Correspondence

EDITED BY LOUISE HOWARD

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Authorship of clinical trial reports

I was most interested to read the report by Baldwin *et al* (1999), particularly as I am listed as a co-author, although I had never seen the text before and did not know it was being submitted for publication! How did such a situation arise and how can its repetition be avoided in future?

In many countries, particularly the USA, it is editorial policy to obtain consent to submission that includes *all* of those who participated directly in the work described; often to the extent of obtaining individual signatures. This is to be applauded. Working at the Feighner Research Institute in London, I personally treated 27 patients who were included in the above trial and informed the company concerned, on more than one occasion, that I wished to see the text of any manuscript submitted for publication prior to the event. But I received no reply.

Individual investigators should be named with their centres, rather than lumped together in an unspecified 'study group'. It would also be helpful, in the context of any statistical conclusions reached, if the number of cases contributed from each individual centre were to be recorded in published reports.

Baldwin, D., Bobes, J., Stein, D. J., *et al* (1999) Paroxetine in social phobia/social anxiety disorder. Randomised, double-blind, placebo-controlled study. *British Journal of Psychiatry*, **175**, 120–126.

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Authors' reply: We are grateful to Dr Wheatley for his comments on the publication of the treatment study with paroxetine, and would like to acknowledge publicly the contribution made by Dr Wheatley and his colleagues within the Feighner Research Institute. We thank all those principal investigators, their research teams and the patients who kindly took part in this investigation.

It is difficult to acknowledge all the clinicians who participate in large multicentre treatment studies, and naturally some investigators will be disappointed when their contribution is not credited as much as that of their colleagues. Ideally, publication plans should be discussed at meetings with potential investigators prior to the start of a study, and then reviewed during the course of the investigation. This is now the policy at SmithKline Beecham, but this study with paroxetine was started before the policy was in place. Due to a change in personnel within SmithKline Beecham, the communication between the sponsoring company and all the participating investigators has been less than optimal, for which we apologise.

In the future, SmithKline Beecham will try to ensure that all the participating investigators are happy with the publication plan before the start of any treatment study. However, the question of contribution to full authorship will remain problematical, for collaborative study groups and journals alike.

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Editor's response: The *Journal* does enforce a policy of obtaining the signatures of all authors before accepting a manuscript for publication. In this instance, confusion has arisen because of uncertainty over the authorship status of members of a study group. This issue has now been clarified in our 'Instructions to authors' as follows:

> The *Journal* does not consider to be authors people thanked in the Acknowledgements or listed as members of a study group on whose behalf a paper is submitted, but whose names do not appear as authors on the title page of the manuscript, or whose signed agreement to the manuscript's submission has not been obtained. It is the responsibility of the corresponding author to ensure that authorship is agreed among the study's workers, contributors of additional data and other interested parties, before submission of the manuscript.

Full text of these Instructions is located on the Royal College of Psychiatrists' web-site (http://www.rcpsych.ac.uk/pub/bjp_ita.htm) and is next scheduled for paper publication in the July issue of the *Journal*.

Psychological model of post-stroke major depression

Gainotti *et al* (1999) concluded that post-stroke major depression may fit with a psychological model rather than with a neurological model based on their findings that post-stroke patients with major depression displayed more reactive symptoms (emotionalism, catastrophic reaction, anxiety) and fewer endogenous symptoms than patients with endogenous depression.

There are some concerns regarding the conception and the methodology of the study. The major drawback in the methodology is the bias in selecting the control group. It is not surprising that patients with endogenous depression will have more endogenous symptoms than patients with post-stroke major depression, as major depression can be diagnosed using DSM–III–R (American Psychiatric Association, 1987) operational criteria in the absence of endogenous or melancholic symptoms. Further, it is also expected that affective symptoms related to brain damage, such as emotionalism and catastrophic reaction, will be more prevalent in post-stroke major depression than in patients with endogenous depression.

Gainotti *et al* seek to create an impression that there is no association between endogenous depression and psychological stressors, and that post-stroke major depression with symptoms such as anxiety and hyperemotionalism are the representation of a psychological reaction to stressful situations. The available literature fails to support the validity of dichotomous endogenous/reactive and endogenous/non-endogenous classifications (Farmer & McGuffin, 1989). Further, emotionalism observed in post-stroke patients is often

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precipitated by non-specific stimuli, and crying or tearfulness observed in emotionalism may not be associated with alterations in mood (Poeck, 1969). No attempt was made to examine whether the symptoms assumed to be stress-related, in stroke patients with major depression, were correlated with the severity of functional impairment or with subjective perception of stressful situations.

It would be of interest to investigate the differences in prevalence of endogenous or melancholic symptoms between post-stroke major depression and major depressive disorder. Moreover, using the same classificatory procedure in sub-typing post-stroke depression and depressive disorders may cause limitations and constraints when phenomenological comparisons are made between these two disorders. To overcome this problem, classification of post-stroke depressive disorders could be approached from the bottom up, with the identification of depressive symptoms in stroke patients. By applying multivariate analysis, these symptoms can be grouped into clusters or syndromes, which can then be validated.

