but bases it on a misreading of our data. When a patient has two diagnoses, each of which has a low BMI as a diagnostic criterion, clearly the concept of a premorbid body weight must be contentious. We, however, have no doubt that our patient had anorexia nervosa. She demonstrated a phobic avoidance of normal body-weight which Crisp (1980) describes as the pathognomonic feature of anorexia nervosa, that is, the feature which clearly demarcates anorexia from all other psychological and physical conditions. In addition, our patient exhibited anorectic behaviour, particularly self-starvation, but the *Journal*'s editorial deletions prevented us from going into details of the psychopathology. (We would not, of course, dispute that a number of psychological factors would be common between the two disorders and act synergistically, and indeed say that in our article.)

Dr Lee states that our patient was "happiest" when 23-years old during her only sexual relationship: we do not say this in the paper and it was not true. Dr Lee chides us, stating that our patient could have had anorexia at this time. There is no need to chide because we totally agree! – and say it in our paper. We believe her anorexia began at 21 years when our patient was 41 kg and had a body mass index of 13.6 (the psychological reasons are given in our paper).

Dr Lee's anxieties arise because of the difficulties inherent in DSM–III–R criteria for anorexia nervosa. Emphasis on a necessary weight loss of 15% of standard body-weight begs questions about what the psychiatrist does with a patient who loses, say, 14.5%, and even more about what "standard body-weight" means. "Amenorrhoea" is useless when a patient, such as ours, describes primary amenorrhoea. We completely agree with Dr Lee that terms such as "intense fear of obesity" or "body image distortion" are difficult to define, non-specific and demonstrated by too many "normal" Western women to have much diagnostic significance. We have ourselves reported this in our own studies (Birtchnell *et al*, 1985; Dolan *et al*, 1987*a*,*b*). It is for these reasons that we strongly urge Crisp's diagnostic criteria. Although these may appear complicated, that is only to be expected for anorexia nervosa is a complicated disorder. Crisp's emphasis on a core psychopathology, with concepts such as body weight, amenorrhoea and disturbed eating patterns taking a necessary second place, give clarity to the diagnosis and would prevent the sort of confusions outlined in Dr Lee's letter.

BIRTCHNELL, S. A., LACEY, J. H. & HARTE, A. (1985) Body image distortion in bulimia nervosa. *British Journal of Psychiatry*, **147**, 408–412.

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—, — & — (1987*b*) Body image distortion in non-eating disordered women and men. *Journal of Psychosomatic Research*, **31**, 513–520.

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Propofol and ECT

SIR: We read with great interest Pippard's audit of ECT in two National Health Service Regions (*Journal*, May 1992, **160**, 621–637). We would like to comment further on his finding of inconsistencies in the choice of anaesthetic. Pippard found propofol was always used in one of 29 ECT clinics he visited and sometimes used in a further two. This causes concern since, although propofol has anaesthetic advantages, namely smooth induction and rapid recovery, it shortens seizure duration in ECT (e.g. Dwyer *et al*, 1988; Simpson *et al*, 1988) and appears to raise the convulsive threshold (Lowson *et al*, 1990), both serious drawbacks for effective ECT. Pippard concludes that propofol should not be used in ECT unless the anaesthetic indications are particularly strong, a sentiment endorsed by the American Psychiatric Association (1990).

In a questionnaire survey of all anaesthetists working in the North West Health Region ($n=460$) carried out between November 1990 and February 1991 we examined views on anaesthetic practice. Completed questionnaires were returned by 261 anaesthetists (57%), all of whom had anaesthetised for ECT at some point in their careers and 128 (49%) who had done so within the last six months. In response to the question 'What anaesthetic do you regard as the drug of choice for ECT?' we obtained the following responses: methohexitone – 74% (192); thiopentone – 11% (30); propofol – 11% (30); methohexitone and propofol equally suitable – 2% (5); no preference between methohexitone, thiopentone and propofol – 2% (4).

A second question asked whether respondents had personal experience of using propofol in ECT; 25% (64) had.

