treatments in properly controlled studies of benzodiazepine withdrawal. The case for buspirone was particularly relevant at the time that our study was planned and initiated (1985/86) since there was a strong likelihood that this recently released drug, with proven anxiolytic activity (Feighner *et al*, 1982), would be widely used in benzodiazepine withdrawal, especially in general practice, where studies had shown it was effective in generalised anxiety disorder (Murphy *et al*, 1989). The study received the approval of the Joint Ethics Committee of the University and Newcastle District.

A fixed benzodiazepine withdrawal time of four weeks was chosen because we were anxious to obtain evidence of the utility or otherwise of buspirone in the shortest possible time in the minimum number of patients. Withdrawal regimes which are individually tailored may take over a year to complete (Ashton, 1987) and make it difficult to compare treatments. Furthermore, we had evidence that outcome is not affected by rate of withdrawal (Ashton, 1987) and that fairly rapid withdrawal may sometimes be appropriate (Ashton, 1984). We limited participation in the study to patients taking low to moderate doses of benzodiazepines, and most patients were referred because they had experienced difficulties during previous attempts with "generally accepted" methods of withdrawal. The study itself showed that 11 out of 12 patients in the placebo group were successful in achieving and maintaining withdrawal with the method used (one patient dropped out for reasons not connected with withdrawal).

The patients chose to take part in the trial; those who declined were still offered treatment at the clinic. Participants were given a full explanation of the aims and methods of the study; they were informed that they may or may not receive buspirone, and that the drug may or may not be helpful. There was no reason to anticipate that the drug might exacerbate withdrawal symptoms. It is not true, as Beeley & Hammersley claim, that the regimen took no account of the patients' response to withdrawal. All patients were able to discontinue the trial at any time. Indeed, many who were taking buspirone dropped out because of increasing symptoms or need for further medication, and the incidence of drop-out was one of the criteria used for assessing the effects of the drug. All patients were assessed at frequent intervals by consultant pharmacologists and psychiatrists experienced in benzodiazepine withdrawal. Patients were free to attend a support group or to be referred for psychological treatment if indicated. Those who dropped out were able to continue attending the clinic (and many were later successful in benzodiazepine withdrawal).

We feel that our study was useful in demonstrating that the use of buspirone in benzodiazepine withdrawal was associated with an increased drop-out rate, and possibly more severe symptoms, compared with dosage reduction under placebo. Our study does not perpetuate the use of a pharmacological short-cut; on the contrary, it shows that one pharmacological treatment is a cul-de-sac.

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Anorexia nervosa across cultures

SIR: I was excited to read the letter by Khandelwal & Saxena (*Journal*, November 1990, **157**, 784) who reported that in India anorexia nervosa (AN) is not only rare but is also not associated with any body image distortion and fear of obesity. As these have been regarded as 'core psychopathology' of AN and are of diagnostic importance in the West, their patients do not fulful DSM–III–R criteria and are placed in the residual category of 'eating disorders not otherwise specified'. However, they were noted to be amenorrheic and rigidly maintain a low body weight just as Western AN patients do. These findings echoed my study of Chinese AN patients in Hong Kong, where a clear distorted body image or an intense fear of obesity is lacking (Lee *et al*, 1989).

AN has often been described as a culture-bound syndrome. While there is more evidence on the greater prevalence of AN in Western than non-Western countries, much less information is available as to whether its clinical patterns also differ across cultures. I believe that they do, and would suggest that more

detailed case histories of these Indian patients should be provided for cross-cultural comparison. Besides, although the three Asian adolescents reported by Bhadrinath (1990) had "considerable fears of getting fat and body image disturbances" and met DSM–III–R criteria, it must be noted that they all came to the UK as a baby or as a child and thus grew up with Western notions of slimness and dietary preoccupations. This phenomenon of acculturation has been studied by Mumford & Whitehouse (1988), who found bulimia nervosa to be less common among white girls than second-generation young British Asians, who are quick to learn fashionable Western styles of reacting to stress.

Diagnostic criteria for AN need to be appreciated in the context of the attitudes to food, eating and body shape of a particular culture. It is vital that psychiatrists from other non-Western cultures do not apply DSM–III–R rigidly, and report on any atypicality in the AN patients they see. This will contribute to important cross-cultural data and a more culture-free understanding of abnormal female fasting, which has a long history, and in Western countries has happened to be defined in 'atheoretical' diagnostic manuals as the AN which most of us recognise today. There is really a difference between 'culture-bound' and 'concept-bound' entities.

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SIR: The recent surge of interest by professionals in eating disorders among Asian people is long overdue. Bhadrinath's highlighting of the condition (*Journal*, April 1990, **156**, 565–568) described an important phenomenon which is probably far more common than has so far been reported. Certainly the EAT score data of Dolan *et al* (*Journal*, October 1990, **157**, 523–528) would suggest this to be so.

From our own recent experience in treating a 13-year-old Asian girl, who fulfilled DSM–III–R criteria for anorexia nervosa, there was evidence to suggest that the condition was precipitated and maintained by features associated with problems relating to cultural conflict. The increased sensitivity to body appearance, common during adolescence, was heightened by the cultural conflict she experienced while negotiating the task of being Asian in the United Kingdom.

One of her main reasons for not eating was the desire to look and dress like 'white models' in fashion magazines and in this way feel more part of western culture at the same time as differentiating herself from that of her parents.

Further work is needed to explore the epidemiology of eating disorders among Asians and the hypothesis that the pursuit of 'westernness' contributes to the aetiology needs to be investigated.

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SIR: I read with interest the comments of Khandelwal & Saxena (*Journal*, November 1990, **157**, 784) in response to Badrinath's article (*Journal*, April 1990, **156**, 565–568).

The variance of anorexia nervosa described by Drs Khandelwal & Saxena highlights an important deficit in the diagnostic practice which fails to distinguish genuine anorexia nervosa from the atypical one. The young female described by them with decreased appetite, excessive weight loss and amenorrhoea, but no clear body image disturbance or fear of becoming fat, may have something else but not 'genuine' anorexia nervosa. Such differentiation becomes more important when we try to understand this disorder in the cross-cultural context.

The concept of anorexia nervosa suggests that the weight loss, emaciation and other characteristics are secondary to a relentless pursuit of thinness which appears to be the primary preoccupation in these patients. Mixing the concepts of genuine anorexia nervosa and the one which is not characterised by this preoccupation about thinness defeats the very purpose of understanding it conceptually and culturally.

When Simmonds (1914), a pathologist, reported a destructive lesion in the pituitary gland, every case of malnutrition was explained as caused by some endocrine pathology (Brusch, 1975). Over the next two decades genuine anorexia nervosa was filtered away from Simmonds disease. Now, when the understanding of its psychopathogenesis and evolution is becoming clearer, it will be a backward step not to