measure is attributable to variability in the other is not disputed and we interpreted it thus in our discussion. We used the regression line to work out correspondence with recognised cut-off points on the BDI taken as a 'gold standard'. This enabled us to identify clinically important thresholds and then calculate sensitivity and specificity.

Yes, D1P does not strongly predict outcome of behaviour therapy, nor does the BDI. This does not invalidate the D1P's utility as a reasonable rough guide to mood during treatment of anxiety disorders.

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The difficult patient – Schreber revisited

Sir: Hinshelwood (1999) proposes that if the care-giver concentrates on understanding his or her countertransference to the difficult patient, this may overcome obstacles in treatment.

There are theoretical difficulties with this psychodynamic approach. Hinshelwood himself has alluded to the potential for coercion in the intersubjective dialogue (Hinshelwood, 1997). This is the case when one of the two subjects involved – the care-giver – is the expert or "the one who knows" ("*le sujet supposé savoir*"; Lacan, 1964). Within mainstream psychiatry, trenchant opposition to the coercive possibility in psychoanalysis is consistently articulated (Clare, 1994). Many psychiatrists believe that there can be no collaborative starting point between psychiatrists and psychoanalysts in patients with severe mental illness.

Faced with this impasse, in re-focusing attention on Schreber, Hinshelwood indicates the way forward, but does not go far enough. Echoing Freud in his 1911 study of the Schreber case, it is my opinion that, rather than countertransference issues, it is the discourse of the person with psychosis that must be prioritised. Schreber himself was a very disturbed individual with paranoid schizophrenia. Despite the bizarre and seemingly senseless character of his delusions, they represented a mythic reconstruction giving meaning to his disastrously altered world. Although normal subjects differ crucially from those with psychosis, the notion of a psychotic mythic reconstruction is not outlandish, given that normal subjects also mediate experience through construction of a myth – the more acceptable neurotic myth (Lacan, 1953).

Schreber demonstrates that the discourse of someone with psychosis is an organised, logically driven process. This logically structured discourse often dictates its own imperatives, sometimes to the detriment of the individual with psychosis or others. This is the phenomenon of *passage-à-l'acte*, where the person with psychosis acts in a sudden, often violent and seemingly unintentional way. Such acts are symbolically determined. It is my argument that because the patient's psychotic structure remains unchanged despite neuropharmacological intervention, psychoanalysis has a place in ameliorating the expression of the myth that drives him. Equally, one must accept that few patients with psychosis would consider – or tolerate – involvement with a psychoanalyst. Despite this, we must set up the conditions that encourage this small minority to engage analytically. This requires dialogue between psychiatrists and psychoanalysts whose views on the treatment of psychosis are not irreconcilable.

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EURODEP study

Sir: The desire to join up dots on graphs must be tempered by reason. In the EURODEP study reported by Copeland *et al* (1999) figures 8 and 9, of depression prevalence by age group, are interpreted as showing "no overall tendency to rise or fall with age, except in extreme old age". Differences in prevalence of any condition between age groups cannot be regarded as functions of 'rising' or 'falling' without repeated measurement in cohorts; joining up dots in cross-sectional surveys encourages almost unconscious assumptions, also manifest in the discussion of the relationship between depression and physical illness. Cohort effects are at least mentioned in another paper by the same authors in the same issue (Prince *et al*, 1999) but, for obvious reasons, care must be used in the use of words such as 'increasing' and 'age effects' which are so easily misunderstood to mean ageing itself.

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Author's reply: Professor Macdonald is right to say that tendencies over time can only be mapped on longitudinal as opposed to cohort studies. Therefore, it would have been more accurate to say that the prevalence of depression varied in the different age groups of the cohorts, older ages not being associated with higher levels of depression.

Clinicians are interested in whether they are missing depression in older patients, and the mistaken public perception still remains that older people are by nature miserable, so prevalence levels by age are not unimportant. Because large samples will be necessary for longitudinal studies of the very old because of the high death rates, we are planning to explore the longitudinal characteristics of the EURODEP samples.

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Psychosis associated with gonadorelin agonist administration

Sir: Puerperal and menstrual psychoses have been associated with hormonal fluctuations

in women, suggesting an oestrogen withdrawal hypothesis in these disorders (Deuchar & Brockington, 1998). Oestrogen release into the peripheral blood is influenced by secretion of the pituitary hormone gonadorelin. We describe a psychotic disorder associated with administration of a long-acting luteinising hormone-releasing hormone agonist (LALHRHA) triptorelin.

