pyramidal reactions (EPRs) have been reported in the literature. Overall, there at least 28 reports involving a minimum of 42 patients who have experienced an EPR associated with an SSRI.[5] In this body of literature, a full range of EPRs have been reported including what appears to be a reversible (tardive-like) dyskinesia. Important risk factors for developing EPRs after starting an SSRI may include concurrent antipsychotic use, using a rapid SSRI dose escalation strategy, treating with high daily SSRI doses, older patients, female patients and patients with PD. Unfortunately, because the available information is largely from anecdotal reports, definitive risk factor guidelines are unavailable.

From cases like the one published by Farragher and Walsh, it has become clear that there is a real possibility that patients being treated with an SSRI may experience EPRs. The true risk of SSRI-related EPRs and the associated risk factors, however, are presently unclear. One potential risk factor may include the concurrent use of an antipsychotic (including an atypical antipsychotic). If an EPR develops while a patient is receiving both an SSRI and an antipsychotic, it is important to realise that there is a potential pharmacodynamic interaction which may occur in addition to a pharmacokinetic interaction.

Charles F Caley, PharmD, BCPP,
Assistant Clinical Professor of Pharmacy,
University of Connecticut – School of Pharmacy
and Psychopharmacology Consultant,
The Institute of Living – Burlingame Center for
Psychiatric Research and Education,
400 Washington Street, Hartford,
CT 06106-3392,
USA

References
1. Farragher B, Walsh N. Delayed extrapyramidal side-effect on combining paroxetine and risperidone [letter]. Ir J Psych Med 1997; 14: 117.
2. Leysen JE, Gommeren W, Eens A *et al.* Biochemical profile of risperidone, a new antipsychotic. J Pharmacol Exp Ther 1988; 247: 661-70.
3. Bymaster FP, Calligaro DO, Falcone JF *et al.* Radioreceptor binding profile of the atypical antipsychotic olanazapine. Neuropsychopharmacology 1996; 14: 87-96.
4. Baldessarini RJ, Marsh E. Fluoxetine and side-effects (letter). Arch Gen Psychiatry 1990; 47: 191-2.
5. Caley CF. Extrapyramidal reactions and the selective serotonin reuptake inhibitors. Ann Pharmacother 1997, in print.

Re: Diogenes syndrome: review and case history

Sir – O'Shea and Falvey report a case of Diogenes Syndrome and in reviewing the literature in the area emphasise the interplay of factors contributory to the presentation, including organic brain disease, psychotic illness, and personality *(Ir J Psych Med 1997; 14(3): 115-6).*[1] We report a case of a 50 year old female patient in which all three factors appeared to contribute to the classic presentation and discuss implications for investigation of the purported syndrome.

A 50 year old woman was admitted to a psychiatric ward in an advanced state of self-neglect. This retired single nurse had a 20 year history of contact with the psychiatric services, initially for peer relationship problems, subsequently being admitted twice for treatment of depressive episodes. Medical history included menorrhagia, hypothyroidism and scoliosis. In the period prior to admission she had stopped all of her regular medication and allowed her home to become extremely dirty. She ate very little, but her cat was well cared for. She agreed to informal admission.

Mental state and physical examination, and blood tests were normal on admission and she improved rapidly without any new treatment. Occupational therapy assessment suggested visuospatial problems, so a MRI scan was performed. This revealed a large sessile meningioma in the left middle cranial fossa, with temporo-parietal mass effect, as well as some cerebral atrophy. Neuropsychological testing showed selective frontal deficits but a high NART-IQ of 122. Spect scan was normal. She had the tumour removed and was discharged home two months later. She had a fluctuating clinical course subsequently, with at least one more admission (with persecutory delusions and self-neglect). Unconcern with her situation was striking throughout.

Our case demonstrated personality features similar to Clark *et al*'s original series,[2] including detachment and poor integration. We felt that organic brain disease had 'released' the behavioural syndrome at an early age. No association of temporo-parietal lobe lesions with neglect has been previously reported. A possible explanation is that the lesion to the parietal lobe led to unawareness of neglect, akin to anosognosia, the unawareness of disease. We feel that organic brain disease should be suspected in all cases of severe neglect, while acknowledging the potential contribution of multiple factors to the phenomenon. Ascription of her neglect to constitutional (personality) factors, or to her previous psychiatric illness, would have been an unfortunate omission.

Bart Sheehan, MRCPsych,
Specialist Registrar in Old Age Psychiatry,
Maudsley Hospital,
Denmark Hill,
London SE5 8AZ,
England.

John Geddes, MD, MRCPsych,
Dept of Psychiatry,
University of Oxford,
Warneford Hospital,
Oxford OX3 7JX,
England.

References
1. O'Shea B, Falvey J. Diogenes syndrome: review and case history. Ir J Psych Med 1997; 14(3): 115-6.
2. Clark ANG, Mankikar GD, Gray I. Diogenes syndrome: a clinical study of gross neglect in old age. Lancet 1975; 1: 366-8.

Serious hypertensive reaction after switching from clomipramine to moclobemide

Sir – Moclobemide is a selective, reversible inhibitor of monoamine oxidase A (MAO-A) which differs from the classical, irreversible monoamine oxidase inhibitors (MAOIs), both in pharmacodynamic and pharmacokinetic properties and has therefore a low propensity for producing drug interaction.[1] Recently, in a doubleblind