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

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## 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.

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## 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

originate from outside the region. This is notably the case for patients with Huntington's chorea, particularly before the initiation of the pre-symptomatic test service in 1986 diminished the customary tendency to concealment of the disease.

Permanent and terminal care was most frequently provided by mental hospitals. Because of the difficulty of nursing and disturbance to other patients, private nursing homes rarely admitted affected patients; except homes run by charities who specialised in Huntington's chorea and necessarily took patients from a wide area. Patients here were therefore frequently outside the health region of their homes from which they were distant, as they were from relatives. Those patients in this situation in the Oxford health region would be notified to Oxford Record Linkage on any admission to an Oxford regional hospital. The surmise that these Huntington patients form a group domiciled outside the Oxford Region who had little or no contact with a responsible family is strongly supported by Shiwach's discovering their paucity of relatives (Shiwach, 1992). Of such patients only those whose domicile can be confirmed to be within the Oxford Region should be included.

Incidentally Shiwach's statement that Watt & Seller's study (1993) "relied entirely on the (Medical genetics) Department's case-notes for the Huntington Disease patients" is not correct. It is true that all the affected patients were referred to the Medical Genetics Department but many other sources of information (e.g. mental hospitals, general hospitals, nursing homes, neurologists, psychiatrists, general practitioners, social workers, other genetic departments et al) were contacted, checked and used as sources of information to eliminate or include subjects.

In his criticism of Watt & Seller's (1993) population figure Shiwach has overlooked the fact that the prevalence he reported was for 1985 whereas Watt & Seller's was for 1988 for which the figure estimated by the Office of Population and Census Services (1994) is 2.512 million.

The two independently calculated prevalence figures of Huntington's chorea for an area where previous estimates vary from 25.0 to 99.5 per million are, three years apart, sufficiently close to support the view that they approach an ideally true figure and are reliable.

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## Anaesthetic technique in the practice of ECT

SIR: Methohexitone shortens seizure duration compared with electroconvulsive therapy (ECT) given without anaesthesia (Ayd, 1961), yet is an ultra-short-acting intravenous anaesthetic drug, the plasma concentration falling rapidly because of its redistribution into body tissues other than brain (Corssen et al, 1988). Recovery of consciousness after a single bolus injection is prompt, suggesting that the concentration of methohexitone in the brain also falls rapidly after injection. If the seizure-shortening property of methohexitone is related to its concentration in the brain and this concentration is falling rapidly after induction, then the time between its intravenous injection and electrical stimulation may influence the length of cerebral seizure activity.

Twenty-two patients (average age 57 years) took part in the study to test the hypothesis that prolongation of the time between induction and electrical stimulation leads to longer cerebral seizure activity. All patients received bilateral ECT for the treatment of a primary depressive illness and all received concomitant psychotropic drug treatment that was standard. Electrical stimulation was given with a prototype of the Ectron Series 5A ECT machine at a dose 75 millicoulombs above the seizure threshold, which had been established empirically at the start of the ECT course. The time from the start of the injection of methohexitone to the start of electrical stimulation was taken as the delay and ventilation was maintained by an anaesthetic circuit that would not reduce alveolar $pCO_2$. The convulsion was timed from the end of electrical stimulation using the cuff technique (Addersley & Hamilton, 1953).

The index treatment was usually the sixth and the average delay was 110 (range 68–176) seconds; at the subsequent treatment this was increased by approximately one minute to 173 seconds. The average length of convulsion at the index treatment was 25.8 seconds, and was 33.2 seconds at the subsequent treatment. The average increase in