Mrs A., 32 years old with two children, had been receiving monthly treatment for 2 months with triptorelin (intramuscular depot formulation: 3 mg/4 weeks) for suspected endometriosis. A few days after the second injection, she experienced persecutory delusions, agitation, and auditory hallucinations, which resolved within two months under neuroleptic medication and were immediately followed by a major depressive episode. This episode fulfilled DSM–IV (American Psychiatric Association, 1994) criteria for schizoaffective disorder. There was no personal history of psychotic disorder (mild baby blues followed the birth of the first child) and no familial history of psychiatric disorder. One year after this episode, she became pregnant. There were no psychiatric symptoms during the pregnancy. Five days after the delivery she presented in a psychotic state clinically similar to the previous one.

Peripheral oestrogen modulates dopaminergic transmission (Fink *et al*, 1996). The oestrogen decrease has been hypothesised as being a causative factor in puerperal and premenstrual psychoses in predisposed individuals (Deuchar & Brockington, 1988). LALHRHAs administered in a depot formulation induce a paradoxical desensitisation of pituitary gonadotrophin secretion, resulting in a reduction in secretion of luteinising hormone and follicle stimulating hormone, and consequently a reduction in oestrogen secretion within one week (Broekmans *et al*, 1992).

As observed in puerperal and premenstrual psychoses, the decrease in oestrogen levels induced by the administration of LALHRHAs is consistent with a modification in the brain oestrogen environment, and is simultaneously associated with the occurrence of acute psychosis. The occurrence of both puerperal psychosis (suggesting an individual predisposition) and LALHRHA-associated psychosis in the same individual suggests a common aetiology. The puerperal and the LALHRHA-associated psychosis showed clinical and hormonal similarities, suggesting that a hormonal mechanism (the delivery-induced and the LALHRHA-induced oestrogen decrease) might be aetiologically relevant in this patient.

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Manic episode due to gabapentin treatment

Sir: Short & Cooke (1995) reported on a case of hypomania associated with gabapentin treatment. In a comment on this report Hauck & Bhaumik (1995) felt that alternating psychosis might be the cause (though electroencephalogram (EEG) was lacking). We report on a 35-year-old woman with a manic episode under gabapentin treatment in whom alternating psychosis as a possible causal factor could be excluded. She suffered from complex partial and tonic–clonic seizures for at least three years. The magnetic resonance imaging scan revealed glio-neuronal hamartia both in the left hippocampus and the right amygdala. A gabapentin monotherapy (3200 mg/day) was achieved five months before admission. Seizure frequency and EEG (within normal range) remained unchanged, however, the patient suffered from labile mood around menstruation and reported a slightly elevated mood in general.

Elevated mood and increased activity with brief episodes of depression over two days led to the patient's admission to hospital. Speech was slightly accelerated. The initial score on the Bech–Rafaelsen Mania scale (BRMAS; Bech *et al*, 1978) was 23. Neurological examination and EEG remained normal. Epileptic seizures had not occurred before admission. She did not have a personal or a family history of psychiatric disorders. Psychiatric symptoms disappeared within five days after discontinuation of gabapentin (BRMAS=5). At that time an anticonvulsant and antimanic treatment with lorazepam (2 mg/day) and oxcarbazepine (1200 mg/day) was established.

Psychic reactions to gabapentin seem to be rare. Only two cases of hypomanic episodes have been reported (Wong *et al*, 1997). Moreover, there is evidence that gabapentin has therapeutic effects in affective psychosis (Young *et al*, 1997). In the case reported here alternating or postictal hypomanic psychosis could be excluded. No other factors possibly contributing to interictal psychosis were detected. Thus, we feel that treatment with gabapentin is related to the patient's manic episode, even more so since she remained without any psychic abnormalities in a two-year follow-up. This may be in line with clinical observations on mood-elevating effects of gabapentin.

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Polydactyly and functional psychosis

Sir: I wish to comment on the report by Cardno *et al* (1998), which described five cases of co-occurrence of polydactyly and functional psychosis. I would like to report on two patients with polydactyly who also presented with mood disorders.

A 48-year-old married male had post-axial polydactyly on the right hand. There was no family history of polydactyly. His daughter had schizophrenia. At the age of 35, he developed elated mood, grandiose delusions and other features suggesting a diagnosis of mania. The episode was successfully treated with neuroleptics for six months. He developed an episode of