

tion of such internalised anger in his essay *Mourning and Melancholia* (1925) fits this piece well. Walter, in the book of that name by David Cook (Penguin, 1980), is a boy with mental handicap. Shakespeare's plays contain characters with psychological or psychiatric issues: Macbeth's guilt, Lear's madness. Hamlet's introspection and perhaps depression are some better known examples. Further discussion of madness in the theatre can be found in Davis (1992).

A list such as this can be no more than idiosyncratic, but might provide an introduction to those wishing to explore the field of madness in literature. Other suggestions from readers would be welcomed.

DAVIS, D. R. (1992) *Scenes of Madness - A Psychiatrist at the Theatre*. London: Routledge.

FREUD, S. (1925) Mourning and melancholia. In *Collected Works*, Vol 14, pp. 239-258. London: Hogarth Press.

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'Taking Liberties'

SIR: I am prompted to write this letter after watching the recent BBC2 documentary, *Taking Liberties* (7 May, 1992). The programme argued a case for death on psychiatric wards being associated with violent and difficult behaviour and poor quality of care. This association with death may be so, but at best the evidence is tenuous (Shah, 1992).

It was a pity that the programme did not concentrate on the more general issue of the established relationship between violent and difficult behaviour, attempted suicides and suicides on psychiatric wards, and staffing levels, agency nursing staff, staff attitudes, under-involvement of medical staff and overcrowding (James *et al*, 1990; Shah *et al*, 1991). Intervention at these levels offers opportunity for primary and secondary prevention, which may ultimately not only reduce behaviour disturbance but improve staff and patient morale and quality of care.

The programme appeared to portray that large Victorian hospitals are the mainstay of treatment. This is no longer the case with the closure of large hospitals and implementation of the community-care policies. However, it has been shown that levels of violence may be increasing in newer psychiatric units in district general hospitals (James *et al*, 1990). Thus, we should concentrate on all types of psychiatric units.

JAMES, D. V., FINEBERG, N. A., SHAH, A. K., *et al* (1990) An increase in violence on an acute psychiatric ward: a study of associated factors. *British Journal of Psychiatry*, 156, 846-852.

SHAH, A. K. (1992) Violence, death and associated factors on a mental handicap ward. *Journal of Intellectual Disability Research* (in press).

—, JAMES, D. V. & FINEBERG, N. A. (1991) Violence among psychiatric in-patients. *Acta Psychiatrica Scandinavica*, 84, 304-309.

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Neuroleptic-induced dislocation of the jaw

SIR: We wish to report a case of neuroleptic-induced orofacial dystonia complicated by dislocation of the jaw, to remind clinicians of this uncommon yet serious occurrence.

Case report. A previously well, unmarried, 21-year-old waiter, of Greek origin, was admitted to hospital with recent onset of bizarre behaviour, elevated mood and formal thought disorder. Over the following four weeks his psychosis was treated with haloperidol to a maximum dose of 30 mg daily and adjunctive use of clonazepam, initially at a dose of 2 mg b.d. Throughout this time he experienced intermittent orofacial dystonia, cogwheel rigidity and festinant gait which responded to benzotropine, 2 mg per day. The anticholinergic medication was withdrawn over a period of two weeks and he was discharged on haloperidol, 7.5 mg at night.

He was readmitted a week later with recurrent symptoms associated with medication non-compliance and cannabis abuse. He received haloperidol, 20 mg in divided doses, and clonazepam, 2 mg during the initial 12 hours but became more agitated and disturbed and received haloperidol, 20 mg, and clonazepam, 2 mg, in the subsequent six-hour period. He developed an orofacial dystonic reaction 18 hours after reintroduction of haloperidol. Bzotropine, 2 mg intramuscularly, was administered six-hourly without effect. He was noted to have facial asymmetry, difficulty swallowing, salivary drooling and was unable to close his mouth. A dislocated jaw was suspected clinically and an anterior dislocation of the right temporomandibular joint was confirmed radiologically. There was no evidence to suggest traumatic dislocation. Haloperidol was discontinued and his jaw was successfully relocated under sedation. This was followed by a diminution of his agitation without evidence of orofacial dystonia.

His psychosis gradually responded to thiothixene, a high potency antipsychotic agent from a different pharmacological class, without recurrence of dystonia. A diagnosis of schizophreniform disorder was reached and he was discharged to his family home five weeks later.

