states that it will not block the withdrawal syndrome often seen with cessation of diazepam therapy. Furthermore it is an ideology that pushes the more immediate question of motivation into the background.

The authors argue that in certain circumstances such as benzodiazepine withdrawal, buspirone displays antagonist rather than agonist effects at the 5-HT1A receptor sites. This is a gross oversimplification of the pharmacodynamic profile of chronically anxious, withdrawing, benzodiazepine-dependent individuals on buspirone. No mention is made of possible interactions causing reduced dorsal raphe nucleus activity and hence reduced behavioural inhibition (Eison *et al*, 1983). In contrast to the benzodiazepines, buspirone increases locus coerulus firing (Sanghera *et al*, 1985). Studies report subjective and objective improvement in alertness and concentration and reduced confusion in patients taking buspirone compared with those taking benzodiazepines (Schweizer *et al*, 1986). It is not known, however, whether the combination of buspirone and a benzodiazepine produces changes in cognition. Thus an isolated discussion of 5-HT1A receptor effects does not do justice to what is known of both buspirone and diazepam.

Any study, no matter how well designed, as was the case here, can suffer from unforeseen errors. Randomisation does not guarantee equivalence and in this study there is no avoiding the fact that the group assigned buspirone were initially more anxious. In retrospect a stratified randomisation might have been a better design. It is my opinion that the more anxious the individual the slower their withdrawal should be. It was a small study and I would have appreciated the inclusion of confidence intervals to provide me with a range of uncertainty (Gardner & Altman, 1990). It has been suggested that weekly rating intervals are inadequate for detecting the full range of relapse, rebound and withdrawal phenomena because symptom increases may be transient (several days) and go undetected with weekly assessments (Rickels *et al*, 1986).

Out of 23 patients, 17 had a good outcome in the study, remaining off benzodiazepines at six and 12 months and here the authors should be congratulated. However, considering the high dropout rate for the buspirone group I am tempted to believe that the better outcome in the control group could in part be due to the authors' continued contact and rapport with these patients, especially since all the buspirone-treated patients who remained in the study achieved a similar good result.

In conclusion, when considering data from studies of adjunctive pharmacotherapy in withdrawal states, it is important to have a conceptual framework that allows specific drug–drug interactions to be viewed as occurring in a dynamic nervous system that has functional plasticity, multiple systems of parallel processing and complex heirarchical association mechanisms. In this way tentative statements of drug agonistic or antagonistic effects can be more realistically appreciated.

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Is psychiatric training still improving?

SIR: We read Brook's article "Is psychiatric training still improving" (*Journal*, September 1990, **157**, 335–338) with interest. As visiting psychiatric trainees from different countries, we hope to learn from psychiatric training in Britain.

The opinions of newly appointed consultants about their training provided the basis of Dr Brook's study. Unfortunately the opinions expressed as to satisfaction were retrospective, with the possibility of a personal 'halo' bias giving a more favourable view of their training. Also respondents were those ex-trainees who have successfully obtained consultant posts; what of the others who failed at this hurdle?

Dr Brook stated that statistical analysis would be inappropriate for 'opinions'. We would disagree. Statistical analysis is particularly important when one is dealing with such data if they are to be useful. Although Dr Brook reiterated that the results reflected opinions about training and that what may have been considered satisfactory ten years ago may not be considered satisfactory today, we feel that the results of the 1982 and 1987 surveys should be subjected to some form of statistical analysis if they are

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