to be compared. Dr Brook's assessments of the results are subjective and hence debatable.

We welcome Dr Brook's suggestions that trainees rate their experiences at intervals as this would remove the bias due to retrospective recall; and trainees should continue to identify inadequacies in their training.

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Are British psychiatrists racist?

SIR: Lewis *et al*'s study of racial bias in psychiatric perception (*Journal*, September 1990, **157**, 410–415) is valuable. While bias may, of course, be elicited with one particular vignette and not with another, it is significant that neither their study nor a similar one of mine (*Journal*, September 1990, **157**, 451–452) found any greater tendency to diagnose schizophrenia among black patients when stated ethnicity was the sole variable. A problem remains, however, of the relationship between the leisurely rating of vignettes and the actual clinical decision-making (in which the differential perception of dangerousness, which Drs Lewis *et al* find, may well be rationalised subsequently through a diagnosis of schizophrenia, on the way to custodial and neuroleptic treatment).

Acute psychotic reactions were rated marginally more frequently in the Afro-Caribbean vignettes than in those of the whites, but I am not certain that Lipsedge and I are as responsible for this as they gently hint. Take the characteristics identified by their respondents, especially in the black vignette: duration less than three months, risk of violence to staff, neuroleptic treatment indicated, and to be charged with criminal damage. Only the first – 'acute' course – corresponds to anything in the clinical profile we derived in the paper (Littlewood & Lipsedge, 1981*b*) accompanying the one they cite (1981*a*). Our profile was based on those patients clinically given a diagnosis of schizophrenia who did not have core symptoms as rated by the research Present State Examination. Furthermore we specified acute 'psychosis' as a stay in hospital of less than three weeks, not three months. In their study, however, schizophrenia is preferentially diagnosed among whites. If there has indeed been a switch in diagnostic preferences among British psychiatrists since 1981 from schizophrenia to acute psychotic reactions for black patients, as a consequence of our paper, this does not seem to have been reflected in recent epidemiological studies. Our use of the term 'acute' was directly related to a Jasperian notion of 'reactivity', not just to duration of the illness. The idea of psychotic reactions of short duration among Afro-Caribbean patients in Britain had been around since at least Tewfik & Okasha's (1965) study. In its stereotyped form this category was criticised for its racism by Lipsedge and myself in a book (Littlewood & Lipsedge, 1982) arguably better known than our papers.

The rather more difficult question is: how and why do psychiatrists use stereotyped judgements? Are they indeed something specific to a psychiatric theory still embedded in imperial fancy, or is it that psychiatric care simply replicates the social power and prejudice located outside medicine? My own vignette study, which showed that medical students before and after they studied psychiatry, and psychiatrists themselves, all had similar perceptions, argues against the ideological power of specific psychiatric theories in themselves.

Both vignette studies would seem to dispute such a power. If transcultural psychiatry in Britain has correctly shifted from its exclusive focus on the black patient to examine the role of the white psychiatrist, we have to be prepared to look at the particular social context of power within which psychiatry operates, which determines the perceptions and responses of both patient and doctor, and their interaction.

Such studies would hardly be independent of an understanding of racism in its wider economic, ideological and coercive forms.

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ECT following clozapine

SIR: The safety of electroconvulsive therapy (ECT) following clozapine therapy has not been documented. The potential for spontaneous seizure phenomena is of particular concern in light of

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clozapine's suspected epileptogenic properties (Heerlen & Kunze, 1979; Juul Povlsen *et al*, 1985). Herein, we report on one patient who developed spontaneous (tardive) seizures following only one electroconvulsive therapy treatment soon after clozapine discontinuation.

Case report: A 26-year-old man with a four-year history of chronic paranoid schizophrenia received up to 800 mg per day of clozapine with limited response as extreme anxiety, fearfulness and paranoia continued to impair his ability to function. At the time that ECT was recommended the patient was receiving clozapine (800 mg), propranolol (60 mg) and diazepam (20 mg) daily, which were then tapered over 14 days and discontinued, with the exception of diazepam which was reduced to 5 mg daily 72 hours before his first ECT session. The patient received bilateral ECT four days after his last dose of clozapine. Seizure duration monitored by two-lead electroencephalography (EEG) was 123 seconds. Recovery was remarkable for significant post-ictal confusion. The patient had two spontaneous *grand mal* seizures witnessed by staff on days four and six following this first and only ECT treatment. An EEG performed on day five after the first seizure and before the prescription of phenytoin was remarkable for mild defused slowing, with a 'questionable' focal abnormality in the right frontotemporal region. Non-contrast computerised tomography and magnetic resonance imaging were normal. The patient was ultimately discharged on maintenance phenytoin and clozapine with minimal improvement in his mental status, but no further seizure activity.

The inferences that can be drawn from this care are, however, unclear. We cannot rule out the possibility that clozapine stopped only four days before ECT in some way facilitated this patient's spontaneous seizures. However, other explanations are equally plausible. The patient had been on long-term benzodiazepine treatment which was reduced from 20 mg to 5 mg over two weeks; this may have been a contributing factor. It is also impossible to tell from an individual case whether this patient's seizure activity is significant in light of the reported one in 500 incidence of tardive seizure phenomena following ECT (Fink, 1977).

As a practical guide, given the suggestion of increased epileptogenic activity with the atypical neuroleptic clozapine, clinicians are advised to permit a drug-free period of 7–10 days following clozapine discontinuation before starting ECT. Theoretical risks of spontaneous seizure activity and prolonged seizure duration are raised which merit further study.

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Buspirone-induced mania

SIR: Buspirone, with its limited liability for abuse (Griffith *et al*, 1986), and lack of withdrawal effects (Tyrer *et al*, 1985), might be considered the drug of choice in the treatment of anxiety in patients with a history of alcohol or drug abuse. Such a patient, who developed mania after two weeks on buspirone, is presented.

Case report: A 28-year-old single man began abusing alcohol and heroin at 14 years of age. From 1984 onward he binged sporadically on alcohol, but remained off heroin. He presented for treatment in November 1989, with severe symptoms of anxiety. He was commenced on buspirone (10 mg b.d.), and also agreed to take disulfiram (400 mg daily). He took the buspirone regularly for two weeks, but took the disulfiram only intermittently. He drank on occasion during this period, but denies any other drug abuse. While on buspirone he described a 'floating feeling', and noted his thoughts going faster. Over the next few weeks he developed pressure of speech, flight of ideas, irritability, elated mood, and overactivity. There was no clouding of consciousness and he denied perceptional disturbances. Physical examination was normal. A drug screen was negative. The patient required high-dose neuroleptic medication to which he responded slowly. Neither he nor his family have any history of affective disorders.

To date there has been three published reports of buspirone causing mania. Two of these (Liegghio & Yeragani, 1988; McDaniel *et al*, 1990) involved patients who already had a diagnosis of bipolar disorder. In both, the introduction of buspirone precipitated a manic swing. In the third (Price & Bielefeld, 1989) a 38-year-old man with resistant depression and anxiety became hypomanic on the two separate occasions that buspirone was introduced.

Buspirone is thought to exert its effect by interaction with 5-HT1A receptors, and enhancement of dopaminergic and noradrenergic activity. These differential effects have been linked to the development of psychotic behaviour. The facilitation of dopaminergic function by buspirone may be more clinically significant than hitherto thought and is possibly the mechanism through which this drug induces mania.

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