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.

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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,

1990). The site of bleeding was assessed by computerised tomography (CT) scanning (41 patients) and intracerebral arteriography (38 patients).

The presence of depression was assessed by means of the Montgomery-Åsberg Depression Rating Scale (Montgomery & Åsberg, 1979), which was modified to decrease the bias involved when using somatic systems to diagnose depression in the medically ill. The modified scale excluded scores on the items for sleep disturbance, poor appetite, lethargy, and poor concentration. Those who scored above seven on this modified depression rating scale during their first week of admission to a regional neurosurgical unit, on average some seven days following the development of their haemorrhage, were categorised as depressed. Using this method, 11 patients were identified as being depressed, and the sites of the arterial haemorrhage in these patients were as follows: anterior communicating artery ($n=3$), right internal carotid artery ($n=4$), basilar or vertebral artery ($n=3$), with no bleeding site being identified in one patient. In the 30 non-depressed patients (some of whom had multiple aneurysms) the site of arterial haemorrhage was anterior communicating artery ($n=13$), right internal carotid artery ($n=1$), left internal carotid artery ($n=5$), right middle cerebral artery ($n=4$), left posterior communicating artery ($n=3$), basilar or vertebral artery ($n=3$), and no site identified ($n=3$). Thus, of the 11 patients who were depressed, 5 had haemorrhages in the right hemisphere and only 2 had a haemorrhage in the left hemisphere.

These results, which do not support the suggestion by Cutting that left hemisphere dysfunction increases the risk of depression, are consistent with studies regarding the incidence of depression following other forms of brain insult. For example, Flor-Henry (1969) suggested that it was right hemispheric dysfunction that may be important in the genesis of depression among patients with epilepsy. Achte *et al* (1969) in a study of cases of affective disorder following brain injury found no relationship between the location of brain damage and the development of depression. More recently, Sharpe *et al* (1990) have found no link between the incidence of depression and the site of cerebrovascular haemorrhage identified using CT-scanning. Thus, at present I do not feel that the balance of evidence supports the suggestion by Cutting that there is a link between depression and changes in left hemisphere function. Instead it is more likely that the incidence of depression is increased following any brain insult, but that damage to one hemisphere rather than the other does not further increase the risk of depression developing.

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The first psychiatric use of lithium

SIR: Scott (*Journal*, May 1992, **160**, 709–710) wrote about S. Weir Mitchell's treatment of epilepsy with lithium bromide and wondered whether this was the first use of lithium in psychiatry. Epilepsy is hardly a psychiatric illness, but Mitchell also gave lithium bromide for emotional lability, hypervigilance, fatigue, reduced appetite, and poor sleep (Mitchell, 1877), thus continuing its use as a tonic (Gibb, 1865) and a sedative (Lévy, 1874). Garrod (1859) had recommended lithium salts for "uric acid diathesis", which included "gouty mania", and Hammond (1871) used lithium bromide for "acute mania with depression". A lithium salt not containing bromine, lithium carbonate, was used on a psychiatric indication by Carl Lange (1886), who gave it to patients with periodic depressions, and by Frederik Lange (1894), who used it for the acute treatment of depression. The early authors based their claims of therapeutic efficacy on theoretical arguments and clinical impressions illustrated by case histories; they did not provide any quantitative documentation. Lithium went out of psychiatry and did not re-enter it until 1949.

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