then has to be reintroduced through focusing, as Professor Leff puts it, on "the societies that are most culturally different from the west, and hence of greater interest". If all societies are becoming increasingly similar, as he argues, then patterns of, say, overdoses presumably become similar and one eventually concludes that overdoses in general are not a social phenomenon. Not surprisingly, two papers in that issue of the *Journal*, both concerned with the perceived consequences of parasuicide, were not in the 'cultural' section.

The newer critiques do not, as Leff suggests, necessarily fault psychiatry's *aspirations* to be a scientific discipline: that is one that seeks explanations independent of the observer's perspective. That at least should be clear from my review. Indeed I myself warn against the too casual neglect of evidence from the biological sciences. The problem is that the wish to be scientific, and the claim to be scientific, are often very different from actually being so. The difficulty with much of the older transcultural psychiatry, carried out by psychiatrists untrained in any social science, is that it mistakes the particular for the universal, the contingent for the necessary, the political for the biological.

To distinguish the two sets of categories is presumably the aim of any attempt at scientific truth. In certain particulars we find that the context of observation determines the observed events to the extent that locating patterns such as overdoses or agoraphobia solely in a person's individual characteristics does not help us to interpret the pattern at all.

These difficulties are not of cause restricted to psychiatry. My speculation as to the value of 'pathology' (or disease) is not, as Leff protests, some sort of Laingian romanticism but a concern as to whether any conception of 'pathology' is truly useful in scientific terms: we are, as the medical student joke has it, on the side of the human not the virus. Fair enough, as humans embedded in our illnesses and in our struggles we are compelled to act, but the prescriptive urge is not necessarily the appropriate ground for understanding. My suggestion in the paper which Leff refers to (Littlewood, 1984) that the experiences associated with what we psychiatrists conventionally refer to as cerebral pathology may at times be taken up by societies in certain situations as meaningful experiences is not an exhortation to consider this as the real meaning, merely that it can occur and that it may have interesting theoretical implications for us. I deal with this possibility at greater length in a forthcoming volume which uses field data including conventional Present State Examination assessments (Littlewood, 1990).

Professor Leff's editorial, its appeal to the demands of the 'practical difficulty' as justification for the validity of the findings, is an instance of the confusion between fact and value which many of us associated with the 'new cross-cultural psychiatry' argue is inevitable. We do not however pretend it can always be avoided, or allow value to masquerade as fact. A little closer examination of epistemology, of actually learning to distinguish the baby from the bathwater, will not come amiss.

Incidentally, in his put down of local knowledge, Professor Leff characterises the Yoruba masculine power Shopana (Shopona, Sopono) as a 'goddess'. I am not sure if he is thinking of the probably cognate Ewe/Fon power Shapata which is sometimes represented as a generic or androgynous emanation of the Mawu/Lisa principle. To characterise these concepts as free-standing deities rather than powers, principles, faculties or even mechanisms is, in any case, problematic. This is not trivial scholasticism but an example of the sort of problem we run into when we interpret others' meanings through our own frameworks. Our own categories of neurosis are hardly independent of assumptions about gender.

ROLAND LITTLEWOOD

Department of Anthropology
University College London
Gower Street
London WC1E 6BT

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Progesterone prophylaxis?

SIR: I was most surprised to read Meakin & Brockington's statement that "Progesterone is widely used in the treatment and prophylaxis of post-natal depression (*Journal*, June 1990, **156**, 910). To my knowledge, progesterone has never been used successfully as a treatment for post-natal depression. In fact, prolonged administration of progestogens may lead to depressive symptoms (Silverstone & Turner, 1982). Dalton (1985) claimed that progesterone prophylaxis was successful in reducing a recurrence rate of post-natal depression from 68% to 10%. However, the study was flawed in two ways. Firstly, it was not double-blind. Also, there was no

attempt to standardise the diagnosis of post-natal depression – cases were divided into mild, moderate and severe pending on how they were treated by their various general practitioners.

The fact that the authors were encouraged to use progesterone as a sole therapy for puerperal mania by their experiences with two patients quite frankly astounds me. The first patient reported a 'subjective calming effect' when progesterone was given (50 mg intramuscularly) before and after neuroleptic therapy was commenced – nothing particularly encouraging about that. The second women's improvement was most likely due to the fact that she was given haloperidol (40 mg intramuscularly – a high dosage) and chlorpromazine (50 mg intramuscularly) during the 48 hours before recovery.

Clearly, hormonal changes in the puerperium may be one of the factors that precipitate a psychotic illness in susceptible individuals but to expect that progesterone might be successful as a therapy for puerperal mania is in my view being rather simplistic.

THERESE O'NEILL

St Brendans Hospital
PO Box 418
Rathdown Road
Dublin 7

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Benzodiazepine withdrawal

SIR: With reference to Ashton *et al*'s paper on buspirone in diazepam withdrawal (*Journal*, August 1990, **157**, 232–238) we would like to make the following points.

We question the clinical relevance of this study. It is now generally accepted that gradual dose reduction with attention to appropriate psychological treatment is the best way to manage benzodiazepine withdrawal (Edwards *et al*, 1990). If this is done at the patient's own rate, pharmacological treatment of symptoms, which may complicate the withdrawal process, as indeed occurred in this study, should be unnecessary.

We think the study was unethical for two reasons: firstly, withdrawal was rapid and took no account of either the starting dose or the patient's response to withdrawal; and secondly, a blind study deprives the patients of the right to determine their own rate of withdrawal and the opportunity to learn from this experience. Did informed consent include telling patients that this was not the best way to come off benzodiazepines, was likely to create unnecessary distress and that buspirone was unlikely to help?

The design of the study is unsuited to small numbers. Unmatched groups, a failure to control for attending a support group or concurrent prescribing, and the high drop-out rate in the buspirone group make it difficult to draw any useful conclusions.

Finally, the study ignores important psychological factors which are crucial to maintaining abstinence. The importance of patients being in control of their withdrawal, learning non-drug alternatives and improving their quality of life makes a psychological approach more appropriate than a pharmacological one.

This study only perpetuates the search down a blind alley for pharmacological short-cuts which fail to respect the patient's right to participate in the decisions, manage the withdrawal and be offered alternative ways of coping.

LINDA BEALEY

Drug and Therapeutics Unit
Queen Elizabeth Hospital
Birmingham B15 2TH

DIANE HAMMERSLEY

515a Bristol Road
Birmingham B29 6AU

Reference

EDWARDS, J. G., CANTOPHER, T. & OLIVIERI, S. (1990) Benzodiazepine dependence and the problems of withdrawal. *Postgraduate Medical Journal*, **66** (suppl. 2), S27–S35.

SIR: I would like to congratulate Cantofer *et al* (*Journal*, March 1990, **156**, 406–411) on their study. Benzodiazepine dependence is a difficult condition to treat and an attrition rate as low as they obtained must indicate considerable enthusiasm. However, I am slightly surprised at the design of the study which appears confounded by having two variables, in that patients were allocated either to abrupt withdrawal and active treatment with propranolol or gradual withdrawal and placebo propranolol. From the study design one could draw the spurious conclusion that propranolol is no benefit for the patient withdrawing from benzodiazepines. I believe there is fairly good evidence that propranolol is of benefit in benzodiazepine withdrawal, at least as far as somatic symptoms are concerned (Halstrom *et al*, 1988). The other main finding of the study, that gradual withdrawal is easier than abrupt withdrawal, is already well supported in existing literature. However, to make such a deduction from the study is an error in