

### **Patient consent to case note perusal during annual visit by the Inspectorate of Mental Hospitals.**

Sir, –

It is now standard practice during the annual visits by the Inspectorate of Mental Hospitals for patients' case notes to be perused by the visiting team. This is to insure that adequate standards of patient care are maintained. Case note perusal is an accepted peer review procedure in many countries and is used by such professional bodies as the Royal College of Psychiatrists during Approval Visits. However, the issue of patient consent to this procedure has rarely been commented on.

In the case of the Inspectorate perusals the problems is more complicated as the members of this team are functioning as agents of the state, albeit for the best of reasons and

under the legal cover of the 1945 Mental Treatment Act. Nonetheless, it would hardly be acceptable to the Irish public if they were to realise that other people and agents of the state at that, had automatic access to their psychiatric records. I imagine an outcry would follow if the full implications of this situation were to sink in. I suggest therefore that during all such perusals the patient's consent should be obtained. This could be done by either the visiting Inspectorate member or by the accompanying consultant of the hospital or service being inspected. I imagine most patients would readily give consent. They would also be impressed at the high value so placed by the Department of Health on patient confidentiality.

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### **Clonazepam in tardive dyskinesia**

Sir, –

Clonazepam has been reported to reduce tardive dyskinesia (1–3). The drug is believed to mimic the action of gamma-amino-butyric acid which is an inhibitory transmitter in the CNS. This explains the effect on abnormal movements and the drowsiness which is a common initial side-effect. An audit of the use of clonazepam in tardive dyskinesia is described.

Nine patients were identified. There were six females and three males. Ages ranged from 28 – 72 years (mean 59.44, SD 12.56). Diagnosis were manic-depressive illness: three patients; schizophrenia: two patients (one had received a leucotomy in the past); mental handicap, brain injury following road accident, cerebrovascular accident and tertiary syphilis: one case each. Three were outpatients.

Overall, six patients lost their dyskinesia on clonazepam and one improved on a combination of clonazepam and tetrabenazine. The latter remained improved when sulpiride was prescribed following a return of her paranoid symptoms. Dyskinesia had been present for less than a year in three cases, of whom two recovered. There was no evidence of differences by sex or age in this sample.

All patients had received neuroleptics in the past, over a year ago in three cases. One patient (with manic-depressive illness) had received procyclidine in the past. She continued to receive a neuroleptic (sulpiride). Her dyskinesia responded well to clonazepam.

All the dyskinesias had been clinically stable for varying periods prior to receiving clonazepam. Additional time free from neuroleptic drugs might account for the improvement seen. However, two of the three patients who had already been free of neuroleptics for more than one year improved, as did five of the six who had received neuroleptics more recently. This difference was not significant (Fisher exact probability test).

Bobruff et al. (1981) reported greater improvement in orofacial dyskinesias. In this study, of the six cases with mainly orofacial dyskinesia, five recovered completely after a short course of clonazepam (four months on 1 mg. daily) and remained well after restarting neuroleptics (fluphenazine). Orofacial and other dyskinesias did not differ significantly in outcome (Fisher exact probability test).

In terms of diagnosis, two patients (tertiary syphilis; cerebrovascular accident) showed no improvement on clonazepam.

This might indicate a worse prognosis for those with tardive dyskinesia and brain lesions, but the first later improved on tetrabenazine, while three other patients with probable organic brain lesions (congenital mental handicap, brain injury following road accident and schizophrenia followed leucotomy) did well on clonazepam alone.

The dose of clonazepam ranged from 1 mg. to 4.5 mg. daily, the maximum recommended by Thaker et al (1990), over periods ranging from three months to two years. The drug had been withdrawn in four cases: two were recovered, one had improved and one had not improved. Mean duration of treatment to withdrawal was 77.43 weeks in the recovered/improved group and 46 weeks in the unimproved group. One patient with a cerebrovascular accident had only limited benefit from clonazepam but tetrabenazine produced unacceptable oversedation.

Thaker et al. (1983) found a 37.1% decrease in dyskinesia scores against placebo in their 19 cases treated with clonazepam. Most of their patients continued to receive neuroleptics throughout the study, as did some of those reported by Bobruff et al (1981) of whom six out of ten showed at least 50% improvement in dyskinesia scores. The clinical assessment reported here shows relief of dyskinesia or significant improvement with the help of clonazepam in seven out of nine patients, one of whom was still receiving a neuroleptic. This supports the case for further study of clonazepam in the management of tardive dyskinesia.

A suitable research design would include detailed drug histories (including anti-Parkinsonian agents); a standard rating of dyskinesia before, during and after treatment with clonazepam; and no neuroleptic administration during the study. A double-blind trial against placebo or tetrabenazine might well be informative.

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#### **References**

1. Bobruff A, Gardos G, Tarsy D, Rapkin RM, Cole JO, and Moore P. Clonazepam and phenobarbital in tardive dyskinesia. *Am J Psychiatry* 1981; 138: 189-93.
2. Chouinard G, Young SN, Annabie L. Antimanic effect of clonazepam. *Biol Psychiatry* 1983; 18: 451-66.
3. Thaker GK, Nguyen JA, Strauss ME, Jacobson R, Kaup BA, and Tamminga CA. Clonazepam treatment of tardive dyskinesia: a practical GABA-mimetic strategy. *Am J Psychiatry* 1990; 147: 445-51.

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**Reference** 1. Dean J.C., Penny J.K. *Epilepsia* 1988; 29 (2): 140-144.

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