position for which no evidence is given, this research, however well intended, may be stigmatising in itself.

Cooksey, E. C. & Brown, P. (1998) Spinning on its axes: DSM and the social construction of psychiatric diagnosis. *International Journal of Health Services*, **28**, 525–554.

Crisp, A. H., Gelder, M. G., Rix, S., *et al* (2000) Stigmatisation of people with mental illnesses. *British Journal of Psychiatry*, **177**, 4–7.

Foucault, M. (1967) *Madness and Civilisation: A History of Insanity in the Age of Reason*. London: Tavistock.

Levine, H. G. (1979) The discovery of addiction: changing conceptions of habitual drunkenness in America. *Journal of Studies on Alcohol*, **15**, 493–506.

McMurran, M. (1994) *The Psychology of Addiction*. London: Taylor and Francis.

D. Heim Department of Psychology, The University of Strathclyde, Graham Hills Building, 40 George Street, Glasgow G1 1QE

Olanzapine and pancreatitis

Two patients with chronic schizophrenia were admitted with acute pancreatitis. Olanzapine was used by both patients and may represent a further example of drug-induced pancreatitis. Olanzapine is a thienobenzodiazepine similar in structure to clozapine; clozapine has been associated with the induction of acute pancreatitis (Frankenburg & Kando, 1992; Gatto *et al*, 1998).

A 34-year-old man with chronic schizophrenia was admitted with abdominal pain. Two years previously he had had acute pancreatitis, but continued to drink alcohol (8 units/day). The patient was taking 20 mg olanzapine daily. Glasgow criteria and computed tomography classified the episode as severe pancreatitis. The patient developed respiratory failure and required artificial ventilation for 11 days. A full recovery was made.

A 29-year-old man with chronic schizophrenia was admitted with abdominal pain. He had a past history of acute pancreatitis, having had an attack 8 years previously. The patient drank at least 15 units of alcohol per week. Regular medication was sulpiride and olanzapine 20 mg daily. Diagnosis was made by computed tomography. The patient made an uncomplicated recovery. The same patient was readmitted 4 months later with another attack of acute pancreatitis – he had continued to take olanzapine and to drink alcohol.

Although both patients were regular consumers of alcohol, a known risk factor for the development of acute pancreatitis, we felt it unusual that two patients on olanzapine should present within such a short period of time. Clozapine has been associated with pancreatitis and has a similar structure to olanzapine; one might therefore suspect that olanzapine might cause pancreatitis. The evidence for olanzapine inducing pancreatitis is strengthened by the fact that in one of the patients a further episode occurred after rechallenge with the drug. It is possible that olanzapine acts in a synergistic fashion with alcohol in the pathogenesis of the disease. We propose that olanzapine should be used with caution with patients who drink alcohol on a regular basis or who have a previous history of pancreatitis.

Frankenburg, F. & Kando, J. (1992) Eosinophilia, clozapine and pancreatitis. *Lancet*, **340**, 251.

Gatto, E., Castronuovo, A. & Uribe Roca, M. (1998) Clozapine and pancreatitis. *Clinical Neuropharmacology*, **21**, 203.

R. Hagger, C. Brown, P. Hurley Mayday University Hospital, London Road, Croydon, Surrey CR7 7YE

Antidepressant augmentation with low-dose olanzapine in obsessive–compulsive disorder

We read with interest the article on obsessive–compulsive disorder (OCD) and delusions, by O'Dwyer & Mark (2000). The suggestion of a continuum of pathological beliefs in OCD from 'none' to 'delusional intensity' suggests the possibility of another continuum of mental disorders from OCD to psychosis.

The authors presented five cases of OCD associated with delusional beliefs. They concluded that these patients are best considered within an OCD management plan. Moreover, they do not recommend use of long-term antipsychotic medication since they consider that such patients are unlikely to respond. However, none of the five patients they reported upon was treated with low doses of an atypical antipsychotic in order to augment the action of serotonin-specific antidepressants, the preferred pharmacotherapy in OCD.

Recently, we have successfully treated a man in his 50s who presented with a 7-year history of typical OCD. His problem was suitable for treatment using exposure and ritual prevention, combined with an antidepressant. He was referred for behavioural psychotherapy at the specialist unit based at the Maudsley Hospital. Although motivated to try this treatment he later found it too difficult to continue with this approach. He therefore remained under the care of his general practitioner (GP) who treated him with fluoxetine 20 mg daily, on which he had only a very slight improvement in his symptoms. When we saw him at the request of his GP he was asking for relief from anxiety symptoms. Small doses of thioridazine produced unacceptable side-effects, so olanzapine 2.5 mg was substituted. After approximately 4 weeks his symptoms were almost completely gone, and at a follow-up appointment he stated that for the first time after 7 years of rituals and obsessions, he felt 90% better.

Initially, our choice of olanzapine was determined by the patient's need for anxiolysis. However, there is emerging evidence that olanzapine may augment the action of fluoxetine in the treatment of individuals with OCD (Weiss *et al*, 1999). Another possibility is that olanzapine was having a direct action on psychotic phenomena. Whatever the case, we would suggest that contrary to the recommendations for management offered by O'Dwyer & Marks (2000) some treatment-resistant cases of OCD might respond to a therapeutic trial with low doses of an atypical antipsychotic in addition to a serotonin-specific antidepressant as usually recommended.

O'Dwyer, A. M. & Marks, I. (2000) Obsessive–compulsive disorder and delusions revisited. *British Journal of Psychiatry*, **176**, 281–284.

Weiss, E. L., Potenza, M. N., McDougle, C. J., *et al* (1999) Olanzapine addition in obsessive–compulsive disorder refractory to selective serotonin reuptake inhibitors: an open-label case series. *Journal of Clinical Psychiatry*, **60**, 524–527.

A. Marušič, A. Farmer SGDP Research Centre, Institute of Psychiatry, De Crespigny Park, London SE5 8AF

Reboxetine-induced spontaneous ejaculation

Delayed or absent ejaculation is a common side-effect of antidepressant drugs. Reboxetine, a selective noradrenaline reuptake inhibitor, is known for its lack of sexual side-effects. We report a case of reboxetine-induced spontaneous ejaculation.

https://doi.org/10.1192/bjp.177.6.567-a Published online by Cambridge University Press