facial Dyskinesia. Advances in Neurology*, Vol. 49 (eds J. Jankovic & E. Tolosa), pp. 199–210. New York: Raven Press.

FITZGERALD, P. M. & JANKOVIC, J. (1989) Tardive oculogyric crises. *Neurology*, **39**, 1434–1437.

LEES, A. J. (1985) The drug-induced dyskinesias. In *Tics and Related Disorders*. pp. 172–234. Edinburgh: Churchill Livingstone.

SACHDEV, P. & TANG, W. M. (1992) Psychotic symptoms preceding ocular deviation in a patient with tardive oculogyric crises. *Australian and New Zealand Journal of Psychiatry*, **26**, 666–670.

THORNTON, A. & MCKENNA, P. J. (1994) Acute dystonic reactions complicated by psychotic phenomena. *British Journal of Psychiatry*, **164**, 115–118.

W. Y. LEE

Tokanui Hospital
Private Bag, Te Awamutu
New Zealand

Huntington's disease in the Oxford region

SIR: Shiwach (*BJP*, September 1994, **165**, 414–415 (letter)) criticises the estimate of the prevalence of Huntington's chorea in the Oxford Health Region given by Watt & Seller (1993) on the grounds that it did not include patients recorded by the Oxford Record Linkage Study, but not referred to the Oxford Department of Medical Genetics; and it used for the population of the Oxford Health Region a prevalence in 2.52 million instead of the figure of 2.437 million used by Shiwach.

Patients recorded by the Oxford Record Linkage Study must have been diagnosed as Huntington's chorea and hospitalised in an Oxford regional hospital. Patients in a regional hospital sometimes