thought relaxation was as credible as exposure.

With respect to Marks' comments about likely active ingredients in our cognitive therapy programme, it would appear that he has not read the article carefully. We make no claims about the relative potency of cognitive and behavioural procedures and explicitly state (p. 224) that the study design did not allow us to determine which of the many cognitive and behavioural procedures that distinguished cognitive therapy from behavioural stress management were responsible for the former's superiority. Marks also appears not to have noticed that assessment of the session tapes detected "no instances of either in-session or homework exposure to avoided illness-related situations (hospitals, television programmes, etc.)" and that "reassurance . . . was not often detected and the two treatments did not differ" in this respect (p. 220). Finally, we did not cite any controlled studies demonstrating that exposure alone or verbal disputation alone are specific treatments for hypochondriasis because none exists. After acceptance of our paper, data that these procedures are better than no treatment were produced by a Dutch group but there is no evidence that they have a specific effect (i.e. are better than an attention placebo condition).

Like Marks we are very interested in the question of which cognitive/cognitive–behavioural procedures are most effective. However, we differ in our views on the best ways to answer this question. Marks *et al* (1998) favour large-scale component analysis treatment trials. Because of their failure to deal with dose response issues, and other logical and variance control problems inherent in their design, we consider such trials insensitive instruments for detecting additive effects of cognitive and behavioural procedures. For this reason, we favour much tighter, single-session experiments (see Salkovskis *et al*, 1999, for a successful example of this methodology).

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Antipsychotic polypharmacy and early death

Sir: The article by Waddington *et al* (1998) is an example of careful audit over a prolonged period. For this, the authors are to be thanked, especially since such 'captive' populations and hence the possibility of such studies, is fast disappearing. However, critical comments are necessary. First, the major conclusions are presented in terms of statistical significance and one of these, the finding that 'polypharmacy' is a contributory cause of early death, is so alarming that it may well have been taken up by a vituperative press seeking to vilify psychiatry and all its works. However, the statistical information presented is so weak as not to be regarded as having significance. It is a curious and contradictory observation that both polypharmacy and withdrawal from medication both contributed, in the same direction, to demise. The article would have been more helpful if actual numbers, or at least median values, had been presented. Means and standard deviations, even with the addition of ranges, provide no clear information. For instance, apparently medication had been stopped in some patients but in how many and for what reason is not stated. I should like to believe that the series of investigations to which I contributed (Andrews *et al*, 1976), which demonstrated that continued medication in such a chronic population was of no value, had had some influence. Then the absence of information regarding clozapine (with its recognised lethal potential and for which careful monitoring is *de rigueur*), is a defect of the study; it is possible that earlier demise occurred in just such a context. Finally, it is a pity that all causes of death were lumped together; the opportunity has been missed to contribute to knowledge as to whether or not high-dose antipsychotic medication is verified as a cause of cardiac disease and death.

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Authors' reply: We appreciate the controversial nature and possible unpalatability of some of the associations that we report, but are disinclined to accept a number of Dr Snaith's strictures. Regarding statistical issues, our major findings are *not* presented in terms of significances but, rather, in terms of relative risks with 95% confidence intervals, in accordance with the 'statistics' section of the *Journal*'s 'Instructions to Authors'. We do not find that antipsychotic polypharmacy is a *contributory cause* of early death; that is one of several interpretations of our finding of an *association* between antipsychotic polypharmacy and early death. Our statistical approach and data presentation are conventional (Altman & Bland, 1998), with Cox proportional hazards modelling accepted as a method of choice for examining a set of variables for independent predictors of survival. There is no contradiction in both antipsychotic polypharmacy and time since final withdrawal of antipsychotics predicting reduced survival. As stated in our article, the index of polypharmacy is the maximum number of antipsychotics given concurrently, to cover instances where this occurred prior to the index evaluation such as when antipsychotics had been withdrawn; both are identified by Cox modelling as independent predictors of reduced survival (i.e. each variable is associated with reduced survival after controlling for the influence of the other). It was not always straightforward to specify on an individual basis the reason(s) for antipsychotic withdrawal (of which there were 20 instances); we accept Dr Snaith's point that a lack of perceived value in continuing antipsychotic treatment may have contributed to its withdrawal in some patients, in addition to our own speculation in terms of terminal physical illness replacing psychiatric disorder as the primary focus of medical care.

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