of the number of right-dominant and left-dominant subjects in the sample. How many left-unilateral, and how many right-sided treatments were administered? Was the probability of a homolateral EEG slowing after unilateral ECT approximately equal in both hemispheres, irrespective of dominance?

We were puzzled by Table III which shows the assessment of type of ECT from a blind comparison of EEG before and after the course of ECT. That table shows that 24 out of 59 records were incorrectly classified, but fails to indicate what these records portrayed. We are particularly interested to know if any of the nine incorrectly classified records after bilateral ECT were lateralized, and to what side. We have examined records before and after a course of unilateral or bilateral ECT in 85 depressed subjects. The electroencephalographer was not aware of the type of ECT administered. The slowing after unilateral ECT ($n = 34$) was pronounced over the side of the placement of treatment electrodes. Bilateral ECT ($n = 51$) elicited left-sided slowing primarily. This expected finding is shown in the attached table.

Another question relates to the comment that the authors did not see any 'evidence to suggest that EEG changes were correlated with clinical improvement or otherwise'. We would like to know how EEG quantification was done to determine this relationship. In earlier studies, EEG slowing was shown to be a necessary, though not sufficient, condition for the behavioural response to ECT (Roth *et al.*, 1952; Fink and Kahn, 1957); and these results were arrived at principally because the authors attempted more than a descriptive estimate.

J. Volavka.
R. Abrams.

Department of Psychiatry,
New York Medical College,
Five East 102nd Street,
New York, N.Y. 10029.

EEG changes with bilateral ECT

Accentuation of the slowing	Pre-ECT	Post-ECT	Total
Left	6	28	34
Symmetric	40	18	58
Right	5	5	10
Total	51	51	102

($\chi^2 = 22{\cdot}6$, $p + 0{\cdot}001$)

References

Fink, M., and Kahn, R. L. (1957). 'Relation of electroencephalographic delta activity to behavioral response in electroshock.' *Arch. Neurol. Psychiat.*, **78**, 516–25.

Roth, M. (1952). 'A theory of ECT action and its bearing on the biological significance of epilepsy.' *J. ment. Sci.*, **98**, 44–59.

Dear Sir,

Thank you for giving us an opportunity to reply to this interesting and important letter.

1. In our sample, in which EEG measurements were completed, we had 57 left-dominant and 2 right-dominant subjects, The 2 right-dominant subjects were given bilateral ECT (quite by the chance of random selection), and consequently all 22 patients given 'dominant ECT' had left-sided ECT and all 18 patients given 'non-dominant ECT' had right-sided ECT.

2. The details of changes after unilateral ECT are shown below:

22 patients given dominant ECT (i.e. left-sided).	18 patients given non-dominant ECT (i.e. right-sided).
14 correct (left-sided slowing).	11 correct (right-sided slowing).
5 bilateral slowing.	2 bilateral slowing.
1 contralateral slowing.	2 contralateral slowing.
2 no change.	3 no change.

3. The changes after bilateral ECT were:
10 correct forecasts (bilateral slowing)
5 showed right-sided slowing (all left-dominant patients)
2 showed left-sided slowing (left-dominant patients)
1 showed no change (left-dominant patient)
1 had a temporal lobe abnormality (left-dominant patient).

The 2 patients who were right-dominant were correctly forecast, i.e. had bilateral slowing after bilateral ECT. We have no evidence, therefore, to support Drs. Volavka and Abrams' finding of dominant slowing after bilateral ECT.

4. Finally, our measurements of EEG changes and clinical improvement were:
EEG changes: minimal, moderate, marked.
Clinical improvement: no improvement, improvement, much improved.

It was found that those patients who showed a marked EEG abnormality after ECT did not necessarily show the greatest clinical improvement, and of 6 patients who showed no detectable EEG change after ECT 4 showed 'improvement', 1 was 'much improved' and 1 showed 'no improvement'.

Diana Knight.
Edmond Sutherland.

St. Crispin Hospital,
Duston,
Northampton, NN5 6UN.