Post-stroke depression

We read with great interest the paper by Gainotti *et al* (1999) and we wish to raise some concerns about their study.

Description of the statistical analysis was omitted from the paper and it is impossible for the reader to know how the data were analysed. The authors did not mention whether the patients were administered drugs usually given to stroke patients (e.g. steroids, beta-blockers and anticonvulsant drugs) and which can induce some depressive symptoms. In the section entitled "Criteria used to make a diagnosis of major depression", the authors state that the validity of their diagnostic criterion (Hamilton Rating Scale for Depression (HAM–D) score >17) has been documented by others (Salzman *et al*, 1994). This assertion is false – we find no scientific support in the Salzman *et al* paper for Gainotti *et al*'s assertion. Also, information on the clinical features of Gainotti *et al*'s sample is limited by their omission to report standard deviations and the mean age of patients with endogenous depression (see Table 1, p. 164).

The most important problem, however, is with regard to the interpretation of HAM–D scores across groups. It is not made clear whether the mean HAM–D scores reported in Table 1 are calculated across the whole sample or only within the group with depression. In the psychiatric literature a HAM–D score of <12 is not generally considered clinically significant, so if Gainotti *et al* have calculated mean HAM–D score only within the group with depression, it is difficult to understand why the mean is so low (11.8 at <2 months post-stroke). Alternatively, if mean HAM–D scores were calculated across the whole sample (i.e. patients with and without depression), the increase in the mean with increasing time post-stroke may be due simply to the increase in the relative number of people with depression (27% at <2 and 2–4 months post-stroke *v.* 40% at >4 months post-stroke). In other words, the increase in the mean HAM–D score does not necessarily imply that the severity of depression in the whole sample increases from the acute to the later post-stroke period, but this tendency may be due simply to an increased proportion of patients with depression within the sample.

Finally, Cohen's κ for diagnostic concordance is not given by Gainotti *et al*. This index is routinely calculated when different diagnostic criteria are adopted for patient classification. Inspection of Table 1 shows that nine well subjects out of 43 were misclassified as having depression using the quantitative criterion of HAM–D score. This begs the question, how many patients with depression were classified as being well?

Gainotti, G., Azzoni, A. & Marra, C. (1999) Frequency, phenomenology and anatomical–clinical correlates of major post-stroke depression. *British Journal of Psychiatry*, **175**, 163–167.

Salzman, C., Schneider, L. S. & Alexopoulos, E. S. (1994) Pharmacological treatment of depression in late life. In *Psychopharmacology: The Fourth Generation of Progress* (eds F. E. Bloom & D. J. Kupfer), pp. 1471–1477. New York: Raven Press.

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Authors' reply: We would like to clarify some aspects of our paper in reply to the points highlighted by Di Michele & Bolino.

First, we would stress the fact that the preliminary analysis of our data had been extensive, but that only data relevant to the specific scope of our study, which consisted in checking the most recent version of Robinson and co-workers' biological theory of post-stroke depression, were included in the manuscript. Our data are clearly inconsistent with this theory.

Regarding our data analysis, continuous data were treated using one-way analysis of variance, whereas frequencies of distribution were analysed by means of χ^2 tests.

The possible influence of drugs was checked in our study by excluding all patients who were taking at the time of examination (or had taken in the previous four weeks) antidepressant drugs. We did not check other drugs (such as steroids, beta-blockers or anticonvulsants) which could induce depressive symptoms, since they were not considered relevant for the specific scope of our study.

The patients with endogenous depression were matched as for age (60.1 years) and educational level (7.9) with the three groups of post-stroke patients. Only a slight difference in gender distribution was observed between stroke patients and those with endogenous depression (a preponderance of females (20 : 10) among the group with endogenous depression). This not unexpected difference was not considered relevant with respect to the scope of the study.

Regarding interpretation of the HAM–D scores across groups, the main scope of our study consisted in determining whether the nature of post-stroke depression is different in the acute and in more chronic post-stroke periods. From this point of view, it was important to evaluate at various time intervals from stroke the qualitative aspects of depression and their anatomical–clinical correlates, whereas the severity of depression in patients with major depression was much less relevant. For this reason, the HAM–D scores were calculated, as the authors of the letter correctly argue, in each group as a whole (including subjects with and without depression) and the increment of the mean depression score across groups mainly reflected the relative increment of subjects with depression. Though this fact is not very relevant to the aim of our research, we must add that even considering only the patients with major post-stroke depression, we could observe a non-significant trend towards an increase in the mean HAM–D score from the acute (20.2) to the post-acute (21.8) and to the more chronic (23.5) post-stroke period.

Concerning the HAM–D score criterion for diagnosis of major depression and correlations between clinical and psychometric criteria, although different cut-off scores have been proposed in the literature, a score of 18 on the HAM–D is the most currently used (Endicott *et al*, 1981; Rapp *et al*, 1990). Furthermore, good concordance exists in our study between clinical (DSM–III–R) and psychometric (score >17 on the HAM–D) criteria. We have measured this concordance on our data by the κ statistic (Holman *et al*, 1982), which gives a numerical measure of chance-corrected categorical agreement. According to this index, which results from the ratio between the chance-corrected observed agreement and the chance-corrected perfect agreement, the perfect agreement corresponds to +1, the complete disagreement corresponds to −1 and the chance level is 0. The chance-corrected level of agreement between DSM–III–R criteria and HAM–D score >17 was quite satisfactory in our study (κ=0.84).

Endicott, J., Cohen, J., Nee, J., *et al* (1981) Hamilton Depression Rating Scale: Extracted from regular and change version of the Schedule for Affective Disorders

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