Dystonic reactions develop in up to 2.5% of patients treated with antipsychotic agents within 48 hours of their commencement (Rupniak, 1986). The majority are either self-limiting or resolve with anticholinergic drug administration and are not

complicated by serious morbidity. Facial distortion, which is a frequent component of dystonic reactions, can be distressing to patients and may add to their level of arousal.

Neuroleptic-induced dystonic reactions are more common in young men (Ayd, 1961; Swett, 1975) and may occur more frequently in patients with a family history of drug-induced dystonic reaction (Rupniak, 1986). They are less likely to occur with neuroleptic agents with high intrinsic anticholinergic activity (Swett, 1975), and this group of agents should be considered for acute and maintenance therapy in vulnerable patients.

AYD, F. J. (1961) A survey of drug-induced extra pyramidal reactions. *Journal of the American Medical Association*, **175**, 1054–1060.

RUPNIAK, N. M. J., JENNER, P. & MARSDEN, C. D. (1986) Acute dystonia induced by neuroleptic drugs. *Psychopharmacology*, **88**, 403–419.

SWETT, C. (1975) Drug-induced dystonia. *American Journal of Psychiatry*, **132**, 532–534.

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Organic mood syndrome in two siblings with Wolfram syndrome

STR: We would like to support the notion of Swift *et al* (1990, 1991) that being a homozygous carrier as well as a heterozygous carrier of the Wolfram syndrome gene predisposes to psychiatric illness. We have observed DSM–III–R organic mood syndrome associated with Wolfram syndrome in two male siblings and psychiatric illness in their pedigree.

Wolfram syndrome is an autosomal recessive neurodegenerative syndrome defined by diabetes mellitus and progressive optic atrophy, first described by Wolfram in 1938. Diabetes insipidus and deafness as well as an atonic bladder and diverse neurological abnormalities are also frequently observed. Increased incidence of psychiatric illness in this syndrome was reported by Swift *et al* (1990). Their conclusion was that the Wolfram syndrome gene predisposes homozygotes to psychiatric illness. A later report by the same group claimed that heterozygous carriers of the gene for the Wolfram syndrome are also predisposed to significant psychiatric illness (Swift *et al*, 1991). They found a larger proportion of psychiatric illness among blood relatives

of those with Wolfram syndrome than among the spouses.

Case 1. A 23-year-old man, the eldest of four brothers, was first admitted to the psychiatric ward at the age of 16 because of manic–depressive episodes. Since then, he has been admitted four times to the same ward. He is now an out-patient at the same hospital.

Case 2. A 22-year-old man, who is the second of four siblings, was examined at the out-patient clinic at the same hospital, because of a manic episode, at the age of 16. He has remained an out-patient since then.

Their psychiatric features, which met DSM–III–R criteria for organic mood syndrome, mixed type, were similar, although those of Case 1 were more severe. In addition, Case 1 sometimes showed delirious state with ideas of reference and persecution during manic episodes. Their episodes were well controlled by carbamazepine and/or a tricyclic antidepressant.

They were first diagnosed as Wolfram syndrome at the age of 16 and 15 respectively, by which time both were totally blind because of their progressive optic atrophy. They also had a sensory deafness and diabetes insipidus, although insulin-dependent diabetes mellitus was diagnosed at the age of 3 and at the age of 1.5 for case 1 and 2 respectively. Psychiatric episodes of the patients did not seem to correlate with exacerbation of physical symptoms.

There are consanguinities in this pedigree, the probands' parents being second cousins, and the probands' paternal and maternal grandparents first cousins. In this pedigree, three members suffer from psychiatric illness – schizophrenia, bipolar affective disorder, and schizoaffective disorder with diabetes insipidus.

SWIFT, R. G., SADLER, D. B. & SWIFT, M. (1990) Psychiatric findings in Wolfram syndrome homozygotes. *Lancet*, **336**, 667–669.

—, PERKINS, D. O., CHASE, C. L., *et al* (1991) Psychiatric disorders in 36 families with Wolfram syndrome. *American Journal of Psychiatry*, **148**, 775–779.

WOLFRAM, D. J. (1938) Diabetes mellitus and optic atrophy among siblings: report of 4 cases. *Mayo Clinic Proceedings*, **13**, 715–718.

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