The patient had previously developed several periods of hyponatraemia while taking diuretics. We have recently submitted a paper reporting a case of hyponatraemia due to the syndrome of inappropriate antidiuretic hormone secretion (SIADH) following commencement of therapy with citalopram in an elderly patient. Her only other medications were aspirin, folic acid, multivitamins and senna, none of which is known to be associated with hyponatraemia and SIADH.

A wide variety of psychotropic drugs have been implicated in causing SIADH, described in a large number of case reports (Spigset & Hedenmalm, 1995) but systematic epidemiological and clinical studies are lacking. While some authors suggest hyponatraemia due to SIADH is an idiosyncratic effect of any antidepressant drug (Committee on Safety of Medicines, 1994), others postulate it is most likely a class effect of the SSRIs (Ball & Herzberg, 1984), although a cross-over effect with TCAs has been reported (Bouman *et al*, 1997, in press). None the less, the exact mechanism remains to be clarified.

Surprisingly, Voegeli & Baumann (1996) state that age does not represent a risk factor for developing SSRI-induced hyponatraemia due to SIADH. We would disagree with this statement. Although the exact nature of age-related changes in sensitivity to ADH remains an area of controversy, elderly people are particularly prone to developing hyponatraemia due to SIADH (Ball & Herzberg, 1994; Committee on Safety of Medicines, 1994; Spigset & Hedenmalm 1995; Bouman *et al*, 1997, in press). In the large majority of published case reports (>90%) the age of the patient is over 65 years, particularly among those treated with SSRIs.

Ball, C. J. & Herzberg, J. (1994) Hyponatraemia and selective serotonin inhibitors. *International Journal of Geriatric Psychiatry*, **9**, 819–822.

Bouman, W. P., Johnson, H., Trescoll-Serrano, C., *et al* (1997) Recurrent hyponatraemia associated with sertraline and lofepramine. *American Journal of Psychiatry*, in press.

Committee on Safety of Medicines (1994) Antidepressant-induced hyponatraemia. *Current Problems in Pharmacovigilance*, **20**, 5–6.

Spigset, O. & Hedenmalm, K. (1995) Hyponatraemia and the syndrome of inappropriate antidiuretic hormone secretion (SIADH) induced by psychotropic drugs. *Drug Safety*, **12**, 209–225.

Voegeli, J. & Baumann, P. (1996) Inappropriate secretion of antidiuretic hormone and SSRIs (letter). *British Journal of Psychiatry*, **169**, 524–525.

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Outcome of panic disorder

Sir: The conclusion of O'Rourke *et al* (1996) in their follow-up study that panic disorder "is a stable clinical entity" is at variance with almost all other comparative studies, which show a wide variation in clinical diagnostic outcome ranging from complete recovery to alcohol dependence, major depression, social phobia, obsessive–compulsive disorder and agoraphobia. The authors defend their single assessment at five years only by the somewhat curious argument that their method avoids the "distorting research effects intrinsic to the prospective method". It is surely relevant that the authors found that an unspecified number of patients "experienced temporary episodes of depression and alcohol misuse" during the follow-up period but this does not enter into the analysis at five years. The choice of the ninth edition of the Present State Examination as a diagnostic bastion for evaluating panic disorder is also odd as this instrument has only one question concerned with panic in its 140 items and does not derive a CATEGO diagnosis of panic disorder. The sample of patients chosen in the study was also not typical of panic disorder as it was a particularly chronic group that had been ill for a mean of five years. As all patients were known to have panic disorder at the time of follow-up there could also have been some tendency for the original symptom pattern to be identified and replicated, particularly if Dr O'Rourke (the assessor) expected this at the outset of the study.

In our own work, diagnostic assessments by structured interview at onset and after 10, 16, 32, 52 and 104 weeks showed that of 66 patients with panic disorder at outset, 60 (91%) had at least one diagnostic change over the next two years, 13 (20%) to a depressive disorder only, 23 (35%) to another anxiety disorder only, and 14 (21%) to both. We should therefore like to suggest that the findings of O'Rourke *et al* make a useful contribution to the debate over the validity of individual diagnoses within the neurotic spectrum but should not be taken as typical of panic disorder as it exists in clinical practice. It would be interesting to know whether the same diagnostic stability in the Galway sample continues to be maintained over the longer time scale as, if the same findings are found with formal diagnostic schedules, the existence of special features such as the higher prevalence of anancastic personality features in Ireland (Kelleher, 1972; Scott *et al*, 1982) might contribute to such unusual stability.

