Obsessive-Compulsive Rituals

SIR: Marks et al (Journal, April 1988, 152, 522-534) have recently reported on the effects of clomipramine and various modalities of behavioural therapy in a double-blind trial involving 49 ritualising obsessive-compulsive patients. We feel that the review of literature and the interpretation of findings provided by the authors may not be fully correct.

The authors suggest that no studies support the therapeutic superiority of clomipramine in comparison with other tricyclic drugs in treating obsessive-compulsive disorder (OCD). This statement overlooks a recent comparative study by Zohar & Insel (1987). A counterbalanced withinsubjects crossover comparison of clomipramine and desipramine found clomipramine to be therapeutically superior to desipramine in treating OCD. We feel that several further comments are justified regarding the methods and conclusions of the study. Findings of clomipramine v. placebo are described in the following manner: "... 26 weeks of clomipramine compared with placebo yielded limited and transient benefit in the first eight weeks only". It is only fair to point out that any effects beyond week 8 are confounded by virtually asymptotic performance of the clomipramine and placebo groups, and a concomitant change in methods. The interested reader is referred to the paper of Kasvikis & Marks (1988), which discusses this trial's methodology further, and notes that therapist-assisted exposure was added following week 8.

Thus, only the initial 8 weeks of this trial offer a relatively unbiased estimate of the therapeutic effect of clomipramine v. placebo. Indeed, inspection of Table 2 and Fig. 2 of the study in fact indicates a significant therapeutic effect of clomipramine in comparison with placebo for the period in question, with respect to target rituals time, global rituals time, target rituals discomfort, level of depression, and social leisure adjustment. As noted, findings do not appear to be transient, as witnessed by the essentially flat slopes of all functions graphed in Fig. 2 beyond week 8. Thus, it remains somewhat unclear what would be adequate in the authors' minds to consider a drug v. placebo difference as clinically significant or durable.

The authors describe clomipramine as having "a limited adjuvant role". It is noteworthy that another aspect of the design of the initial 8-week phase of this study may have masked any more direct role. During the initial period of assessment (weeks 1-8) all patients were preselected to ensure that they would be responsive to behavioural intervention, and all patients except those in group Cé (i.e. the antiexposure group) were required to undergo up to 3 hours of

self-exposure therapy each day. It is unlikely that any other treatment would have much effect, or for that matter could have much effect, given this magnitude of exposure.

The method used for the trial makes it unfit to hierarchise the therapeutic factors. The claim that self-exposure comes first, followed by clomipramine and by therapist-aided exposure is not warranted, as the design was indeed set up as a comparative study of the adjuvants to exposure. Thus, it merely shows that clomipramine is the best ancillary therapeutic factor once the decision was made to use exposure as the axis of the treatment.

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References

KASVIKIS, Y. & MARKS, I. (1988) Clomipramine in obsessive compulsive ritualizers treated with exposure therapy: relations between dose, plasma level, outcomes and side effects. *Psychopharmacology*, 95, 113–118.

ZOHAR, J. & INSEL, T. (1987) Obsessive compulsive disorder: psychological approaches to diagnosis, treatment, and pathophysiology. *Biological Psychiatry*, 22, 667-687.

Depersonalisation and Self-Perception

SIR: I share with Dr O'Shea (Journal, November 1988, 153, 709) the belief handed down by Mayer-Gross (1935) that depersonalisation is attributable to a "preformed functional response of the brain". My argument is teleological. Why does this response exist? What is its purpose? The answer lies, I believe, in its occurrence in life-threatening situations (Noves & Kletti, 1977), in which it probably has significant survival value. The victims of such situations experience emotional, cognitive, and somatic detachment; a dissociation between the observing and participating 'self'. Thus, they are able to take action that enhances their chance of survival at a time when they might be expected to be paralysed by fear. While this form of depersonalisation appears to be beneficial, this experience also occurs in otherwise normal subjects under certain conditions when it is innocuous or at most a minor inconvenience. The question of the relationship between these benign manifestations and the disabling depersonalisation experienced by psychiatric patients I cannot presume to answer, except at a descriptive level. The difference is that