American Psychiatric Association (1987) *Diagnostic and Statistical Manual of Mental Disorders* (3rd edn, revised) (DSM–III–R). Washington, DC: APA.

Farmer, A. & McGuffin, P. (1989) The classification of the depressions. Contemporary confusion revisited. *British Journal of Psychiatry*, **155**, 437–443.

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.

Poeck, K. (1969) Pathophysiology of emotional disorders associated with brain damage. In *Handbook of Clinical Neurology* (Vol. 3) (eds P. J. Viken & G. W. Bruyn), pp. 343–366. New York: N. Holland.

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Author's reply: Dr Ramasubbu argues against our suggestion that psychological rather than neurological factors mostly account for post-stroke depression (PSD). His/her main argument is that our results could be due to a bias in selecting a control group of patients with endogenous depression. He/she remarks that it is not surprising that endogenous symptoms were more frequent in this control group, whereas affective symptoms related to brain damage prevailed in patients with major PSD. This argument could certainly be appropriate if the aim of our study consisted in matching the main symptoms of patients with major PSD and with major endogenous depression, but this was clearly not the scope of our paper. Patients with major endogenous depression were, in fact, the group against which we matched various subgroups of patients with major PSD observed at various time intervals from stroke. The scope of these comparisons consisted in assessing whether patients with endogenous depression are more similar to patients with major PSD observed immediately after the stroke than to those observed in more chronic post-stroke periods. A similar strategy had been used in previous studies of this field conducted by our research group, since the distinction between various forms of PSD plays a critical role in the model of PSD proposed by Robinson *et al* (see Starkstein & Robinson, 1989, for review). This model is, in fact, substantially based on the distinction between two forms of PSD: (a) the major form, due to a lesion encroaching upon the left frontal lobes and indistinguishable from the clinical and pathophysiological points of view from the major forms of endogenous depression; and (b) the minor form, considered as a form of reactive (dysthymic) depression and having no specific anatomical substrate. In a previous paper (Gainotti *et al*, 1997) aiming to test this original version of the Robinson *et al* model, the symptomatological profiles of patients affected by endogenous depression were matched with those in three groups of stroke patients, on the basis of DSM–III–R criteria, as having no depression, minor PSD, or major PSD. The following predictions were made: (a) if major PSD is indistinguishable from endogenous depression, whereas minor PSD is a reactive form of depression, then the symptomatological profile of patients with a major form of PSD should be more similar to that of patients with endogenous depression than to that of patients with a form of minor PSD; (b) if, on the contrary, no qualitative difference exists between major and minor forms of PSD, then patients with major PSD should be more similar to those with a minor form of PSD than to those with a form of endogenous depression. Our results clearly supported the second prediction showing that a continuum exists between major and minor forms of PSD.

To account for these and other data also inconsistent with the Robinson *et al* model, Herrmann & Wallesch (1993) proposed a restricted version of the model which assumes that only the forms of major PSD observed in the acute post-stroke periods are very similar to endogenous depression, whereas those observed in more chronic stages must be considered as reactive forms, mainly due to psychosocial factors.

To test this new version of the Robinson *et al* model with a strategy similar to that used in our previous paper, we matched the symptomatological profiles of patients with major PSD observed at various time intervals from stroke with those of patients with endogenous depression. Since the profiles of patients with major PSD observed at various time intervals from stroke were very similar, and were very different from those in patients with endogenous depression, we concluded that even the restricted version of the Robinson *et al* model is inconsistent with our data.

We therefore think that no methodological defect exists in this or in our previous study and that our data allow us to conclude that no qualitative difference exists either between minor and major forms of PSD or between forms of major PSD that arise at various time intervals from stroke.

Let us pass now to our suggestion that psychological factors, rather than neurological factors, mostly account for PSD. This suggestion was mostly due to the distinction that we have more analytically described in our previous study (Gainotti *et al*, 1997) between motivated and unmotivated affective patterns. The term 'motivated reactions' refers not only to the reactive symptoms of anxiety, emotionalism and catastrophic reactions, whose prevalence in patients with PSD could be due (as Dr Ramasubbu suggests) to a bias in the selection of the control group, but also to a qualitative analysis of the responses given by patients in sections of the Post-Stroke Depression Rating Scale (PSDRS) devised to evaluate 'depressed mood', 'guilt feelings' and 'thoughts of death and/or suicide'. In these sections, patients were requested to qualify their response by saying whether their bad mood, guilt feelings and thoughts of death were related to their actual condition or were independent from it. Patients with major or minor forms of PSD usually attributed depression to the consequences of stroke, felt guilty because they considered their previous lifestyle as partly responsible for their actual disease, and had stroke-related death worries, whereas patients with endogenous depression attributed guilt feelings to their moral

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