studies in the literature but not all. While several studies have demonstrated elevated MAO activity in agoraphobia with panic attacks (DSM-III criteria) there are methodological shortcomings with these studies (Norman et al, 1988). Furthermore, in the largest study undertaken to date (Khan et al, 1986) no difference was found between panic disorder, agoraphobia or generalised anxiety disorder (GAD) and a control group. More recently, Norman et al (1988) found no differences in either  $K_m$  (affinity constant) or  $V_{\text{max}}$  (maximum uptake velocity) of platelet MAO between patients with panic disorder or agoraphobia with panic attacks (DSM-III criteria) and normal controls. These conflicting results are difficult to explain but clearly studies in small numbers of patients using diverse methodological refinements correctly require circumspect interpretation.

The finding of increased platelet 5-HT uptake in 'neurotic' depression is of interest in the context of a hypothesised decreased 5-HT availability in depressive illness. Most reports find decreased 5-HT uptake in endogenous depression but no difference from controls in non-endogenous (neurotic (sic)) depression (Langer et al, 1987). This is again a finding which requires cautious interpretation given the small number of patients involved. In the agoraphobic group it was stated that there was "no association between the 5-HT uptake kinetics and the central symptoms of agoraphobia". It would have been interesting to have known the relationship with panic attacks, particularly as a significant negative correlation between  $V_{\text{max}}$  of uptake and the tense-panicky dimension of the fear questionnaire was reported (r = -0.44; P < 0.05). We have previously reported an elevated  $V_{\text{max}}$  of platelet serotonin uptake in panic disorder and agoraphobia with panic attacks (Norman et al, 1989). Certainly, these studies have implications for a serotonin-overactivity hypothesis of panic disorders, which is supported by some neuroendocrine challenge tests (Khan et al, 1988). It does not necessarily follow that unchanged <sup>3</sup>Himipramine binding in panic disorder supports the unchanged platelet serotonin kinetics. It is notable that dissociation of 5-HT uptake and <sup>3</sup>H-imipramine binding sites has been observed. The more likely association between 5-HT uptake and 3H-imipramine binding site is that of an allosteric control exerted by the latter over the 5-HT transporter (see Langer et al, 1987). While <sup>3</sup>H-imipramine binding may be normal, 5-HT uptake can be altered, a finding we noted in patients with panic attacks (Norman et al, 1989).

It is unusual that in the group of agoraphobic patients studied by Dr Flaskos *et al* (1989) the presence or absence of panic attacks is neither com-

mented on nor quantified. This additional information would facilitate the comparison of their results with those that already exist in the literature.

TREVOR R. NORMAN FIONA K. JUDD IAIN M. McIntyre

Department of Psychiatry University of Melbourne Austin Hospital Heidelberg Victoria 3084, Australia

#### References

AMERICAN PSYCHIATRIC ASSOCIATION (1978) Diagnostic and Statistical Manual of Mental Disorders (3rd edn) (DSM-III). Washington, DC: APA.

KHAN, A., LEE, E., DAGER, S., et al (1986) Platelet MAO-B activity in anxiety and depression. Biological Psychiatry, 21, 847-857.

 ASNIS, G. M., WETZLER, S., et al (1988) Neuroendocrine evidence for serotonin receptor hypersensitivity in panic disorder. Psychopharmacology, 96, 360-364.

Langer, S. Z., Galzin, A. M., Poirier, M. F., et al (1987) Association of [<sup>3</sup>H]-imipramine and [<sup>3</sup>H]-paroxetine binding with the 5-HT transporter in brain and platelets: relevance to studies in depression. *Journal of Receptor Research*, 7, 499-521.

NORMAN, T. R., ACEVEDO, A., McIntyre, I. M., et al (1988) A kinetic analysis of platelet monoamine oxidase activity in patients with panic attacks. *Journal of Affective Disorders*, 15, 127-130.

—, BURROWS, G. D., JUDD, F. K., et al (1989) Serotonin and panic disorders: a review of clinical studies. *International Journal of Clinical Pharmacology Research*, 9, 151-157.

# 'Le Suicide'

SIR: I would like to thank Berrios & Mohanna (Journal, January 1990, 156, 1-9) for their cogent and illuminating critique of Durkheim's use of psychiatric texts in Le Suicide. The main thrust of their argument is that Durkheim is both 'selective' and 'idiosyncratic' in his choice of psychiatric material and terminology. I am left wondering, however, just how many contemporary articles would stand up to so rigorous an appraisal, as I am sure it cannot be only late 19th century social scientists who are selective and idiosyncratic in their choice of material. For example (and not because of particular dismerit), many World Health Organisation (WHO) transcultural studies play down or de-emphasise anthropological explanations or accounts of the phenomena they study (Kleinman, 1987).

