host of clinical studies which he mixes up (like apples and pears) with the handful of community based studies that exists. One of the few methodologically acceptable community based studies of anorexia nervosa (see Treasure, 1990; Patton & King, 1991) – the one performed in Göteborg, Sweden in the 1980s (Råstam *et al*, 1989) – was excluded from Fombonne's analysis for "obvious reasons". It seems these "obvious reasons" were (1) that the material of the Göteborg study was presented in sufficient detail to allow specific analysis of whether DSM–III or DSM–III–R criteria applied, (2) that partial syndromes – later meeting full DSM–III–R criteria (Gillberg *et al*, 1994) – were included as a separate group in the original study, and (3) that the birth-cohort was followed up for a few years leading to the appearance of new cases. The findings were presented in a way which has made it possible for Fombonne to calculate all sorts of rates needed for a thorough review. He himself complains that several studies have not provided enough information about the diagnostic criteria used, and that few authors have looked at cohorts in a longitudinal fashion, so we had some difficulty understanding what was so "obvious" about the reasons for excluding this study.

The prevalence rate of anorexia nervosa in the community based studies was considerably higher than the median rate calculated by Fombonne. Again, this should not be taken as evidence that there has been an increase in prevalence rate over the years. However, it is essential that conclusions be based on the most reasonable data sets rather than those that, according to the standards set out by the author of a review/meta-analysis, are less than adequate.

FOMBONNE, E. (1995) Anorexia nervosa, No evidence of an increase. *British Journal of Psychiatry*, **166**, 462–471.

GILLBERG, I. C., RÅSTAM, M. & GILLBERG, C. (1994) Anorexia nervosa outcome: six-year controlled longitudinal study of 51 cases including a population cohort. *Journal of the American Academy of Child and Adolescent Psychiatry*, **33**, 729–739.

PATTON, G. C. & KING, M. B. (1991) Epidemiological study of eating disorders: time for a change of emphasis. *Psychological Medicine*, **21**, 287–291.

RÅSTAM, M., GILLBERG, C. & GARTON, M. (1989) Anorexia nervosa in a Swedish urban region: a population-based study. *British Journal of Psychiatry*, **155**, 642–646.

TREASURE, J. (1990) Anorexia nervosa and bulimia nervosa. *Current Opinion in Psychiatry*, **3**, 211–214.

M. RÅSTAM
C. GILLBERG

University of Göteborg
S-41345 Göteborg, Sweden

Periodic psychosis of puberty

SIR: The article by Abe & Ohta (1995) regarding adolescent onset brief periodic psychoses raises a number of important issues which must be clarified before this condition can be so confidently defined. It is not clear from the report whether the cases they describe met criteria (ICD–10 or DSM–IV) for other psychiatric diagnoses. Certainly, if the subjects cross-sectionally met criteria for depression or mania, it should come as no surprise that these disorders would recur.

Without a description of family history, natural course, comorbid symptoms/diagnoses, or other external validators, it is difficult to assign any diagnostic validity to the concept of a "periodic psychosis of puberty". The reported relationship of worsening psychotic symptoms associated with menses is well recognised in adolescent in-patient units in which severely ill teenage girls with mania or depression are treated. This phenomenon does not of itself qualify for a unique diagnostic label.

In ten consecutive years of adolescent in-patient practice, we have only ever seen one teenager (white, male, age 16) with a non-substance induced, non-affective or non-schizophreniform brief psychosis. This is one of about 1000 admissions. Perhaps there is a cultural diversion to this type of presentation in psychiatrically disturbed teenagers that differentiates Canadian from Japanese youth.

ABE, K. & OHTA, M. (1995) Recurrent brief episodes with psychotic features in adolescence: periodic psychosis of puberty revisited. *British Journal of Psychiatry*, **167**, 507–513.

S. P. KUTCHER

Dalhousie University
Halifax, Nova Scotia
Canada

Treatment of PTSD

SIR: Busuttil *et al* (1995) suggest that their case series strongly endorses the use of psychological debriefing (PD) in the treatment of PTSD. However, the 63 hours of "formal work sessions" in a residential setting described in their paper seems excessive and difficult to justify for PTSD sufferers who have not first tried out-patient treatment. Briefer therapies can work. For example, Foa *et al* (1991) described a randomised controlled trial in which PTSD sufferers experienced a marked reduction in symptoms after nine 90 minute exposure therapy sessions at three and a half month follow-up.