Clearly a significant proportion of anaesthetists are using propofol, thereby increasing the chance that patients will experience inadequate seizures or failure to convulse. This risk is exacerbated since early constant current machines are still in use which

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are incapable of delivering effective stimuli to patients with high seizure thresholds. Furthermore, as Pippard commented, individual preference for different anaesthetics would make stimulus quantification for individual patients more complicated should such a policy be introduced in this country.

Anaesthetic ignorance of the problems inherent in using propofol in ECT is the most likely explanation for its frequent use. In turn this can be seen as a consequence of psychiatrists neglecting ECT and of their poor liaison with anaesthetists. We suggest that closer liaison between the two professions is added to Pippard's list of recommendations, and furthermore stress that at present propofol should be avoided, where possible, in ECT anaesthesia.

AMERICAN PSYCHIATRIC ASSOCIATION (1990) *Task Force on ECT: The Practice of ECT: Recommendations for Treatment, Training and Privileging*. Washington DC: APA.

DWYER, R., MCCAUGHEY, J., LAVERY, J., *et al* (1988) Comparison of propofol and methohexitone as anaesthetic agents for electroconvulsive therapy. *Anaesthesia*, **43**, 459–462.

LOWSON S., GENT, J. P. & GOODCHILD, C. S. (1990) Anticonvulsant properties of propofol and thiopentone: comparison using two tests in laboratory mice. *British Journal of Anaesthesia*, **64**, 59–63.

SIMPSON, K. H., HALSALL, P. J., CARR, C. M. E., *et al* (1988) Propofol reduces seizure duration in patients having anaesthesia for electroconvulsive therapy. *British Journal of Anaesthesia*, **61**, 343–344.

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Ventricular size in schizophrenia

SIR: Van Horn & McManus's meta-analysis of ventricular enlargement in schizophrenia (*Journal*, May 1992, **160**, 687–697) provides a valuable review of the evidence, but I would like to take issue with their conclusions. They point out that all the studies have shown a wide variance around the means – in other words there is a very considerable overlap between the scores of 'controls' and of 'schizophrenics'. This is true even of the important study of discordant identical twins by Suddath *et al* (1990), which they mention in their text but do not include in their table.

The conclusion should be *not* that "schizophrenics indubitably have larger ventricles than controls", but that "while *some* schizophrenics have larger ventricles than controls, *most* schizophrenics' VBRs are within the normal range." The difference is important, because the issue is not one of specificity of diagnosis but of inferences about aetiology. The simple statement 'schizophrenics have larger ventricles than controls' should be answered 'No' in any MRC Psych MCQ question.

SUDDATH, R. L. CHRISTISON, G. W., TORREY, E. F., *et al* (1990) Anatomical abnormalities in the brains of monozygotic twins discordant for schizophrenia. *New England Journal of Medicine*, **322**, 789–794.

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Hemisphere dysfunction in psychiatric disorders

SIR: Dr Cutting's interesting article on the role of hemisphere dysfunction in psychiatric disorders (*Journal*, May 1992, **160**, 583–588), although generally informative and balanced, did appear to set up poor Flor-Henry as an Aunt Sally. His arguments, especially in his later writings, are more sophisticated than one would gather from this article. In particular, he makes clear distinctions between the consequences of actively discharging lesions compared with those of areas of passive neuronal destruction, and he uses the concept of reciprocal inhibition especially between corresponding areas in opposite hemispheres. Both concepts have their origin in the writings of Hughlings Jackson. If used, Dr Cutting's anti Flor-Henry argument from the evidence provided by the results of temporal lobectomy turns out to be in fact a pro Flor-Henry one.

Moreover, some of the earliest work on the distinction between right and left hemisphere depressions was carried out by Fromm-Auch who was working in very close association with Flor-Henry at the time.

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SIR: In the review by Cutting of the role of hemispheric cerebral dysfunction in the genesis of psychiatric disorders (*Journal*, May 1992, **160**, 583–588) he suggests that disorders of the left cerebral hemisphere are related to an increased incidence of severe depressive disorder. In order to address this question I have recently analysed the results from 41 consecutive patients admitted with subarachnoid haemorrhage to compare the site of the subarachnoid haemorrhage with the development of depression, something that has not previously been done in this group of patients. These patients were assessed as part of a study investigating the incidence of depression in acutely ill medical patients (Silverstone,

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