Drs Berrios & Mohanna have themselves been selective, if not idiosyncratic, in choosing "not to deal, in any way, with Durkheim's sociological views", explaining instead that they will do this in a separate publication. Durkheim's undoubted methodological sleights-of-hand become more understandable when seen in the broader context of his other works. I would suggest that Le Suicide can be seen as an,

admittedly flawed, sociological and statistical study of suicide rates as an index of pathological forms of the division of labour. This stems from a positivist desire to study rates, rules and types of social phenomena rather than enquiring about (unknowable) individual intentionality. This is one solution to a dilemma not unknown to psychiatrists attempting to make sense of phenomenological data through the use of epidemiology and operational criteria.

KEITH LLOYD

Bethlem Royal Hospital Beckenham Kent BR3 3BX

#### Reference

KLEINMAN, A. (1987) Anthropology and psychiatry: the role of culture in cross-cultural research on illness. *British Journal of Psychiatry*, 151, 447-454.

### There are none so double-blind . . .

SIR: Oxtoby et al (Journal, November 1989, 155, 700–701) raise important points in their significant and elegant article.

Surely journal editors should follow the policy they imply? No article should be accepted for publication, which includes the claim to have been a 'double-blind' drug trial, unless it contains clear data establishing the validity of that claim. It is decidedly odd that hitherto, editors have required only the *simulation* of double-blinding, rather than the reality of how the technique is effectively used.

The experience reported by Oxtoby et al, of failing to gain publication of their valid and important critiques, raises another most important caution concerning the scientific literature. In my experience there is in practice a significant rate of rejection of articles and letters critical of substantial methodological flaws in studies; a rejection rate that is unrelated to the truth or relevance of the criticisms, or to the quality of the written submission. There may be several reasons for this. Editors and reviewers (consciously and unconsciously) might not be well disposed to submissions that demonstrate serious flaws in articles they have accepted for publication; especially if the authors of the faulty articles are significant establishment figures.

Another problem arises from the attempts to seek 'peer' review. Items obviously need to be reviewed by experts. However, especially within some fields, a regular ring of reviewers develops, whose personal and emotional investment in particular views lead them to urge rejection of contrary ones. One should be very cautious of using reviewers too prominent or dominant within their field.

This was well demonstrated recently, when this Journal (Simpson, Journal, October 1989, 155, 565) published a letter in which I criticised some of the many flaws in the burgeoning fad literature on multiple personality disorder (MPD). I have since received numerous sustaining and supportive letters from American readers, delighted to have seen criticism of this sacred cow of the psychosocial literature actually appear in print. In North America, it is almost impossible for such critical views on MPD to appear in print because all such submissions are reflexly sent for review to a small circle of devotees of MPD, who reject them.

As scientists, authors, reviewers and editors, we should value sincere and informed critics far more highly, for they are a valuable but endangered species. Our disciplines need to treasure iconoclasts and there are more than enough of these about.

MICHAEL A. SIMPSON

PO Box 51, Pretoria 0001 South Africa

## Malaria presenting as atypical depression

SIR: We report here a case of a patient with cerebral malaria who presented in the UK with a hysterical stupor occurring in the context of an atypical depression.

Case Report: A 30-year-old woman was admitted under Section 2 of the Mental Health Act (1983) following a domiciliary visit. She had been stuporose for 48 hours and had stopped eating and drinking. On returning from a day trip to the seaside she had been quite unable to get out of the car because of her semistuporose condition and resisted any attempts to move her, even by force. Prior to this episode she had complained of bouts of extreme tiredness, depression and irritability for about four months. She also had sleep reversal, with daytime sleepiness and nocturnal insomnia. There was no weight loss, but appetite was impaired. Her family history was negative for all psychiatric disorders, but seven years previously the patient had taken an overdose following the break-up of a relationship. Early life and schooling were unremarkable and she had been successful in her career as an information officer. At the time of the onset of depression, she had been in dispute with a girlfriend with whom she had shared a flat. In addition, six weeks prior to admission she had got married and reported some difficulties in forming a relationship with her stepson. It was thought that her recent life events were sufficient cause for her depression. Her general practitioner (GP) had started treatment with imipramine with a good initial response. However, she stopped the drug because of side effects and this appeared to coincide with the onset of deterioration in her clinical state and the onset of stupor.

During the admission to hospital, she took to her bed for two days every 3-4 days, complaining of severe exhaustion