In a study of similar design (and hence with similar flaws) to that of Busuttil *et al* we (Bisson & Jones, 1995) reported a similar dramatic improvement at three month follow-up in a group of 18 military and non-military personnel with post-traumatic stress reactions. Nine of the subjects' symptoms related to experiences in the Gulf War. The treatment technique was that of taped imaginal exposure (TIE) which employs many elements of psychological debriefing. In TIE the personal account is audiotaped and then listened to as "homework". The average number of 60–90 minute out-patient therapy sessions in our study was 4.2 range 3–7).

Brief effective out-patient therapies will prove more attractive to patients and purchasers in the NHS. They are likely to be more cost-effective and to allow more PTSD sufferers to receive treatment than intensive residential programmes. Such programmes should be reserved as second line treatment for patients who have failed to improve with out-patient interventions.

BISSON, J. I. & JONES, N. (1995) Taped imaginal exposure as a treatment for post-traumatic stress reactions. *Journal of the Royal Army Medical Corps*, **141**, 20–25.

BUSUTTIL, W., TURNBULL, G. J., NEAL, L. A., *et al* (1995) Incorporating psychological debriefing techniques within a brief group psychotherapy programme for the treatment of post-traumatic stress disorder. *British Journal of Psychiatry*, **167**, 495–502.

FOA, E. B., OLASOV ROTHBAUM, B., RIGGS, D. S., *et al* (1991) Treatment of post traumatic stress disorder in rape victims: a comparison between cognitive–behavioural procedures and counselling. *Journal of Consulting and Clinical Psychology*, **59**, 715–723.

J. I. BISSON

University of Wales College of Medicine
Heath Park, Cardiff CF4 4XN

SSRI and sympathomimetic interaction

SIR: Interactions between specific serotonin re-uptake inhibitors and sympathomimetics, while a theoretical possibility, are apparently uncommon because the latter are rarely prescribed. Such interactions may, however, occur if patients self-administer sympathomimetics. We report two such cases caused by popular pharmacomythology.

Case 1

A 31-year-old man presented to a casualty department with symptoms he recognised as being those of a large amphetamine overdose. He was extremely restless and agitated, over-talkative, anxious, hyperventilating and described ideas of reference. These symptoms subsided over the course of five hours in the department, his mental state returning eventually to normal. His GP had prescribed fluoxetine 20 mg daily for depression, but he had been taking a dose of fluoxetine 60 mg daily until a week before his presentation. He had intentionally trebled his prescribed dose because associates and the media led him to believe that pleasant psychological effects might result.

Shortly before presentation he had taken a dose of amphetamines that was small by his usual standards, but which was presumably augmented by the high levels of fluoxetine still present in his circulation because of the very long half-life of the drug.

Case 2

A 32-year-old man was admitted to an adult general psychiatric ward presenting first rank symptoms of schizophrenia. As a result of a business failure, he became depressed, and attended his GP, who prescribed fluoxetine 20 mg daily. Four days prior to admission he took two doses of amphetamines. He became very energetic and complained of insomnia. He also reported hearing voices talking to him and believed that the TV and radio were sending him messages and signals indicating the directions he had to follow when driving his car. On admission the patient received a single dose of 100 mg of chlorpromazine and he was discharged free of abnormal perceptual experiences and psychotic symptoms three days later.

This patient did not experience psychotic symptoms on previous exposure to amphetamines. However, the combination of amphetamines and fluoxetine was associated with a brief, but severe psychotic episode which lasted for approximately three days.

Sympathomimetic–fluoxetine interaction has been described in a bulimic patient who combined a proprietary appetite suppressant with the drug (Walters, 1992). The interaction with illicit amphetamines is more dangerous, and is likely in a population that perceives fluoxetine to be a potentially rewarding drug of abuse. Clinicians should take this potential interaction into account when prescribing for depressed drug abusers.

WALTERS, A. M. (1992) Sympathomimetic–fluoxetine interaction. *Journal of the American Academy of Child and Adolescent Psychiatry*, **31**, 565–566.

J. BARRETT
O. MEEHAN
T. FAHY

Maudsley Hospital
Denmark Hill
London SE5 8AZ