Kelleher, M. J. (1972) Cross-national (Anglo-Irish) differences in obsessional symptoms and traits of personality. *Psychological Medicine*, **2**, 33–41.

O'Rourke, D., Fahy, T. J., Brophy, J., *et al* (1996) The Galway study of panic disorder. III: Outcome at 5 to 6 years. *British Journal of Psychiatry*, **168**, 462–469.

Scott, A., Kelleher, M. J., Smith, A., *et al* (1982) Regional differences in obsessionality and obsessional neurosis. *Psychological Medicine*, **12**, 131–134.

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Authors' reply: Tyrer *et al* wrongly assert that we concluded that panic disorder is a stable clinical entity. We said that the mirror image of Present State Examination (PSE) test–retest after six years, although not conclusive, was difficult to reconcile with the notion that panic disorder is but one facet only of a general neurotic syndrome. That PSE does not define panic disorder with precision is very much to the point: PSE CATEGO-derived diagnoses are thereby less likely to be influenced by diagnostic bias. Their suggestion that our single rater cooked the PSE data is difficult to absolutely refute: if so, then the data were cooked to a turn – to a correlation of 0.92 to be exact, a culinary achievement all the more remarkable since baseline PSE data were not available until after follow-up was complete.

Our sample of panic-disordered patients was not a "particularly chronic group": most reviewers, including Argyle & Roth (1990), estimate mean chronicity at index as five to 10 years (five years in our sample). Tyrer *et al* ignore this notorious chronocity of panic disorder before treatment in their insistence on diagnostic musical chairs over time *after* treatment. They suggest that the reason why some results do not tally with theirs is that the former are biased by hospital practice. This time, however, the objection has no validity; our patients were treatment-naive, unpaid and were referred by general practitioners, as we have pointed out (Fahy *et al*, 1992; O'Rourke *et al*, 1996). If our patients were in any way special, it was in the notable absence of severe

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personality disorder and primary major depressive illness. Our patients were carefully and reliably diagnosed by traditional clinical methods as well as by structured diagnostic schedules at baseline and at follow-up.

We perceive their final suggestion that Irish panic-disordered patients are somehow ethnically anancastic as a racist joke in poor taste.

Finally, we note that our data on Axis I comorbidity are consistent with the arguments for a general vulnerability factor in the aetiology of panic disorder, so ably rehearsed by Andrews (1996): he showed that patients themselves discriminate between primary disorder (e.g. panic disorder) and secondary or derivative disorders (e.g. depression, substance abuse, etc.), which may complicate the long-term course of the primary illness. This return to clinical common sense may clarify the reasons why 'different' Axis I disorders have appeared in recent surveys to cluster together in individual patients.

Andrews, G. (1996) Comorbidity and the general neurotic syndrome. *British Journal of Psychiatry*, **168** (suppl. 30), 76–84.

Argyle, N. & Roth, M. (1990) The phenomenological study of 90 patients with panic disorder. *Psychiatric Developments*, **7**, 187–209.

Fahy, T. J., O'Rourke, D., Brophy, J., *et al* (1992) The Galway study of panic disorder. I: Clomipramine and lofepramine in DSM–III–R panic disorder: A placebo controlled trial. *Journal of Affective Disorders*, **25**, 63–76.

O'Rourke, D., Fahy, T. J., Brophy, J., *et al* (1996) The Galway study of panic disorder. III: Outcome at 5 to 6 years. *British Journal of Psychiatry*, **168**, 462–469.

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Body dysmorphic disorder

Sir: We read with interest the paper by Veale *et al* (1996) on body dysmorphic disorder. In the field of orthodontics and maxillofacial surgery we also encounter a number of patients suffering from body dysmorphic disorder. In contrast to Veale *et al* we have not found a higher proportion of female patients to be affected. Phillips (1991) stated that the ratio of women to men in reported cases was approximately 1.3:1, and in a later paper (Phillips *et al*, 1994) this ratio was quoted as approximately 1:1. A study in Japan by Fukuda (1977) found 62% of affected patients were male, although this may reflect ethnic variations.

It would appear that the large female component in the study by Veale *et al* is almost certainly due to the self-referral pattern, with females having higher figures for self-referral and consulting doctors generally. This ratio may also be heavily influenced by the authors having advertised in a women's magazine (*Cosmopolitan*).

Fukuda, O. (1977) Statistical analysis of dysmorphophobia in out-patient clinic. *Japanese Journal of Plastic and Reconstructive Surgery*, **20**, 569–577.

Phillips, K. A. (1991) Body dysmorphic disorder: The distress of imagined ugliness. *American Journal of Psychiatry*, **148**, 1138–1149.

—, McElroy, S. L., Keck, P. E., *et al* (1994) A comparison of delusional and nondelusional body dysmorphic disorder in 100 cases. *Psychopharmocology Bulletin*, **30**, 179–186.

Veale, D., Boocock, A., Gournay, K., *et al* (1996) Body dysmorphic disorder. A survey of fifty cases. *British Journal of Psychiatry*, **169**, 196–201.

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Steroid-induced prepartum psychosis

Sir: We were interested to read Johnson's letter (1996) on steroid-induced prepartum psychosis. We have encountered two similar patients.

A 25-year-old primigravida was treated with dexamethasone at 31 weeks' gestation, because of intrauterine growth retardation of one twin, necessitating very early delivery. She promptly developed delusional mania. One week later she gave birth to twins, one of whom proved to have Down syndrome. Her illness continued into the puerperium.

A 29-year-old primigravida, with insulin-dependent diabetes, was delivered by caesarian section at 36 weeks' gestation. She developed laryngeal spasm and was thought to have angioneurotic oedema as a reaction to the anaesthetic. She was given dexamethasone and developed a delusional psychosis within 36 hours of the birth.

Johnson kindly refers to previous Birmingham work on the association of prepartum and puerperal psychosis, but we were not the first to report it: there are at least seven earlier reports making (with Johnson's case) 15 in all – quite a strong association. There are also several other reports of puerperal and steroid-induced psychoses occurring in the same women (five in all). Johnson may be right in suggesting that adrenal steroids *predispose* to postpartum psychosis. Another view is that late pregnancy, the puerperium and exposure to excessive adrenal steroids are independent triggers of manic–depressive psychosis, i.e. the predisposition is inborn. The association of pregnancy-related and steroid-induced psychosis could be a clue to their shared aetiology, because progesterone is a precursor of adrenal steroids as well as oestrogen.

Johnson, I. (1996) Steroid-induced prepartum psychosis (letter). *British Journal of Psychiatry*, **169**, 522.

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In-patient psychotherapy

Sir: We write in support of in-patient psychotherapy units (Norton & Hinshelwood, 1996). Although Haigh & Stegen (1996) are right to point out that day units provide an important service, there will always be patients desperately in need of psychotherapy who can only be contained in in-patient settings.

At Francis Dixon Lodge the majority of patients are admitted directly from general psychiatry wards, many of them having recently been detained under the Mental Health Act. They often exhibit active suicidal behaviour, on-going serious self-harm, and are on a variety of neuroleptics, antidepressants, mood stabilisers and benzodiazepines when first admitted. By providing 24 hour support, in an environment where a crisis meeting can be called at any hour of the day or night, massive levels of anxiety can be more contained, and residents gradually encouraged to find more constructive ways of coping with distress.

The day unit Haigh & Stegen describe has very different admission criteria: patients have to stop psychotropic medication; survive out of hospital for three months; and self-harm is a dischargeable offence. It is unlikely, therefore, that they are talking about the same clinical population as Norton & Hinshelwood (1996) or Francis Dixon Lodge.

It is ironic that at a time when services for the mentally ill are being prioritised that psychotherapy is being pushed in the opposite direction. There is enormous pressure to offer brief therapies and see as many patients as quickly as possible. In many areas there is nothing for the patient with personality disorder between brief therapies and secure units for severe forensic cases.